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Update: Blood Lead Levels — United States, 1991–1994

Lead is an environmental toxicant that may deleteriously affect the nervous, hematopoietic, endocrine, renal, and reproductive systems (*1*). Lead exposure in young children is a particular hazard because children absorb lead more readily than do adults and because the developing nervous systems of children are more susceptible to the effects of lead (*2*). Blood lead levels (BLLs) at least as low as 10 μg/dL can adversely affect the behavior and development of children (*2*). CDC's National Health and Nutrition Examination surveys (NHANES), an ongoing series of national examinations of the health and nutritional status of the civilian noninstitutionalized population, have been the primary source for monitoring BLLs in the U.S. population. From NHANES II (conducted during 1976–1980) to Phase 1 of NHANES III (conducted during October 1988–September 1991), the geometric mean (GM) BLL for persons aged 1–74 years declined from 12.8 μg/dL to 2.9 μg/dL, and the prevalence of elevated BLLs (BLLs ≥10 μg/dL) decreased from 77.8% to 4.4% (*3*).* This report updates national BLL estimates with data from Phase 2 of NHANES III (conducted during October 1991–September 1994), which indicate that BLLs in the U.S. population aged ≥1 year continued to decrease and that BLLs among children aged 1–5 years were more likely to be elevated among those who were poor, non-Hispanic black, living in large metropolitan areas, or living in older housing.

In NHANES III, blacks, Mexican Americans, children aged 2 months–5 years, and persons aged ≥60 years were oversampled to increase the reliability of estimates for these groups (*4*). A household interview and a physical examination were conducted for each survey participant. During the physical examination, 1 mL of whole blood was collected by venipuncture from examinees aged ≥1 year. Graphite furnace atomic absorption spectrophotometry was used to measure BLLs at a detection limit of 1 μg/dL (*5*); BLLs below the level of detection were assigned a value of 0.7 μg/dL.

In this analysis, income categories were defined using the poverty-income ratio (PIR; the ratio of total family income to the poverty threshold for the year of the interview); low income was defined as PIR ≤1.300; middle, as PIR 1.301–3.500; and high, as PIR ≥3.501. Urban status was based on U.S. Department of Agriculture codes

*The BLL value assigned to persons with BLLs below the level of detection and the sample examination weights were revised slightly in the NHANES III data set after publication of BLLs from Phase 1. Therefore, some values for Phase 1 data reported here do not match previously published values.

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that classify counties by total population and proximity to major metropolitan areas (*6*); the two categories used were metropolitan areas with a population ≥1 million and metropolitan and nonmetropolitan areas with a population <1 million. Data on the age-of-housing variable were collected by self-report using three categories (built before 1946, during 1946–1973, and after 1973); these cutpoints closely correspond to years in which the amount of lead contained in residential paint was altered (*2*)†. The sample included 13,642 persons; 2392 were children aged 1–5 years. Data for racial/ethnic groups other than non-Hispanic black, non-Hispanic white, and Mexican American were too small for reliable estimates. Statistical analyses were performed using Software for Survey Data Analysis, which accounted for the complex sample design. Asymmetric 95% confidence intervals were calculated using the natural logarithmic transformation (*7*).

During 1991–1994, the overall GM BLL of the U.S. population aged ≥1 year was 2.3 μg/dL (Table 1). GM BLLs varied by age and were highest among children aged 1–2 years and persons aged ≥50 years. Among those aged ≥1 year, approximately 2.2% had BLLs ≥10 μg/dL (Table 1). Among those aged 1–5 years, approximately 4.4% had BLLs ≥10 μg/dL (Table 1), representing an estimated 930,000 children aged 1–5 years in the United States with BLLs ≥10 μg/dL. In addition, among children aged 1–5 years, approximately 1.3% had BLLs ≥15 μg/dL, and 0.4% had BLLs ≥20 μg/dL.

