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Determination of Numbers of Lead-Exposed U.S. Children by Areas of the United States: An Integrated Summary of a Report to the U.S. Congress on Childhood Lead Poisoning

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In response to Congressional mandate and under the aegis of the Federal Agency for Toxic Substances and Disease Registry (ATSDR), a comprehensive report to Congress on childhood lead poisoning in the United States was prepared. We have examined numbers of lead-exposed U.S. children by socioeconomic/demographic strata for children 0.5 to 5 years of age; by children in U.S. lead-screening programs; and by enumerations of children 0.5 to 5 years old in the oldest (i.e., highest paint lead and lead plumbing) housing. Using blood lead (PbB) prevalence projection modeling and data of the Second National Health and Nutrition Examination Survey (NHANES II), it is estimated for 1984 that 2.4 million black and white children 0.5 to 5 years old in metropolitan U.S. had PbB levels > 15 μg/dL. For all races and the entire nation, we estimate 3 to 4 million children will have PbB levels > 15 μg/dL. Inner-city, low-income children have the highest prevalences of PbB levels above this criterion level, but sizable numbers of all strata of children have elevated PbB levels when considering both base populations and prevalences for the specific strata (total of 30 strata). Lead screening programs indicate much lower numbers of exposed children compared to NHANES II-based projections, for various reasons that allow programs to underestimate true prevalences. Analysis of 1980 U.S. Census Bureau housing data for 318 standard metropolitan statistical areas show that 4.4 million children 0.5 to 5 years old live in the oldest U.S. housing (pre-1950). Of these, most are actually in the more affluent socioeconomic strata.

Introduction

Despite the overt societal importance of the problem, the true quantitative dimensions of both lead exposure and lead effects among high risk segments of the U.S. populations, especially young children and fetuses, have remained difficult to identify. The second National Health and Nutrition Survey (NHANES II) conducted by the U.S. National Center for Health Statistics (*1*) yielded prevalences of blood lead (PbB) levels by socioeconomic/demographic strata within the nation as a whole for 1976 to 1980.

A more current prevalence assessment and one including numbers of children having such elevated lead exposures was required, particularly since evidence has continued to accumulate showing that lower PbB levels than previously recognized are associated with subtle, adverse health effects in risk groups (*2–5*). In legislative recognition of these developments, the U.S. Congress mandated an examination by the Federal government of the quantitative aspects of childhood lead poisoning in the United States.

In response to Section 118(f) of the 1986 Superfund reauthorization legislation, the Agency for Toxic Substances and Disease Registry (ATSDR) submitted a comprehensive report to Congress in 1988 which included a thorough examination of area-stratified lead exposure among preschool U.S. children (*6*). This examination involved various approaches and methodologies and yielded a number of important quantitative results, all of which are provided here as an analytical summary and overview by the authors of that portion of the Congressional report (*7*).

This examination of area-stratified numbers of lead-exposed children consisted of both enumeration (actual counting) and estimation methodologies. It also involved both numbers of children exposed at levels to yield prevalences of preselected PbB criterion levels and those children whose environmental setting would be expected to provide a significantly elevated risk of systemic exposure, despite the

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absence of specific blood lead prevalence data. The specific studies in the report were *a*) estimation of numbers of black and white U.S. metropolitan children in various socioeconomic/demographic strata having PbB levels above selected criterion values; *b*) counts of young children identified through current U.S. lead screening programs in various U.S. communities; and *c*) numbers of U.S. children in each of the 318 U.S. standard metropolitan statistical areas (SMSAs) who live in the oldest housing (pre-1950) and have the highest potential exposures from lead paint, lead in plumbing, and contiguous lead sources present as dust/soil contamination.

Strategies and Methods

Information accurately identifying the numbers of children with any particular PbB level and in any geographical area of the nation does not currently exist as such. Consequently, we were compelled to use various data sets that represent combinations of projection estimates of specific PbB prevalences and enumerations of either base and/or exposed child population groups.

Stratified Estimates of Both Selected PbB Prevalences and Numbers of Children with Selected PbB Levels

This section includes estimates of the number of children living in metropolitan areas of the U.S. who are exposed to lead at levels adequate to pose an unacceptable health risk. In this report, the metropolitan population is defined as that fraction of the total population residing in the 318 U.S. SMSAs as identified by the U.S. Census Bureau. The three specific PbB criterion values used as biological markers of adverse health risk were those defined and discussed elsewhere in the Congressional report (*2–5*). These levels reflect current scientific and public health concerns about early effect onset. Because the levels of lead exposure of concern in terms of blood lead concentrations are continuously being lowered in response to new evidence of low-level lead effects, these indices may decline even further.

The most recent year for which all required Census and other reference child population data were available to the authors was 1984. These base populations were further subdivided by strata, and each stratum's base population was multiplied by a projected (to 1984) prevalence of a given PbB criterion value. Socioeconomic and demographic stratification of the exposed child population is necessary because these strata are associated with widely varying prevalences of elevated PbB levels and yield more meaningful information than a single national figure.

The original NHANES II data set provided the basis for projected PbB prevalences from the NHANES II period, 1976 to 1980, to 1984. Projection was required to accommodate recognized declines in stratified PbB levels because of ongoing reduction in exposure due to declining lead content of gasoline.

Three basic steps were involved in the estimates: *a*) enumerating the total number of children in each SMSA and allocating them to the selected strata as defined by age, race, income, and, where possible, urbanization categories to match the strata employed in the NHANES II analysis; *b*) summing specific SMSA strata populations to obtain national totals for each stratum; and *c*) multiplying each stratum population (national total) by the adjusted NHANES II percentages for the three selected criterion PbB levels.

The NHANES II survey reported prevalence of PbB values as a function of socioeconomic/demographic strata of children across the nation, but not for specific geographic population clusters, such as SMSAs or cities or legal entities, such as municipalities, counties, or states. For example, we can consider, in the aggregate, white or black children residing inside the central city of an SMSA with a population of over 1 million who were aged 0.5 to 5 years and in a family with an annual income of less than $6000, but we cannot estimate the actual number of exposed children in, for instance, the Springfield, IL, SMSA.

To apply the NHANES II prevalences in a statistically valid manner, we sorted out the SMSA child populations as provided by 1980 Census computer tapes into the population strata used by NHANES II. However, since we were interested in child populations in 1984, a more recent year, we proceeded as follows. The actual counts from the 1980 Census are available on data tapes from the U.S. Bureau of the Census, and a specific user tape, "Public Use Microdata Samples," contained the counts for all the 318 SMSAs defined for 1980. This Census Bureau tape omitted some of the detail for some of the data units, posing several problems.

