Prevalence of Blood Lead Levels ≥5 μg/dL Among US Children 1 to 5 Years of Age and Socioeconomic and Demographic Factors Associated With Blood of Lead Levels 5 to 10 μg/dL, Third National Health and Nutrition Examination Survey, 1988–1994

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ABSTRACT. *Objectives.* **As part of an investigation into the impact of a potential revision in federal childhood lead poisoning prevention policy that would result in screening children for blood lead levels (BLLs) ≥5 μg/dL rather than the current 10 μg/dL, we analyzed the most recent available, nationally representative data to identify prevalence of BLLs ≥5 μg/dL and socioeconomic and demographic characteristics of 1- to 5-year-old children with BLLs ≥5 but <10 μg/dL.**

Methods. **We performed statistical analyses on data from the Third National Health and Nutrition Examination Survey (NHANES III) (1988–1994) to describe trends in BLLs ≥5 μg/dL overall and among subpopulations of children <6 years old and to compare risk factors for falling within 1 of 3 groups of children (those with BLLs ≥5 but <10 μg/dL; ≥10 but <20 μg/dL; and ≥20 μg/dL) using the group reported as 0.7 to <5 μg/dL as the referent.**

Results. **Overall prevalence of BLLs ≥5 μg/dL among 1- to 5-year-old children was 25.6%, although most (76%) of these children had BLLs <10 μg/dL. Children with BLLs ≥5 μg/dL included 46.8% of non-Hispanic black children, 27.9% of Mexican American children, and 18.7% of non-Hispanic white children; 42.5% of children in housing built before 1946, 38.9% of children in housing built between 1946 and 1973, and 14.1% of children in housing built after 1973 had BLLs ≥5 μg/dL. Compared with non-Hispanic white children, non-Hispanic black children were 3 times more likely to have a BLL ≥5 but <10 μg/dL, 7 times more likely to have a BLL of 10–20 μg/dL, and 13.5 times more likely to have a BLL ≥20 μg/dL. Similar increases in the association between risk factor and BLL were seen with respect to other known risk factors including age of housing, region of the country, and poverty.**

Conclusions. **The high prevalence of BLLs ≥5 μg/dL overall and within US subpopulations will be an important variable in any change in screening and intervention criteria. However, most children with BLLs ≥5 μg/dL are below the current intervention level of 10 μg/dL. Exposure to lead from multiple sources is suggested by the prevalence of BLLs ≥5 μg/dL but <10 μg/dL among children with uncertain risk factors. The probable presence of one or more known risk factors for childhood lead poisoning increases as BLL increases.** ***Pediatrics*** **2003;112:1308–1313;** ***lead, child, prevalence, environment, NHANES III.***

ABBREVIATIONS. BLL, blood lead level; NHANES, National Health and Nutrition Examination Survey; CDC, US Centers for Disease Control and Prevention; CI, confidence interval; PIR, poverty income ratio; OR, odds ratio.

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BACKGROUND

Past uses of lead in interior and exterior paints, plumbing, food and beverage containers, and gasoline in the United States resulted in ubiquitous environmental lead exposure during the 20th century and continuing into the 21st.[1,2] Although these uses have largely been prohibited, childhood lead exposure persists because of the continuing presence of leaded paint in housing and residual lead contamination in soil resulting from industrial and transportation emissions.[3]

As a result of federal regulatory measures to reduce population exposure to environmental lead, overall childhood blood lead levels (BLLs) have since declined. Screening data from the late 1960s and early 1970s found that 20% to 45% of children tested had BLLs ≥40 μg/dL. Between 1976 and 1980, the weighted geometric mean BLL among 1- to 5-year-old children in the US was 14.9 μg/dL.[2] Almost all (95%) the children had BLLs ≥8 μg/dL and 88.2% had BLLs ≥10 μg/dL. Data from the Third National Health and Nutrition Examination Survey (NHANES III), phase 1 (1988–1991) showed a decline in the geometric mean BLL in 1- to 5-year-old children to 3.6 μg/dL.[2] NHANES III, phase 2 data (1991–1994) showed a further decline in geometric mean BLL to 2.7 μg/dL.[4] The NHANES III, phase 2 data indicated that ~4.4% of 1- to 5-year-old children (~890 000 children) had BLLs ≥10 μg/dL.[4]