For children aged 1–5 years, the prevalence of BLLs ≥10 μg/dL was higher among those who were non-Hispanic blacks or Mexican Americans, from lower-income families, living in metropolitan areas with a population ≥1 million, or living in older housing (Table 2). The differences in risk for an elevated BLL by race/ethnicity, income, and urban status generally persisted across age-of-housing categories. Similarly, the higher risk for an elevated BLL associated with older age of housing generally persisted across race/ethnicity, income, and urban status categories. Therefore, the risk for an elevated BLL was higher among non-Hispanic black children living in housing built before 1946 (21.9%) or built during 1946–1973 (13.7%), among children in low-income households who lived in housing built before 1946 (16.4%), and among children in areas with populations ≥1 million who live in housing built before 1946 (11.5%) when

†Residential paint containing up to 50% lead was in widespread use through the 1940s; lead usage in residential paint declined thereafter and was banned in 1978.

TABLE 1. Weighted geometric mean (GM) blood lead levels (BLLs) and percentage of population aged ≥1 year with BLLs ≥10 μg/dL, by age group — United States, Third National Health and Nutrition Examination Survey–Phase 2, 1991–1994

Age group (yrs)	Sample size	GM BLL (μg/dL)		% with BLLs ≥10 μg/dL	
		BLL	(95% CI*)	%	(95% CI)
1– 5	2,392	2.7	(2.5–3.0)	4.4%	(2.9%–6.6%)
1–2	*987*	*3.1*	*(2.8–3.5)*	*5.9%*	*(3.7%–9.2%)*
3–5	*1,405*	*2.5*	*(2.3–2.7)*	*3.5%*	*(2.2%–5.4%)*
6–11	1,345	1.9	(1.8–2.1)	2.0%	(1.2%–3.3%)
12–19	1,615	1.5	(1.4–1.7)	0.8%	(0.3%–1.9%)
20–49	4,716	2.1	(2.0–2.2)	1.5%	(1.0%–2.2%)
50–69	2,026	3.1	(2.9–3.2)	2.9%	(2.1%–3.8%)
≥70	1,548	3.4	(3.3–3.6)	4.6%	(3.4%–6.0%)
Total	**13,642**	**2.3**	**(2.1–2.4)**	**2.2%**	**(1.6%–2.8%)**

*Confidence interval.

TABLE 2. Percentage of children aged 1–5 years with blood lead levels (BLLs) ≥10 μg/dL, by year housing built and selected characteristics, and weighted geometric mean (GM) BLLs, by selected characteristics — United States, Third National Health and Nutrition Examination Survey–Phase 2, 1991–1994*

	Year housing built†						Total			
	Before 1946		During 1946–1973		After 1973				GM BLL (μg/dL)	
Characteristic	%	(95%CI§)	%	(95% CI)	%	(95% CI)	%	(95% CI)	BLL	(95% CI)
Race/Ethnicity¶										
Black, non-Hispanic	21.9%	(9.4%–51.1%)	13.7%	(9.1%–20.6%)	3.4%	(1.4%–7.9%)	**11.2%**	**(6.7%–18.7%)**	**4.3**	**(3.7–5.0)**
Mexican American	13.0%	(5.7%–29.8%)	2.3%	(1.1%– 5.1%)	1.6%	(0.5%–5.2%)	**4.0%**	**(2.2%– 7.2%)**	**3.1**	**(2.7–3.5)**
White, non-Hispanic	5.6%	(2.2%–14.4%)	1.4%	(0.3%– 6.0%)	1.5%	(0.3%–7.0%)	**2.3%**	**(1.0%– 5.0%)**	**2.3**	**(2.1–2.5)**
Income**										
Low	16.4%	(9.9%–27.2%)	7.3%	(4.6%–11.4%)	4.3%	(2.1%–9.1%)	**8.0%**	**(5.4%–11.7%)**	**3.8**	**(3.3–4.2)**
Middle	4.1%	(1.3%–12.8%)	2.0%	(1.0%– 4.1%)	0.4%	(0.1%–1.3%)	**1.9%**	**(1.1%– 3.2%)**	**2.3**	**(2.1–2.5)**
High	0.9%	(0.1%– 6.5%)	2.7%	(0.6%–11.3%)	0††		**1.0%**	**(0.3%– 3.4%)**	**1.9**	**(1.7–2.1)**
Urban status§§										
Population ≥1 million	11.5%	(6.5%–20.2%)	5.8%	(3.2%–10.4%)	0.8%	(0.3%–2.1%)	**5.4%**	**(3.0%– 9.8%)**	**2.8**	**(2.4–3.2)**
Population <1 million	5.8%	(2.0%–16.8%)	3.1%	(0.9%–10.1%)	2.5%	(0.7%–9.6%)	**3.3%**	**(1.5%– 7.0%)**	**2.7**	**(2.3–3.0)**
Total	**8.6%**	**(5.2%–14.2%)**	**4.6%**	**(2.9%– 7.5%)**	**1.6%**	**(0.6%–4.4%)**	**4.4%**	**(2.9%– 6.6%)**	**2.7**	**(2.5–3.0)**