First, the population figures, originally exact counts, had been rounded off to the nearest hundred, creating a problem when using data for black children, since numbers of black children in many SMSAs is quite small, and their distribution among the required socioeconomic strata results is often below 100 in a given stratum. In these cases, the tape showed zero as the number in one or more of the race/age/urban status/family income categories, and we could not estimate the numbers of children at selected criterion values of PbB for these particular cases. On the other hand, this particular problem of allocation is rendered less significant when SMSAs are examined collectively, as they were in this report.

Second, the available data tapes depict 34 of the SMSAs as merged into 17 pairs. This made it impossible to separate the data for the pairs. These 17 pairs are listed in the introduction to Appendix C of the Congressional report (*6*).

Finally, the population in some SMSAs could not be separated by urban status, i.e., "inside central city," and "outside central city." One SMSA did not contain any population center meeting the census definition of "central city," and the tape failed to distinguish central city residents from all others for the 17 merged pairs. This necessitated presentation of our findings for three SMSA sets of estimates and also required reanalyzing NHANES II data to accommodate this problem. Originally, the NHANES II data required stratification by these variables.

The data presented are limited to young white and black children, 0.5 to 5 years old. The NHANES II survey did not include enough children of Hispanic and "other race"

origins for calculating reliable prevalences. The age band selected was obtained through a merging of two NHANES II age bands, 0.5 to 2 years and 3 to 5 years.

Tabulating tape data for 1980 yielded the distribution of young children into two race categories, by urban status and family income groups. A further stratification variable, the size of an SMSA's total population as either over or under 1 million, was also known. We applied these 1980 distributions to the number of children established as the 1984 child population 0.5 to 5 years for the two races.

The most recent natality statistics available were for 1984, and the white and black metropolitan child population 0.5 to 5 years was determined as of 1984 by the following steps for each SMSA. Census counts of children up to 2 years old in 1980 were determined.

Added to these counts were the resident live births minus infant deaths for 1981. This yielded the age group 3 to 5 years. Natality and infant death data were obtained for 1982, 1983, and 1984. The numbers for 1984 were divided in half to yield the 0.5- to 1-year-old group. The census counts and natality and infant mortality data were all available by race. The Division of Vital Statistics of the National Center for Health Statistics supplied the published data for 1981 (*8,9*) and printouts of the as yet unpublished data for the more recent years.

Further stratifications (other than race, age, and size of SMSA) that were employed by NHANES II were accomplished by applying the distributions for inside/outside central city and three categories of family income as found in the 1980 Census counts for children who were then 0.5 to 5 years old.

For the percentages of children in each SMSA living inside the central city in the 1980 Census counts, we established the percentages for each of the three family income categories for either black or white children and applied them to children in the given age band in 1984. We repeated this process for the percentage of children outside the central city. Using this process, we could establish the 1984 child population strata by NHANES II characteristics in each SMSA. We established a total of 30 socioeconomic/demographic strata for large and small SMSAs, 24 strata and 6 strata, respectively.

The NHANES II analyses originally summarized and published PbB levels that did not include all the levels of interest to this report, namely, >10, >15, >20, and >25 μg/dL. The Congressional report and earlier comments here gave the rationale for selecting these levels. Furthermore, the original NHANES II prevalences were calculated for 1978, and applying those rates to 1984 populations would overestimate children at risk owing to declines in PbB levels.

The NHANES II data have already shown through time trend analysis (*10*) the impact of the gasoline lead phasedown over the survey period, 1976 to 1980. Because the amount of lead from this source continues to decrease, a method for projecting prevalences from 1978 to our reference year of 1984 was necessary. This was done through EPA's Office of Policy Analysis, using statistical procedures that included logistic regression analysis and resulting in prevalences at the selected criterion values of >5, >10, >15, >20, and >25 μg/dL for SMSA populations of children in the required strata. A detailed discussion of this logistic regression/projection methodology is presented in Appendix G of the Congressional report (*6*) and EPA's 1985 impact analysis report on lead phase down actions (*11*).

Numbers of Lead-Exposed Children by Community-Based Screening Programs

Until 1981, the nation's childhood lead poisoning screening programs were administered and supported through the Federal government by the U.S. Centers for Disease Control (CDC). The various community screening programs employed common criteria (*12*) for classification of poisoning risk depending on the joint results of both a PbB measure and a blood erythrocyte protoporphyrin (EP) level. In practice, EP was first measured and if a level of >50 μg/dL whole blood was obtained, it was followed by measuring PbB. A PbB of >30 μg/dL and an EP of >50 μg/dL whole blood was taken as a positive test for reporting purposes, and positive results were assigned to one of three categories of risk. Results for the nation were reported annually (*13*).

After 1981, screening was turned over to the various states under the aegis of the Public Health Foundation (*14*), the administrative unit of the Association of State and Territorial Health Officials (ASTHO). Screening funding was incorporated into the Maternal and Child Health (MCH) Block Grant program of the U.S. Department of Health and Human Services' Bureau of Health Care and Delivery Assistance.

Since any CDC screening results on a U.S. community-by-community basis would not be current and since the ASTHO data would be difficult to examine for completeness and adherence to uniform screening criteria, a more current and complete method was required to enumerate children with elevated lead exposure in those U.S. communities which at least had screening programs. The method chosen for enumeration of current screening results in the report was a systematic canvassing by ATSDR staff of all programs still active in December 1986, when ATSDR was doing the survey. In the survey, all relevant summary results were requested from various program staff, and qualifying information was also sought, such as whether current screening results were based on older CDC screening risk criteria (*12*) or newer, more protective guidelines issued by CDC in January 1985 (*15*). In this survey, data were provided for various time frames. In some cases, calendar year 1985 was the time period; in other programs, fiscal years that bridged both 1985 and 1986 were used. In tabulating and interpreting these data, the authors indicate the specific time period represented by the screening results.

Enumeration and Ranking of Children with Potential Exposure to High Lead Sources in U.S. Housing

The final section of the report to Congress dealt with enumerations of children in an exposure setting reliably associated with a high exposure risk, regardless of absence or availability of actual PbB prevalences. This would describe

children in the oldest segment of U.S. housing, i.e., pre-1950, having the highest lead levels in painted surfaces, lead plumbing, the highest probability of deterioration and proximity to high levels of dust/soil lead.

The data for U.S. housing-based child distributions is based on 1980 U.S. Census Bureau enumerations included in the Microdata Sample tapes, which permitted us to extract information for each of the 318 U.S. SMSAs with respect to U.S. housing stratified into three housing age bands for each SMSA; the total number of children, regardless of race/ethnic origin, in each of the three housing age bands for each SMSA; and the number of young children, regardless of race/ethnic origin, in each of three family income categories and in one of two urban status categories, i.e., in or outside of central city for each SMSA's housing age band.

