

## **Trends in Blood Lead Levels and Blood Lead Testing Among US Children Aged 1 to 5 Years, 1988-2004**

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### Abbreviations:

BLL - blood lead level

CDC - Centers for Disease Control and Prevention

CI - confidence interval

NHANES - National Health and Nutrition Examination Survey

PIR - poverty income ratio

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## ABSTRACT

### OBJECTIVES

To evaluate trends in children's blood lead levels and the extent of blood lead testing of children at risk for lead poisoning from national surveys conducted during a 16-year period in the United States.

### METHODS

Data for children aged 1 to 5 years from the National Health and Nutrition Examination Survey III Phase I, 1988–1991, and Phase II, 1991–1994 were compared to data from the survey period 1999–2004.

### RESULTS

The prevalence of elevated blood lead levels,  $\geq 10$   $\mu\text{g/dL}$ , among children decreased from 8.6% in 1988–1991 to 1.4% in 1999–2004, which is an 84% decline. From 1988–1991 and 1999–2004, children's geometric mean blood lead levels declined in non-Hispanic black (5.2–2.8  $\mu\text{g/dL}$ ), Mexican American (3.9–1.9  $\mu\text{g/dL}$ ), and non-Hispanic white children (3.1  $\mu\text{g/dL}$  to 1.7  $\mu\text{g/dL}$ ). However, levels continue to be highest among non-Hispanic black children relative to Mexican American and non-Hispanic white children. Blood lead levels were distributed as follows: 14.0% were  $< 1.0$   $\mu\text{g/dL}$ , 55.0% were 1.0 to  $< 2.5$   $\mu\text{g/dL}$ , 23.6% were 2.5 to  $< 5$   $\mu\text{g/dL}$ , 4.5% were 5 to  $< 7.5$   $\mu\text{g/dL}$ , 1.5% were 7.5 to  $< 10$   $\mu\text{g/dL}$ , and 1.4% were  $\geq 10$   $\mu\text{g/dL}$ . Multivariable analysis indicated that residence in older housing, poverty, age, and being non-Hispanic black are still major risk factors for higher lead levels. Blood lead testing of Medicaid-enrolled children increased to 41.9% from 19.2% in 1988–1991. Only 43.0% of children with elevated blood lead levels had previously been tested.

### CONCLUSIONS

Children's blood lead levels continue to decline in the United States, even in historically high-risk groups for lead poisoning. To maintain progress made and eliminate remaining disparities, efforts must continue to test children at high risk for lead poisoning, and identify and control sources of lead. Coordinated prevention strategies at national, state, and local levels will help achieve the goal of elimination of elevated blood lead levels.

### INTRODUCTION

The adverse health effects of lead are well documented,<sup>1,2</sup> and no threshold for adverse effects has been specified.<sup>3–5</sup> Because overt clinical symptoms are rare at blood lead levels (BLLs) of  $< 70$   $\mu\text{g/dL}$ , blood lead testing is necessary to identify asymptomatic children with elevated BLLs of  $\geq 10$   $\mu\text{g/dL}$ . The United States Department of Health and Human Services and the Centers for Disease Control and Prevention (CDC) have targeted BLLs of  $\geq 10$   $\mu\text{g/dL}$  for elimination in the United States by 2010.<sup>6</sup> Childhood lead poisoning prevention programs have focused on young children aged  $< 6$  years, because these children are especially vulnerable to the adverse health effects of lead. The nervous systems of young children are still developing and the hand-to-mouth behaviors common at these ages increase their risk for ingesting lead in their environment.

Nationally, BLLs in children have been declining.<sup>7–11</sup> Some children, however, continue to be at greater risk for exposure to lead than others.<sup>12–14</sup> Since 1976, blood lead data from the National Health and Nutrition Examination Surveys (NHANES) have been used to characterize children's BLLs. Children at highest risk are non-Hispanic black, live in housing built before 1950, and their families are poor.<sup>15,16</sup> From 1991–1994, Medicaid enrollees accounted for 60% of US children who had elevated BLLs, yet only 19% of Medicaid-enrolled children had a blood lead test before their participation in the NHANES III.<sup>17,18</sup> The decline in the prevalence of elevated BLLs over time has been most pronounced among children belonging to high-risk groups, especially non-Hispanic black children. They experienced a 72% decline in the prevalence of elevated BLLs between the 1991–1994 and 1999–2002 NHANES.<sup>7</sup> Nevertheless, the geometric mean BLL remained higher for non-Hispanic black children compared with Mexican American and non-Hispanic white children in the 1999–2004 NHANES (2.8 vs 1.9 and 1.7  $\mu\text{g/dL}$ , respectively) (Table 1), indicating that differences in risk for exposure to lead still exist as seen from previous reports of NHANES data: 1976–1980, 1988–1991, and 1999–2002.<sup>7–10</sup>

**TABLE 1. Distribution of BLLs According to Selected Demographic Characteristics, Children Aged 1 to 5 Years, NHANES 1999–2004**

	n	Geometric mean (95% CI), µg/dL	<1 µg/dL, % (95% CI)	1 to <2.5 µg/dL, % (95% CI)	2.5 to <5 µg/dL, % (95% CI)	5 to <7.5 µg/dL, % (95% CI)	7.5 to <10 µg/dL, % (95% CI)	≥10 µg/dL, % (95% CI)
Overall	2532	1.9 (1.8-2.0) <sup>a</sup>	14.0 (11.6-16.6) <sup>b</sup>	55.0 (52.1-57.9)	23.6 (21.1-26.1)	4.5 (3.3-5.9)	1.5 (1.0-2.1)	1.4 (1.0-2.0) <sup>b</sup>
Gender								
Females	1211	1.9 (1.7-2.0) <sup>a</sup>	14.1 (10.8-17.7) <sup>b</sup>	54.5 (51.1-57.8)	23.9 (20.3-27.8)	4.5 (3.3-5.8)	1.4 (0.8-2.3)	1.7 (0.9-2.6) <sup>b</sup>
Males	1321	1.9 (1.7-2.0) <sup>a</sup>	14.0 (11.4-16.7) <sup>b</sup>	55.5 (51.4-59.5)	23.2 (20.3-26.3)	4.6 (3.0-6.5)	1.5 (0.9-2.3)	1.3 (0.7-2.6) <sup>b</sup>
Age <sup>c</sup>								
1-2 y	1231	2.1 (2.0-2.2) <sup>a,c</sup>	10.6 (7.7-13.9) <sup>b,d</sup>	51.0 (46.7-55.3) <sup>c</sup>	27.9 (24.9-31.0) <sup>d</sup>	6.7 (5.0-8.6) <sup>d</sup>	1.4 (0.8-2.2)	2.4 (1.4-3.5) <sup>b,d</sup>
3-5 y	1301	1.7 (1.6-1.9) <sup>a,c</sup>	16.2 (12.9-19.9) <sup>b,d</sup>	57.6 (53.8-61.4) <sup>c</sup>	20.7 (17.9-23.7) <sup>d</sup>	3.1 (1.9-4.6) <sup>d</sup>	1.5 (0.8-2.3)	0.9 (0.4-1.5) <sup>e,b,d</sup>
Race/ethnicity <sup>c</sup>								
Non-Hispanic black	755	2.8 (2.5-3.0) <sup>a,f,g</sup>	4.0 (2.5-5.7) <sup>f,g</sup>	42.5 (37.8-47.2) <sup>f,g</sup>	36.2 (33.1-39.3) <sup>f,g</sup>	9.4 (6.9-12.2) <sup>f,g</sup>	4.6 (3.0-6.5) <sup>f,g</sup>	3.4 (1.8-5.5) <sup>b</sup>
Mexican American	812	1.9 (1.7-2.0) <sup>a,f</sup>	10.9 (8.6-13.4) <sup>b,f,h</sup>	61.0 (56.9-65.1) <sup>f</sup>	22.1 (18.0-26.5) <sup>f</sup>	3.4 (2.2-5.0) <sup>f</sup>	1.3 (0.6-2.2) <sup>f</sup>	1.2 (0.4-2.6) <sup>e</sup>
Non-Hispanic white	731	1.7 (1.6-1.8) <sup>a,g</sup>	17.6 (14.0-21.5) <sup>b,g,h</sup>	57.1 (52.4-61.7) <sup>g</sup>	19.7 (16.1-23.5) <sup>g</sup>	3.6 (1.9-5.8) <sup>g</sup>	0.8 (0.3-1.6) <sup>e,g</sup>	1.2 (0.6-2.0) <sup>b</sup>
PIR <sup>c</sup>								
≤1.3	1302	2.4 (2.2-2.5) <sup>a,c</sup>	6.7 (4.6-9.2) <sup>b,d</sup>	49.3 (44.9-53.7) <sup>d</sup>	32.5 (28.6-36.4) <sup>d</sup>	6.9 (5.2-8.8) <sup>d</sup>	2.8 (1.7-4.1) <sup>d</sup>	1.8 (1.1-2.7) <sup>b</sup>
>1.3	1070	1.5 (1.4-1.6) <sup>a,c</sup>	19.9 (16.3-23.8) <sup>b,d</sup>	60.4 (56.9-63.8) <sup>d</sup>	16.0 (12.9-19.3) <sup>d</sup>	2.3 (1.2-3.7) <sup>d</sup>	0.6 (0.1-1.4) <sup>d,e</sup>	0.8 (0.3-1.6) <sup>b,e</sup>
Medicaid Status <sup>c</sup>								
Yes	990	2.6 (2.2-2.5) <sup>a,c</sup>	5.8 (3.6-8.6) <sup>b,d</sup>	49.5 (45.2-53.8) <sup>d</sup>	32.4 (28.4-36.5) <sup>d</sup>	7.7 (5.7-10.0) <sup>d</sup>	2.7 (1.6-4.2) <sup>d</sup>	1.9 (0.9-3.1) <sup>b</sup>
No	1500	1.7 (1.6-1.8) <sup>a,c</sup>	17.6 (14.0-21.4) <sup>b,d</sup>	57.9 (54.3-61.5) <sup>d</sup>	19.3 (16.2-22.6) <sup>d</sup>	3.2 (2.1-4.6) <sup>d</sup>	0.9 (0.4-1.6) <sup>d,e</sup>	1.1 (0.6-1.7) <sup>b</sup>
Year housing built (risk of lead exposure) <sup>c,i</sup>								
Low risk	674	1.5 (1.3-1.6) <sup>a,j,k</sup>	22.1 (17.3-27.3) <sup>b,k</sup>	62.8 (57.4-68.1) <sup>k</sup>	13.0 (9.6-16.8) <sup>h,k</sup>	1.4 (0.6-2.5) <sup>e,k</sup>	0.3 (0.1-0.4) <sup>k</sup>	0.4 (0.0-1.3) <sup>e,k</sup>
Medium risk	555	1.8 (1.7-1.9) <sup>a,j,l</sup>	14.4 (10.2-19.3) <sup>b,l</sup>	57.9 (52.0-63.8)	23.4 (18.5-28.6) <sup>h</sup>	2.3 (1.1-3.9) <sup>e,l</sup>	1.1 (0.4-2.1) <sup>e</sup>	0.8 (0.3-1.7) <sup>b,e</sup>
High risk	335	2.5 (2.2-2.8) <sup>a,k,l</sup>	5.5 (2.4-9.7) <sup>e,k,l</sup>	47.4 (40.2-54.7) <sup>k</sup>	32.0 (25.0-39.4) <sup>k</sup>	8.7 (5.3-12.9) <sup>k,l</sup>	3.1 (1.5-5.2) <sup>k</sup>	3.3 (1.5-5.8) <sup>e,k</sup>
Not known	968	2.3 (2.1-2.6)	7.6 (5.2-10.4)	46.3 (40.6-52.0)	33.2 (29.5-37.0)	8.2 (5.9-10.7)	2.4 (1.3-4.0)	2.3 (1.1-3.8)