The NHANES III data showed that certain children, particularly non-Hispanic black and Mexican American children, children from low-income families, and children living in urban areas, were at higher risk for BLLs ≥10 μg/dL (28.4%).[3,4] The decline in prevalence of BLLs ≥10 μg/dL from phase 1 to phase 2 was somewhat higher in these subpopulations than in the total 1- to 5-year-old group.[4]

Previously published analyses of NHANES III data with respect to childhood blood lead do not focus on trends in BLLs <10 μg/dL, which are below the current level to which federal lead screening and intervention activities are tied.[5] The question of whether to lower the screening and intervention level to 5 μg/dL is under discussion within the childhood lead poisoning prevention community. In this article we evaluate 1) the prevalence of BLLs ≥5 μg/dL, which is critical to developing an appropriate screening strategy (eg, in determining the need for universal versus targeted screening); and 2) socioeconomic and demographic trends among US 1- to 5-year-old children with BLLs ≥5 but <10 μg/dL (ie, the population that would be newly defined as at-risk).

METHODS

NHANES III was a stratified, multistage probability sample survey of the noninstitutionalized US population aged ≥2 months conducted by the Centers for Disease Control and Prevention (CDC) National Center for Health Statistics between 1988 and 1994.[3,6] NHANES III provides the most recent primary source of national blood lead data.[5]

The sample population included a total of 5787 children 1–5 years old who participated in a household interview (of a parent/guardian) and in a standardized physical examination, including the taking of a venous blood sample, conducted in a "mobile examination center." Details of the data collection and analysis are provided elsewhere.[6–10] Of the total sample, 1163 or 21% (weighted) refused or did not have valid blood lead samples, leaving 4624 children in the sample. Possible bias attributable to differences between children from whom blood was drawn and other children in the sample population are discussed below.

We explored the significance of a number of variables concerning the child's age and sex; nutritional status; health care status; family poverty and characteristics of parent/guardian such as education level; race/ethnicity (under NHANES III, available categories were non-Hispanic white, non-Hispanic black, and Mexican American)[6]; residence in a metropolitan area with a population ≥1 million; region of the country (Northeast, Midwest, South, or West); residence in current home or city/town/area >1 year; and age of housing.[11] Because mobile examination center examinations took place in the Northeast and Midwest (the regions with the highest BLLs) during the summer and fall (the seasons with the highest BLLs),[3] we could not investigate the association between season and BLLs found in some studies.[3,12–15]

We incorporated variables significantly associated in bivariate analyses with elevated BLLs (see Table 1) into analyses stratified separately by race/ethnicity, poverty, and age of housing. When stratified by race/ethnicity, having a regular clinic or physician and having private insurance were no longer significant, and a history of anemia was only marginally significant. When stratified by poverty, having a regular clinic or physician, having private insurance, having a history of anemia, and reported incidence of food insufficiency were no longer significant. All variables remained significant when stratified by age of housing.

Finally, we performed a multivariate logistic regression to identify independent, significant risk factors and odds ratios (ORs) for belonging in each of 3 elevated blood lead groups (BLLs ≥5 but <10 μg/dL; ≥10 but <20 μg/dL; and ≥20 μg/dL, as compared with a reference group with BLLs reported as 0.7 to <5 μg/dL). A sample population of 2529 of the full 5787 children was included in this regression, in which race/ethnicity, age, housing age, poverty, Medicaid coverage, education level of the reference adult, region of the country, and the presence of an adult smoker in the home were fitted into the model.