The three housing age groups were pre-1950, 1950 to 1969, and 1970 to 1980. The age band for the children was that of the NHANES II-based estimates in the first section, i.e., 0.5 to 5 years old. Stratification by income and urban status were as described in the methodology for the stratification analyses.

Tabulated stratified counts for the three groups of SMSAs referred to earlier were prepared and are given in Appendices A, B and C in the report to Congress (*6*). For each SMSA, we provided numbers of children in the three age bands and the percentage of SMSA child total in each housing band. These numbers were then stratified further by income and urban status, where the latter was appropriate (*6*).

We then ranked each SMSA with regard to the number of children in pre-1950 housing. When several SMSAs were found to have the same number of children, the percentage of pre-1950 housing counts served as a second ranking criterion. This is the basis of Appendix D of the report to Congress (*6*).

Results and Discussion

Stratified Estimates of Selected PbB Prevalence and Numbers of Children with Selected PbB Levels

Tables 1 and 2 give the projected prevalences of PbB levels in children above three selected levels, 15, 20, and 25 μg/dL, within the strata. These prevalence rates were stratified into inside central city (Table 1) and outside central city (Table 2). Shown are three family income levels to indicate relative poverty, and each income category has two classifications by race. Each of these six strata is further divided into "SMSA with population ≥1 million" and "SMSA with population <1 million." Among 122 SMSAs, 12 strata were available, and each stratum gave us three prevalence estimates.

Table 3 shows the more limited number of strata and the relevant sets of prevalence estimates when inside/outside central city status could not be ascertained. This applied to 196 SMSAs: 34 paired SMSAs, 161 with populations below 200,000 (with very few exceptions), and Nassau-Suffolk, New York, with a population over 1 million but no central city.

Table 1. Projected percentages of children 0.5 to 5 years old estimated to exceed selected PbB criterion values by family income, race, and urban status[a] who live inside central city of SMSAs, 1984.

Family income/ race	PbB, μg/dL					
	>15		>20		>25	
	<1 M	≥1 M	<1 M	≥1 M	<1 M	≥1 M
< $6,000						
White	25.7	36.0	7.6	11.2	2.1	3.0
Black	55.5	67.8	22.8	30.8	7.7	10.6
$6,000–14,999						
White	15.2	22.9	4.0	6.1	1.1	1.5
Black	41.1	53.6	14.1	19.9	4.1	5.9
≥ $15,000						
White	7.1	11.9	1.5	2.5	0.4	0.5
Black	26.6	38.2	6.8	10.4	1.5	2.2

[a]SMSA (standard metropolitan statistical area) with population <1 million (<1 M) and SMSA with population ≥ 1 million (≥1 M).

Table 2. Projected percentages of children 0.5 to 5 years old estimated to exceed selected PbB criterion values by family income, race, and urban status[a] who live outside central city of SMSAs, 1984.

Family income/ race	PbB, μg/dL					
	>15		>20		>25	
	<1 M	≥1 M	<1 M	≥1 M	<1 M	≥1 M
<$6,000						
White	19.2	27.7	5.6	8.4	1.6	2.3
Black	45.9	57.8	17.9	24.5	6.1	8.4
$6,000–14,999						
White	10.9	16.8	2.9	4.5	0.8	1.2
Black	41.1	53.6	14.1	19.9	4.1	5.9
≥$15,000						
White	4.7	8.1	1.0	1.7	0.2	0.4
Black	9.5	28.9	4.9	7.6	1.1	1.7

[a]SMSA with population <1 million (<1 M) and SMSA with population ≥ 1 million (≥1 M).

Table 3. Projected percentages of children 0.5 to 5 years old estimated to exceed selected PbB criterion values by family income and race who live in small SMSAs, 1984.[a]

Family income/ race	PbB, μg/dL		
	>15	>20	>25
<$6,000			
White	23.9	6.9	1.8
Black	56.5	22.9	7.4
$6,000–14,999			
White	13.2	3.4	0.9
Black	40.3	13.6	4.0
≥$15,000			
White	5.8	1.2	0.3
Black	25.4	6.3	1.4

[a]SMSAs with < 1 million population.

The NHANES II PbB levels used in calculating prevalences for criterion levels are based on PbB determinations for all subjects in the survey; the rates are not influenced by initial EP determinations, as is the case in U.S. community lead screening programs.

Statistically, the rates in Tables 1 through 3 are national, urbanized, composite prevalences and cannot be disaggregated to produce region-specific or SMSA-specific, stratified lead exposure figures with any degree of reliability.

Table 4. Estimated numbers of children, 0.5 to 5 years old, who are projected to exceed three levels of blood lead by family income and race, living inside central cities in SMSAs, 1984.[a]

	SMSAs <1 M				SMSAs ≥1 M		
	PbB, μg/dL				PbB, μg/dL		
Family income/race	Number	>15	>20	>25	Number	>15	>20	>25
<$6,000								
White	130,900	33,600	9,900	2,700	259,400	93,400	29,000	7,800
Black	142,200	78,900	32,400	10,900	346,400	234,900	106,700	36,700
Total[b]	273,100	112,500	42,300	13,600	605,800	328,300	135,700	44,500
$6,000–14,999								
White	287,300	43,700	11,500	3,200	493,300	113,000	30,000	7,400
Black	138,900	57,100	19,600	5,700	345,000	184,900	68,700	20,400
Total[b]	426,200	100,800	31,100	8,900	838,300	297,900	98,700	27,800
≥$15,000								
White	648,500	46,000	9,700	2,600	1,046,800	124,600	26,200	5,200
Black	157,000	41,800	10,700	2,400	395,300	151,000	41,100	8,700
Total[b]	805,500	87,800	20,400	5,000	1,442,100	275,600	67,300	13,900
National total[b]	1,504,800	301,100	93,800	27,500	2,886,200	901,800	301,700	86,200

[a]Data for SMSAs in this table permit separation of population residing inside/outside central cities; SMSAs with total population less than 1 million (<1 M) and SMSAs with total population of 1 million or more (≥1 M).
[b]Totals by addition, not estimation.

Table 5. Estimated numbers of children, 0.5 to 5 years old, projected to exceed three levels of blood lead by family income and race, living not inside central cities in SMSAs, 1984.[a]

	SMSAs <1 M				SMSAs ≥1 M		
	PbB, μg/dL				PbB, μg/dL		
Family income/race	Number	>15	>20	>25	Number	>15	>20	>25
<$6,000								
White	143,000	27,400	8,000	2,300	256,600	71,100	21,600	5,900
Black	32,300	14,800	5,800	2,000	77,200	4,600	18,900	6,500
Total[b]	175,300	42,200	13,800	4,300	333,800	115,700	40,500	12,400
$6,000–14,999								
White	428,200	46,700	12,400	3,400	716,500	120,400	32,200	8,600
Black	54,000	17,500	5,800	1,700	114,300	49,900	17,600	5,300
Total[b]	482,200	64,200	18,200	5,100	830,800	170,300	49,800	13,900
≥$15,000								
White	1,300,400	61,100	13,000	2,600	2,977,400	241,200	50,600	11,900
Black	73,700	14,400	3,600	800	222,800	64,400	16,900	3,800
Total[b]	1,374,100	75,500	16,600	3,400	3,200,200	305,600	67,500	15,700
National total[b]	2,031,600	181,900	48,600	12,800	4,364,800	591,600	157,800	42,000

[a]Data for SMSAs in this table permit separation of population residing inside/outside central cities; SMSAs with total population less than 1 million (<1 M) and SMSAs with total population of 1 million or more (≥1 M).
[b]Totals by addition, not estimation.

