Exposure of the U.S. Population to Lead, 1991–1994

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Blood lead measurements were obtained on 13,642 persons aged 1 year and older who participated in Phase 2 of the Third National Health and Nutrition Examination Survey (NHANES III) from 1991 through 1994. NHANES III is a national representative survey of the civilian, noninstitutionalized U.S. population. The overall mean blood lead level for the U.S. population aged 1 year and older was 2.3 µg/dl, with 2.2% of the population having levels ≥10 µg/dl, the level of health concern for children. Among U.S. children aged 1–5 years, the mean blood lead level was 2.7 µg/dl, and 890,000 of these children (4.4%) had elevated blood lead levels. Sociodemographic factors associated with higher blood lead levels in children were non-Hispanic black race/ethnicity, low income, and residence in older housing. The prevalence of elevated blood lead levels was 21.9% among non-Hispanic black children living in homes built before 1946 and 16.4% among children in low-income families who lived in homes built before 1946. Blood lead levels continue to decline in the U.S. population, but 890,000 children still have elevated levels. Public health efforts have been successful in removing lead from population-wide sources such as gasoline and lead-soldered food and drink cans, but new efforts must address the difficult problem of leaded paint, especially in older houses, as well as lead in dust and soil. Lead poisoning prevention programs should target high-risk persons, such as children who live in old homes, children of minority groups, and children living in families with low income. *Key words*: air pollution, food contamination, gasoline lead, lead, leaded paint, lead poisoning, water pollution. *Environ Health Perspect* 106:745–750 (1998). [Online 19 October 1998]
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Human exposure to lead remains a serious public health problem. Lead adversely affects the nervous, hematopoietic, endocrine, renal, and reproductive systems of the body (*1,2*). Of particular concern are the effects of relatively low levels of lead exposure on cognitive development of children (*1,2*). Since the 1970s, federal regulations and abatement efforts have mainly focused on reducing the amount of lead in gasoline, paint, and soldered cans (*1,3*). In addition, federal programs have supported screening for lead poisoning in children by state and local health departments and physicians and lead abatement in housing (*1*). Currently, lead exposure usually results from lead in deteriorating household paint, lead at the workplace, lead used in hobbies, lead in some "folk" medicines and cosmetics, and lead in crystal or ceramic containers that leaches into water or food (*1*).

Since the late 1970s, the extent of lead exposure in the U.S. population has been assessed by the National Health and Nutrition Examination Surveys (NHANES) (*3–8*). These national surveys have measured blood lead levels (BPb) of tens of thousands of children and adults and assessed the extent of lead exposure in the civilian population by age, sex, race/ethnicity, income, and degree of urbanization. The surveys have demonstrated an overall decline in BPbs since the 1970s, but they also have shown that a large number of children continue to have elevated blood lead levels (≥10 µg/dl) (*3*).

We report here on the most recent blood lead data for the U.S. population from Phase 2 of the third NHANES (NHANES III), which sampled the U.S. population from 1991 to 1994. The BPbs are presented by sociodemographic characteristics for persons aged 1 year and older.

Methods

Survey design. NHANES III was a stratified, multistage probability cluster sample of households whose target population was civilian, noninstitutionalized persons residing in the United States. The 6-year survey consisted of two nationally representative samples: one in Phase 1, from October 1988 to September 1991, and a second in Phase 2, from October 1991 to September 1994. In NHANES III, non-Hispanic blacks, Mexican Americans, children aged 2 months–5 years, and persons aged 60 years and older were oversampled to increase the reliability of estimates for these groups (*9*). A detailed description of the sample design has been published (*10*). The survey included a household interview and a standardized physical examination conducted in a mobile examination center. Blood lead results from Phase 1 have previously been published (*3,4*), and selected findings from Phase 2 have been reported (*5*).

Laboratory methods. During the physical examination, a 1-ml sample of EDTA-anticoagulated whole blood was obtained by venipuncture from participants aged 1 year and older. Blood specimens were frozen and shipped on dry ice for analysis to the NHANES Laboratory, Division of Environmental Health Laboratory Sciences, National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, Georgia. Blood specimens remained frozen at -20°C until analyzed.

Lead was measured by graphite furnace atomic absorption spectrophotometry (GFAAS) using the method of Miller et al. (*11,12*). The GFAAS method included deuterium background correction and had a limit of detection of 1.0 µg/dl. The lead result is the mean of duplicate measurements. The blood lead measurements were calibrated by using standards prepared from lead nitrate Standard Reference Material 928 obtained from the National Institute of Standards and Technology, Gaithersburg, Maryland.