We used in our analysis public access data files,[16] imported into a SAS dataset (SAS Institute, Inc, Release 8.1, Cary, NC) using Statistical Export and Tabulation System (SETS) Version 1.22a retrieval software. Statistical analyses were performed by using SAS and SUDAAN (Research Triangle Institute, Release 7.5.4, Research Triangle Park, NC) software. Use of the SUDAAN software is recommended because of the complex sample design of NHANES III to enable analysts to use sample weights.[6,17] Sample weighting was used in NHANES III to compensate for oversam-

TABLE 1. Variables Significantly Associated in Bivariate Analysis With Elevated BLL Groups Among Children 1–5 Years Old, NHANES III 1988–1994

Variable*	*N*	% Response (Weighted)	*n* (+ Blood Lead)	*P* Value
Race/ethnicity†	5496	89.1	4381	<.001
Age‡	5787	100	4624	<.001
Maternal age at birth§	5751	99.4	4596	<.001
Adult smoker in home	5782	99.9	4620	<.001
Education of reference person‖	5787	100	4545	<.001
Poverty level¶	5285	94	4273	<.001
Family income#	5285	94	4273	<.001
Medicaid coverage	4163	70.5	3435	<.001
Private insurance	4891	90.2	3954	<.001
Regular health clinic	5787	100	4624	.03
Regular physician	5269	94.5	4209	.04
Take vitamin supplement	5766	99.8	4608	<.001
History of anemia	5764	99.7	4606	.01
Reported hunger	5780	99.9	4619	<.001
Participation in WIC**	5775	99.9	4615	<.001
Prior lead test	5676	97.6	4534	<.001
Age of housing††	4914	87.6	3912	<0.001
Time at home address‡‡	5759	99.6	4608	<0.001
Region of country§§	5787	100	4624	<0.001

* Where not specified, yes/no response.
† Non-Hispanic black, non-Hispanic white, and Mexican American. Excludes "other" category.
‡ 1–2 years old; 3–5 years old.
§ ≤21 years; >21 years old.
‖ High school graduate; non-high school graduate.
¶ Below or at PIR; above PIR.
# <$10 000/y; $10 000 to <$30 000/y; $30 000 to <$50 000/y; $50 000+/y.
** Program on Women, Infants, and Children.
†† Before 1946; 1946–1973; 1974 to present.
‡‡ ≤1 year; >1 year.
§§ Northeast, South, Midwest, and West.

pling among some subgroups, reduce nonresponse biases, bring sample data in line with target population totals, compensate for inadequacies in sample design, and reduce variances in the estimation procedure.[6]

RESULTS

Of the total weighted population of 1- to 5-year-old children, 25.6% had BLLs ≥5 μg/dL (see Table 2). Forty-seven percent of non-Hispanic black 1- to 5-year-old children and 28% of Mexican American children had BLLs ≥5 μg/dL compared with 19% of non-Hispanic white children. Among the 1- to 2-year-old children, 32% had BLLs ≥5 μg/dL compared with 21.5% of 3- to 5-year-old children. The proportion of children with BLLs ≥5 μg/dL increased with age of housing: 42.4% of children in housing built before 1946 had BLLs ≥5 μg/dL compared with 26.4% living in housing built between 1946 and 1973 and 14% of children living in housing built since 1973. Yet overall and within each of these age, race/ethnicity, and housing subcategories, most of the children with BLLs ≥5 μg/dL were below the 10 μg/dL level. For example, although almost 26% of children overall had BLLs ≥5 μg/dL, many fewer (6.3%) had BLLs ≥10 μg/dL. Thus, as Table 2 indicates, a change in the CDC's recommended threshold screening and intervention level from 10 μg/dL to 5 μg/dL would result in a broad change in the number of children identified as at-risk, although the majority of those children (76%) remain below the current screening and intervention level.

Given that such a change in intervention level would be targeted at the children whose BLLs are ≥5 μg/dL but <10 μg/dL, it is also useful to look at how those children are similar to or different from children with higher or lower BLLs. Table 3 shows the proportion of children within each risk category falling into 1 of the 4 groups of BLL (including the <5 μg/dL control group) (indicating that, for example, 30% of the children in the Northeast and 12% of the children in the West have BLLs ≥5 but <10 μg/dL). Table 4 shows, conversely, the proportion of children within each blood lead grouping who fall within one of the risk variables. For example, although 69% of the weighted sample population was non-Hispanic white, 75% of the children with BLLs <5 μg/dL were non-Hispanic white. In contrast, 19% of the weighted sample population was non-Hispanic black, but 65% of the group with BLLs ≥20 μg/dL were non-Hispanic black children.