This is due to such factors as community-specific differences in source-specific exposure intensity. Such differences are merged in a national, stratified sampling design such as for NHANES II.

Tables 4, 5, and 6 present the results of applying the estimated prevalences of the 30 strata of children to the respective base populations in the SMSA segments of the U.S. child population, 0.5 to 5 years of age. Table 4 depicts children living inside central cities for the SMSAs where such division was possible, and Table 5 shows the children outside central cities in these SMSAs. Table 6 shows the findings for smaller and paired SMSA child populations. A partial summary of these three tables for children with PbB levels above 15 μg/dL is presented in Table 7.

Table 8 presents the summary data for all three PbB criterion levels for the SMSA segment of U.S. black and white children, 0.5 to 5 years of age. In this table, the overall findings show a 1984 child population of about 13,840,000

Table 6. Estimated number of children 0.5 to 5 years old, projected to exceed three levels of blood lead by family income and race in small SMSAs, 1984.[a]

		PbB, μg/dL		
Family income/race	Number	>15	>20	>25
<$6,000				
White	249,700	59,700	17,200	4,500
Black	100,200	56,600	23,000	7,400
Total[b]	349,900	116,300	40,200	11,900
$6,000–14,999				
White	741,000	97,800	25,200	6,700
Black	141,100	56,900	19,200	5,600
Total[b]	882,100	154,700	44,400	12,300
≥$15,000				
White	1,670,800	96,900	20,000	5,000
Black	143,000	36,300	9,000	2,000
Total[b]	1,813,800	133,200	29,000	7,000
Unstratified				
Total	6,800	—	—	—
National total[b]	3,052,600	404,200	113,600	31,200

[a]SMSAs with less than 1 million population except Nassau-Suffolk, NY, which has more than 1 million but no central city.
[b]Totals by addition, not estimation.
[c]No estimates possible.

Table 7. Estimated numbers of children, 0.5 to 5 years old, projected to exceed 15 μg/dL PbB, by family income and race in all SMSAs, 1984.

Family income/race	Population base	Inside central city[a]		Not inside central city[a]		Small SMSAs[b]	Total
		<1 M	≥1 M	<1 M	≥1 M		
<$6,000							
White	1,039,600	33,600	93,400	27,400	71,100	59,700	285,200
Black	698,300	78,900	234,900	14,800	44,600	56,600	429,800
Total[c]	1,737,900	112,500	328,300	42,200	115,700	116,300	715,000
$6,000–14,999							
White	2,666,300	43,700	113,000	46,700	120,400	97,800	421,600
Black	793,300	57,100	184,900	17,500	49,900	56,900	366,300
Total[c]	3,459,600	100,800	297,900	64,200	170,300	154,700	787,900
≥$15,000							
White	7,643,900	46,000	124,600	61,100	241,200	96,900	569,800
Black	991,800	41,800	151,000	14,400	64,400	36,300	307,900
Total[c]	8,635,700	87,800	275,600	75,500	305,600	133,200	877,700
National total[c]	13,840,000[d]	301,100	901,800	181,900	591,600	404,200	2,380,600

[a] SMSAs with total population less than 1 million (<1 M) and SMSAs with total population of 1 million or more (≥1 M).
[b] SMSAs with less than 1 million population except Nassau-Suffolk, New York, which has more than 1 million but no central city.
[c] Totals by addition, not estimation.
[d] Includes 6,800 children from small SMSAs who could not be stratified by family income.

living in SMSAs. Of these, 2,381,000 are expected to have PbB levels above 15 μg/dL, indicating that about 17% of the SMSA-based target population is at risk for adverse health impacts from lead exposure. About 5% of the children would be expected to have PbB levels above 20 μg/dL and about 1.5% would have PbB levels above 25 μg/dL.

Although not presented in the report to Congress, we also calculated prevalence rates at >5 and >10 μg/dL and numbers of SMSA black and white children estimated to exceed these levels. Since EPA's Science Advisory Board has recently identified 10 μg/dL as the new PbB of concern for early effects (*16*), we also estimated that 6.4 million black and white SMSA U.S. children had PbB levels >5 μg/dL in 1984. Prevalence rates for the 30 strata >10 μg/dL PbB level were considerably above those at 15 μg/dL, notably in the higher base-population strata (see above tables). In many of the highly urbanized strata, prevalences >5 μg/dL were at/about 100%, and the lowest, for white affluent children in smaller SMSAs outside of city centers, was 78%, all for 1984.

Before discussing these findings, their limitations as national totals must be emphasized. These limitations include both underestimates and overestimates due to the methodologies employed. These estimation uncertainties can result from four sources. First, different numbers of children in each cell of the original NHANES II data base introduce different levels of precision into the prevalences. Second, the logistic regression analysis only accounted for the reduction of lead from a single source, leaded gasoline. Any reduction due to lowered levels in foods cannot be accounted for from available information.

Third, the total number of all U.S. lead-exposed children has been underestimated due to the exclusion of sizable national population segments, especially children of Hispanic and "all other races" origins who live in SMSAs. Even though no reliable prevalences could be calculated for them, they often account for even larger totals than black children in the West and Southwest regions of the nation. It is reasonable to assume that the association between high PbB levels and poverty would hold for such groups. Other cultural/ethnic differences are still undefined in terms of PbB. However, it should be realized that birthrates in these race/ethnic groups are relatively high, and consequently, these children will constitute an ever increasing proportion of the total child population in the future.

Finally, children residing in SMSAs account for about 80% of the total child population. However, the Congressional mandate confined the analysis to metropolitan children in SMSAs.

In summary, it is impossible to define precisely the various

Table 8. Summary of estimated numbers of children 0.5 to 5 years old in all SMSAs, projected to exceed selected levels of blood lead, by urban status, 1984.