<sup>a</sup> Difference between 1988–1991 and 1999–2004 statistically significant at  $P < .05$  after Bonferroni adjustment.

<sup>b</sup> Difference between 1988–1991 and 1999–2004 within lead categories BLL <1 µg/dL and/ BLL ≥10 µg/dL statistically significant at  $P < .05$  after Bonferroni adjustment.

<sup>c</sup> Differences between strata for categorical blood lead variable in 1999–2004 statistically significant at  $P < .05$ .

<sup>d</sup> Difference between strata in 1999–2004 statistically significant at  $P < .05$ .

<sup>e</sup> Relative SE of point estimate ≥ 30%; does not meet standard of statistical reliability and precision.

<sup>f</sup> Difference between non-Hispanic black children and Mexican American children in 1999–2004 statistically significant at  $P < .05$  after Bonferroni adjustment.

<sup>g</sup> Difference between non-Hispanic black children and non-Hispanic white children in 1999–2004 statistically significant at  $P < .05$  after Bonferroni adjustment.

<sup>h</sup> Difference between Mexican American children and non-Hispanic white children in 1999–2004 statistically significant at  $P < .05$  after Bonferroni adjustment.

<sup>i</sup> Risk of lead exposure by year housing built defined as follows:

NHANES 1999–2004: low risk, built 1978 and later; medium risk, built between 1950 and 1977; high risk, built before 1950.

NHANES III phases 1 and 2: low risk, built 1974 and later; medium risk, built between 1946 and 1973; high risk, built before 1946.

<sup>j</sup> Difference between low-risk housing and medium-risk housing in 1999–2004 statistically significant at  $P < .05$  after Bonferroni adjustment.

<sup>k</sup> Difference between low-risk housing and high-risk housing in 1999–2004 statistically significant at  $P < .05$  after Bonferroni adjustment.

<sup>l</sup> Difference between medium-risk housing and high-risk housing in 1999–2004 statistically significant at  $P < .05$  after Bonferroni adjustment.

With increasing evidence that adverse health effects occur at BLLs of <10 µg/dL,<sup>5, 19–21</sup> little is known about the distribution of and risk factors associated with BLLs of <10 µg/dL. Although in at least 1 study, well-established risk factors associated with BLLs of ≥10 µg/dL were also predictive of BLLs of ≥5 µg/dL.<sup>22</sup> However, this study also found that for a number of children with BLLs 5 to 9 µg/dL, multiple sources of lead exposure seemed likely given the prevalence of these levels among children without obvious risk factors.<sup>22</sup> In this study, we augmented previous work by updating information on the distribution of children's BLLs, the extent of blood lead testing of children at risk for lead poisoning, and risk factors for higher BLLs among children 1 to 5 years of age from 2 separate NHANESs (1988–1994 and 1999–2004).

## METHODS

### Survey design

The National Center for Health Statistics of the CDC conducts the NHANES, which measure health and nutrition in a representative sample of the US noninstitutionalized civilian population aged 3 months and older by using a multistage probability design. Since 1999, the NHANES has been inclusive of all ages and has been a continuous survey as previously described.<sup>23</sup> Beginning in 1999, ~ 5000 people were recruited annually to participate in the NHANES. Of these ~ 5000 individuals, ~ 550 were children between the ages of 1 to 5 years. A medical examination included blood lead testing as part of the laboratory component and a household interview contained questions on health, demographics and nutritional characteristics, as well as whether a child had been previously tested for lead poisoning, and health insurance status, including Medicaid enrollment. Participants ages 1 year and older were eligible for blood lead testing. A family member provided questionnaire responses for children <16

years of age. The NHANES III (1988–1994) design and the blood lead component have been described previously.<sup>9, 15–16, 24</sup>

### **Laboratory methods**

Whole blood samples were drawn into prescreened ethylenediaminetetraacetic acid-anticoagulated evacuated tubes by venipuncture of all participants.<sup>24</sup> The analysis of lead was conducted in the Inorganic Toxicology Laboratory, Division of Laboratory Sciences, National Center for Environmental Health, CDC, Atlanta, Georgia. Blood lead samples were stored frozen at  $-20^{\circ}$  C or lower until analyzed. The laboratory methods used to analyze blood lead in the NHANES III have been described in detail previously.<sup>9,16</sup> During the survey years of 1999–2002, the blood samples were measured for lead by graphite furnace atomic absorption spectrometry, (CDC method No. ITB002A) by using a modification of the method of Miller et al.<sup>25</sup> During the survey years of 2003–2004, the blood samples were measured for lead by inductively coupled plasma mass spectrometry (CDC method No. ITB001A) by using a modification of the method of Nixon et al.<sup>26</sup> The blood lead limit of detection (LOD) for graphite furnace atomic absorption spectrometry and inductively coupled plasma mass spectrometry is  $0.3\mu\text{g/dL}$ . Analytical quality control was monitored by using 4 concentrations of bench quality control material and 2 levels of “blind” quality control materials.

### **Statistical Analysis**

This analysis included data from the NHANES III, Phase 1 (1988–1991), NHANES III, Phase 2 (1991–1994), and the first 6 years of the current NHANES (1999–2004) for children 1 to 5 years of age.

