VI. EXAMINATION OF NUMBERS OF LEAD-EXPOSED U.S. CHILDREN BY LEAD SOURCE

A. GENERAL ISSUES

Three general points need to be discussed before source-specific childhood lead exposure can be addressed. First, "exposure" must be defined according to the available data and the intent of the Congressional directive. Second, a relationship must be established between the lead source and the exposure population to assess the biological diversity of human population response. Third, behavioral characteristics and other covariates influencing the degree to which exposure to lead in the external environment results in internal (systemic) exposure must be studied.

1. The Level of Exposure Risk in Human Populations Characterized and Quantified by Lead Source

Source-specific exposures are difficult to delineate because of multimedia exposures to lead. When exposures come from several sources, how should they be ranked? For example, children exposed to lead in paint (by either direct chewing or swallowing paint chips) often simultaneously contact dust from chalked or weathered paint. Therefore, one source of lead may be the dominant but not the sole source. For rural or suburban adults, food or water can be a major source of lead. It is difficult to quantify exposure once the important sources have been determined. Section 118(f) places no statutory restraints on defining exposure and estimating the number of children exposed. As noted in Chapter II, human exposure can be indexed either by external or internal means--that is, environmental or biological monitoring.

General environmental monitoring can provide external exposure in the population. However, these estimates are overbroad because they estimate the number of subjects at the lead source, regardless of the level of contact with

the source. Although these estimates produce the largest numbers, they are the least accurate when associating exposure with the actual risk of toxicity. For example, when leaded paint exposure is estimated in this way, houses containing lead paint are counted and the number of children from the U.S. Census count is distributed proportionally among them.

A more accurate exposure assessment can be achieved if individuals are exposed only to a source containing lead levels exceeding that needed to elevate Pb-B levels. This estimate is determined by some empirical relationship, usually through use of regression equations. Lead levels in all old leaded paint are high enough for this estimate; so are lead levels in some dust and soil contaminated from lead fallout in air or weathering paint (U.S. EPA, 1986a). Blood lead from water and food can also be assessed using this method.

The most accurate way to assess exposure for source-specific lead is to study elevations in a biological indicator. For example, determining if elevated Pb-B levels can be traced to the intake or uptake of source-specific lead.

Reliable information is needed when using biological monitoring to estimate lead-exposed populations by source. This reliable information requires some means of apportioning a given Pb-B level among input sources. When available, this information would be invaluable in reporting the true scope of source-based lead poisoning in the United States. In view of this, we need to discuss the relationship of Pb-B level to sources in more detail.

The levels of precision in assessing population exposures by lead source are given in Table VI-1. This table summarizes the type of exposure analysis needed for each level of precision within a given source category. From the table, one can determine the approximate exposure level associated with each type of estimation. Those children estimated to be exposed internally (in vivo, systemically) at unacceptable levels of Pb-B are of greatest concern. Those children estimated to have a measurable increase in systemic exposure, i.e., elevated Pb-B levels, but who have not been characterized by risk level for adverse effects achieved by the increase are of second greatest concern. Finally, those children estimated to be in an external lead environment and who risk internal contact with the toxicant, although no data indicating systemic uptake exist, are the third concern. Children with potential exposure are at higher risk than children in environments with no lead

This hierarchical array of types of exposure assessment is illustrated in Table VI-1 for lead in drinking water. In Section F of this chapter, estimates are given for: (1) number of children in an environment likely to have potential exposure to elevated levels of lead in water; (2) number of children who will have some degree of actual exposure due to elevated lead levels in water, but exposures not necessarily high enough to produce lead poisoning. Finally, (3) number of children who have varying elevated lead exposures (Pb-B elevations) to lead levels in water above the proposed maximum contaminant level (MCL) of 20 µg/dl.

2. Relationships of External to Internal Lead Exposure on a Total Population Basis

Before applying any biological indicator for lead (such as Pb-B) to population surveys or to categorize risk, its quantitative dimensions--especially the distribution of Pb-B levels among that population--must be understood. Populations show a range of responses, including a range of Pb-B levels, rather than a single uniform response to lead exposure. This variability of responses occurs because individuals within a population react in different ways to the same exposure, because of host factors. Also, the inherent nature of toxicant distribution, as affected by toxic cellular or organ responses, can internally increase toxicant levels enough to affect their distribution, which is called dose-dependent distribution.

In humans, Pb-B values are distributed in a log-normal rather than a normal manner. A log-normal distribution, unlike a normal distribution, is skewed and not symmetrically bell-shaped. One consequence of this skewing, which is crucial to public health, is that the segment with highest blood lead levels, the upper tail of this distribution, includes a larger fraction of the entire population than it would in a normal distribution. The actual fraction of these individuals in a given population can be defined by the geometric standard deviation (GSD) and the geometric mean (GM). Risk assessors or risk managers consider this segment when defining some desirable cut-off value in a cumulative frequency to protect a risk population at some selected exposure level, such as some specified Pb-B level.

From the GSD, percentages of populations at risk can be calculated as well as the mean or median population lead burden, which is required for the

TABLE VI-1. CATEGORIES OF ESTIMATION METHODS FOR CHILDREN EXPOSED TO LEAD BY SOURCE

Source Category	Level of Precision	Method of Exposure Measurement
1. Lead in paint	Potential exposure	Determination of numbers of children in housing with highest likely lead-paint burdens; complements Chapter V data
	Potential exposure with a better indication of actual exposure risk	Number of children estimated to be in deteriorated housing with leaded paint: peeling paint, broken plaster, other damage
	Likely actual exposure	Use of a specifically determined prevalence for an NHANES II stratum matching such children; other, regional survey data
2. Lead in gasoline	Potential exposure (Pb-B changes) in a subset of U.S. urban child population	Total number of young children in the 100 largest U.S. cities
	Actual exposures based on leaded gasoline combustion	Logistic regression analysis to estimate numbers of children below selected Pb-B criterion values
3. Lead from stationary sources	Potential exposure	Total of young children in communities near lead operations
	Actual exposure	Prevalence of indicated Pb-B levels, at or above some criterion level in actual field studies of stationary sources
4. Lead in dusts and soils	Potential exposure	Summation of potential exposure numbers from the above three categories

(continued on following page)

TABLE VI-1. (continued)

Source Category	Level of Precision	Method of Exposure Measurement
	Actual exposure	Summation of corresponding actual exposure numbers from first three actual exposure categories, or use of multimedia regression equations (not possible with present data)
5. Lead in drinking water	Potential exposure	Numbers of young children in homes with either old lead plumbing or with lead solder in new home
	Actual exposure that is measurable but not highest toxicity risk	Numbers of young children in homes with lead levels in drinking water above 20 µg/l
	Actual exposure at or near toxic levels	Number of children estimated from NHANES II prevalences of projected toxic Pb-B levels
6. Lead in food	Potential exposure at or near toxic levels	Number of U.S. children within selected age group
	Actual exposure	Fraction of those potentially exposed children whose food lead intake may raise Pb-B high enough to cause concern

next step in establishing regulations. The maximum amount of lead that regulators permit in a lead source without exceeding these mean or median values can also be computed. These regulated levels usually correspond to the amount of anthropogenic activity, such as emissions from industry and auto exhaust. If, by using these calculations, it is determined that, for example, 99% of the U.S. population lies below a specific Pb-B ceiling level, about 2.4 million individuals would still be considered to have an unacceptable level of toxicity risk.