Characteristic	Population base	Blood lead level, μg/dL		
		>15	>20	>25
In SMSAs ≥1,000,000	7,251,000	1,493,400	459,500	128,200
In central city	2,886,200	901,800	301,700	86,200
Not in central city	4,364,800	591,600	157,800	42,000
In SMSAs <1,000,000	3,536,400	483,000	142,400	40,300
In central city	1,504,800	301,100	93,800	27,500
Not in central city	2,031,600	181,900	48,600	12,800
In small SMSAs	3,052,600[a]	404,200	113,600	31,200
National total	13,840,000	2,380,600	715,500	200,700

[a] Total includes 6,800 children who could not be stratified by income and were not included in estimates for three PbB levels.

elements of overestimations and underestimations. Therefore, the estimates presented should be characterized conservatively as best estimates that can be based on available scientific data.

Table 7 summarizes the distribution of children predicted to show PbB levels >15 μg/dL by the strata for all SMSAs. The residential distribution of children is reflected in this table: black children are overrepresented in the poverty and low income strata as well as in the inner city areas of the SMSAs. But the ubiquity of the exposure to lead >15 μg/dL, and certainly at lower PbB criterion values of >10 and >5 μg/dL as also estimated, is the striking finding. There are no strata of children totally free of this potential health risk, which holds true for higher PbB levels as well.

The findings summarized in Table 8 indicate the extent of the problem at all three PbB levels, but they obscure insights into demographic/socioeconomic characteristics that have been associated with varying prevalences, and these can be observed for one PbB level (> 15 μg/dL) as indicated in Table 7. The tables showing the estimates of prevalences for the strata (Tables 1, 2, and 3) show the expected negative association of socioeconomic status and PbB level. A positive association is found for density of population. Residence in the central cities and race also are associated with the variations in prevalence.

Again, the most important finding is that no strata of these children are totally exempt from risk of PbB levels high enough to represent a potentially adverse health impact. To illustrate, a numerically very large stratum of children, characterized by family income above the poverty level and predominantly white, is found to be of suburban residential status (not in central city). Although the estimated prevalences in these children are relatively low, estimates of those at risk should not be ignored when planning screening and case finding programs because such a large number of children are in the stratum. White children in the highest income group, living outside inner city and estimated to have PbB levels above 15 μg/dL totaled about 350,000 nationwide. (For the small SMSAs, we added half the estimated numbers of white high-income children with that PbB level.)

Numbers of Lead-Exposed Children by Community-Based Screening Programs

Screening results obtained by ATSDR in December 1986 are presented in Table 9. The responses ATSDR received indicate that the results are derived mainly from the old CDC classification scheme if fiscal year 1985 was employed. Other programs, reporting for a calendar year of screening, sent tabulations reflecting the old classification and the change over to the new 1985 CDC classification scheme during 1985. This latter case is typified by the programs of St. Louis and New York City. For 1985–1986 screenings reported to ATSDR, shown in Table 9, a total of 785,285 children were screened in 40 or more programs, and the overall rate of positive toxicity risk cases was 1.5%.

Toxicity responses for 1985–1986 ranged from 0.3% for four programs to 11.0% for the city of St. Louis program. The five highest rates of positive cases are 11.0% (St. Louis, Missouri); 9% (Augusta and Savannah, Georgia); 4.9% (Harrisburg, Pennsylvania); 3.5% (Washington, DC), and 3.5% (Merrimac Valley, Massachusetts).

The number of children screened in the reports to ATSDR for 1985–1986 (785,285) is higher than the figure of 675,571 reported by state-based screenings (*14*) and the 535,730 children noted in the CDC national statistics for fiscal year 1981 (*13*). However, it is not possible to compare these numbers in any statistically meaningful way because of such differences as screening protocols, variation in central administration, etc.

Within each screening program period and between the different screening programs, several factors have influenced, and continue to influence, screening results. First, the screening risk classifications have changed since CDC set forth its first systematic risk classifications in 1978 (*12*). Most notably, the updated 1985 CDC guidelines (*15*) reduced the PbB action level to 25 μg/dL from the preceding value of 30 μg/dL, with concomitant reduction of the tandem marker of toxicity, EP level, from 50 to 35 μg/dL in whole blood.

Expected changes in prevalence, which resulted from lowering PbB and EP levels used for screening, can theoretically be seen in a program with stable protocols and targeting criteria. Changes in lead toxicity rates were assessed in New York City for the last quarter of 1985 relative to the last quarter of 1984 (*17*).

For the October–December quarter of 1985, the number of New York City lead toxicity cases using the previous classification of CDC was compared with the number found using the new classification. When the previous classifications are used to compare the numbers for the fourth quarter of 1984 and 1985, the numbers of positives do not materially differ statistically. But when the new classification was used, the number of cases for the fourth quarter of 1985 increased 61.4% over the number for the fourth quarter of 1984 (502 cases compared with 311). Furthermore, the increase (the difference between 292 cases with the previous levels and 502 with the new, or 210 cases) is 42% of the cases for the fourth quarter of 1985.

Second, targeting of high-risk exposure populations may well have changed over the years. From fiscal year 1972 to 1981, the strategies for screening populations, under CDC guidance, were uniform. The main goal was to screen groups of children in the community judged to be at high risk and having high prevalence rates for elevated PbB levels and for lead poisoning serious enough to warrant medical or public health action. The extent to which criteria of CDC have been preserved in the many state-centered Block Grant successor programs remains unknown.

To examine any national and program-specific trends with time in rates of positive toxicity risk cases, i.e., CDC classifications of II–IV (*12,15*), we compared the positive response rates over time as reported by CDC (*13*), ASTHO (*14*), and in the most recent survey of ATSDR. These data are tabulated in Table 10. In examining this table, the various qualifications noted above should be kept in mind.

National screening results suggest a moderate decline in cases of lead toxicity over time. This is true even when

Table 9. Lead screening activities (1985–1986) reported by state and local programs to ATSDR[a]