### **Race/Ethnicity**

We reported only 3 categories of race/ethnicity (non-Hispanic white, non-Hispanic black, and Mexican American) in the race/ethnicity section of the tables, because there were small numbers of children in the other Hispanic, other race, and multiracial categories. For all other sections of the tables, results were included for all participants in the survey.

### **Housing**

Because responses for “year housing was built” used different categories between the NHANES III and the NHANES 1999–2004, we defined the most closely similar categories as highest risk (built before 1946 [NHANES III] and before 1950 [NHANES 1999–2004]), medium risk (built between 1946 and 1973 [NHANES III], and 1950 and 1977 [NHANES 1999–2004]), and low risk (built 1974 and after [NHANES III]) and 1978 and after [NHANES 1999–2004]). Observations for which information was not available about when the housing unit was built were defined as “not known,” this category was reported in the tables but was not used in any statistical comparisons.

### **Socio-Economic Status**

The poverty income ratio (PIR) (defined as the ratio of total family income to the poverty threshold for the year of the interview) was stratified as  $\leq 1.3$  (corresponding to low income) and  $> 1.3$  (corresponding to middle to high income). These categories were selected in part to be consistent with major government food assistance programs that use a PIR of 1.3 to determine eligibility.<sup>15</sup> The Medicaid status variable was defined as whether a child was enrolled in Medicaid (yes) or not enrolled in Medicaid (no).

### **Analytic Strategy**

Statistical analysis was conducted by using SAS 9.1.3 (SAS Institute, Inc, Cary, NC) and SUDAAN 9.0 (Research Triangle Institute, Research Triangle Park, NC). NHANES-specific sample weights were used for all analyses to adjust for the differential probabilities of selection, nonresponse, and noncoverage. All statistical test results were evaluated by using an overall significance level of  $P < .05$ . BLLs were not rounded before statistical analysis. Because of small proportions, the arcsine method was used to construct 2-sided confidence intervals (CIs).<sup>27, 28</sup> We computed geometric mean BLLs by taking the antilog of the mean of  $\log_{10}$  of the BLLs.

We also analyzed BLLs by examining their distribution across 6 categories:  $< 1.0$ ,  $1.0$  to  $< 2.5$ ,  $2.5$  to  $< 5$ ,  $5$  to  $< 7.5$ ,  $7.5$  to  $< 10$ , and  $\geq 10\mu\text{g/dL}$ . These categories were selected to provide a relatively fine breakdown of BLLs of  $< 10$

µg/dL. These fine breakdowns show the changes in distributions across these categories in subpopulations between NHANESs as well as the status of these subpopulations within the current NHANES. Because no BLL has been identified as safe for children, the use of several fine cut points of <10 µg/dL also allows assessment at several levels that may be of interest. For example some analyses have used a cut point of 5 µg/dL. Less than 1 µg/dL was chosen as a cut point, because the LOD in BLL measurements in the current NHANES is 0.3 µg/dL. These cut points may be more easily understood than statistically derived cut points (eg, quartiles).

**TABLE 2. Distribution of BLLs According to Selected Demographic Characteristics, Children Aged 1 to 5 Years, NHANES III, Phase 1 (1988–1991)**

	n	Geometric mean, (95% CI) <sup>a</sup> µg/dL	< 1 µg/dL, % (95% CI)	1 - < 2.5 µg/dL, % (95% CI)	2.5 - < 5 µg/dL, % (95% CI)	5 - < 7.5 µg/dL, % (95% CI)	7.5 - < 10 µg/dL, % (95% CI)	≥ 10 µg/dL, % (95% CI)
Overall	2232	3.6 (3.2-3.9) <sup>a</sup>	4.4 (2.6-6.6) <sup>b</sup>	25.6 (20.9-30.5)	38.7 (34.2-43.2)	15.7 (13.5-18.0)	7.1 (5.6-8.8)	8.6 (5.0-13.0) <sup>b</sup>
Gender								
Females	1144	3.5 (3.2-3.9) <sup>a</sup>	4.8 (2.5-7.6) <sup>b</sup>	25.6 (19.0-32.9)	38.3 (32.7-44.1)	15.9 (13.2-18.9)	6.9 (4.9-9.2)	8.5 (5.2-12.5) <sup>b</sup>
Males	1088	3.6 (3.2-4.0)	4.1 (2.4-6.2) <sup>b</sup>	25.5 (21.4-29.8)	39.0 (33.6-44.4)	15.5 (12.5-18.8)	7.3 (5.4-9.5)	8.7 (4.5-14.1) <sup>b</sup>
Age								
1-2 y	924	4.0 (3.6-4.5) <sup>a</sup>	3.3 (2.0-4.9) <sup>b</sup>	19.1 (14.6-24.1)	38.6 (32.0-45.4)	20.5 (16.6-24.6)	7.4 (4.9-10.4)	11.1 (7.2-15.6) <sup>b</sup>
3-5 y	1308	3.3 (2.9-3.7) <sup>a</sup>	5.1 (2.8-8.0) <sup>b</sup>	29.5 (23.9-35.4)	38.7 (34.3-43.2)	12.8 (10.5-15.3)	6.9 (5.0-9.1)	7.0 (3.3-12.0) <sup>b,c</sup>
Race/ethnicity								
Non-Hispanic black	679	5.2 (4.7-5.7) <sup>a</sup>	2.9 (1.1-5.5) <sup>c</sup>	11.8 (9.1-14.8)	32.2 (25.4-39.4)	20.4 (17.1-23.9)	14.0 (10.7-17.7)	18.6 (13.3-24.6) <sup>b</sup>
Mexican American	803	3.9 (3.0-4.8) <sup>a</sup>	3.3 (0.2-9.5) <sup>b,c</sup>	19.6 (11.5-29.2)	42.3 (37.6-46.9)	19.3 (13.2-26.1)	8.4 (3.9-14.5)	7.2 (4.3-10.9)
Non-Hispanic white	658	3.1 (2.9-3.4) <sup>a</sup>	5.4 (3.1-8.3) <sup>b</sup>	30.4 (25.2-35.9)	39.6 (34.0-45.4)	14.0 (11.1-17.1)	5.1 (3.1-7.5)	5.5 (2.9-8.9) <sup>b</sup>
PIR								
≤1.3	1019	4.7 (4.0-5.4) <sup>a</sup>	2.1 (0.8-3.9) <sup>b,c</sup>	13.4 (8.3-19.5)	39.0 (31.9-46.2)	20.4 (17.0-24.0)	10.2 (7.4-13.3)	15.0 (9.0-22.2) <sup>b</sup>
>1.3	1004	3.1 (2.8-3.3) <sup>a</sup>	6.0 (3.5-9.2) <sup>b</sup>	31.5 (26.9-36.2)	39.0 (33.6-44.6)	13.1 (10.8-15.6)	5.4 (3.6-7.6)	5.0 (2.5-8.3) <sup>b</sup>
Medicaid Status								
Yes	626	5.3 (4.2-6.3) <sup>a</sup>	1.9 (0.5-4.2) <sup>b,c</sup>	9.7 (4.4-16.8)	37.5 (28.7-46.8)	18.8 (14.1-24.4)	11.1 (7.3-15.6)	21.0 (12.0-31.7) <sup>b</sup>
No	1266	3.1 (2.9-3.4) <sup>a</sup>	5.6 (3.3-8.3) <sup>b</sup>	30.9 (25.9-36.2)	39.6 (34.1-45.2)	13.9 (11.4-16.6)	5.0 (3.6-6.6)	5.0 (2.8-7.8) <sup>b</sup>
Year housing built (risk of lead exposure) <sup>d</sup>								
Low risk	602	2.9 (2.5-3.3) <sup>a</sup>	5.3 (2.0-9.9) <sup>b,c</sup>	34.9 (27.6-42.6)	40.3 (33.8-46.9)	10.0 (7.1-13.3)	4.7 (2.6-7.3)	4.8 (1.6-9.8) <sup>c</sup>
Medium risk	931	3.6 (3.2-4.0) <sup>a</sup>	5.0 (3.3-7.1) <sup>b</sup>	23.2 (17.0-30.0)	37.7 (32.0-43.6)	19.2 (15.0-23.7)	6.5 (4.8-8.5)	8.3 (5.2-12.0) <sup>b</sup>
High risk	378	5.2 (4.4-5.9) <sup>a</sup>	1.2 (0.1-3.6) <sup>c</sup>	12.2 (7.6-17.8)	37.3 (28.9-46.2)	17.5 (11.2-24.8)	13.6 (8.7-19.4)	18.2 (12.0-25.5)
Not known	170	4.9 (3.8-5.9)	0.6 (0.0-2.4) <sup>c</sup>	14.6 (6.3-25.6) <sup>c</sup>	38.6 (25.4-52.8)	20.9 (13.1-30.1)	11.3 (4.3-21.1) <sup>c</sup>	14.0 (4.3-28.1) <sup>c</sup>

<sup>a</sup> Difference between 1988–1991 and 1999–2004 statistically significant at  $P < .05$  after Bonferroni adjustment.