These observations are relevant for lead sources that are ubiquitous in the United States, but which show "moderate" concentrations when given as the

average Pb-B resulting from a specific source. The propensity to assess the extent of the lead problem with these averages can be misleading. Because lower averages apparently would suggest a lower public health risk, they can result in misleading interpretations when studying inherently serious effects or when there are tens to hundreds of millions in the population being discussed. The reasons for why this is so follow from the discussion below.

Figure VI-1 illustrates a hypothetical blood lead distribution for a human population. The quantitative usefulness of such a figure can be seen in the 1986 Draft Report of the World Health Organization for air quality guideline recommendations in the European community (WHO, 1986). In developing guidelines for lead levels in air for Europe, the WHO Working Group for lead took several approaches; one approach determined the following parameters.

(1) Based on available evidence at the time (September 1984), the group determined that a Pb-B level of 20 µg/dl was the exposure level of action (WHO, 1986).
(2) The group also determined that this Pb-B 20 µg/dl value was to be prevented in 98% of the European community populations.
(3) Because only data on adults were available, the requisite GM and GSD data were employed to determine that a 98% cumulative frequency for a 20 µg/dl Pb-B level required a median Pb-B value of 10.5 µg/dl in adult populations. In other words, for the Pb-B level in 98% of an adult population to remain below 20 µg/dl, that population requires a median Pb-B level of 10.5 µg/dl.

These three parameters are depicted in Figure VI-1. The shaded area represents subjects in the remaining 2% of the distribution above the level of 20 µg/dl.

If we were to apply this approach to the U.S. population, we would first determine--using the lowest of the three projection formulae of the U.S. Census Bureau--that the 1985 projected adult population is 175 million. If we also allow 98% to have levels below 20 µg/dl, with a 10.5 µg/dl median, the remaining 2% still gives 3.5 million individuals at some risk of lead toxicity. If we expand the protection (cumulative frequency) band to 99.5% and/or reduce the acceptable Pb-B level to 15 µg/dl or less, then this median will be considerably below 10.5 µg/dl.

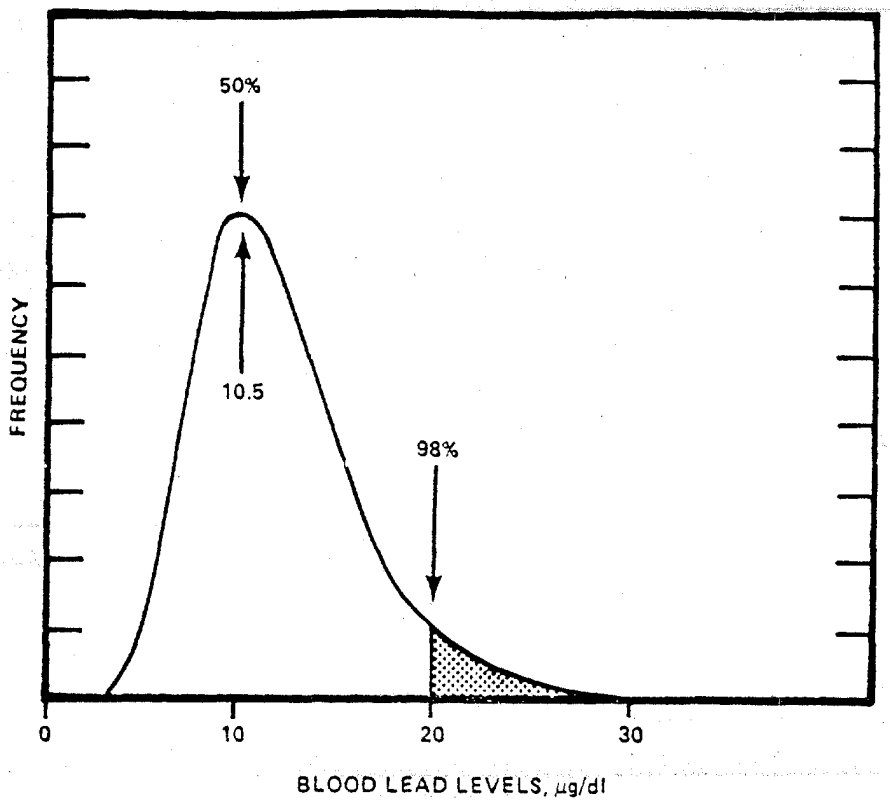

Figure VI-1. Illustrative log-normal Pb-B distribution curve. Values from WHO (1986): Median = 10.5 μg/dl., 98% = 20.0 μg/dl. Shaded area = 2%.

How do these estimates fit available information on the recent or current median Pb-B values for adults in the U.S. population? In the WHO/UNEP world survey of Pb-B levels, included in the Global Environmental Monitoring Survey (GEMS), the U.S. adult median Pb-B level in 1981 was 7.5 μg/dl (Friberg and Vahter, 1983). Judging from the various projections and other data from the U.S. EPA (1985, 1986a), this median Pb-B value has declined in recent years.

For a given Pb-B level and a given cumulative frequency, the values for children would probably be higher than for an adult population, because distributions of Pb-B levels among members of a population are also age-dependent; that is, young children have a different distribution from adults, especially in heterogeneous populations. Adult/child differences occur even in rather homogeneous populations. U.S. EPA (1986a) shows these differences using NHANES II data (Table VI-2).

TABLE VI-2. SUMMARY OF Pb-B LEVELS (μg/dl) OF A RELATIVELY HOMOGENEOUS WHITE POPULATION IN THE UNITED STATES[a,b]

Age Group (yr)	Geometric Mean (μg/dl)	Median (μg/dl)	99th Percentile (μg/dl)	Geometric Standard Deviation (μg/dl)
0.5-6	12.9	13.0	32.0	1.43
6-18	10.6	10.0	24.0	1.46
18+ (Men)	14.7	15.0	35.8	1.44
18+ (Women)	10.0	10.0	23.0	1.46

[a]Adapted from U.S. EPA (1986a). EPA internal analysis of available NHANES II data sets.

[b]Sample size (vertical order): 752, 573, 922, and 927.

From a toxicological standpoint, the distribution of lead levels within target organs (the central nervous system of children, for example), as a function of a given Pb-B value is important for illustrating the distribution phenomena. These distributions are not found in present epidemiological approaches to lead exposure. However, their importance can be seen by studying another metal toxicant, cadmium. Kjellstrom (1985) discusses target organ distributions of cadmium (measured in vivo) including biological indicators and population dose-response curves.

The distribution of lead in major body components such as bone is another important biological factor, because lead can become mobilized and re-enter the blood (Chapters III and IV). The characteristics of this distribution in young children over time are of particular interest.