Demographic and socioeconomic covariates. Race/ethnicity was categorized as non-Hispanic black, non-Hispanic white, Mexican American, and other. Because of small sample size, persons not defined by the three largest U.S. race/ethnicity groups were included only in the estimates for the overall population. Income was categorized using the poverty–income ratio (PIR), defined as the ratio of total family income to the poverty threshold for the year of the interview. Low income was defined as a PIR ≤1.300, middle income as a PIR 1.301–3.500, and high income as a PIR ≥3.501. 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 (*13*).

Urban status was based on the U.S. Department of Agriculture codes that classify counties by total population and proximity to major metropolitan areas (*14*). The two categories were metropolitan areas with population ≥1 million and metropolitan and nonmetropolitan areas with a population <1 million. These urban status categories did not separate residence in central city areas from noncentral city areas. Year of housing construction was obtained from self-report

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using three categories: before 1946, 1946–1973, and after 1973.

Statistical analysis. Statistical analyses were performed using SAS software (*15*) and SUDAAN (*16*), a software package that incorporates the sample weights and adjusts the analyses for the complex sample design of the survey. Survey sample weights were used in all analyses to produce estimates that were representative of the noninstitutionalized, civilian U.S. population.

Geometric mean blood lead levels were calculated by taking the antilog of the mean of $\log_{10}$ of the BPb levels. Multivariate linear regression analyses were performed to determine the relationship between BPb levels and sociodemographic variables. Separate models were run for five age categories: 1–5 years, 6–11 years, 12–19 years, 20–49 years, and 50 years and older. Log_{10} of the BPbs was used as the dependent variable. Independent variables included age, sex, race/ethnicity, PIR, urban status, and age of housing categories. Both backward and forward stepwise regression were used to be sure the final model was independent of the stepwise regression approach.

Results

Blood lead levels of children. The geometric mean BPb for children ages 1–5 years was 2.7 µg/dl, and 4.4% of these children (890,000 in the U.S. population) had elevated BPbs (i.e, ≥10 µg/dl). Children aged 1–2 years had a geometric mean BPb of 3.1 µg/dl and 5.9% had elevated BPbs. For children aged 3–5 years, BPbs were slightly lower, with a geometric mean of 2.5 µg/dl, and 3.5% of these children had elevated BPbs.

Table 1 shows BPbs for children by selected demographic characteristics. Males and females had similar geometric mean BPbs. A greater percentage of males had elevated BPbs, but the difference was not statistically significant. Blood lead levels were highest in non-Hispanic black children, followed by Mexican–American children, with non-Hispanic white children having the lowest levels.

Children living in homes built before 1946 had higher BPbs, as did children living in lower income families. Children living in metropolitan areas with a population ≥1 million had similar geometric mean BPbs to children living in metropolitan and nonmetropolitan areas with a population <1 million. However, children living in the more populated metropolitan areas were more likely to have elevated BPbs, though this difference did not achieve statistical significance.

Table 2 presents the geometric mean BPbs and percentage of elevated BPbs for children stratified by selected demographic characteristics and age of housing. The prevalence of elevated BPbs was highest (21.9%) among non-Hispanic black children living in homes built before 1946. The prevalence of elevated BPbs among children living in older homes whose families had low incomes was also high (16.4%). Geometric mean BPbs were consistently higher in persons living in older homes and in non-Hispanic black, low income, and large urban population groups. In general, the trends in percentage of children with elevated BPbs that were observed by race/ethnicity and income persisted when these categories were stratified by age of housing.

Results of multiple regression analysis of children's BPbs are shown in the first column of Table 3. Final regression models showed that BPbs of children aged 1–5 years were significantly associated with age, non-Hispanic black race/ethnicity (compared to non-Hispanic whites), low income

Table 1. Blood lead levels of children aged 1–5 years by selected demographic characteristics: United States, Third National Health and Nutrition Examination Survey, Phase 2, 1991–1994