<sup>b</sup> Difference between 1988–1991 and 1999–2004 within lead categories BLL <1 µg/dL and/ BLL ≥10 µg/dL statistically significant at  $P < .05$  after Bonferroni adjustment.

<sup>c</sup> Relative SE of point estimate ≥30%; does not meet standard of statistical reliability and precision.

<sup>d</sup> Risk of lead exposure by year housing built defined as follows:

NHANES 1999–2004: low risk, built 1978 and later; medium risk, built between 1950 and 1977; high risk, built before 1950.

NHANES III phases 1 and 2: low risk, built 1974 and later; medium risk, built between 1946 and 1973; high risk, built before 1946.

We evaluated differences in geometric means and proportions for 1999–2004 by computing the  $t$  statistic, by using the Bonferroni method to adjust for multiple comparisons across categories of race-ethnicity and housing risk. For 1999–2004, we evaluated the differences in the categorical BLL variable across levels of age, race-ethnicity, PIR, Medicaid status, and housing risk, by using the  $\chi^2$  test of independence. For 1999–2004, we also evaluated differences in the proportion of children with BLLs of <1 µg/dL (lowest category) and the proportion of children with BLLs of ≥10 µg/dL (highest category) across the covariates mentioned previously. We evaluated changes over time for geometric means and proportions by using a 2-tailed  $t$  test to test for differences between the periods of 1988–1991 and 1999–2004, by using the Bonferroni method to adjust for multiple comparisons within variables. We used a comparable approach to assess changes over time for overall geometric means and overall prevalence of elevated BLLs in the current NHANES data, assessing change from the 1999–2000 period to the 2003–2004 period.

To assess risk factors associated with higher BLLs, we also fit multivariable logistic and linear regression models by using the RLOGIST and LINEAR procedures in SUDAAN. Multivariable logistic regression was used to assess various risk factors associated with an elevated BLL (≥10 µg/dL). Multivariable linear regression was used to assess risk factors associated with higher BLLs. The dependent variables were the probability of having a BLL of ≥10 µg/dL for the logistic model and a natural log-transformed BLL for the linear model. For both modeling approaches, the main effects were fitted that included gender of the child (male, female [referent]), age of the child (1–2 years, 3–5 years [referent]), race/ethnicity (non-Hispanic black, Mexican American, non-Hispanic white [referent]), PIR (≤1.3, >1.3 [referent]), housing risk (high risk, medium risk, low risk [referent]), and NHANES (NHANES 1999–2004, NHANES III 1991–1994, NHANES III 1988–1991 [referent]). Gender of child was not a predictor of BLLs in the logistic model nor in the linear model and thus was not included in the final models. We also evaluated all possible 2-way interaction terms for the final main effects model by adding them, 1 at a time, to

the model and assessing their statistical significance. None of the 2-way interaction terms achieved statistical significance in either the logistic or linear analyses. No 3-way interactions were evaluated.

## RESULTS

Geometric means and distribution of BLLs across 6 blood lead categories both overall and by analytical variables, for NHANES III Phase 1 (1988–1991), NHANES III Phase 2 (1991–1994), and NHANES 1999–2004 are presented in Tables 1 to 3. Overall, the distribution of BLLs for US children shifted toward lower BLL categories from the 1988–1991 period to the 1999–2004 period, as evidenced by the lower geometric mean for the latter period ( $P < .0001$ ). The overall prevalence of BLLs of  $\geq 10$   $\mu\text{g}/\text{dL}$  declined dramatically, from 8.6% in 1988–1991 to 1.4% in 1999–2004, a decrease of 84%. Among the race/ethnicity groups examined, the prevalence of BLLs of  $\geq 10$   $\mu\text{g}/\text{dL}$  declined 84% in Mexican American children, 82% in non-Hispanic black children, and 78% in non-Hispanic white children between the 1988–1991 period and 1999–2004 period.

**TABLE 3. Distribution of BLLs According to Selected Demographic Characteristics, Children Aged 1 to 5 Years, NHANES III, Phase 2 (1991–1994)**

	n	Geometric mean, (95% CI) <sup>a</sup> $\mu\text{g}/\text{dL}$	< 1 $\mu\text{g}/\text{dL}$ , % (95% CI)	1 - < 2.5 $\mu\text{g}/\text{dL}$ , % (95% CI)	2.5 - < 5 $\mu\text{g}/\text{dL}$ , % (95% CI)	5 - < 7.5 $\mu\text{g}/\text{dL}$ , % (95% CI)	7.5 - < 10 $\mu\text{g}/\text{dL}$ , % (95% CI)	$\geq 10$ $\mu\text{g}/\text{dL}$ , % (95% CI)
Overall	2392	2.7 (2.5-3.0)	8.2 (6.4-10.3)	37.7 (33.4-42.1)	33.1 (29.5-36.8)	12.1 (9.1-15.6)	4.4 (3.0-6.1)	4.4 (2.7-6.5)
Gender								
Females	1181	2.7 (2.4-2.9)	7.1 (5.0-9.4)	42.3 (36.2-48.5)	31.7 (26.2-37.6)	11.5 (8.3-15.3)	4.1 (2.4-6.2)	3.3 (2.0-4.9)
Males	1211	2.8 (2.5-3.1)	9.3 (6.6-12.4)	33.5 (28.4-38.7)	34.3 (28.5-40.5)	12.7 (9.4-16.4)	4.7 (2.9-6.9)	5.5 (3.1-8.4)
Age								
1-2 y	987	3.1 (2.8-3.5)	6.1 (4.1-8.6)	30.3 (25.9-34.9)	36.9 (31.5-42.5)	14.8 (11.8-18.1)	6.0 (3.6-8.8)	5.9 (3.4-9.0)
3-5 y	1405	2.5 (2.3-2.7)	9.6 (7.4-12.0)	42.6 (37.4-47.8)	30.6 (27.1-34.2)	10.4 (6.9-14.5)	3.4 (2.2-4.9)	3.4 (2.0-5.3)
Race/ethnicity								
Non-Hispanic black	783	4.3 (3.7-5.0)	1.9 (0.6-4.0) <sup>a</sup>	17.9 (12.3-24.3)	38.3 (32.2-44.6)	22.0 (15.8-29.0)	8.6 (6.0-11.7)	11.2 (5.9-18.0)
Mexican American	827	3.1 (2.7-3.5)	5.7 (3.1-8.9)	30.1 (24.8-35.8)	41.3 (37.6-45.2)	13.6 (10.1-17.4)	5.3 (3.3-7.6)	4.0 (1.8-6.9) <sup>a</sup>
Non-Hispanic white	631	2.3 (2.1-2.5)	10.2 (7.6-13.1)	44.7 (39.8-49.7)	31.6 (28.2-35.0)	8.9 (5.6-12.8)	2.4 (0.9-4.7)	2.3 (0.8-4.5) <sup>a</sup>
PIR								
$\leq 1.3$	1249	3.7 (3.3-4.2)	1.8 (0.7-3.5) <sup>a</sup>	27.2 (20.6-34.4)	35.3 (29.6-41.3)	19.4 (13.4-26.1)	8.3 (5.9-11.1)	7.9 (5.0-11.5)
$> 1.3$	1001	2.1 (2.0-2.3)	13.2 (10.0-16.8)	45.6 (41.7-49.6)	31.3 (28.0-34.7)	6.7 (4.4-9.6)	1.6 (0.7-2.9) <sup>a</sup>	1.5 (0.8-2.6)
Medicaid Status								
Yes	984	3.9 (3.5-4.3)	2.0 (0.7-4.0) <sup>a</sup>	23.0 (17.7-28.9)	36.5 (28.9-44.4)	20.8 (14.7-27.8)	9.1 (6.2-12.5)	8.5 (4.9-13.0)
No	1403	2.3 (2.1-2.5)	11.0 (8.5-13.9)	44.4 (40.0-48.7)	31.6 (28.1-35.2)	8.2 (6.4-10.1)	2.3 (1.3-3.6)	2.5 (1.2-4.3)
Year housing built (risk of lead exposure) <sup>b</sup>								
Low risk	744	2.0 (1.9-2.2)	13.7 (9.6-18.5)	49.4 (42.6-56.2)	27.7 (23.0-32.6)	6.0 (3.9-8.7)	1.6 (1.1-2.0)	1.6 (0.4-3.8) <sup>a</sup>
Medium risk	889	2.8 (2.6-3.0)	6.5 (4.0-9.7)	39.0 (31.5-46.8)	33.4 (27.4-39.8)	13.4 (8.9-18.8)	2.9 (1.5-4.9)	4.6 (2.6-7.2)
High risk	368	3.8 (3.1-4.5)	5.4 (1.5-11.6) <sup>a</sup>	20.9 (14.5-28.3)	36.3 (27.9-45.2)	18.3 (13.0-24.3)	10.4 (6.7-15.0)	8.6 (4.6-13.6)
Not known	351	3.6 (3.0-4.2)	1.5 (0.3-3.8) <sup>a</sup>	27.2 (17.7-37.8)	40.5 (32.5-48.9)	17.0 (10.9-24.2)	8.1 (4.0-13.5)	5.6 (1.3-12.6) <sup>a</sup>