3. Human Behavior and Other Factors in Source-Specific Population Exposures to Lead

In the relevant literature, discussed in detail by U.S. EPA (1986a) and in critical studies and reviews cited in the EPA document, we find that, given a specified and significant degree of external lead contamination, a number of socioeconomic/demographic variables can affect the relationship between lead in the environment and blood in children. The interrelationships of several of these variables can amplify the degree of adverse interaction between the

child and the contaminated environment. Such factors affect the relative results that investigators find in relating such exposure indices as Pb-B to some measure of adverse effect. For example, one can understand that if children are residing in a heavily contaminated environment and are at the age when they are orally exploring their environment, then the degree of lead exposure via such exploration will be influenced by parental attention to child activity, extent of mouthing, and ingestion of lead-containing material. We might then expect inverse relationships between quality of parental care and degree of lead exposure and some measure of outcome, at least under conditions of moderate lead exposure. Such studies, however, do not imply that lead exposure does not occur or does not significantly contribute to an adverse effect. They simply imply that the degree of exposure interacts with other factors.

Examining modifying factors for scientific reasons is appropriate and necessary, but we should not assume that it will neutralize the effect of low-level exposure in the overall lead problem. These assumptions would be illogical and can detract from a simple rule of health risk management: abating the lead sources removes or reduces the risk for all children, whatever their socioeconomic or demographic status. Such caveats against misinterpretations of the above-noted studies are even stronger when we examine the significant rise in the number of lead toxicity cases associated with urban "gentrification," where children of upper socioeconomic status families reside in lead-contaminated environments formerly occupied by children of lower socioeconomic status (Rabinowitz et al., 1985).

4. Organization of the Chapter

In the main body of this section we estimate and discuss the numbers of children exposed to six different lead sources: paint, gasoline combustion, stationary emissions, soil or dust, water and food. We do not include relatively limited sources of lead (such as exposure from painted toys or hobbies) or contact specific to an ethnic group (as seen in some types of folk medicine). This approach does not imply that these sources are unimportant in certain circumstances, particularly with newly arrived ethnic groups. However, these sources are difficult to quantify and do not affect the overall effect of the major sources of lead described below. For some of these six categories--lead

in food, for example--we cannot identify any specific inputs; we can only say that human activity, collectively, adds considerably to lead levels. The relative impact of these lead sources varies greatly, both by source and by different geographic/demographic/socioeconomic strata. These strata refer to numbers of subjects and not necessarily to the intensity of exposure at a contaminated site. Any population of children having significant contact with lead in dust and soil is also highly likely to have significant contact with lead in air and paint. This category, however, is mainly included to identify a significant pathway for childhood lead toxicity and to evaluate the source for dust and soil in linkage with its primary sources.

B. NUMBERS OF CHILDREN EXPOSED TO LEAD IN PAINT

Many reports address the role of leaded paint in lead poisoning and consider paint lead poisoning to be a public health issue (see, for example, CDC, 1985; U.S. EPA, 1986a). The cause-and-effect relationship of leaded paint to severe lead poisoning dates back many decades. Evidence has long been available to show radio-opaque (lead) paint chips in the abdomens of children who had both high Pb-B levels and severe poisoning and who had not been in contact with any other source of lead. Although the total number of acute, very severe U.S. cases of lead poisoning has declined greatly, the basic epidemiological picture characterizing paint lead-associated toxicity has not materially changed for chronic interaction. The problem can still be described as it has been in some recent studies.

In their prospective study of inner-city children in Cincinnati, OH, Clark et al. (1985) found that child Pb-B levels varied across housing categories and children who lived in the worst housing had the highest Pb-B levels. The housing-quality category accounted for more than 50% of the Pb-B variability in 18-month-old children. In a prospective examination of Baltimore children treated for lead toxicity, Chisolm et al. (1985) observed that children returned to housing where work had been done to remove leaded paint showed significantly higher Pb-B levels than children returned to public housing free of leaded paint. Furthermore, little decline was noted in the Pb-B levels of children who lived in lead-abated units over an extended period--indicating that current efforts to abate lead fall short of public health goals. Both lead paint-contaminated and deteriorated housing units are included in the lead

problem. These units have the largest exposures and they also represent affordable housing for a sizable fraction of inner-city children and parents.

Public health officials have long viewed leaded paint as a lead source in the child's home. However, they should also consider older public buildings used as day-care centers, kindergartens, elementary schools, etc., as potentially serious exposure hazards.

1. Estimation Strategies and Methods

As indicated in Table VI-1, estimates of U.S. children exposed to lead in paint are based on degrees of potential risk and on estimates of the numbers of children predicted to have actual elevated risk because of paint-associated elevations in their Pb-B levels. For potential risk of lead exposure via leaded paint, estimates are given for children living in units with leaded paint and children living in units with an elevated probability of actual exposure because of peeling paint, broken plaster, or other deterioration. The data sets used include calculations by Pope (1986) and the estimates of categories and numbers of lead-painted units with problems from the American Housing Survey of the U.S. Bureau of the Census, 1983 (U.S. Bureau of the Census, 1986).

Pope (1986) first determined a child density factor for each unit (i.e., numbers of children per lead-painted residence) by examining the child population under 7 years of age and the number of housing units in the nation. The child-density factor is specifically the ratio of children under 7 years of age per 1000 housing units. This number, given by Pope (1986), is 287/1000 or 0.287. National figures for housing yielded a value for the fraction of housing units containing leaded paint as a function of age: pre-1940, 1940-1959, and 1960-1974. Furthermore, data from the American Housing Survey, U.S. Bureau of the Census, provide three criteria for unsound units that are relevant for lead paint exposure: peeling paint, broken or cracked plaster, and holes in walls. The data source also provides the fractions of the total units that these units represent. We therefore have estimates of: (1) the total number of children in homes with lead paint; and (2) the number of children in homes with leaded paint that are in disrepair, thus maximizing lead exposure. In addition to Pope's best estimate, we also used Pope's national upper bound. Pope also estimated children by four major regions: Northeast, Midwest, West, and South.

There is a general dearth of nationwide studies that estimate the number of children living in lead paint-containing homes who have elevated Pb-B levels, and much of the information relating leaded paint in the environment to Pb-B levels is not in a form suitable for our analyses. Reasonable data for our needs are available in two forms. The first is a comprehensive unit-by-unit screening conducted in Chicago in 1978 as part of the city's lead-screening program for that year. Although screening was confined to one metropolitan area, it was a comprehensive study, involving more than 80,000 housing units, to determine both Pb-B levels and the presence of leaded paint in the children's houses. The second approach projected (to 1984) prevalences from NHANES II data for Pb-B levels in those socioeconomic/demographic strata where paint is likely to be the major, if not entire, source of exposure. These prevalences were presented in the previous chapter.

EPA's Office of Policy Analysis (U.S. EPA, 1985) used the Chicago data to estimate the likely percentage of children in Chicago (under 6 years old) who would have a Pb-B level greater than 30 μg/dl due to leaded paint exposure. Using estimates of the probability of lead in paint occurring in a home with a child having lead toxicity and the probability of lead in paint occurring in the survey housing in general--both parameters were determined in the Chicago survey--EPA employed Bayes' theorem to determine the probability of elevated Pb-B at the then current toxicity risk level. This prevalence value, 12.8% of all children in the survey, has limited use since it is a dated estimate, represents a Pb-B level too high for our present purposes, and may not represent a best estimate for that year.