The results of the multiple logistic regression are set forth in Table 5. Our analysis indicated that the association between known risk factors and BLL in-

TABLE 2. Prevalence (%) of BLLs Elevated ≥5 μg/dL and ≥10 μg/dL, NHANES III (1988–1994)

Variable	Category	≥5 μg/dL	≥10 μg/dL
Race/ethnicity	Non-Hispanic black	47	14
	Mexican American	28	5
	Non-Hispanic white	19	4
Age	1–2 y	32	8
	3–5 y	22	5
Age of housing	Pre-1946	43	13
	1946–1973	26	6
	1974 to present	14	3
Medicaid participants	Yes	42	13
	No	16	3
Private insurance	Yes	20	5
	No	33	8
Region	Northeast	43	13
	Midwest	31	10
	South	22	4
	West	14	2
Total		26	6

TABLE 3. Percent of the Population Within Each Variable That Falls Within Each of the 4 Blood Lead Group Categories, NHANES III, 1988–1994

Variable	Blood Lead Groupings (μg/dL)			
	<5	5–10	10–20	≥20
Race/ethnicity				
Non-Hispanic black	53	32	12	2.5
Mexican-American	72	23	5	0.5
Non-Hispanic white	81	15	4	0.3
Age				
1–2 y	68	24	7	1
3–5 y	78	16	5	0.5
Housing				
Pre-1946	58	30	11	1.7
1946–1973	74	20	6	0.6
1974 to present	86	11	3	<0.1
Health insurance				
Medicaid	58	30	11	2
Private insurance	80	15	4	0.5
Poverty				
At or below PIR	58	30	11	1.5
Above PIR	82	14	3	0.3
Total family income				
<$10 000/y	53	34	12	1.4
$10	72	22	6	0.7
$10 000–<$130 000/y				
$30 000–<$50 000/y	83	14	3	0.4
≥$50 000/y	90	8	2	0.3
Region				
Northeast	57	30	11	1.3
Midwest	69	21	8	1.4
South	78	18	4	0.2
West	86	12	2	0.2

Totals may not exactly match totals in Table 2 because of rounding.

TABLE 4. Proportion of Blood Lead Grouping Comprised of a Particular Variable Subset

Variable	Blood Lead Groupings (μg/dL)			
	<5	5–10	10–20	≥20
Race/ethnicity				
Non-Hispanic black (19%)	14	32	43	65
Mexican American (12%)	11	14	11	8
Non-Hispanic white (69%)	75	54	46	26
Housing				
Pre-1946 (21%)	16	34	41	56
1946–1973 (39%)	38	43	38	39
1974 to present (40%)	46	24	22	5
Health insurance				
Medicaid (39%)	30	60	74	88
Private insurance (73%)	77	63	64	51
Poverty				
Below PIR	41	63	73	88
At or above PIR	60	38	27	13
Region				
Northeast (16%)	13	25	33	32
Midwest (24%)	22	26	36	51
South (35%)	37	33	23	9
West (25%)	29	15	8	8

Numbers will not add up to 100% because of rounding.

TABLE 5. Odds and 95% CIs of Being in 1 of 3 Ordinal BLL Groups Versus in Lowest BLL Group Among Children 1–5 Years Old (NHANES 1988–1994)