Agency program	Number of children screened (year)	Cases of confirmed Pb toxicity (%)
Delaware	5,818 (FY 85/July–June)	130 (2.2)
Washington, DC	17,000 (FY 85/Oct–Sept)	595 (3.5)
Georgia		
Augusta	2,960 (FY 85/Oct–Sept)	808/8,644 (9.0)[b]
Savannah	5,684 (FY 85/Oct–Sept)	
Illinois		
Chicago	37,409 (CY 85)[c]	693 (1.8)[d]
Indiana	3,770 (FY 85)	17 (0.5)
Iowa		
12 Counties	2,143 (CY 85)	28 (1.3)
Scott County	897 (Jan–Nov 86)	9 (1.0)
Kansas		
Wyandotte	5,098 (CY 85)	16 (0.3)
Worcester	8,161 (FY 85)	71 (0.9)
	9,658 (FY 86)	72 (0.8)
Michigan		
Detroit	20,248 (CY 85)	371 (1.8)
	13,132 (Jan–Aug 86)	392 (3.0)
Minnesota		
Hennepin Co. (Minneapolis)	3,563 (Jan 85–June 86)	26 (0.7)
St. Paul	8,555 (CY 85)	64 (0.8)
	8,553 (Jan–Nov 86)	41 (0.5)
Mississippi	3,628 (FY 86, Oct–Sept)	29 (0.8)
Missouri		
St. Louis City	12,308 (CY 85)	1,356 (11.0)
	9,758 (Jan–Oct 86)	1,653 (16.0)
Nebraska		
Douglas County	3,167 (FY 86/Oct–Sept)	29 (0.8)
Maryland		
Baltimore	30,583 (CY 85)	504 (1.7)
Remainder of state	18,132 (CY 85)	46 (0.3)
Massachusetts		
Statewide	143,000 (FY 85)	1,531 (1.0)
	166,900 (FY 86)	1,011 (0.6)
Maternal and child health projects		
Boston	29,925 (FY 85)	507 (1.7)
	29,356 (FY 86)	337 (1.2)
Holyoke	1,547 (FY 86)	23 (1.5)
Merrimac Valley	5,050 (FY 85)	177 (3.5)
	3,619 (FY 86)	42 (1.2)
North Shore	4,038 (FY 86)	46 (1.2)
Southeastern Massachusetts	4,745 (FY 85)	54 (1.1)
University	3,775 (FY 86)	35 (0.9)
Springfield	1,735 (FY 85)	34 (2.0)
	352 (FY 86)	3 (0.9)
New Hampshire	5,021 (FY 85/July–June)	24 (0.5)
	6,483 (FY 86/July–June)	46 (0.7)
New Jersey	58,080 (CY 85)	1,690 (2.9)
New York		
New York City[f]	206,467 (FY 85)	1,337 (0.7)
Bronx	44,501	28 (0.7)
Brooklyn	72,314	720 (1.0)
Manhattan	47,456	154 (0.3)
Queens	38,604	154 (0.4)
Richmond	3,256	24 (0.7)
(Unknown)	(356)	
North Carolina	15,567 (FY 85/Oct–Sept)	66 (0.4)
Pennsylvania	22,894 (FY 85)	631 (0.3)
Philadelphia	15,133	357 (2.3)
N.E. Philadelphia	983	8 (0.8)
Allegheny County	2,092	32 (1.6)
Harrisburg	2,026	101 (4.9)
Erie County	1,080	9 (0.8)
Rhode Island	14,640 (CY 85)	280 (2.0)
South Carolina	64,993 (FY 86)	920 (1.2)
Texas		
Dallas City	35,000 (average for 2 years)	350–700 (1–22)[f]
Vermont	402 (Jan–Aug 1985)	1 (0.3)
Total	785,285	11,739 (1.5)[g]

[a] Fiscal year 1985 (FY 85) programs mainly using CDC 1978 statement classification scheme. Calendar year 1985 (CY 85) programs use 1985 scheme in some cases. FY 86 and CY 86 summaries mainly use 1985 CDC scheme.
[b] Sum of the two cities.
[c] First screens only.
[d] 1985 CDC scheme starting July 1985.
[e] 1985 CDC scheme starting October 1985; confirmed cases refer to actually medically managed, not class II–IV positives.
[f] Estimated.
[g] Includes upper estimates of cases for Dallas, Texas: 700.

Table 10. Temporal variation in lead toxicity cases in selected lead poisoning screening programs, 1973–1985.[a]

Year	Pb toxicity cases (% of screened)		
	National	New York City[b]	St. Louis
1973	19,059 (6.4)	761 (0.6)	2,396 (32.3)
1974[c]	24,443 (5.4)	494 (0.4)	1,577 (27.0)
1975[c]	30,343 (7.2)	1,559 (1.4)	2,530 (22.9)
1976	33,043 (8.1)	984 (1.0)	3,709 (28.0)
1977	28,072 (7.4)	652 (0.7)	3,519 (24.0)
1978[c]	26,734 (6.5)	802 (0.7)	2,080 (15.2)
1979	32,362 (6.8)	931 (0.8)	1,560 (12.5)
1980	25,293 (5.0)	976 (0.7)	1,422 (11.4)
1981	18,272 (3.6)	1,538 (1.2)	1,422 (12.4)
1982	10,144 (2.0)	1,259 (0.9)	1,278 (10.9)
1983	9,317 (1.6)	1,201 (0.8)	869 (7.6)
1984	5,035 (1.1)	979 (0.6)	1,066 (8.2)
1985[c]	11,739 (1.5)	1,337 (0.6)	1,356 (11.0)

[a]National screening figures given by CDC, 1973–1981 (*13*), by ASTHO for fiscal years 1982–1984 (*14*), and by ATSDR survey for 1985 results (*7*). New York and St. Louis figures as provided to ATSDR by respondents.

[b]Positive cases refer to children hospitalized and given chelation therapy. The number is considerably less than total positive screens, above 40 μg/dL or all classes in 1978 classification scheme. See CDC statistics (*13*) for total positive screening cases in New York City for fiscal year 1981 (n=5010, 4.3%).

[c]Year in which CDC criteria for toxicity changed.

complicating factors are considered, such as changes in protocols for laboratory quality assurance, new lead toxicity criteria, differences in levels of adherence to target population criteria, and the moderate decline in toxicity cases caused by reducing the lead content of gasoline.

Table 10 shows this for both local and national program results. The national figures for 1973 through 1981 are from CDC annual statistics (*13*); for fiscal year 1982 through fiscal year 1984 from ASTHO (*14*), and for 1985–1986, from the December 1986 ATSDR surveys. Please note that the New York City rates of toxicity refer to medically managed children and not total class II-IV positives [see CDC (*13*) for fiscal year 1981 totals].

However, Table 10 also clearly shows that many children still suffer from lead toxicity. In St. Louis, for example, the number of lead toxicity cases has declined from the early 1970s, but the percentage of screening tests that are positive for toxicity is still unacceptable, and this percentage has not essentially changed for the past 6 to 7 years. For FY 1981, CDC figures of total positives (*13*) for New York City give a rate of 4.3%.

The St. Louis program illustrates the difficulty in eliminating the prevalence of lead poisoning by community-wide intervention, even though the overall program efforts were effective over the extended time frame shown in Table 10. Results from this city's program may also indicate outcomes to be expected in other cities if equivalent intensities of screening were pursued (*6*).

Comparison of Elevated Lead Exposure Risk in Children Assessed by Screening Programs and NHANES II-Based National Projections

When comparing the screening results with PbB prevalence modeling data reported here, the latter yield higher rates at the comparison PbB value of 25 μg/dL and for those specific NHANES II strata that overlap those high-risk children targeted in screening programs, e.g., inner-city, high-density population, low-income status groups.