One can, alternatively, use prevalences for more appropriate Pb-B levels than the rather high Chicago survey criterion value of 30 μg/dl. In Chapter V, prevalences updated to 1984 are tabulated at Pb-B levels of >15, >20, and >25 μg/dl for young children in various socioeconomic/demographic strata. We have taken the number of young children enumerated by the U.S. Census as living in deteriorated housing with 100% high lead paint, and applied the most logical prevalences for the stratum that would apply to children in deteriorated housing. We assumed that children in 100% deteriorated, high lead-paint housing conform to the stratum that is in the inner city, in the densest population areas, and in the lowest income category. We also assumed that many of these children would be black.

From the relevant tabulation in Chapter V, the Pb-B prevalences (Po-B level percentage) for the 0.5 to 5-year-old/inner-city/higher urban density/lowest income/black stratum are: >15 μg/dl, 67.8%; >20 μg/dl, 30.8%; >25 μg/dl, 10.6%.

2. Results

To estimate the total numbers of young children living in lead-paint housing, we can first estimate the percentages of housing having paint with lead greater than or equal to 0.7 mg/cm^2 as: pre-1940, 99%; 1940-1959, 70%; and 1959-1974, 20% (Pope, 1986). Given a total housing inventory of 80,390,000 in 1980 (U.S. Bureau of the Census, 1983 Survey), we arrive at a final tally of 41,964,000, or 52% of all residential housing units have lead paint greater than or equal to 0.7 mg/cm^2. This figure for the lead-paint concentration is based on the 1985 CDC statement (CDC, 1985). A count of children less than 7 years old in homes with lead paint is shown in Table VI-3, as given by Pope (1986). The national best estimate of the number of children in all lead-painted housing, regardless of the age or state of repair, is 12,043,000. The national upper-bound estimate is higher--13,579,000. When compared to the number of children ranked by age of housing and SMSA as given in Chapter V, and taking into account some differences in children's age in these two sets

TABLE VI-3. NATIONAL BEST ESTIMATE AND UPPER BOUND OF NUMBERS OF CHILDREN UNDER 7 YEARS OLD IN LEAD-BASED PAINTED U.S. HOUSING BY AGE OF UNITS[a]

Estimate Type	Housing Age	Number of Lead-based Painted Houses (Thousands)	Number of Children (Thousands)
Best Estimate			
	Pre-1940	20,505	5,885
	1940-1959	16,141	4,632
	1960-1974	5,318	1,526
Total	Pre-1980	41,964	12,043
Upper Bound			
	Pre-1940	20,712	5,944
	1940-1959	20,753	5,956
	1960-1974	5,850	1,679
Total	Pre-1980	47,315	13,579

[a]Adapted from Pope (1986).

of data, we then can obtain the percentage of all young children who reside in urbanized housing. This percentage is about 80%.

In considering the numbers of children who live in deteriorated housing that contain leaded paint, Pope (1986) classified the housing according to Census Bureau designations for unsound housing within the three house age groups. Table VI-4 shows the best national estimate and the national upper bound for children in deteriorated, lead-painted houses and the number of these houses as a function of age and condition, along with the totals. From Table VI-4, we can calculate that the best national estimate and the national upper bound estimate of children under 7 years old living in unsound lead-painted housing are 1,772,000 and 1,996,000, respectively. Table VI-5 shows young children exposed to peeling paint (the only sign of deterioration) in lead-based painted homes by the four major geographic regions, as given by Pope (1986). As expected, the older developed areas, specifically urban areas in the Northeast and Midwest, have the highest and next highest figures: Northeast, 174,000; Midwest, 139,000 for children in homes with peeling leaded-paint as the survey criterion for deterioration. The South with 130,000 ranks third, while the West has the lowest figure, 77,000.

TABLE VI-4. NUMBERS OF U.S. CHILDREN RESIDING IN UNSOUND AND LEAD-BASED PAINTED HOUSING RANKED BY AGE AND CRITERIA FOR DETERIORATION[a,b,c]

Unsound Category	Age of Home	Number of Unsound Lead-Based Painted Houses	Number of Children
Peeling paint	Pre-1940	964,000	277,000
	1940-1959	758,000	218,000
	1960-1974	250,000	72,000
Total	Pre-1980	1,972,000	567,000
Broken plaster	Pre-1980	1,594,000	458,000
Holes in walls	Pre-1980	2,602,000	747,000
Grand Totals	Pre-1980	6,199,000 (6,954,000)[d]	1,772,000 (1,996,000)[d]

[a]Adapted from Pope (1986).

[b]Housing data from 1983 Housing Survey, (U.S. Bureau of the Census, 1986).

[c]Children under 7 years old.

[d]National upper bound to the numbers.

TABLE VI-5. REGIONAL BEST ESTIMATE OF NUMBERS OF CHILDREN IN UNSOUND, LEAD-BASED PAINTED HOUSING BY AGE AND NUMBERS OF PEELING PAINT UNITS[a,b;c,d]

Region	Age	Number Peeling Paint Lead-Based Painted Houses	Number of Children
Northeast	Pre-1940	432,000	110,000
	1940-1959	203,000	51,000
	1960-1974	51,000	13,000
Total	Pre-1980	686,000	174,000
Midwest	Pre-1940	264,000	74,000
	1940-1959	159,000	47,000
	1960-1974	47,000	14,000
Total	Pre-1980	479,000	139,000
West	Pre-1940	92,000	27,000
	1940-1959	127,000	37,000
	1960-1974	44,000	13,000
Total	Pre-1980	236,000	77,000
South	Pre-1940	156,000	46,000
	1940-1959	203,000	60,000
	1960-1974	80,000	24,000
Total	Pre-1980	439,000	130,000

[a]Adapted from Pope (1986).

[b]Estimates of housing from U.S. Census Bureau (1983).

[c]Children under 7 years of age.

[d]These figures do not include children in units meeting other criteria for unsoundness but only for peeling paint.

One can ask whether these figures are not actually lower bounds when compared with estimates that might be made by summing, across SMSAs, all young children in the known socioeconomic/demographic risk categories. Sums of the SMSA-specific tabulation in Chapter V, Section C, indicate that this is not the case. The estimates of housing (based on Pope) used here include non-SMSA housing stock. Non-SMAS housing should not be in any better condition than SMSA housing. For example, the fraction of substandard homes in rural America is about 41% (2+ million/5 million), according to Lerman (Economic Research Service, USDA, 1986). "Substandard" is technically different from "unsound" in these surveys, but the fraction of houses with peeling paint and cracked plaster as well as lead-based painted surfaces is probably significant in substandard rural housing. Toxicologically, these factors determine the level of lead exposure, not definitions of housing, *per se*. We may be placing a

lower bound to the child density per unit by simultaneously increasing the number of units, thereby offsetting distortions. The above estimates of numbers of children potentially exposed to leaded paint minimize the contributions to the total numbers that would arise from children exposed to lead because older housing is being renovated--the so-called urban gentrification phenomenon. Reliable figures for quantifying this aspect of childhood lead exposure are not available.