Population group	Sample size	Geometric mean blood lead level (µg/dl)	CI	% Children with blood lead levels ≥10 µg/dl	CI
All	2,392	2.7	2.5–3.0	4.4	2.9–6.6
Sex					
Female	1,181	2.7	2.4–2.9	3.3	2.1–5.0
Male	1,211	2.8	2.5–3.1	5.5	3.5–8.7
Race/ethnicity					
Black, non-Hispanic	783	4.3	3.7–5.0	11.2	6.7–18.7
Mexican American	827	3.1	2.7–3.5	4.0	2.2–7.2
White, non-Hispanic	631	2.3	2.1–2.5	2.3	1.0–5.0
Year housing built					
Pre-1946	368	3.8	3.1–4.5	8.6	5.2–14.2
1946–1973	889	2.8	2.6–3.0	4.6	2.9–7.5
After 1973	744	2.0	1.9–2.2	1.6	0.6–4.4
Income					
Low	1,249	3.8	3.3–4.2	8.0	5.4–11.7
Middle	740	2.3	2.1–2.5	1.9	1.1–3.2
High	261	1.9	1.7–2.1	1.0	0.3–3.4
Urban status					
Population ≥1 million	1,323	2.8	2.4–3.2	5.4	3.0–9.8
Population <1 million	1,069	2.7	2.3–3.0	3.3	1.5–7.0

CI, 95% confidence interval.

Table 2. Percentage of children aged 1–5 years with blood lead levels ≥10 µg/dl, by year housing was built and selected demographic characteristics: United States, Third National Health and Nutrition Examination Survey, Phase 2, 1991–1994

	Year housing built								
	Before 1946			1946–1973			After 1973		
Population group	Geometric mean	%	CI	Geometric mean	%	CI	Geometric mean	%	CI
Race/ethnicity									
Black, non-Hispanic	5.8	21.9	9.4–51.1	4.5	13.7	9.1–20.6	3.3	3.4	1.4–7.9
Mexican American	3.9	13.0	5.7–29.8	3.0	2.3	1.1–5.1	2.6	1.6	0.5–5.2
White, non-Hispanic	3.3	5.6	2.2–14.4	2.4	1.4	0.3–6.0	1.8	1.5	0.3–7.0
Income									
Low	5.5	16.4	9.9–27.2	3.6	7.3	4.6–11.4	3.0	4.3	2.1–9.1
Middle	2.9	4.1	1.3–12.8	2.4	2.0	1.0–4.1	1.9	0.4	0.1–1.3
High	3.1	0.9	0.1–6.5	1.9	2.7	0.6–11.3	1.6	–[a]	–[a]
Urban status									
Population ≥1 million	4.1	11.5	6.5–20.2	2.9	5.8	3.2–10.4	2.1	0.8	0.3–2.1
Population <1 million	3.5	5.8	2.0–16.8	2.7	3.1	0.9–10.1	2.0	2.5	0.7–9.6

CI, 95% confidence interval.
[a]Sample size too small for valid estimate.

(compared to high income), and residence in homes built before 1946 or from 1946 to 1973 (compared to homes built after 1973). As in the stratified analysis, sex and urban status were not significantly associated with BPbs in children. Season of the year was not a significant predictor of BPbs in children.

Blood lead levels of persons aged ≥1 year. The geometric mean BPb for persons aged ≥1 year was 2.3 µg/dl, and 2.2% of these persons had elevated BPbs. Table 4 presents BPbs for this age range by selected demographic characteristics. Geometric mean BPbs follow a U-shape, with BPbs higher among the very young and the very old. The prevalence of elevated BPbs had a similar U-shaped distribution. Males had higher mean levels than females and a greater prevalence of elevated BPbs, especially among those aged 12 and older (Fig. 1). The BPb trend with race/ethnicity followed the same pattern as for children, with highest levels seen in non-Hispanic blacks. Blood lead levels were also found to be higher among persons living in older homes and persons living in lower income households. Persons living in urban residences in more populated metropolitan areas had slightly higher geometric mean BPbs than those from less populated areas; the difference was of borderline statistical significance.

Figure 1 shows geometric mean BPbs stratified by age group and sex. The overall difference between males and females noted in Table 4 is seen in this figure to be more pronounced in persons aged 12 and older. Figure 2 shows geometric mean BPbs stratified by age group and race/ethnicity. In general, non-Hispanic blacks had the highest levels, with differences most pronounced in the age groups 1–2 years, 3–5 years, 50–59 years, 60–69 years and ≥70 years. Except for persons 60 years and older and those 20–29 years, Mexican Americans had BPbs intermediate between non-Hispanic blacks and non-Hispanic whites.