<sup>a</sup> Relative SE of point estimate  $\geq 30\%$ ; does not meet standard of statistical reliability and precision.

<sup>b</sup> Risk of lead exposure by year housing built defined as follows:

NHANES 1999-2004: low risk, built 1978 and later; medium risk, built between 1950 and 1977; high risk, built before 1950.

NHANES III phases 1 and 2: low risk, built 1974 and later; medium risk, built between 1946 and 1973; high risk, built before 1946.

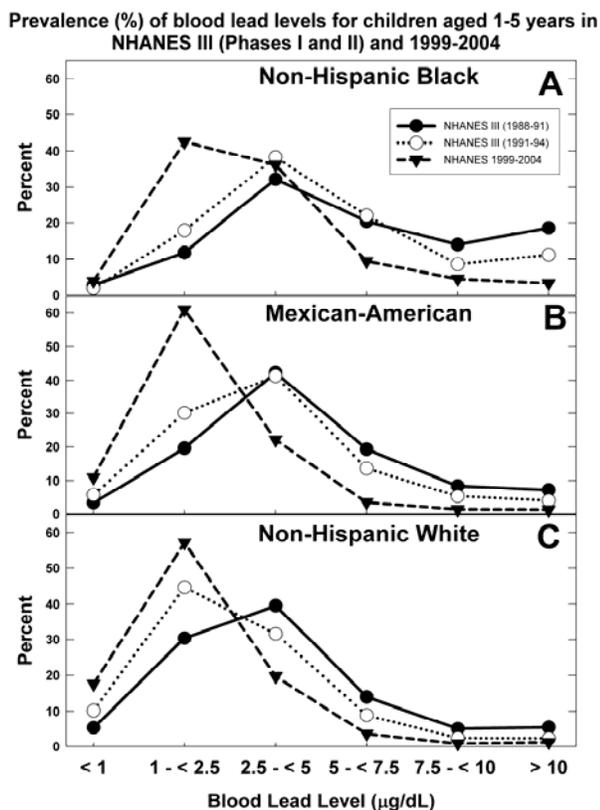
### Distribution of BLLs

In the 1999–2004 period, 14.0% of children ages 1 to 5 years had BLLs of  $< 1.0$   $\mu\text{g}/\text{dL}$ , 55.0% had BLLs of 1.0 to  $< 2.5$   $\mu\text{g}/\text{dL}$ , 23.6% had BLLs of 2.5 to  $< 5$   $\mu\text{g}/\text{dL}$ , 4.5% had BLLs of 5 to  $< 7.5$   $\mu\text{g}/\text{dL}$ , 1.5% had BLLs of 7.5 to  $< 10$   $\mu\text{g}/\text{dL}$ , and 1.4% had BLLs of  $\geq 10$   $\mu\text{g}/\text{dL}$  (Table 1). Differences in the distribution of BLLs across the 6 BLL categories were statistically significant across strata of age, race/ethnicity, income, Medicaid status, and housing risk (Table 1). A higher percentage of children with BLLs of  $\geq 10$   $\mu\text{g}/\text{dL}$  were non-Hispanic black (3.4% vs 1.2% for Mexican American and 1.2% for non-Hispanic white children), 1 to 2 years of age (2.4% vs 0.9% for 3 to 5 years of age), and enrolled in Medicaid (1.9% vs 1.1% for not enrolled in Medicaid); however, none of these differences achieved statistical significance ( $P > .05$ ).

Children with BLLs of  $< 1$   $\mu\text{g}/\text{dL}$  were more likely to be non-Hispanic white (17.6% vs 4.0% for non-Hispanic black children [ $P < .0001$ ] and 10.9% for Mexican American children [ $P < .01$ ]); not enrolled in Medicaid (17.6% vs 5.8% for enrolled in Medicaid;  $P < .0001$ ), and from higher income (PIR  $> 1.3$ ) families (19.9% vs 6.7% for being from low income families [PIR  $\leq 1.3$ ];  $P < .0001$ ). The geometric mean BLL was higher for non-Hispanic black children compared with non-Hispanic white children ( $P < .001$ ) and Mexican American children ( $P < .0001$ ); lower

income children compared with higher income children ( $P < .0001$ ); children enrolled in Medicaid compared with those not enrolled in Medicaid ( $P < .0001$ ); and children living in high-risk housing compared with those living in medium-risk ( $P < .0001$ ) and low-risk housing ( $P < .0001$ ) (Table 1).

Figure 1 provides a visualization of the distribution of BLLs using the categories defined for the analysis for each of the 3 race/ethnicity groups (non-Hispanic white, Mexican American, non-Hispanic black) for the 3 most recent cycles of the NHANES. By the 1999–2004 survey, the distributions of BLLs for non-Hispanic white and Mexican American children were similar. Although the geometric mean BLL for non-Hispanic black children in the 1999–2004 was still higher than those of non-Hispanic white children and Mexican American children (Table 1-3), the prevalence of elevated BLLs in non-Hispanic black children declined dramatically between NHANES III Phase I and the current NHANES, as noted above. The prevalence of BLLs of  $\geq 10 \mu\text{g/dL}$  in most of the high-risk groups (low income, Medicaid eligible, and high-risk housing) also declined dramatically.



**FIGURE 1.** The distribution of BLLs for the 3 race/ethnicity groups (A, non-Hispanic black; B, Mexican American; and C, non-Hispanic white) for the 3 most recent NHANES: 1988–1991, 1991–1994, and 1999–2004.

Likewise, the distributions of BLLs by markers of poverty status (PIR and Medicaid enrollment status) were lower (Table 1-3) by 1999–2004, although the prevalence in all categories for lower income children did not decline to the same extent as did prevalences for higher-income children. Similarly, BLLs for children living in high-risk housing also were lower, although not to the same extent as for children living in low- and medium-risk housing.

For the 1999–2004 NHANES, the overall geometric means were  $2.2 \mu\text{g/dL}$  (95% CI: 2.0-2.5) for 1999–2000,  $1.7 \mu\text{g/dL}$  (95% CI: 1.6-1.8) for 2001–2002, and  $1.8 \mu\text{g/dL}$  (95% CI: 1.6-1.9) for 2003–2004. The difference in geometric mean for 2003–2004 compared with 1999–2000 was statistically significant ( $P < .05$ ). Prevalences of elevated BLLs ( $\geq 10 \mu\text{g/dL}$ ) were 2.2% (95% CI: 1.2-3.4) in 1999–2000, 1.1% (95% CI: 0.6-2.0) in 2001–2002, and 1.2% (95% CI: 0.4-2.4) in 2003–2004. The relative SEs of the prevalence estimates for 2001–2002 and 2003–2004 was between 30% and 40% (data not shown). The prevalence of elevated BLLs for 2003–2004 did not differ statistically from that for 1999–2000 ( $P > .05$ ).