Variable	Category	5 < 10 μg/dL	10 < 20 μg/dL	≥20 μg/dL	*P* Value (Wald χ^2)
Race/ethnicity	Non-Hispanic black	3.3 (2.1, 5.4)	7.3 (3.4, 15.4)	13.5 (4.7, 38.5)	<.001
	Mexican American	2.4 (1.4, 4.2)	2.5 (0.9, 6.7)*	1.8 (0.3, 9.4)*	
	Non-Hispanic white	Reference	Reference	Reference	
Age	1–2 y	1.9 (1.4, 2.6)	2.6 (1.7, 4.2)	2.1 (1.0, 4.6)	<.001
	3–5 y	Reference	Reference	Reference	
Age of housing	Before 1946	4.4 (2.9, 6.8)	5.1 (2.7, 9.8)	15 (2.8, 80.8)	<.001
	1946–1973	2.2 (1.5, 6.8)	0.9 (1.7, 3.9)*	3.7 (0.8, 17.5)*	
	1974 to present	Reference	Reference	Reference	
Poverty	At or below PIR	1.6 (1.0, 2.5)	2.7 (1.2, 6.0)	2.1 (0.7, 6.7)*	<.04
	Above PIR	Reference	Reference	Reference	
Adult smoker in home	Yes	1.3 (0.9, 1.9)*	1.6 (1.1, 2.4)	2.8 (1.3, 6.3)	<.01
	No	Reference	Reference	Reference	
Education level of reference adult	Non-high school graduate	1.5 (1.0, 2,2)	2.2 (1.3, 3.8)	1.9 (0.8, 4.6)*	<.03
	High school grad	Reference	Reference	Reference	
Medicaid coverage	Yes	1.9 (1.2, 3.2)	2.4 (1.2, 4.8)	5.2 (1.4, 19.5)	<.001
	No	Reference	Reference	Reference	
Region of country	Northeast	5.8 (2.0, 16.2)	10.7 (2.6, 43.42)	12.5 (1.7, 94.2)	<.001
	Midwest	3.9 (1.6, 9.3)	12.8 (4.6, 35.6)	10.5 (1.8, 61.2)	
	South	2.4 (1.1, 5.2)	2.8 (1.0, 7.9)	1.2 (0.1, 10.3)*	
	West	Reference	Reference	Reference	

Reference group = children with BLLs <5 μg/dL (0.24 μmol/L).
* Nonsignificant.

creased with blood lead content even when controlling for other risk factors. For example, non-Hispanic black children were 3.3 times more likely to be in the 5 to 10 μg/dL group (95% confidence interval [CI]: 2.1, 5.4), 7.3 times more likely to be in the 10 to 20 μg/dL group (95% CI: 2.4, 15.4), and 13.5 times more likely to be in the ≥20 μg/dL group (95% CI: 4.7, 38.5) than they were to fall within the <5 μg/dL group compared with non-Hispanic white children. In contrast, although Mexican American children were 2.4 times more likely to be in the 5 to 10 μg/dL group than in the <5 μg/dL (95% CI: 1.4, 4.2), children with BLLs ≥10 μg/dL were no more likely to be Mexican American than to be non-Hispanic white.

As expected, toddlers are at higher risk of having elevated BLLs than 3- to 5-year-old children, but for BLLs ≥20 μg/dL, the age-group difference declines and becomes only marginally significant.

Living in housing built before 1946 greatly increased a child's likelihood of elevated BLLs at all levels. Children in older housing were 4.4 times more likely than children in newer housing to have BLLs between 5 and 10 μg/dL (95% CI: 2.9, 6.8); 5.1 times more likely to have BLLs between 10 and 20 μg/dL (95% CI: 2.7, 9.8); and 15 times more likely to have BLLs ≥20 μg/dL (95% CI: 2.8, 80.8). Children living in housing built between 1946 and 1973 were twice as likely than children in newer housing to have BLLs ≥5 μg/dL (95% CI: 1.5, 6.8) but were no more likely to have BLLs ≥10 μg/dL.

There were strong regional differences in risk at all levels of elevated BLL; these differences remained even when age of housing and poverty was at least partially controlled for and are likely caused by historic and economic differences among communities.

Being poor (as measured by family income at or below poverty income ratio [PIR]) was associated with an approximately doubled risk of BLLs ≥5 μg/dL, with the OR highest when comparing the 10 to 20 μg/dL group to the <5 μg/dL referent (OR: 2.7; 95% CI: 1.2, 6.0). Poverty as measured by PIR was not strongly associated with increased risk of elevated BLLs in this model, perhaps because of the inclusion of other variables associated with poverty. Efforts to include various interaction variables and/or substituted income variables in the model did not improve model fit.