Table 5 shows BPbs for persons aged ≥1 year by age group, sex, and race/ethnicity. In these three characteristic stratifications, trends are similar to those seen when age group, sex, and race/ethnicity were examined separately (Table 4). The highest geometric mean BPbs were found in non-Hispanic black males (5.4 µg/dl for ages 1–2 years, 5.9 µg/dl for ages 50–69 years, and 6.6 µg/dl for ages ≥70 years). Table 6 provides a similar stratification by age, sex, and age of housing. In every age and sex subgroup, except ≥70 years, BPbs are higher in persons living in older homes. The U-shaped association of BPbs with age is found in each of the subgroups defined by sex and age of housing.

Results of multiple regression analysis for age groups 6–11 years, 12–19 years, 20–49 years, and ≥50 years are presented in Table 3. The multiple regression results for age,

Table 3. Regression coefficients (standard errors) from linear regression analysis of $\log_{10}$ blood lead by age group: United States, 1991–1994

	Age (years)				
Covariates	1–5	6–11	12–19	20–49	≥50
Intercept	0.359 (0.034)*	0.268 (0.052)*	0.015 (0.022)	-0.174 (0.031)*	0.140 (0.054)*
Age	-0.046 (0.008)*	-0.021 (0.006)*	NS	0.010 (0.001)*	0.003 (0.001)*
Race					
Non-Hispanic black	0.190 (0.032)*	0.180 (0.024)*	NS	0.077 (0.017)*	0.118 (0.030)*
Mexican American	NS	NS	NS	0.071 (0.018)*	NS
Non-Hispanic white[a]	0	0	0	0	0
Sex					
Male	NS	NS	0.188 (0.024)*	0.247 (0.012)*	0.172 (0.015)*
Female[a]	0	0	0	0	0
Income					
Low	0.201 (0.025)*	0.166 (0.034)*	0.199 (0.024)*	0.068 (0.012)*	0.094 (0.025)*
Middle	NS	NS	NS	NS	0.060 (0.019)*
High[a]	0	0	0	0	0
Year housing built					
Before 1946	0.258 (0.040)*	0.241 (0.038)*	0.070 (0.031)*	0.057 (0.018)*	NS
1946–1973	0.095 (0.021)*	0.096 (0.030)*	NS	NS	NS
After 1973[a]	0	0	0	0	0
Urban status					
Population ≥1 million	NS	NS	NS	NS	NS
Population <1 million[a]	0	0	0	0	0
Season of year					
Spring	NS	NS	NS	NS	NS
Summer	NS	NS	NS	NS	NS
Fall	NS	NS	NS	NS	0.061 (0.023)**
Winter[a]	0	0	0	0	0

NS, not statistically significant ($p>0.05$).
[a]Reference category for variables in group.
*$p< 0.01$; **$0.01<p<0.05$.

Table 4. Blood lead levels of the population by selected demographic characteristics: United States, Third National Health and Nutrition Examination Survey, Phase 2, 1991–1994

Population group	Sample size	Geometric mean blood lead level (µg/dl)	CI	% Persons with blood lead levels ≥10 µg/dl	CI
All	13,642	2.3	2.1–2.4	2.2	1.6–2.8
Age (years)					
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
Sex					
Female	7,384	1.9	1.8–2.0	0.9	0.6–1.3
Male	6,258	2.8	2.6–2.9	3.5	2.6–4.6
Race/ethnicity					
Black, non-Hispanic	4,458	2.8	2.6–3.0	5.2	4.0–6.9
Mexican American	3,923	2.4	2.3–2.6	2.9	2.1–4.0
White, non-Hispanic	4,553	2.2	2.0–2.3	1.5	0.9–2.3
Other	708	2.3	2.1–2.6	3.0	1.7–5.1
Year housing built					
Before 1946	2,551	2.6	2.4–2.8	3.3	2.3–4.7
1946–1973	5,532	2.3	2.2–2.5	2.2	1.6–3.0
After 1973	3,735	1.9	1.8–2.1	1.2	0.8–1.8
Income					
Low	4,390	2.6	2.5–2.8	4.5	3.7–5.4
Middle	4,460	2.2	2.1–2.3	1.8	1.3–2.6
High	2,188	2.1	2.0–2.2	0.7	0.4–1.2
Urban status					
Population ≥1 million	6,797	2.4	2.2–2.5	2.3	1.7–3.0
Population <1 million	6,845	2.2	2.0–2.4	2.0	1.3–3.3

CI, 95% confidence interval.