In considering estimates of these children exposed to lead in paint who have elevated Pb-B levels because of this exposure, we first chose to combine the numbers in Tables VI-4 and VI-5 for children in unsound, lead-based painted housing with the 12.8% of children >30 μg/dl Pb-B calculated by EPA for all children residing in these units who represent a large urban area. This approach gives an estimate of about 230,000 children; however, the relative accuracy is unknown for reasons already stated. Next, the results of using selected lower Pb-B criterion values--15, 20, and 25 μg/dl--using NHANES II projected prevalences for values that would be plausible for a group of children living in 100% deteriorated, high lead-based painted housing combined with base numbers of such children are tabulated in Table VI-6. The numbers for Table VI-6 are reasonable estimates but are still likely underestimates (see next paragraph). The rationale for assuming the demographic/socioeconomic profile of children likely to reside in such housing is also reasonable. The number of children in such housing having Pb-Bs above 15 μg/dl is around 1,200,000 while the corresponding figures for Pb-B limits of above 20 and 25 μg/dl are around 545,000 and 188,000, respectively.

Numbers in Table VI-6 do not give us an estimate of exposed children in old housing with high paint lead levels but lacking specific criteria for deterioration. The totals in this case may be substantial, since Section C, Chapter V noted that many families in old housing are not in the central city and not in poverty and the homes of these children are not in a deteriorating state. For this reason, these figures in Table VI-6 should be viewed as possible lower bounds (or underestimates) to the true count. Similarly, the stratum of NHANES II selected as appropriate for assignment of these children in order to obtain actual prevalences may represent percentages, after projection to 1984, which are actually from a mix of housing quality. In other words, the true projected Pb-B prevalences for present-day children in 100% deteriorated, high lead paint housing may be considerably above that set of Pb-B prevalences actually selected for estimates in Table VI-6.

TABLE VI-6. ESTIMATED NUMBERS OF U.S. CHILDREN LIVING IN UNSOUND, LEAD-BASED PAINTED HOUSING ABOVE INDICATED Pb-B CRITERION VALUES[a;b]

Category	Housing Age	Total Children	Children With Pb-B (μg/dl) >15	>20	>25
Peeling paint	Pre-1940	277,000	187,800	85,200	29,400
	1940-1959	218,000	147,800	67,100	23,100
	1960-1974	72,000	48,800	22,200	7,600
Total	Pre-1980	567,000	384,400	174,500	60,100
Broken plaster	Pre-1980	459,000	310,500	140,900	48,500
Hole in wall	Pre-1980	747,000	506,500	229,800	79,500
Grand Total		1,772,000	1,201,400	545,200	188,100

[a]Total child count from Table VI-4.

[b]Selection of NHANES II stratum for use of specific prevalences is discussed in text. Prevalences are from Table V-1.

Using both of these factors, the true count of children with elevated Pb-B levels could be underestimated considerably. On the other hand the estimates may overlap in Table VI-6. Units with peeling paint may also have been counted as having broken plaster, etc., in a number of instances.

C. NUMBERS OF CHILDREN EXPOSED TO LEAD FROM LEADED GASOLINE

The combustion of leaded gasoline by motor vehicles and the dispersal of lead from exhausts have had a major role in the status of lead as a public health issue. The recent debate on air quality criteria, health effects, and the leaded gasoline phasedown is only the latest episode of a controversy dating back to 1925, when lead additives had just been introduced. The Hamilton et al. (1925) review of the new leaded gasoline problem from a public health standpoint voiced many concerns that applied equally to the 1950s, 1960s, or 1970s (for a public health perspective on 60 years of leaded gasoline, see Rosner and Markowitz, 1985).

Since about 60 years ago when lead additives for gasoline were introduced, millions of tons of lead from the combustion and dissipation of leaded gasoline in the United States have entered the environment. Much of this quantity has

been lodged in ecosystems where it can lead to human exposure, for example, through dust and soil. A chronological look at such inputs is provided in Table VI-7. For the 10 years shown in Table VI-7, more than 1 million metric tons of gasoline lead were dispersed just in the United States.

A comparison of leaded paint and leaded gasoline indicates the full, insidious nature of the lead problem. Leaded paint can cause very high exposure, with overt poisoning, in a rather confined area and can also induce chronic toxicity due to lower persistent exposure. In contrast, lead from vehicular exhausts can cause sufficient exposure, related to chronic health effects, over a large area.

Evidence showing that leaded gasoline was enough of a human health risk to require further regulatory changes in the existing ambient air standard was compiled in the 1977 EPA Air Quality Criteria for Lead (U.S. EPA, 1977). In addition to assessing the scientific literature related to leaded gasoline, the document also included concepts and perceptions that were then beginning to figure in health risk assessment and biomedical practice as applied to environmental health. Relevant environmental health phenomena, such as blood lead distribution in the population, were discussed. Aggregate exposure to pollutants was defined and discussed. In addition, subtle adverse effects of lead--particularly as noted by the pediatric medical community--were examined.

TABLE VI-7. RECENT CONSUMPTION OF LEAD IN GASOLINE[a]

Calendar Year	Leaded Gasoline Volume (10^9 gal)	Lead Consumed (10^3 ton)[b]
1975	92.5	167.4
1976	87.0	171.4
1977	79.7	168.9
1978	75.0	153.0
1979	68.1	129.4
1980	57.5	78.8
1981	51.0	60.7
1982	52.5	59.9
1983	47.5	52.3
1984	43.8	46.0
Total	654.6	1,087.8

[a]From U.S. EPA (1986a).

[b]Consumption in metric tons.

Between the 1977 and 1986 EPA Air Quality Criteria for Lead Documents, information on the lead problem accumulated; much of it concerned lead from the combustion of leaded gasoline. For example, the quantitative relationships of blood lead to this source were studied; the knowledge about the adverse effects of lead at lower levels was expanded; and the mechanisms of toxicological action were examined. Most of these subjects lie beyond the scope of this report.

Gasoline lead makes a sizable contribution (about 90 to 95%) to the total atmospheric lead burden in developed countries such as the United States. By using lead isotope ratio tagging for lead in gasoline, we can follow that fraction of lead not only into the environment but also into humans. The best estimate of leaded gasoline contributions, using isotope ratios in urban Italy, is about 90% (Fachetti and Geiss, 1982).

As expected, air lead levels related to gasoline combustion and auto density are highest in areas of highest traffic volume, urban and suburban commuting, and commercial activity zones. The most extensive data set of U.S. ambient air levels over the years was compiled by the National Filter Analysis Network and its predecessors. Such surveys have shown that ambient air-lead levels in remote parts of the United States are 2 ng/m^3 and that in urban areas, levels are often 1 to 3 $\mu g/m^3$, some 1,000-fold higher. The trend in these air-lead levels is downward, particularly with the leaded gasoline phasedown that EPA implemented in about 1975. The current allowable lead content of leaded gasoline is 0.1 g/gallon (F.R., 1985, March 7). From 1975 to 1984 U.S. gasoline lead consumption decreased 73%, and estimated lead levels in ambient air showed a similar decrease.

Dispersal of gasoline-based lead from air into food, soils, and dust via fallout has been amply documented and critically evaluated (U.S. EPA, 1986a; WHO, 1986). Airborne lead fallout associated with traffic, as well as lead levels in exterior dust, house dust, soil, and plants are highest near traffic arteries in urban and suburban areas. This observation parallels the findings for ambient air lead from urban stationary sources, such as secondary smelters and municipal incinerators.