## Multivariable Regression Results

Results of the multivariable logistic and linear regression analyses are presented in Table 4. All risk factors associated with BLLs of  $\geq 10$   $\mu\text{g}/\text{dL}$  in bivariable analyses (Table 1–3) (being ages 1–2 years, being non-Hispanic black, having a PIR of  $\geq 1.3$ , and living in a moderate–risk [built ~ 1950–1977] or high–risk house [built before 1950]) were statistically significant in the multivariable logistic model. For housing risk, the odds of having a BLL of  $\geq 10$   $\mu\text{g}/\text{dL}$  was 1.8 times higher in children living in a moderate–risk house compared with a low–risk house (built ~ 1978 and later) and 5.9 times higher in children living in a high–risk house compared with a low–risk house. The decline in the proportion of children with BLLs of  $\geq 10$   $\mu\text{g}/\text{dL}$  across the 3 NHANES study periods was also statistically significant, with children in the NHANES III 1991–1994 being 60% less likely to have a BLL of  $\geq 10$   $\mu\text{g}/\text{dL}$  than children in the NHANES III 1988–1991; children in NHANES 1999–2004 were 90% less likely to have a BLL of  $\geq 10$   $\mu\text{g}/\text{dL}$  than children in the NHANES III 1988–1991. Likewise, in the multivariable linear model, all risk factors associated with higher geometric mean BLLs in bivariate analyses (Table 1-3) (the same risk factors as in the logistic models) were statistically significant. Non-Hispanic black children had higher BLLs than non-Hispanic white children, whereas Mexican American children had BLLs comparable to non-Hispanic white children across all 3 surveys. The increasingly negative linear regression coefficients ( $\beta$ ) corresponding to later NHANES survey periods (Table 4) reflected the decline seen in mean population BLLs from 1988 to 2004.

**TABLE 4. Summary of Multivariate Logistic and Linear Regression Models for BLLs, Children Aged 1 to 5 Years, NHANES 1988–2004**

Variable	Final Logistic Model (N = 4817)	Final Linear Model <sup>b</sup> (N = 4817)	
	Odds Ratio (95% CI) <sup>a</sup>	$\beta$ (SE)	P
Age			
1-2 y	2.7 (1.8-3.9)	0.21 (0.02)	< .0001
3-5 y	1.0	0.0	
Race/ethnicity			
Non-Hispanic black	3.2 (2.1-4.8)	0.38 (0.03)	<.0001
Mexican American	1.0 (0.6-1.8)	0.03 (0.04)	.45
Non-Hispanic white	1.0	0.0	
PIR			
$\leq 1.3$	2.8 (1.8-4.4)	0.37 (0.03)	< .0001
$> 1.3$	1.0	0.0	
Year housing built (risk of lead exposure)			
Low risk	1.0	0.0	
Medium risk	1.8 (1.0-3.1)	0.15 (0.03)	<.0001
High risk	5.9 (3.2-10.8)	0.51 (0.05)	<.0001
Survey			
NHANES III Phase 1 (1988–1991)	1.0	0.0	
NHANES III Phase 2 (1991–1994)	0.4 (0.2-0.7)	-0.31 (0.05)	<.0001
NHANES 1999–2004	0.1 (0.1-0.2)	-0.67 (0.05)	<.0001

<sup>a</sup> Dependent variable in logistic model was children with BLLs  $\geq 10$   $\mu\text{g}/\text{dL}$ .

<sup>b</sup> Dependent variable in linear models was natural log-transformed BLL;  $R^2$  of final model = 0.36.

With all main effects in the model, data from 4817 sampled children (67% of total number originally available for analysis) were available to compute estimates. These data included 321 sampled children with BLLs of  $\geq 10$   $\mu\text{g}/\text{dL}$  (70% of the total number with BLLs of  $\geq 10$   $\mu\text{g}/\text{dL}$  available for analysis). This smaller number of observations was primarily due to missing data on the year the housing was built. This categorical variable was missing for 14% in NHANES III 1988–1991, 16% in NHANES III 1991–1994, and 38% in NHANES 1999–2004. To assess the impact of the smaller samples that had missing information on the year housing was built, we used 2 strategies: (1) we compared the bivariable results from Table 1 through 3 to a bivariable analysis by using the 4817 children included in the multivariable analysis, and (2) we computed multivariable regression models that omitted the housing risk variable. In the bivariable analysis, the percentage of children with BLLs of  $\geq 10$   $\mu\text{g}/\text{dL}$  were affected more by the smaller sample (with these percents tending to be smaller than in the original bivariable analysis) than were geometric means, with differences being more evident for NHANES 1999–2004. However, inferences from the bivariable analysis using the smaller sample were comparable to the original bivariable analysis for both percent of children with BLLs of  $\geq 10$   $\mu\text{g}/\text{dL}$  and for geometric means. In the regression analyses that excluded housing risk, the primary impact on the logistic model was a reduced odds ratio for children ages 1 to 2 years compared with children ages 3 to 5 years. However, inferences were comparable to those from the models containing year housing built. In the linear models, excluding year housing built from the model reduced the model  $R^2$  from 0.36 to 0.27, but had little impact on the computed main effects compared with the models containing year housing built.

These analyses indicate that the year housing built category is an important predictor for elevated BLLs. However, missing data on this variable did not impact inferences for other risk factors.

### Children Previously Tested for Lead

Because the information on previous lead testing was first obtained in the NHANES 1988–1991, the percentage of 1- to 5-year-old children tested increased by 274% from 8.9% to 33.3% ( $P < .0001$ ) (Table 5). For non-Hispanic black children, the percentage tested increased from 21.0% in 1988–1991 to 43.6% in 1999–2004 ( $P < .0001$ ). In Mexican American children, the percentage tested increased dramatically, from 1.2% in 1988–1991 to 28.0% in 1999–2004 ( $P < .0001$ ). Among children enrolled in Medicaid in 1988–1991, 19.2% of children had had a blood lead test at some point before the NHANES examination; in 1999–2004 this percentage had increased to 41.9% ( $P < .001$ ). Among the small percentage of children with elevated BLLs ( $\geq 10$   $\mu\text{g/dL}$ ) in the NHANES, the percentage previously tested was 43.0% in 1999–2004, compared with 30.1% in 1988–1991; however, this difference was not statistically significant ( $P > .05$ ).

**TABLE 5. Percent With a Reported Previous Test for BLL According to Selected Demographic Characteristics, Children Aged 1 to 5 years, NHANES III Phase 1 (1988–1991), NHANES III Phase 2 (1991–1994), and NHANES 1999–2004.**

Population Group	NHANES III Phase 1 (1988–1991)		NHANES III Phase 2 (1991–1994)		NHANES 1999–2004	
	n <sup>a</sup>	% (95% CI)	n <sup>a</sup>	% (95% CI)	n <sup>a</sup>	% (95% CI)
All	2975	8.9 (5.3–13.4)	2701	11.5 (7.6–16.0)	3281	33.3 (29.1–37.7) <sup>p</sup>
Gender						
Female	1537	8.0 (4.4–12.5) <sup>b</sup>	1334	12.0 (7.7–17.2)	1613	33.6 (28.8–38.7) <sup>p</sup>
Male	1438	9.8 (5.9–14.6) <sup>b</sup>	1367	11.0 (7.1–15.6)	1668	33.1 (28.8–37.5) <sup>p</sup>
Age						
1–2 y	1311	8.1 (5.0–11.8) <sup>b</sup>	1166	11.8 (7.4–17.0)	1670	29.0 (24.8–33.4) <sup>b,c</sup>
3–5 y	1664	9.5 (5.3–14.7) <sup>b</sup>	1535	11.3 (7.1–16.2)	1611	36.3 (31.4–41.5) <sup>b,c</sup>
Race/ethnicity						
Non-Hispanic black	884	21.0 (13.7–29.3) <sup>b</sup>	865	24.6 (16.9–33.2)	954	43.6 (37.2–50.2) <sup>b,d,e</sup>
Mexican American	1078	1.2 (0.4–2.6) <sup>b,f</sup>	914	9.7 (5.8–14.6)	1021	28.0 (22.5–33.8) <sup>b,d</sup>
Non-Hispanic white	899	6.5 (2.2–12.9) <sup>b,f</sup>	750	7.6 (4.1–12.1)	992	31.8 (26.3–37.6) <sup>b,e</sup>
PIR						
$\leq 1.3$	1332	13.6 (9.4–18.4) <sup>b</sup>	1367	16.5 (10.4–23.8)	1574	38.8 (32.6–45.2) <sup>b,c</sup>
$> 1.3$	1322	6.4 (2.6–11.9) <sup>b,f</sup>	1164	7.8 (4.7–11.6)	1471	29.1 (24.4–34.1) <sup>b,c</sup>
Medicaid Status						
Yes	817	19.2 (12.0–27.7) <sup>b</sup>	1079	19.2 (13.1–26.0)	1214	41.9 (35.2–48.8) <sup>b,c</sup>
No	1693	6.3 (2.8–11.2) <sup>b,f</sup>	1616	8.3 (5.3–11.9)	2007	29.7 (25.7–33.9) <sup>b,c</sup>
Year housing built (risk of lead exposure) <sup>g</sup>						
Low risk	784	4.3 (1.2–9.1) <sup>b</sup>	855	5.4 (3.1–8.3)	891	25.9 (19.9–32.3) <sup>b,h</sup>
Medium risk	1267	9.4 (5.7–13.9) <sup>b,f</sup>	990	10.8 (7.0–15.4)	722	29.1 (23.6–35.0) <sup>b,i</sup>
High risk	507	18.0 (11.4–25.5) <sup>b</sup>	416	20.9 (13.5–29.4)	434	45.1 (36.0–54.3) <sup>b,h,i</sup>
Not known	211	20.4 (12.4–29.8)	396	17.2 (10.3–25.4)	1234	40.5 (35.3–45.9)
BLL, $\mu\text{g/dL}$						
$< 10$	1922	6.3 (3.4–10.1) <sup>b</sup>	2211	10.3 (6.5–14.8)	2330	32.6 (28.6–36.9) <sup>p</sup>
$\geq 10$	254	30.1 (17.7–44.1)	145	37.4 (24.2–51.8)	53	43.0 (22.9–64.5)

<sup>a</sup> Total *n* includes children both tested and not tested for blood lead in referent NHANESs.