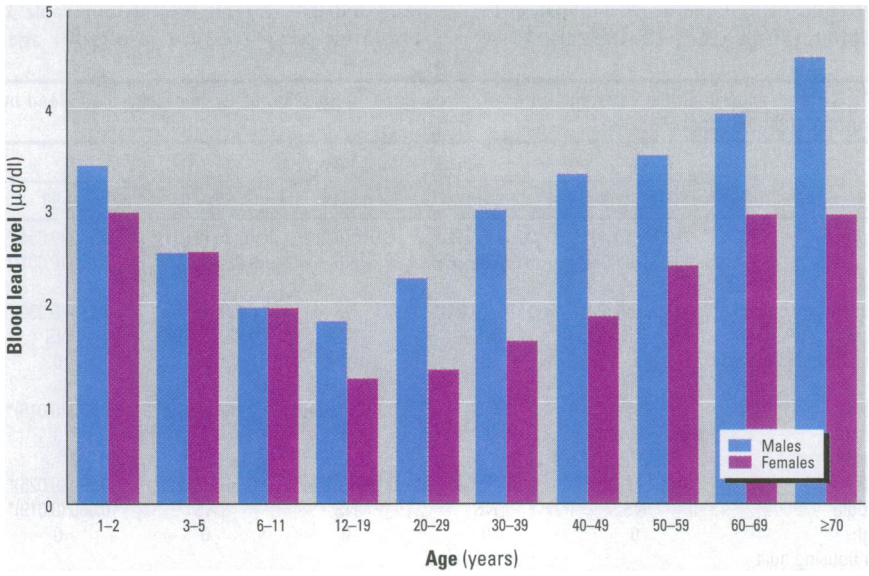

Figure 1. Geometric mean blood lead levels by age category and sex: United States, 1991–1994.

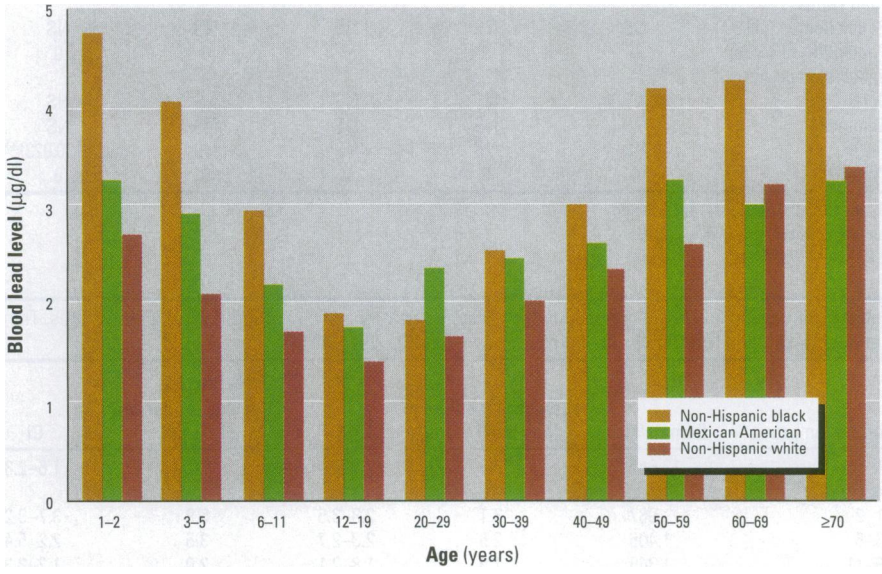

Figure 2. Geometric mean blood lead levels by age category and race/ethnicity: United States, 1991–1994.

race/ethnicity, and sex were similar to results for these variables in the stratified analysis (Tables 4 and 5; Fig. 1 and 2). For example, the regression coefficients for age are negative in age groups 1–5 years and 6–11 years, non-significant in age group 12–19 years, and positive in age groups 20–49 years and ≥50 years. Low income was significantly associated with high BPbs in every age group. Persons living in homes built before 1946 had higher BPbs in each age group except ≥50 years. In the five different age group regression models, season of the year was not significantly associated with BPbs except for the fall season in persons ≥50 years of age. Seasonal variation in BPbs has been noted in the late 1970s and early 1980s (*6,17*). The most likely explanation for the lack of significant seasonal variation in the current survey (1991–1994) is the minimal amount of lead remaining in gasoline. Lead in gasoline was a major source of lead in exposure in the late 1970s and early 1980s (*6*).