The quantitative relationships between airborne lead from leaded gasoline combustion or other sources and a biological indicator such as Pb-B have been the object of numerous studies, and they are discussed at some length in Chapter 11 of U.S. EPA (1986a). This report focuses on the relative impact of

airborne lead inhaled directly and air lead ingested after fallout, such as by children who take in dust and soil.

Given the continuing interest in the impact of airborne lead on Pb-B levels, numerous studies have been conducted on the blood lead to airborne lead ratio; that is, the amount of change in Pb-B one might expect from a unit change ($\mu g/m^3$) in airborne lead. Basically, such a ratio is an oversimplified, only partially integrative depiction of systemic exposure. It permits a quick, but imprecise, look at the effects of changes in a community's airborne lead levels on the systemic exposure of child and adult populations.

The blood lead/airborne lead ratio is lowest when (1) the ratios are examined experimentally in test chambers where the only exposure pathway is inhaled airborne lead, or (2) the ratios are examined for adults, a group with little secondary entry of airborne lead via dust and soil. This ratio rises considerably when children are comprehensively examined for all impacts of airborne lead (Brunekreef, 1984). Direct inhalation of airborne lead yields ratios of about 1 to 2. When children are examined for both direct (inhalation due to emitted lead particulates) and indirect (entrained dust/soil inhalation or ingestion of dust and soil) effects, a ratio of 5 to 6 or even higher is measured. That is, for each 1 $\mu g/m^3$ increase, the Pb-B level rises 5 to 6 $\mu g/dl$. Because dust ingestion and gut absorption vary so much among children, the contribution to individual children varies widely around these figures.

Since internal, or systemic, lead exposure comes from several sources, it is necessary to determine how much exposure comes exclusively from airborne lead. This measurement was conducted using an isotope tracing method, the isotope lead experiment (ILE, Fachetti and Geiss, 1982). Such studies indicate that airborne lead contributes at least 20 to 25% of total Pb-B in adults with inhalation as a principal route. Such estimates are considered lower bounds since a significant fraction of lead within this isotopic ratio, when absorbed, moves to the bone, where it joins a large lead burden. This mixture now loses any isotope identity in blood by a significant "isotope dilution." A sizable fraction of this short-term lead deposit will move from the bone back into blood (U.S. EPA, 1986a).

Collectively, the above data indicate (1) that past gasoline lead inputs produced airborne lead that added significantly to atmospheric and soil/dust/food burdens; (2) airborne lead added significantly to blood lead by direct and indirect routes, yielding 20 to 25%, as a lower bound, based on isotope

tracing and up to 50% based on NHANES II data. In children, blood lead/airborne lead ratios of 5 to 6 and even higher indicate that the airborne lead input to blood lead can be very significant. Therefore, we would expect alterations in airborne lead, paralleling reductions of lead in gasoline, to reduce lead in blood. This relationship is indeed the case, and major support for this statement is the very high correlation between the decrease in Pb-B levels in the general population seen in the NHANES II survey for all segments of the population and declines in the use of leaded gasoline. Specifically, the correlation indicates that decreased use of leaded gasoline over 1976-1980 is the reason for the lower Pb-B levels. Regional data supporting the above national trend were presented by Rabinowitz and Needleman (1982) for a large sampling of newborn cord blood levels in the Boston, MA, area.

1. Estimation Strategies and Methods

If we examine the total potential of direct (inhalation) and indirect (fallout) childhood exposure to leaded gasoline, we mainly examine large urban-suburban areas with denser traffic, i.e., urban population centers, in which airborne lead levels have been high enough to add a potentially significant burden to dust and soil. Since such parts of the environment can retain lead for long periods (U.S. EPA, 1986a), a population of children can be exposed to lead lingering from this source long after airborne lead levels have started to decline.

To examine the number of children 6 years old or younger who were potentially exposed to airborne lead via inhalation or dust/soil lead, children less than 7 years old in the 100 largest U.S. cities were counted from Census Bureau 1984 estimates of total population, and 11% of the population was determined to be under 7 years old.

No children are exposed to lead exclusively from leaded gasoline, and we estimate imprecisely the fraction of children exposed to lead via this source. We can conclude, however, that children whose blood lead levels have changed due to the decreased use of leaded gasoline can be said to have sufficient contact with lead by this route to meet the intent of Section 118(f). As noted earlier, Pb-B levels are declining, and efforts have been directed to quantitate this decline as a function of the decreased use of leaded gasoline. As part of this effort, EPA's Office of Policy Analysis (U.S. EPA, 1985) performed projection analyses of the number of children whose Pb-B levels will fall

below selected criterion values due to continued declines in leaded gasoline use over a number of years.

The EPA Office used logistic regression analyses based on NHANES II data. These regressions were estimated for both black and white children and for the rather broad age band of children at risk, 6 months to 13 years old. Assuming that a log-normal Pb-B distribution would occur with the decreased use of leaded gasoline, EPA generated estimates of the mean and variance of transformed (normal) distribution for determining percentages above specific Pb-B levels, logistic regression estimates of the children with Pb-B equal to or above 30 μg/dl, and computer estimates of the mean of the log-normal distribution (SAS/SURREGR). A more detailed discussion of this method is beyond the scope or purpose of this report, and appears in the original EPA document (1985). Since much of this methodology is relevant to Chapter V, these techniques also appear in Appendix G of this report.

2. Results

As indicated, we restricted our count of children at risk of exposure to leaded gasoline to the 100 largest U.S. cities. These cities had a 1984 estimated total population of 50,597,300, of which 5,565,700, or 11%, were children less than 7 years old. This estimate of some 5.6 million children at risk cannot be related directly to the following discussion on the impact of leaded gasoline on Pb-B levels, because the children are different ages and possess inherent differences.

Table VI-8 shows the numbers of U.S. children, 6 months to 13 years old, whose Pb-B levels will fall below selected toxicity levels when projected to 1990. The numbers are significant, showing expected increases as the Pb-B level is lowered. Smaller numbers of children occur at higher Pb-B criterion levels, since the numbers originally above these levels were smaller. These values represent nationwide projections for a 13-year age band in children. Since Pb-B distributions are age-dependent in this age band, especially in younger children, it is not easy to divide out these figures into narrower age bands from the values in Table VI-8. One difficulty with the broad age range for the number of affected children is that the numbers of very young children are not available to the reader. A second factor, evident in Table VI-8, is the prevalence of the numbers of children with Pb-B levels below expected Pb-B levels in the years long after the the leaded gasoline phasedown: gasoline

TABLE VI-8. ESTIMATED NUMBERS OF U.S. CHILDREN (THOUSANDS) FALLING BELOW INDICATED Pb-B (μg/dl) LEVELS AS A RESULT OF Pb-GASOLINE PHASEOUT[a,b]

Blood lead (μg/dl)	1985	1986	1987	1988	1989	1990
25	72	172	157	144	130	119
20	232	563	518	476	434	400
15	696	1,726	1,597	1,476	1,353	1,252

[a]From U.S. EPA (1985). Based on regulatory action beginning January 1, 1986, to achieve 0.1 g/gal by January 1, 1988.