The presence of an adult smoker in the home and the education level of the reference adult were both significant in this model. In addition to their association with poverty, these variables may be independently associated with risk of lead exposure, perhaps because they reflect a guardian's lack of awareness of information about health risks of certain environmental exposures.[18] Home exposure to environmental tobacco smoke was not associated with BLLs <10 μg/dL. Conversely, the education of the reference adult (high school graduate or not) had an impact on risk of being in the 5–10 μg/dL group (OR: 1.5 [95% CI: 1.0, 2.2]) and the 10 to 20 μg/dL group (OR: 2.2 [95% CI: 1.3, 3.8]) but not on the risk of being in the ≥20 μg/dL group.

Children whose families reported Medicaid coverage were more likely than those whose families did not report Medicaid coverage to have BLLs elevated to any level. The odds of being in the 5 to 10 μg/dL group were 1.9 times higher for Medicaid participants than for non-Medicaid participants (95% CI: 1.2, 3.2); in the 10 to 20 μg/dL group, 2.4 times higher (95% CI: 1.2, 4.8); and in the ≥20 μg/dL group, 5.2 times higher (95% CI: 1.4, 19.5).

DISCUSSION

It is well-established that the burden of lead exposure above the current intervention level of 10 μg/dL is borne disproportionately by children who are poor, Medicaid-eligible, non-Hispanic black, resident in older housing, and/or located in the Northeast and Midwest.[1] This analysis, which examined more closely how many and which children are at risk of BLLs ≥5 μg/dL but <10 μg/dL, identified 3 patterns significant to future childhood lead poisoning prevention policy.

First, the prevalence of BLLs ≥5 μg/dL is high overall (more than one fourth of US 1- to 5-year-old children) and very high within certain subpopulations (eg, nearly half of all non-Hispanic black children <6 years old have BLLs of at least 5 μg/dL). Although most of these children do not have BLLs that reach the current intervention level of ≥10 μg/dL, they would be considered at risk if the threshold screening and intervention level were lowered.

Second, sources of exposure for many of the children with BLLs ≥5 but <10 μg/dL are not well-defined and could not be investigated by using the NHANES III data. A number of potential sources beyond residential and nonresidential[19] lead paint exposures have been identified.[3,20–25]

Third, the higher a child's BLL, the greater the odds that the child had one or more of the previously known risk factors. The fact that risk factors increase in degree, not kind, with increasing BLL suggests that intervention strategies that effectively target the most at-risk members of disproportionately affected populations (eg, by making lead abatement measures in older housing either legally required or economically advantageous) might have a beneficial effect on children within the targeted populations or locations where blood lead is elevated to lower levels. The public health and policy ramifications of these findings have been analyzed elsewhere.[26]

There was a high nonparticipation rate (21%) in blood laboratory analysis among the 1- to 5-year-old NHANES III participants.[6] Kaufman et al reported that data were more likely to be missing for children in the Northeast and for the younger children, both of whom generally have higher BLLs, but also for children falling within lower risk categories (children sampled in the fall and winter in warmer regions of the country, children who were not anemic, and children with a household income ≥$20 000.[10] Thus, the direction of any bias is not clear. Such a bias in particular may have affected the regression analysis, in which complete data were available for 44% of the study population of 1- to 5-year-old children. Some bias is controlled for because of the use of the SUDAAN statistical program, which estimates variance based on the entire sampling design and thus minimizes the risk of error in variance estimation caused by sample subset creation.

Some questions in the NHANES interview changed over time, and differences likely affected responses or the amount of valid responses entered. For example, 3 versions of health insurance questions were asked over the course of the survey, with the main difference being in time frame ("last month," "now," and "past 12 months").[6,27] In this analysis, we used the questions that asked about private or Medicaid insurance coverage for any member of the child's family (rather than solely for the child), because the response rates were higher.