Discussion

The BPb levels of persons surveyed in NHANES III, Phase 2 (1991–1994) indicate continued success in reducing the exposure of the U.S. population to lead, but they also show that 4.4%, or 890,000, of children aged 1–5 years in the U.S. population still have BPb levels of health concern. The geometric mean BPb levels for children ages 1–5 years has declined from 3.6 µg/dl in NHANES III, Phase 1 (1988–1991) to 2.7 in Phase 2 (1991–1994). Over the same time interval, the percentage of children ages 1–5 years with elevated BPbs decreased from 8.6% to 4.4%. Corresponding changes for persons aged ≥1 year were a decrease in the geometric mean from 2.9 to 2.3 µg/dl and a decrease in the percentage of persons with BPbs ≥10 µg/dl from 4.4% to 2.2%.

The decline in BPbs in the U.S. population since the late 1970s is attributable to multiple efforts to reduce exposure to lead. The reductions in the amount of lead in gasoline, residential paint, food and soft-drink cans, and plumbing systems greatly contributed to decreased lead exposure for the overall population (*1,3,18–22*). From 1976 to 1990, the amount of lead in gasoline decreased by 99.8% (*18*). In 1980, 47% of food and soft-drink cans were lead soldered. By 1991, lead-soldered food and soft-drink cans were no longer manufactured in the United States (*20,21*). In addition, treatment of water to make it less corrosive to pipes, regulations to protect workers from lead exposure, and childhood lead poisoning prevention programs have helped to decrease lead exposure. Continuing decreases in air lead levels and in the percentage of the population that lives in pre-1950 housing have likely contributed to the continuing decline in BPbs since NHANES III, Phase 1 (*23,24*).

The data reported here indicate that some population groups continue to be disproportionately at high risk for elevated lead exposure. In general, these are people with low income, people of non-Hispanic black race, and people who live in older housing. In previous studies (*3,4*), residence in a central city with a population of more than 1 million people was also found to be a risk factor for lead exposure. In this study, residence in an urban area was defined more broadly to be consistent with the U.S. Department of Agriculture codes that classify counties by total population and proximity to major metropolitan areas (*14*). The two residence categories were metropolitan areas with population ≥1 million and metropolitan and non-metropolitan areas with a population <1 million. Urban residence defined this way was only of borderline significance as a risk factor.

These high-risk population groups are important to recognize for efficient targeting of public health efforts in lead poisoning prevention (*1,25*). Our results also emphasize the importance of leaded paint, especially in older homes, as a continuing source of lead exposure. 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 (*26*). Between 5 and 15 million homes contain paint that has deteriorated to the point of being a health hazard (*27*). Hazard control costs are highly variable, depending on the extent of the

Table 5. Geometric means and 95% confidence intervals (CI) of blood lead levels (μg/dl) for persons 1 year of age and older by age category, sex, and race/ethnicity: United States, 1991–1994

		Non-Hispanic white			Non-Hispanic black			Mexican American		
	Age (years)	Sample size	Geometric mean	CI	Sample size	Geometric mean	CI	Sample size	Geometric mean	CI
Males	1–2	163	2.8	2.5–3.2	148	5.4	4.6–6.3	172	3.6	3.1–4.1
	3–5	153	2.1	1.7–2.4	246	4.3	3.6–5.0	248	3.1	2.6–3.5
	6–11	162	1.7	1.5–1.9	291	3.2	2.7–3.5	201	2.2	1.8–2.7
	12–19	149	1.7	1.4–1.9	315	2.5	2.1–2.8	246	2.4	2.2–2.5
	20–49	526	2.7	2.4–2.9	663	3.3	3.0–3.6	732	3.3	3.1–3.5
	50–69	399	3.4	3.1–3.6	245	5.9	5.2–6.7	249	4.3	3.9–4.8
	≥70	423	4.4	4.2–4.6	112	6.6	5.5–7.8	105	4.0	3.3–4.7
Females	1–2	139	2.6	2.0–3.2	145	4.2	3.4–5.0	158	3.0	2.4–3.5
	3–5	176	2.1	1.9–2.3	244	3.9	3.2–4.6	249	2.9	2.4–3.3
	6–11	148	1.7	1.4–2.0	294	2.8	2.4–3.1	178	2.2	1.9–2.5
	12–19	192	1.2	1.0–1.3	360	1.5	1.3–1.7	260	1.4	1.2–1.5
	20–49	801	1.5	1.4–1.6	1,001	1.8	1.7–2.0	770	1.7	1.6–1.8
	50–69	483	2.5	2.3–2.7	278	3.3	2.7–3.8	255	2.4	2.1–2.7
	≥70	639	2.9	2.7–3.1	116	3.3	2.8–3.8	100	2.7	2.4–3.0