[b]Tabulations in original U.S. EPA (1985) analysis were extended only to 1990 for this table.

lead phasedown alone will not bring all Pb-B levels down to acceptable low levels. Table VI-8 shows, even in terms of the 25 μg/dl level, that sizable numbers of children are projected to fall below these various Pb-B ceiling levels from 1986-1990 and that large declines are expected for criterion levels of 15 and 20 μg/dl.

D. NUMBERS OF CHILDREN EXPOSED TO LEAD FROM STATIONARY EMISSION SOURCES

As noted in Chapter II, stationary sources mainly refer to fixed operations that emit lead into the atmosphere and, consequently, into other ecological areas. Such sources include primary and secondary smelters, incinerators, and operations involved in coal and waste oil combustion. In terms of impact, these operations mainly affect neighboring communities, but they can cause severe lead contamination.

The United States has 11 mines, 5 primary smelters and refineries, 60 secondary smelters, and 132 plants where lead-acid batteries are manufactured. For these sources, we have to consider that contamination occurs even after the facilities have closed. Over the years, a lead-emitting operation will add a heavy ecological burden to nearby areas. Of particular concern is lead fallout from smelters transferring to nearby soil, dust, and forest cover. The evidence linking lead emissions from stationary operations to the elevated body lead burdens of young children is well established. These connections have been derived from studies of a number of major U.S. smelter operations (including ones in Idaho, Montana, and Nebraska) that have been extensively examined

by U.S. EPA (1986a). Airborne lead levels near lead smelters and refineries, and in some cases up to 5 to 10 km away, reached 5 to 15 $\mu g/m^3$ in past periods, particularly before emission controls were installed in the 1970s. In impact zones, lead levels ranged up to 100,000 ppm (10% by weight) where emission controls were minimal, and near smelters in Missouri, soil lead levels reached levels up to 60,000 ppm. Today, soil and dust levels range between 500 and 5,000 ppm in areas near point sources. The levels decrease exponentially with distance from the operation.

Results of numerous studies document that children sustain marked increases in blood lead and body lead burdens when they live near stationary lead emitters, particularly lead smelters. This relationship can be seen in the investigations of Yankel et al. (1977), who evaluated Pb-B levels in children 1 to 9 years old living near a smelting operation in Silver Valley, ID, in 1974-75. Table VI-9 shows the results of the biological and environmental monitoring in this smelter area. These overall blood lead results were extremely high, especially the percentage of levels over 40 µg/dl. Airborne lead levels were also very high, even at 10 km from the operation. In the zone adjacent to the smelter, about 100% of the Pb-B levels were above 40 µg/dl. Elevated Pb-B levels have been found in children living near other smelter sites, both in the United States and elsewhere (U.S. EPA, 1986a).

TABLE VI-9. GEOMETRIC MEAN Pb-B LEVELS (µg/dl) BY DISTANCE FROM SMELTER (AREAS 1-6) FOR CHILDREN[a] NEAR IDAHO SMELTER[b]

Area and km from Source		Airborne Lead ($\mu g/m^3$)	Geometric Mean Pb-B (GSD) (µg/dl)	% Pb-B >40
1	0-1.6	18.0	65.9 (1.30)	98.9
2	1.6-4.0	14.0	47.7 (1.32)	72.6
3	4.0-10.0	6.7	33.8 (1.25)	21.4
4	10.0-24.0	3.1	32.2 (1.29)	17.8
5	24.0-32.0	1.5	27.5 (1.30)	8.8
6	~75	1.2	21.2 (1.29)	1.1

[a]Ages 1-9 years.

[b]EPA analysis of Yankel et al. (1977) data.

Recent surveys carried out in two smelter communities, Montana (CDC, 1986a) and Idaho (CDC, 1986b), as a joint effort by CDC, EPA, and the respective states, indicate that considerable levels of residual dust and soil contamination linger after former active, high atmospheric inputs.

As a consequence of this lingering exposure problem in a smelter community in Idaho (CDC, 1986b), the survey found that:

(1) Children who lived close to the smelter in the Silver Valley area of Idaho had a higher geometric mean Pb-B (20 μg/dl) than children farther away (11 μg/dl).
(2) Detailed statistical analysis of the data base showed that the only significant environmental contributor was lead in the soil, and its contribution was via lead in household dust. Soil lead near the smelter had a geometric mean level of 3,472 ppm, while a mean level of 481 ppm was measured for sites farther away; the corresponding geometric mean for lead dust was 3,933 near the smelter and 1,138 ppm farther away.
(3) At the time of the survey, the ambient air level had mean values, by area, of 0.10 to 0.28 $\mu g/m^3$.
(4) The percentage of children living near the smelter whose Pb-B and EP levels exceeded the CDC risk criteria for 1985, i.e., Pb-B ≧25 μg/dl and EP ≧35 μg/dl, was 26%, while the corresponding figure for those farther away was 2%.

These two comprehensive studies conclusively document that previous lead fallout remains a main contributor to lead exposure in general, and contributes to body lead burden of children in particular.

We turn now to estimates of U.S. children who are either potentially at risk to lead exposure from fixed operations or actually have elevated Pb-B levels due to exposure from stationary sources. Such population data are surprisingly meager. Only a limited number of reports have helped us to quantify this aspect of the U.S. lead problem. One preliminary report, for EPA's Office of Air Quality Planning and Standards (OAQPS) (GCA Corporation, 1986), provides the larger of the two data sets for the total number of children living near stationary sources in potential exposure to lead. The data set of the Lead Industries Association (TRC Environmental Consultants, Inc., 1986) provides a markedly different estimate of children from that of EPA. These data sets are for potential exposure.

1. Estimation Strategies and Methods

The interim OAQPS numbers are derived from dividing the stationary sources into three categories; primary smelters, secondary smelters, and lead-acid battery plants. Estimates for each group were collected for different radii around the operations, which reflect differences in lead dispersal patterns. The numbers are for total subjects and the fraction of children less than 7 years old, 10.4%.

The LIA study differs from the OAQPS study in terms of inventory of units still operating, quadrants surveyed, and radii around the operations. The LIA study mainly made its estimates based on ambient airborne lead levels. The LIA assessment did not count closed facilities. But in assessing the net and continuing lead exposure of children around stationary operations, closed facilities must be included because past lead emissions continue to have an impact. These sources must be included to avoid underestimating the risk of lead exposure.

The LIA study narrowed the radius of exposure population considerably, compared with the OAQPS model. The LIA considered airborne lead movement for the dominant wind direction at the emission point, but did not allow for changes in wind direction on soil and dust levels in sectors not in the dominant path.

Data in Table VI-9 show that at 10 to 24 km away from a smelter, with an airborne lead level of 3.1 $\mu g/m^3$, the geometric mean Pb-B level in children was 32.2 µg/dl, with almost a fifth of the lead levels above 40 µg/dl. If children are also examined for uptake pathways 10 to 24 km from the smelter--distances well beyond both the LIA and the OAQPS plottings--the Pb-B/Pb-Air ratio is almost 6 when the levels are normalized to those of a group of control children in this zone. This ratio strongly suggests heavy additional input into Pb-B above that contributed by inhalation, i.e., dust and soil lead contributions. Angle et al. (1984), for an Omaha smelter area, reported a value of 6 to 7 for total inputs to children's Pb-B levels by lead exposure through air inhalation, dust, and soil.