* Sample size=2392, and includes racial/ethnic groups in addition to those listed separately.
† Age of housing was unknown by the household respondent for 11.7% of children aged 1–5 years; approximately 5.6% of these children had BLLs ≥10 μg/dL.
§ Confidence interval.
¶ Data for other racial/ethnic groups were too small for reliable estimates.
** Income categories were defined using the poverty-income ratio (PIR; the ratio of total family income to the poverty threshold for the year of the interview): low income was defined as PIR ≤1.300; middle, as PIR 1.301–3.500; and high, as PIR ≥3.501. Persons with data missing for income were not included in the analysis of income.
†† No children in the sample had these characteristics; however, the true estimate for this population group is probably larger than zero.
§§ Urban status was based on U.S. Department of Agriculture codes that classify counties by total population and proximity to major metropolitan areas (*6*) and divided into two categories: metropolitan areas with a population ≥1 million and metropolitan and nonmetropolitan areas with a population <1 million.

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compared with children in other categories. Based on a multivariate logistic regression model, non-Hispanic black race/ethnicity, low income, and living in housing built before 1946 were independent predictors of elevated BLLs in children aged 1–5 years. Living in urban areas was not an independent predictor of elevated BLLs when controlling for race/ethnicity, income, and age of housing.

For the total population, GM BLLs decreased by 21.7% from Phase 1 to Phase 2 with minimal variation within age, sex, race/ethnicity, income, age-of-housing, and urban status groups (range: 17.4%–26.4%). Among children aged 1–5 years, the overall absolute decrease in the prevalence of elevated BLLs from Phase 1 to Phase 2 was 4.1 percentage points. The percentage point decrease was generally greater among those groups with higher prevalences of elevated BLLs during Phase 1: children aged 1–2 years (5.2), non-Hispanic black children (7.4), children from low-income families (6.9), children living in areas with a population <1 million (5.3), and children living in housing built before 1946 (9.6). Conversely, the percentage decrease of elevated BLLs from Phase 1 to Phase 2 was 48.4% among all children aged 1–5 years and generally was smaller among those groups at highest risk for elevated BLLs.

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Editorial Note: The findings in this analysis of NHANES III data indicate that the GM BLL for the U.S. population aged ≥1 year decreased by 22% from Phase 1 to Phase 2, and the prevalence of BLLs ≥10 μg/dL decreased by 51% over the same period. However, constraints of the survey design of NHANES III precluded statistical testing for the differences in GM BLLs and the prevalences of elevated BLLs from Phase 1 to Phase 2. The decrease in BLLs observed from Phase 1 to Phase 2 follow even larger decreases from NHANES II (1976–1980) to Phase 1 of NHANES III. Among persons aged 1–74 years, the GM BLL declined 77% from NHANES II to Phase 1 of NHANES III, and the prevalence of BLLs ≥10 μg/dL decreased by 94% (*3*).

The dramatic decline in BLLs in the U.S. population since the late 1970s is probably a direct consequence of the regulatory and voluntary bans enacted during this period on the use of lead in gasoline, household paint, food and drink cans, and plumbing systems (*2*). The effects of these changes benefited all U.S. population groups studied. In addition, BLLs may have been reduced in some groups as the result of childhood lead poisoning-prevention efforts undertaken by public health agencies, lead paint-abatement programs, and the promulgation of a standard for lead exposure in industry.

Despite the recent and large declines in BLLs, the risk for lead exposure remains disproportionately high for some groups, including children who are poor, non-Hispanic black, Mexican American, living in large metropolitan areas, or living in older housing. Although confidence intervals for elevated BLL prevalence estimates overlapped across age-of-housing and urban status categories for all children aged 1–5 years, the overall direction of the risk differentials is consistent with results from previous years (*8*). In addition, with the exception of urban status—which was too broadly defined in this study to reflect gradations of risk associated with residence in a central city versus residence in outlying metropolitan or suburban areas—each of these factors was an independent contributor to the risk for elevated BLLs among children.