Table 6. Geometric means and 95% confidence intervals (CI) of blood lead levels (μg/dl) for persons 1 year of age and older by age category, sex, and year housing built: United States, 1991–1994

		House built before 1946			House built 1946–1973			House built after 1973		
	Age (years)	Sample size	Geometric mean	CI	Sample size	Geometric mean	CI	Sample size	Geometric mean	CI
Males	1–2	93	4.1	3.3–4.8	184	3.6	3.0–4.3	389	2.6	2.2–3.1
	3–5	113	3.8	2.9–4.8	260	2.5	2.1–2.8	197	1.7	1.5–1.9
	6–11	112	2.6	2.2–3.0	262	2.0	1.7–2.2	207	1.4	1.3–1.6
	12–19	126	2.1	1.6–2.5	293	1.9	1.5–2.3	221	1.6	1.4–1.8
	20–49	327	3.2	2.8–3.5	749	2.8	2.4–3.1	645	2.5	2.2–2.7
	50–69	188	4.1	3.6–4.6	478	3.7	3.3–4.1	190	3.3	3.0–3.6
	≥70	213	4.7	4.3–5.0	319	4.3	3.9–4.7	82	4.9	4.4–5.5
Females	1–2	60	5.2	4.2–6.1	177	2.9	2.5–3.4	152	2.2	1.9–2.4
	3–5	102	2.9	2.3–3.5	268	2.6	2.3–2.9	232	2.0	1.7–2.2
	6–11	112	2.6	2.1–3.2	232	1.9	1.5–2.2	196	1.4	1.2–1.7
	12–19	147	1.5	1.1–1.9	338	1.3	1.1–1.4	261	1.1	0.9–1.2
	20–49	430	1.7	1.5–1.8	1,026	1.6	1.5–1.7	853	1.5	1.3–1.6
	50–69	242	2.9	2.5–3.3	557	2.5	2.4–2.7	197	2.5	1.9–3.0
	≥70	286	2.9	2.6–3.2	389	2.8	2.6–3.0	139	2.9	2.5–3.2

intervention, existing market conditions (such as degree of competition among contractors and licensing laws), type of housing, and associated housing rehabilitation work. However, median costs range from several hundred dollars for spot repairs to around $10,000 for complete abatement of paint with window replacement (*28,29*).

Because of the high cost of abatement, the scarcity of adequately trained lead-abatement professionals, and the absence (until 1995) of federal guidelines for implementing less costly methods of leaded paint hazard containment, residential lead-paint-abatement efforts have focused on homes in which there is a resident child with an elevated BPb (i.e., secondary prevention), rather than on homes that have the potential to expose a child to lead (i.e., primary prevention). Similarly, publicly funded lead poisoning prevention programs have focused on screening children to identify those who already have elevated BPbs so that they may receive interventions, rather than on preventing future lead exposure among children without elevated levels. However, at least some of the adverse health effects that occur even at relatively low levels of lead may be irreversible (*30*). As the number of children with elevated BPbs has decreased, lead poisoning prevention programs may now be able to redirect some of their resources to primary prevention, thus protecting many young children from needless health damage. Still, secondary prevention efforts through screening will remain vitally important to ensure that children with elevated BPbs receive prompt and effective interventions to reduce further lead exposure and minimize health consequences.

As this analysis demonstrates, the risk for lead exposure is not spread evenly throughout the pediatric population. Rather, lead hazards can be localized within neighborhoods, largely due to such factors as housing conditions, industrial emissions, or dust and soil contamination; more rarely, lead hazards occur sporadically as a result of such factors as parental activities and use of leaded ceramics, cosmetics, or folk medicines (*1*). In accordance with these findings, the Centers for Disease Control and Prevention recently advised health departments to evaluate the distribution of risk for lead poisoning in their communities and devise locally appropriate screening recommendations (*25*). Universal blood lead screening is recommended only in high-risk areas; in generally low-risk areas, screening should be targeted to those individual children at high risk. Physicians are advised to follow the recommendations of their state and local health departments to determine which of their pediatric patients should be screened.

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