<sup>b</sup> Difference between 1988–1991 and 1999–2004 statistically significant at  $P < .05$  after Bonferroni adjustment.

<sup>c</sup> Difference between strata in 1999–2004 statistically significant at  $P < .05$ .

<sup>d</sup> Difference between non-Hispanic black children and Mexican American children in 1999–2004 statistically significant at  $P < .05$  after Bonferroni adjustment.

<sup>e</sup> Difference between non-Hispanic black children and non-Hispanic white children in 1999–2004 statistically significant at  $P < .05$  after Bonferroni adjustment.

<sup>f</sup> Relative SE of point estimate  $\geq 30\%$ ; does not meet standard of statistical reliability and precision.

<sup>g</sup> Risk of lead exposure by year housing built defined as follows:

NHANES 1999–2004: low risk - built 1978 and later; medium risk - built 1950–1977; high risk - built before 1950.

NHANES III Phases 1 and 2: low risk - built 1974 and later; medium risk - built 1946–1973; high risk - built before 1946.

<sup>h</sup> Difference between low-risk housing and high-risk housing in 1999–2004 statistically significant at  $P < .05$  after Bonferroni adjustment.

<sup>i</sup> Difference between medium-risk housing and high-risk housing in 1999–2004 statistically significant at  $P < .05$  after Bonferroni adjustment.

## DISCUSSION

BLLs in US children continue to decrease most likely as a result of an intense coordinated effort to control or eliminate lead sources in children’s environments by government officials, health care and social service providers, and the communities most at risk. Although disparities have lessened, the mean BLLs and distribution of BLLs continue to be higher for low-income children, non-Hispanic black children, and children living in older housing stock (built before 1950). The analysis also indicated that the vast majority of US children still have some low-level

exposure to lead. Given that no "safe" BLL in children has been identified,<sup>3-5</sup> primary prevention of lead poisoning will play an important role in continuing lead control efforts.

Since the 1970s, the NHANES have provided valuable information on children's BLLs and risk factors for elevated BLLs in the United States. Because these surveys are based on a nationally representative sample, estimates can be generalized only to the US population; the sample is not designed to provide estimates for smaller geographic areas or specific populations where the risk of elevated BLLs is high. For example, 1 inner-city prevalence study in 2001 found that nearly 33% of children in 1 community had elevated BLLs<sup>12</sup> much higher than the national prevalence of 1.6% reported here. Therefore, it may not be appropriate to assume that local BLLs would be similar to the NHANES estimates. State and local surveillance data are needed to monitor local trends. For this reason, the CDC funds childhood lead poisoning prevention programs to include surveillance of BLLs. Data from CDC-funded surveillance programs consistently have shown that the risk for exposure to lead is not evenly distributed through the pediatric population.<sup>8,13,29</sup> When health care providers are determining which children to test for lead poisoning, they should assess whether a child has any known risk factors for lead poisoning.<sup>30-32</sup>

The percentage of children who have had a previous BLL test increased almost fourfold in the NHANES 1999–2004 compared with NHANES III Phase 1 (1988–1991). More importantly, markedly larger percentages of highest-risk children (eg, Medicaid-enrolled children, children from low-income families, and children from the NHANES with elevated BLLs) reported having had a previous blood lead test in the NHANES 1999–2004 compared with NHANES III Phase 1, although the increase was not statistically significant in children with elevated BLLs. Since 1997, the CDC has recommended that states develop plans to target testing to children at high risk (state testing plans can be accessed at [www.cdc.gov/nceh/lead](http://www.cdc.gov/nceh/lead)). The CDC and the Centers for Medicare and Medicaid Services also recommend that states link blood lead surveillance and Centers for Medicare and Medicaid Services claims data to identify children and areas where testing is inadequate.<sup>33</sup>

The data suggest that the recommendation for targeted rather than universal blood lead testing for preschool children has not resulted in a decrease in testing among children at highest risk. Nevertheless, fewer than half of children enrolled in Medicaid had been tested for lead poisoning. Federal regulations require that all children enrolled in Medicaid must receive a blood lead screening test at ages 12 and 24 months. All children aged 36 to 72 months who have not previously been screened must also receive a blood lead test. The American Academy of Pediatrics<sup>30</sup> and the CDC's Advisory Committee for Childhood Lead Poisoning Prevention<sup>31</sup> concur. No state is exempt from this testing requirement. A limitation of the testing prevalence estimates is that they are parent reported and may be biased. However, testing may be improved by monitoring testing by various providers and working to improve testing rates. For example, testing of children enrolled in Medicaid varies by the child's usual place of health care. A recent Rhode Island study found that although the percentage of enrolled children tested was high (80% had at least 1 blood lead test), testing varied by provider site: 68% for office-based physicians, 86% for neighborhood health centers, 86% for hospital-based clinics, and 91% for staff-model health maintenance organizations.<sup>34</sup>

Children can be exposed to lead from multiple sources. Because leaded house paint is a common high-dose source of exposure for children living in the United States, the focus of US public health efforts should continue to be on reducing exposure to leaded house paint and the dust and soil it contaminates.<sup>35-38</sup> However, there are other less-common sources of lead in the United States that also have high-lead content. Some CDC-funded childhood lead poisoning prevention programs have documented that lead in consumer products, imported toys, imported and traditional medicines and house wares, and "take-home" exposure for children whose parents work with lead have been identified for as many as 15% to 30% of children with elevated BLLs.<sup>39,40</sup> The single most important step to reduce children's BLLs is to identify and remove or control lead sources.<sup>41</sup>

Lead poisoning and other public health issues arising from environmental problems are often complex, costly, controversial, and require creative solutions. It is critical to incorporate human health concerns into environmental policy-making, as public health problems arise from, and solutions must be sought, beyond the health sector (eg, environmental, social, commercial, economic and political sectors).<sup>42</sup> The challenge is to develop strategies that can prevent children and adults from ever becoming poisoned by lead. Successful efforts combine epidemiologic

surveillance, source identification and reduction, regulatory enforcement, and a long-term government commitment to eliminating lead as a public health threat, especially to children.

## CONCLUSIONS

Children's BLLs continue to decline in the United States, even in historically high-risk groups for lead poisoning. To maintain progress made as well as eliminate remaining disparities, efforts must continue to test children at high risk for lead poisoning and identify and control lead sources that can poison children. Coordinated prevention strategies that are implemented at national, state, and local levels will help achieve the goal of elimination of elevated BLLs.