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The risk for lead exposure in children is primarily determined by environmental conditions of the child's residence. The most common source for lead exposure for children is lead-based paint that has deteriorated into paint chips and lead dust (*2*). In the United States, approximately 83% of privately owned housing units and 86% of public housing units built before 1980 contain some lead-based paint (*9*). In addition, soil and dust contaminated with residual lead fallout from vehicle exhaust contribute to exposure; concentrations of lead in soil and dust are highest in central urban areas (*10*). For adults, the most common high-dose exposure sources are occupational (*1*). Other exposure sources for adults and children can include lead dust brought into the home on clothing from workplaces, lead used for some hobbies, lead contained in some "folk" medicines and cosmetics, and lead in plumbing and in crystal and ceramic containers that leaches into water or food (*2*).

Despite the substantial progress in eliminating sources of lead in the United States, the NHANES data indicate that nearly 1 million children aged 1–5 years had elevated BLLs during 1991–1994. In addition to efforts to reduce or eliminate sources of lead and exposure to lead, screening efforts are necessary for early identification of children with elevated BLLs to enable prompt and appropriate environmental, educational, and medical interventions.

Because the distribution of risk for childhood lead exposure varies widely within the United States, prevention activities must be conducted at the local level and must be appropriate to local conditions. In areas where the risk for elevated BLLs is low, screening efforts should be targeted to children who remain at elevated risk for lead exposure. CDC is developing guidelines to assist state and local health departments in designing screening recommendations appropriate to their jurisdictions. A draft of these guidelines is available for public review and comment through April 7, 1997; copies can be obtained by calling (888) 232-6789 or accessing the World Wide Web at http://www.cdc.gov/nceh.

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Trends in Ischemic Heart Disease Deaths — United States, 1990–1994

In 1994, a total of 481,458 persons died as a result of ischemic heart disease (IHD), which comprises two thirds of all heart disease—the leading cause of death in the United States. This report presents trends in IHD mortality in the United States for 1990–1994 (the latest year for which data are available) and compares these trends by race, sex, and state. These findings indicate IHD death rates decreased from 1990 through 1994; however, the rate of decline was slower than rates of previously observed declines.

Age-adjusted IHD death rates for persons aged ≥35 years were calculated using mortality data tapes compiled by CDC and population estimates from the Bureau of the Census. IHD death rates were directly age-adjusted to the 1980 U.S. standard population aged ≥35 years. IHD deaths were defined as those with the underlying cause of death listed on the death certificate as *International Classification of Diseases, Ninth Revision* [ICD-9], codes 410–414.9. The average annual percentage change in IHD mortality from 1990 through 1994 was calculated as the 1994 rate minus the 1990 rate divided by the 1990 rate divided by 4 multiplied by 100. Data are presented only for blacks and whites because numbers for other racial/ethnic groups were too small for meaningful analysis.

From 1990 through 1994, age-adjusted IHD death rates for the U.S. population aged ≥35 years decreased 10.3%, from 416.3 deaths per 100,000 to 373.6 deaths per 100,000. However, the rate of decrease varied by race and sex; rates of decline were faster for whites than for blacks and for men than for women (Figure 1). The largest average annual percentage decrease occurred among white men (2.9% per year), followed by white women (2.5%), black men (2.3%), and black women (1.6%).

IHD death rates varied substantially among the states (Table 1). In 1994, the rates for both women and men residing in the states with the highest IHD death rates were approximately two times higher than for persons residing in the states with the lowest IHD death rates. For women, IHD death rates in 1994 ranged from 156.7 per 100,000 (Montana) to 406.3 per 100,000 (New York) and, for men, ranged from 289.4 per 100,000 (New Mexico) to 638.8 per 100,000 (New York).

From 1990 through 1994, IHD death rates declined in nearly all 50 states and the District of Columbia (Table 1). However, the magnitude of change over time varied widely; some states had small declines (e.g., Nevada, 0.1% per year and Hawaii, 0.9% per year) while other states experienced larger declines (e.g., Alaska, 5.5% per year and Montana, 5.6% per year). Sex-specific IHD death rates for both men and women declined for each state except Idaho and Nevada (small increase for women only) and the District of Columbia (small increase for men only).

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