First, screening programs are conducted with methods that may underreport the true screening prevalences of PbB levels. Since an EP level is the first step in assessing lead exposure in young children monitored in screening programs, children who have a normal EP level but an elevated PbB level will not be counted as a subject risking toxicity. The rate of these false negatives was reported to be considerable in a detailed analysis of the 1976–1980 NHANES II data (*18*).

However, this problem may not be as significant in high-risk, inner-city children as it is found to be in a national, stratified statistical sampling such as the NHANES II survey. It is these high-risk children who are the subject of screening programs. Since prevalences of iron deficiency are higher in such high-risk children, and since increases in PbB is highly correlated with iron deficiency (*2,18*), the EP false negative rate would, perforce, be less. Second, there is the epidemiological factor of the underreporting of rates of positive tests when children are tested only at the time of clinic visits or the equivalent, as compared with the intensive, door-to-door canvassing which is done in NHANES II-targeted communities.

Over a rather long time frame, from the 1930s to the present, there have been clear reductions in the rates of severe lead poisonings of children, i.e., requiring emergency medical management and often producing severe neuropsychological sequelae (*2,12,15,19*). With regard to more subtle, chronic lead poisoning in high-risk U.S. children, the available screening data described above suggest moderate declines in prevalences of elevated PbB levels, but any clear evidence of trends is obscured by the various changes noted above. On the other hand, the positive toxicity rates for stable programs over time, e.g., the city of St. Louis program, do show marked persistence of exposures in children at worrisome rates (Table 10).

We also compared changes in exposure or toxicity evaluated in case studies with changes detailed in the usual screening program summaries. Chisolm et al. (*20*) have pointed out the average PbB values in Baltimore may not be declining significantly among subjects at highest risk. For example, in 1956, the mean PbB level for a sizable group of children (n = 330) at highest risk was 43 μg/dL. In 1975, 19 years later, the mean PbB level for 155 children at highest risk was 38 μg/dL. These two means are both high and virtually indistinguishable.

A related question concerns whether the distribution of lead-intoxicated children among the three CDC risk categories changes over time independent of changes in the classification schemes.

The U.S. EPA, in its initial cost-benefit analysis of reducing lead in gasoline (*21*), examined quarterly data from CDC screening programs for the percentage of children with lead toxicity who were in classes III and IV for the years 1977 to 1981 and found no change in the 20 quarters for these nationwide data.

Finally, the numbers of reportedly asymptomatic children with high PbB levels entering the health care system for

the first time should be examined. Of interest are the rates of these child admissions over the years along with the corresponding mean PbB values in the groups of children admitted.

Such relationships have been described by Schneider and Lavenhar (*22*), who examined the medical records of Newark, New Jersey, inner-city children hospitalized for treatment of lead toxicity over the period 1972 to 1980, with particular attention to the group mean PbB levels. In Newark, New Jersey, the rate of child admissions for chelation therapy declined from 1972 until 1976, after which it increased significantly through 1980. This rise does not appear to be due to changes in chelation treatment criteria, and no movement downward in the PbB index signaled such a change. The rise in rate does coincide with declines in funding starting in 1976 and continuing through 1980.

Ranking of Children by SMSAs with Potential Exposure to Lead Paint and Plumbing Lead in Housing

In the previous sections, estimates were given for the number of young white and black children in all SMSAs predicted to have PbB levels above selected criterion values, and enumerations were provided through findings from local screening programs. In those estimates, specific sources of lead were not considered.

Since the age of housing indicates the degree of exposure to lead in paint and plumbing, we analyzed the distribution of children living in SMSAs by the age of their housing units. This approach, then, represents a combination of both area-based exposure and source-specific exposure. The latter topic was presented in Chapter VI of the report to Congress (*6*) and is being published elsewhere (*23*).

Table 11 summarizes the ranking of the 50 largest U.S. SMSAs with reference to the number of young children in pre-1950 housing. This ranking does not automatically correlate with the size of the SMSA populations nor does it correlate with the total SMSA population of young children. For example, in the complete ranking of all 318 SMSAs by numbers of children in pre-1950 housing, presented as Appendix Table D in the report to Congress (*6*), Phoenix, Arizona, was ranked 76th, and San Jose, California, was ranked 85th, even though both are in the group of 38 SMSAs with total populations of over 1 million; nationally, Phoenix ranks 26th and San Jose ranks 30th in terms of total populations. It is not unexpected that the older U.S. population centers contain larger proportions of children living in older housing.

The chronology of urban and suburban growth varied enormously among SMSAs, as Appendix Table D of the report to Congress shows (*6*). Some SMSAs grew most rapidly between 1950 and 1969, and others between 1970 and 1980. In general, for the older SMSAs, the children living in housing built from 1970 to 1980 represented a small percentage of those living in the central city. However, this finding is not true of SMSAs that have achieved maximum growth since the 1950s, as is apparent, for example, when comparing Buffalo, New York, and Houston, Texas, ranked 17th and 20th, respectively, in Table 11.

Table 11. Ranking of 50 1980 census SMSAs by largest number of children 0.5 to 5 years old living in pre-1950 housing and total numbers of young children in the SMSA.

		Children 0.5–5 years		
Rank	SMSA	Number in pre-1950 housing	Percent of total in SMSA	Total number in SMSA
1	New York, NY-NJ	422,800	60.1	703,500
2	Chicago, IL	271,500	43.2	628,800
3	Los Angeles-Long Beach, CA	225,700	33.5	628,800
4	Philadelphia, PA	172,500	46.4	371,800
5	Detroit, MI	141,900	37.8	375,800
6	Boston, MA	110,400	62.7	176,100
7	Newark, NJ	80,300	53.1	151,200
8	Cleveland, OH	75,100	49.1	153,000
9	San Francisco-Oakland, CA	74,800	32.3	231,500
10	Pittsburgh, PA	71,300	47.4	150,500
11	St. Louis, MO-IL	69,200	34.0	203,600
12	Minneapolis-St. Paul, MN-WI	60,000	34.2	186,600
13	Baltimore, MD	56,700	34.2	165,700
14	Milwaukee, WI	53,100	43.5	122,100
15	Nassau-Suffolk, NY	51,100	26.5	192,500
16	Washington, DC-MD-VA	49,900	20.9	234,400
17	Buffalo, NY	46,800	51.3	91,300
18	Cincinnati, OH-KY-IN	44,300	35.4	125,000
19	Dallas-Fort Worth, TX	40,900	15.3	267,400
20	Houston, TX	36,400	12.4	293,400
21	Rochester, NY	35,900	45.4	79,000
22	Jersey City, NJ	34,200	72.6	47,100
23	Albany-Schenectady-Troy, NY	34,000	54.3	62,600
24	Providence-Warwick-Pawtucket, RI-MA	33,300	53.3	62,500
25	Portland, OR	32,900	45.0	71,300
26	Toledo, OH-MI	32,100	45.0	71,300
27	Columbus, OH	31,500	30.4	103,600
28	Kansas City, MO-KA	31,500	28.0	112,300
29	New Orleans, LA	28,600	24.2	118,000
30	Seattle-Everett, WA	28,600	23.3	122,600
31	Indianapolis, IN	27,700	27.6	100,400
32	Riverside-San Bernardino-Ontario, CA	25,900	16.5	156,600
33	Denver-Boulder, CO	25,300	17.7	142,900
34	Syracuse, NY	24,700	45.7	54,000
35	Northeast Pennsylvania	24,400	53.0	46,000
36	Gary-Hammond-East Chicago, IN	24,400	37.3	65,500
37	San Diego, CA	23,900	15.4	154,800
38	San Antonio, TX	23,000	20.7	111,200
39	Akron, OH	22,900	40.2	56,900
40	Dayton, OH	22,800	32.9	69,400
41	Hartford, CT	21,900	12.4	176,400
42	Atlanta, GA	21,900	12.4	176,400
43	Allentown-Bethlehem-Easton, PA-NJ	21,600	50.6	42,700
44	Grand Rapids, MI	21,000	37.0	56,800
45	Paterson-Clifton-Passaic, NJ	20,600	59.4	37,400
46	Salt Lake City-Ogden, UT	20,600	14.7	140,500
47	Louisville, KY-IN	19,800	24.7	80,100
48	Norfolk-VA Beach-Portsmouth, VA-NC	19,100	25.5	72,900
49	Flint, MI	19,000	36.1	52,600
50	Birmingham, AL	18,600	25.5	72,900