## REFERENCES

1. Agency for Toxic Substances and Disease Registry (ATSDR). *Toxicological profile for lead*. Atlanta, GA: ATSDR; 1999
2. Lidsky TI, Schneider JS. Lead neurotoxicity in children: basic mechanisms and clinical correlates. *Brain*. 2003;126(pt 1):5–19
3. Schwartz J. Low-level lead exposure and children's IQ: a meta-analysis and search for a threshold. *Environ Res*. 1994;65(1):42-55
4. Rogan WJ and Ware JH. Exposure to lead in children—how low is low enough? *N Engl J Med*. 2003;348(16):1515-1516
5. Lanphear BP, Hornung R, Khoury J, et al. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. *Environ Health Perspect*. 2005;113(7):849-899
6. US Department of Health and Human Services. *Healthy People 2010* (conference edition, 2 volumes). Washington, DC: US Department of Health and Human Services; 2000. Available at: [www.healthypeople.gov/document/html/objectives/08-11.htm](http://www.healthypeople.gov/document/html/objectives/08-11.htm). Accessed November 10, 2008
7. Centers for Disease Control and Prevention. Blood lead levels—United States, 1999–2002. *MMWR Morb Mortal Wkly Rep*. 2005;54(20):513-516
8. Meyer PA, Pivetz T, Dignam TA, Homa DM, Schoonover J, Brody D. Surveillance for elevated blood lead levels among children: United States, 1997—2001. *MMWR Surveill Summ*. 2003;52(10):1-21
9. Pirkle JL, Brody DJ, Gunter EW, et al. The decline in blood lead levels in the United States: the National Health and Nutrition Examination Surveys (NHANES). *JAMA*. 1994;272(4):284–291
10. US Department of Health and Human Services, Centers for Disease Control and Prevention. The Third National Report on Human Exposure to Environmental Chemicals, July 2005. Available at: [www.cdc.gov/exposurereport](http://www.cdc.gov/exposurereport). Accessed November 10, 2008
11. Iqbal S, Muntner P, Batuman V, Rabito FA. Estimated burden of blood lead levels  $\leq 5$   $\mu\text{g}/\text{dL}$  in 1999-2002 and declines from 1988–1994. *Environ Res*. 2008;107(3):305-311
12. Dignam TA, Evens A, Eduardo E, et al. High-intensity targeted screening for elevated blood lead levels among children living in 2 inner-city Chicago communities. *Am J Public Health*. 2004; 94(11):1945-1951
13. Robin LF, Beller M, Middaugh JP. Statewide assessment of lead poisoning and exposure risk among children receiving Medicaid services in Alaska. *Pediatrics*. 1997;99. Available at: [www.pediatrics.org/cgi/content/full/99/4/e9](http://www.pediatrics.org/cgi/content/full/99/4/e9)
14. Brown MJ, Shenassa E, Tips N. *Small Area Analysis of Risk for Childhood Lead Poisoning*. Washington, DC: Alliance to End Childhood Lead Poisoning; 2001
15. Pirkle JL, Kaufmann RB, Brody DJ, Hickman T, Gunter EW, Paschal DC. Exposure of the U.S. population to lead, 1991–1994. *Environ Health Perspect*. 1998;106(11):745–750

16. Brody DJ, Pirkle JL, Kramer RA, et al. Blood lead levels in the US population: phase I of the Third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991). *JAMA*. 1994;272(4):277-283
17. Kaufmann RB, Clouse TL, Olson DR, Matte TD. Elevated blood lead levels and blood lead screening among US children aged one to five years: 1988-1994. *Pediatrics*. 2000;106(6). Available at: [www.pediatrics.org/cgi/content/full/106/6/e79](http://www.pediatrics.org/cgi/content/full/106/6/e79)
18. United States General Accounting Office. *Medicaid: Elevated Blood Lead Levels in Children*. Washington, DC: US General Accounting Office, 1998:GAO publication no. (HEHS) 98-78
19. Canfield RL, Henderson CR, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentrations below 10 µg per deciliter. *N Engl J Med*. 2003;348(16):1517–1526
20. Rogan WJ, Dietrich KN, Ware JH, et al. The effect of chelation therapy with succimer on neuropsychological development in children exposed to lead. *N Engl J Med*. 2001;344(19):1421–1426
21. Lanphear BP, Dietrich K, Auinger P, Cox C. Cognitive deficits associated with blood lead concentrations <10 µg/dL in US children and adolescents. *Public Health Rep*. 2000;115(6):521–529
22. Bernard SM, McGeehin MA. Prevalence of blood lead levels  $\geq 5$  µg/dL among US children 1 to 5 years of age and socioeconomic and demographic factors associated with blood lead levels 5 to 10 µg/dL, Third National Health and Nutrition Examination Survey, 1988-1994. *Pediatrics*. 2003;112(6 pt 1):1308-1313
23. National Center for Health Statistics. *National Health and Nutrition Examination Survey, 2001–2002*. Hyattsville, MD: US Department of Health and Human Services, CDC. Available at: [www.cdc.gov/nchs/about/major/nhanes/nhanes01-02.htm](http://www.cdc.gov/nchs/about/major/nhanes/nhanes01-02.htm). Accessed November 10, 2008
24. Gunter EW, Lewis BL, Koncikowski SM. Laboratory methods used for the Third National Health and Nutrition Examination Survey (NHANES III), 1988-1994. In: CD-ROM 6-1078, *NHANES III Reference Manuals and Reports*. Hyattsville, MD: US Department of Health and Human Services, CDC; 1996
25. Miller DT, Paschal DC, Gunter EW, Stroud PE, D'Angelo J. Determination of lead in blood using electrothermal atomization atomic absorption spectrometry with L'vov platform and matrix modifier. *Analyst*. 1987;112(12):1701-1704
26. Nixon DE, Burritt MF, Moyer TP. The determination of mercury in whole blood and urine by inductively coupled plasma mass spectrometry. *Spectrochim Acta Part B At Spectrosc*. 1999;54(8):1141-1153
27. Brown LD, Cai TT, DasGupta A. Interval estimation for a binomial proportion. *Stat Sci*. 2001;16(2):101-133
28. Wolter KM. *Variance Estimation*. New York, NY: Springer; 1990
29. Centers for Disease Control and Prevention. Childhood lead poisoning prevention program. Available at: [www.cdc.gov/nceh/lead/lead.htm](http://www.cdc.gov/nceh/lead/lead.htm). Accessed November 10, 2008
30. American Academy of Pediatrics, Committee on Environmental Health. Lead exposure in children: prevention, detection and management. *Pediatrics*. 2005;116(4):1036-1046
31. Centers for Disease Control and Prevention, Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP). Recommendations for blood lead screening of young children enrolled in Medicaid: targeting a group at high risk. *MMWR Recomm Rep*. 2000;49(RR-14):1-13
32. Centers for Disease Control and Prevention. *Screening Young Children for Lead Poisoning; Guidance for State and Local Public Health Officials*. Atlanta, GA: CDC; 1997
33. Alliance for Healthy Housing. Lead screening for children in the Medicaid program: a tool kit (June 2001). Available at: [www.afhh.org/res/res\\_pubs/res\\_pubs\\_pv/medicaidtoolkit\\_pv.htm](http://www.afhh.org/res/res_pubs/res_pubs_pv/medicaidtoolkit_pv.htm). Accessed November 10, 2008
34. Vivier PM, Hogan JW, Simon P, Leddy T, Dansereau LM, Alario AJ. A statewide assessment of lead screening histories of preschool children enrolled in a Medicaid managed care program. *Pediatrics*. 2001;108(2). Available at: [www.pediatrics.org/cgi/content/full/108/2/e29](http://www.pediatrics.org/cgi/content/full/108/2/e29)

35. Bornschein RL, Succop P, Kraft KM, et al. Exterior surface dust lead, interior house dust lead and childhood lead exposure in an urban environment. In: Hemphill DD, ed. *Trace Substances in Environmental Health, XX. Proceedings of University of Missouri's 20<sup>th</sup> Annual Conference, June 1986*. Columbia, MO: University of Missouri; 1987
36. Lanphear BP, Matte TD, Rogers J, et al. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels. *Environ Res*. 1998;79(1):51-68
37. Lanphear BP, Weitzman M, Winter NL, et al. Lead-contaminated house dust and urban children's blood lead levels. *Am J Public Health*. 1996;86(10):1416-1421
38. Lanphear BP, Roghmann KJ. Pathways of lead exposure in urban children. *Environ Res*. 1997;74(1):67-73
39. Courtney JG, Ash S, Kilpatrick N, et al. Childhood lead poisoning associated with tamarind candy and folk remedies—California, 1999-2000. *MMWR Morb Mortal Wkly Rep*. 2002;51(31):684-686
40. Gletman PL, Brown MJ, Cochran J. Lead poisoning among refugee children resettled in Massachusetts, 1995-1999. *Pediatrics*. 2001;108(1):158-162
41. Centers for Disease Control and Prevention. Managing elevated blood lead levels among young children: recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention (2002). Available at: [www.cdc.gov/nceh/lead/CaseManagement/caseManage\\_main.htm](http://www.cdc.gov/nceh/lead/CaseManagement/caseManage_main.htm). Accessed November 10, 2008
42. Centers for Disease Control and Prevention; Agency for Toxic Substances and Disease Registry. *Global Health Activities Annual Report FY 1998*. Washington, DC: US Department of Health and Human Services; 1999