The race/ethnicity groupings specified in our analysis use the categories provided in the NHANES III dataset: non-Hispanic white, non-Hispanic black, and Mexican American. These categories exclude data on children coded as "other" (10.9% of the total number of 1- to 5-year-old children [weighted proportion]). The sample size of this "other" group was too small to be used analytically, and the category, which included other Hispanics, Asians, and Native Americans, cannot be labeled accurately.[6] As a result, this analysis does not provide specific information about risks of lead poisoning to non-Mexican Hispanic populations, which may be at elevated risk because of general risk factors (poverty, urban residence, and living in the Northeast or Midwest) or because of culture-specific exposures.

The NHANES III survey was conducted over a 6-year period beginning 14 years ago and ending 8 years ago (1988–1994). Deliberate and unintended reductions in childhood lead exposure have occurred since then. It is likely that the NHANES III data are elevated above those that would be found in the current population of 1- to 5-year-old children.[28]

We assumed that any lowering of the intervention level would be to 5 μg/dL for 2 reasons: 1) all past revisions to the intervention level have involved a 5-point (or multiple of 5-point) change in BLL;[29] and 2) 5-point differences in a child's BLL define the recommended level of primary, secondary, or tertiary intervention.[5] However, any BLL above the limit of detection (1 μg/dL during NHANES III[4,10] and 0.3 μg/dL during NHANES 1999[28]) could also be used as an intervention level.

CONCLUSIONS

We identified 2 patterns significant for future childhood lead poisoning prevention policy. First, children with well-established risk factors are most likely to have BLLs ≥5 μg/dL, ≥10 μg/dL, and ≥20 μg/dL, with the probable presence of one or more risk factors becoming greater with increasing BLL. Second, exposure to lead from multiple sources is suggested by the prevalence of BLLs ≥5 μg/dL but <10 μg/dL overall and among children with uncertain risk factors such as those living in newer housing unlikely to have lead paint. The impact of these patterns on public health policy will have to be determined if the CDC take steps to lower the intervention level.

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REFERENCES

1. President's Task Force on Environmental Health Risks and Safety Risks to Children. *Eliminating Childhood Lead Poisoning: A Federal Strategy Targeting Lead Paint Hazards*. Washington, DC: 2000. Available at: http://www.hud.gov/utilities/intercept./cfm?/offices/lead/reports/fedstrategy2000.pdf. Accessed October 8, 2003
2. 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) [see comments]. *JAMA*. 1994;272:284–291