Thirty-three SMSAs and one paired SMSA of the entire data set of 313 SMSAs in the full report showed 50% or more of all children living in pre-1950 housing units, with Jersey City, New Jersey, showing 72.6%. Among the 122

SMSAs for which we could separate inside central city/outside central city status, we found 14 SMSAs where 70% or more of the inner-city children lived in pre-1950 housing. In Buffalo, New York, 84.8% of inner-city children lived in the oldest housing. As noted in the full report, 4.37 million children, 0.5 to 5 years old, of all SMSA children lived in pre-1950 housing. This figure is 30.6% of the 14.3 million children aged 0.5 to 5 years who lived in the 318 U.S. SMSAs.

Appendix Tables A, B, and C in the report to Congress (*6*) showed the relationship of family income and housing age for the children in all U.S. SMSAs. They indicate, interestingly, that children in the highest income group constitute the majority of the children in the SMSAs. Consequently, these children frequently are a large portion of residents in each of the three ages of housing categories. Exceptions are SMSAs of more recent growth where such children tend to live in the suburban areas and are found relatively infrequently in recently constructed housing in the central city. In the other SMSAs, children tend to be distributed more in relation to their proportions in the child population. We found that while the children from the highest income population lived disproportionately more frequently in the newest and newer housing, they still represent a significant portion of the residents in the oldest housing as well.

Overview

The report to Congress, including the material on which this article is based, represents the first systematic effort to quantify the extent of the U.S. child lead poisoning problem and to place such numbers of children in some context of *a*) the socioeconomic/demographic numerical distributions of these lead-exposed children, *b*) the various sources of their exposure, *c*) the adverse human health consequences of lead in these children, and *c*) strategies for lead reduction or removal. This paper analyzes and integrates the stratified components of item *a* and is as important for the numbers provided as it is for helping answer the obvious questions, Which children have a problem with excessive lead exposure? and What can we start to do about it?

Among the many key findings noted here are estimates showing that the total 1984 number of black and white metropolitan (SMSA-based) U.S. children exposed to lead above the new ceiling PbB >10 μg/dL, amounts to 6.4 million, 46% of the SMSA child total. Similarly, the number of such children with PbB levels above 15 μg/dL, used as the then-current lowest toxicity level for the 1988 report to Congress, amounted to 2.4 million children, 17% of the total. Additional estimates, also not noted in the report to Congress, showed that 12.7 million such children, 92% of the SMSA total, had PgB levels >5 μg/dL. These figures arise from many socioeconomic and demographic strata.

It was to be expected that the long-recognized high-risk exposure groups, e.g., poor, inner-city black children, would have figured prominently in the estimation outcomes, and they do. These high-risk groups are usually defined in terms of high prevalence rates of elevated PbB levels. Less well understood, perhaps, is the fact that the totals for children in these exposure strata in this report are derived from both a prevalence for a given PbB and from the base population by which the prevalence fraction is multiplied to give a stratum final total.

The consequences of including estimates of stratified exposure totals is simply that large numbers in a stratum's base population can have quite low prevalences for certain PbB levels and still yield numbers that are comparable to those obtained from high-risk strata, i.e., those having smaller base populations of children but quite high prevalences of elevated PbB levels.

In this section, a variety of estimating strategies were employed and provide quite different numbers for exposure estimates. These differences were explained earlier in the report and support the point that conventional screening approaches may understate the true magnitude of the lead exposure problem in high-risk children.

Furthermore, some of the totals complement each other, providing different views of the same total population of U.S. children. For example, examining the very detailed U.S. Census Bureau counts (not estimates) of children in the 318 SMSAs reported in terms of housing age and family income produced the unexpected finding that more children in older housing (high paint lead and plumbing lead levels) were also in noncentral city, nonpoverty families than were children associated with the typical risk groups.

This observation generally is consistent with the stratified distributions of this report's projected numbers of the nation's children with elevated PbB levels. These demonstrated surprising numbers of upper-income, noncentral city children with elevated lead exposure. Put differently, the distribution of children in the oldest, highest risk housing may account for why certain PbB prevalences in the otherwise lower-risk strata of U.S. SMSA children are as high as they are. Clearly, however, other sources also have an impact on PbB levels and their prevalences in the general child population, and this point is established in the section of the report dealing with source-specific lead exposure (*23*).

Estimates of exposure and toxicity based on data gathered at isolated points in time, such as the estimates and enumerations given in the report to Congress, greatly understate the cumulative risk for a population that is posed by a uniquely persistent and ubiquitous pollutant such as lead. This cumulative toll over extended time is of much greater magnitude than the prevalence or total exposure estimates for a given year.

One clear example of this cumulative risk population problem can be readily given. One can amplify those numbers in this report that characterize SMSA children in old, higher-risk housing, i.e., 4.37 million children 0.5 to 5 years, by many-fold if there is no removal or reduction in the sources of exposure in such dwellings over the decades. If one assumes that a given cohort of highly mobile, preschool children in such housing, which is typical of urban rental housing, has an occupancy period of 3 to 5 years, then perhaps 10 times as many children as indicated above would be potentially exposed over a 30- to 50-year period.

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