3. Brody DJ, Pirkle JL, Kramer RA, et al. Blood lead levels in the US population: phase 1 of the Third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991) [see comments] [published correction appears in *JAMA*. 1995;274:130]. *JAMA*. 1994;272:277–283
4. Centers for Disease Control and Prevention. Update: blood lead levels—United States, 1991–1994 [published correction appears in *MMWR Morb Mortal Wkly Rep.* 1997;46:607]. *MMWR Morb Mortal Wkly Rep*. 1997;46:141–146
5. Centers for Disease Control and Prevention. Fatal pediatric lead poisoning—New Hampshire, 2000. *MMWR Morb Mortal Wkly Rep*. 2001; 50:45745–45749
6. Centers for Disease Control and Prevention (US). *Screening Young Children for Lead Poisoning: Guidance for State and Local Public Health Officials*. Atlanta, GA: US Department of Health and Human Services; 1997
7. National Center for Health Statistics. *Analytic and Reporting Guidelines: The Third National Health and Nutrition Examination Survey, NHANES III (1988–94)*. Hyattsville, MD: National Center for Health Statistics, Centers for Disease Control and Prevention; 1996
8. Gunter EW, Lewis BG, Koncikowski SM. *Laboratory Procedures Used for the Third National Health and Nutrition Examination Survey (NHANES III), 1988–1994*. Hyattsville, MD: US Department of Health and Human Services, Centers for Disease Control and Prevention; 1996
9. National Center for Health Statistics. *Plan and Operation of the Third National Health and Nutrition Examination Survey, 1988–1994*. Hyattsville, MD: Centers for Disease Control and Prevention; 1994
10. 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: http://www.pediatrics.org/cgi/content/full/106/6/e79
11. Ballew C, Khan LK, Kaufmann R, et al. Blood lead concentration and children's anthropometric dimensions in the Third National Health and Nutrition Examination Survey (NHANES III), 1988–1994. *J Pediatr*. 1999; 134:623–630
12. Gemmel D. Association of housing age and condition with blood lead levels. In: *25th Public Health Conference on Records and Statistics and the National Committee on Vital and Health Statistics 45th Anniversary Symposium.* Washington, DC: US Department of Health and Human Services; 1995
13. Flaherty EG. Risk of lead poisoning in abused and neglected children. *Clin Pediatr*. 1995;34:128–132
14. Hayes EB, McElvaine MD, Orbach HG, Fernandez AM, Lyne S, Matte TD. Long-term trends in blood lead levels among children in Chicago: relationship to air lead levels. *Pediatrics*. 1994;93:195–200
15. Melman ST, Nimeh JW, Anbar RD. Prevalence of elevated blood lead levels in an inner-city pediatric clinic population. *Environ Health Perspect*. 1998;106:655–657
16. Yiin LM, Rhoads GG, Lioy PJ. Seasonal influences on childhood lead exposure. *Environ Health Perspect*. 2000;108:177–182
17. National Center for Health Statistics. *National Health and Nutrition Examination Survey, III 1988–94*. Hyattsville, MD: US Department of Health and Human Services; 1997
18. Shah BV, Barnwell BG, Bieler GS. *SUDAAN Software for the Statistical Analysis of Correlated Data: User's Manual. Release 7.5, Volume 1*. Research Triangle Park, NC: Research Triangle Institute; 1997
19. Johnson DL, Swank PR, Baldwin CD, et al. Adult smoking in the home environment and children's IQ. *Psychol Rep*. 1999;84:149–154
20. Shorten CV, Hooven MK. Methods of exposure assessment: lead-contaminated dust in Philadelphia schools. *Environ Health Perspect*. 2000;108:663–666
21. Rothenberg SJ, Manalo M, Jiang J. Maternal bone lead contribution to blood lead during and after pregnancy. *Environ Res*. 2000;82:81–90
22. Hu H, Hashimoto D, Besser M, Levels of lead in blood and bone of women giving birth in a Boston hospital. *Arch Environ Health*. 1996;51: 52–58
23. Lynch RA, Boatright DT, Moss SK, Lead-contaminated imported tamarind candy and children's blood lead levels. *Public Health Rep*. 2000; 115:537–543
24. Azcona-Cruz MI, Rothenberg SJ, Schnaas L, Zamora-Munoz JS, Romero-Placeres M. Lead-glazed ceramic ware and blood lead levels of children in the city of Oaxaca, Mexico. *Arch Environ Health*. 2000;55: 217–222
25. Rothenberg SJ, Schnaas L, Perroni E, Hernandez RM, Ortega JF. Blood lead secular trend in a cohort of children in Mexico City. II. 1990–1995. *Arch Environ Health*. 2000;55:245–249
26. Patel AB, Williams SV, Frumkin H. Blood lead in children and its determinants in Nagpur, India. *Int J Occup Environ Health*. 2001;7: 119–126
27. Bernard SM. Should the Centers for Disease Control and Prevention's childhood lead poisoning intervention level be lowered? *Am J Public Health*. 2003;93:1253–1260
28. National Center for Health Statistics. *Third National Health and Nutrition Examination Survey, 1988–1994: Youth Notes*. Hyattsville, MD: National Center for Health Statistics, US Centers for Disease Control and Prevention; 1996
29. Centers for Disease Control and Prevention. Blood lead levels in young children—United States and selected states, 1996–1999. *MMWR Morb Mortal Wkly Rep*. 2000;49:1133–1137
30. Centers for Disease Control and Prevention (US). *Preventing Lead Poisoning in Young Children*. Atlanta, GA: US Department of Health and Human Services; 1991

TRIALISTS HAVE A WORD FOR IT

"The 'nocebo effect' is the term invented to describe symptoms reported by patients assigned to placebo groups. They read informed consent forms, which detail all the possible symptoms that can arise from taking the active drug under test, and then report these suggested side effects."

Groopman J. Sick with worry. *New Yorker Magazine*. August 11, 2003

Submitted by Student