The above estimates by LIA and OAQPS were for potential exposure subjects. Actual Pb-B prevalence data for such sites as primary and secondary smelters also exist. The recent smelter community studies in Montana and Idaho (CDC, 1986a, 1986b), as cited earlier, show that both dust and soil levels and Pb-B

levels of children remain elevated even when airborne lead levels have dropped to very low levels (0.10 to 0.28 $\mu g/m^3$). These results showed that about 1% (1/98) of the children within 1 mile (1.6 km) of the East Helena smelter and 26% (11/43) of the children within 1 mile (1.6 km) of the Kellogg smelter met the 1985 CDC criteria for some level of lead toxicity: a Pb-B level of at least 25 µg/dl and an EP level of at least 35 µg/dl. Further, results of a systematic survey in Dallas, TX showed 4% of the children living near secondary smelters in the area had Pb-B levels above 20 µg/dl (City of Dallas, 1985).

2. Results

Table VI-10 shows the values from the LIA study (TRC, Inc., 1986) for potential numbers of exposed children; Table VI-11 shows the interim OAQPS estimates of the same potential exposure group (GCA, 1985). The OAQPS data are probably more useful in accounting for the indirect impact of stationary source emissions on present and past levels in dust and soil. If results of other studies, such as the Yankel et al. (1977) Silver Valley, ID, study and the CDC East Helena, MT, and Silver Valley, Idaho studies (CDC, 1986a, 1986b), are examined together, the OAQPS tabulation may be a conservative estimate of the total exposure population for primary smelters. Dispersion radii for secondary smelters are usually shorter than for primary smelters, but such operations tend to be in densely populated urban areas. OAQPS is reanalyzing and updating information on impact zones around different point sources.

Table VI-11 provides the best guide for estimating the total number of children who may have potential lead exposure due to proximity to stationary sources. To estimate actual exposures, we used the rates of 1% and 26% for primary smelters and 4% for secondary smelters, as noted earlier. These rates give differences among the point source emission characteristics, geography, neighboring communities, etc. Extrapolating the results of the East Helena primary smelter study--where 1% of the children had Pb-B levels over 25 µg/dl--yields a total of 210 children for all primary smelters. The corresponding prevalence recently found in Idaho, 26%, gives a figure of around 5,500. In 1984, in Herculaneum, MO, 18% of the children living within 1.5 miles of the primary lead smelter had Pb-B levels above 25 µg/dl (communication, OAQPS/EPA to ATSDR). Using this factor yields a total of around 3,800. Likewise, an estimate of 7,500 is the corresponding figure for children sufficiently affected by secondary smelters to have Pb-B levels above 20 µg/dl,

TABLE VI-10. EXTRAPOLATED PEDIATRIC POPULATION ESTIMATES FOR STATIONARY LEAD SOURCES: TRC/LIA AND EPA APPROACHES[a,b]

Source	Operating Facilities	Number of Children Extrapolated for TRC Distances[c] 1.0 μg/m³	0.5 μg/m³	Extrapolation for EPA Distances[c]
Primary smelter	5	616	1,946	6,154
Primary refinery	1	7	553	8,961
Secondary smelter	23	141	2,377	19,738
Tetraethyl lead	1	37	122	365
Battery Plants	98	1,647	3,293	15,827
Total		2,448	8,291	51,045

[a]Adapted from TRC, In: LIA study as submitted to OAQPS/EPA (TRC, 1986).

[b]Using 8,291 as the best estimate of potentially exposed subjects.

[c]Distances from point source as defined in EPA and TRC reports.

TABLE VI-11. GCA/OAQPS ESTIMATES OF TOTAL AND CHILD (<7 YEARS) POPULATIONS EXPOSED TO STATIONARY SOURCES OF LEAD[a]

Source	Radius Around Plant (km)	Total Population	Number of Children
Primary lead smelters	5	200,000	21,000
Secondary lead smelters	2	1,800,000	187,000
Lead-acid battery plants	1	240,000	25,000
Total		2,240,000	233,000

[a]As tabulated and submitted to OAQPS/EPA, April 8, 1985. Radii to estimate potentially affected population are preliminary and are under re-examination.

assuming a 4% prevalence above 20 μg/dl reported for the Dallas survey. We cannot estimate the numbers with elevated Pb-B levels near the lead-acid battery plants because data do not exist.

E. NUMBERS OF CHILDREN EXPOSED TO LEAD IN DUSTS AND SOILS

In previous sections, the three major contributors to lead in dusts and soils were evaluated according to a ranking of childhood lead exposure by source: lead paint, gasoline lead, and stationary lead emissions. Dusts can be further classified into soil dusts, street dusts, and household dusts. In various exposure environments, the relative importance of these types for childhood exposure differs. In summer, street dusts are probably more important because of the amount of time children spend outdoors; in colder weather, household dusts are probably more important. Children may be further exposed to lead in dust via dust brought home on the clothing of working parents and relatives. These occupational dusts may have high lead content, reflecting fairly concentrated amounts of substances such as lead oxide (Milar and Mushak, 1982). As noted earlier, lead levels in household and street dusts vary as a function of their primary contributors; these levels can range well above 1,000 ppm in many urban areas (see U.S. EPA, 1986a, for a detailed discussion). Brunekreef et al. (1983) determined, in a study in the Netherlands at sites without major point sources, that household dust increased in lead content by 400 to 700 ppm for each 1 $\mu g/m^3$ increase in airborne lead.

A major problem with reported studies of lead in dust and soil in relation to body lead burdens is the absence of any current standard method for collecting samples at the test site. For soils, current core samples or surface scrapings can be taken; multiple sites can be sampled or hot spots can be emphasized.

Near stationary lead sources, lead levels in dust respond more dramatically to changes in airborne lead levels (Yankel et al., 1977; CDC, 1986a, 1986b). Soil lead content is also considerably affected by airborne lead fallout from mobile and stationary sources. Lead levels in soil can rise considerably with fallout, but the levels generally are lower than in dusts because the nonlead fraction dilutes the soil samples more (see, e.g., CDC, 1986b). An important aspect of soil contamination is the uptake of lead onto plants, which is mainly deposited on the surface with some uptake through the root system. Plant uptake becomes significant when assessing lead exposure in plants that are an

important part of the human diet. Lead in soil also impacts lead levels in livestock; when the animals forage lead-contaminated crops or ingest soil lead, lead may enter the food chain.

A number of reports have addressed the quantitative relationships of lead in soil and dust to Pb-B, and U.S. EPA (1986a) has summarized these reports. In general, lead in dust and soil at levels of 500 to 1,000 ppm begins to affect children's Pb-B levels (Baker et al., 1977; Mielke et al., 1984); a number of investigators have found a highly significant correlation between Pb-B levels and lead levels in dust and soil (see, for example, Angle et al., 1984; Roels et al., 1980).

Results from U.S.-based investigations of these relationships have been confirmed and extended to other countries (Duggan and Inskip, 1985).

In the study of the Silver Valley, ID smelter reported by CDC (1986b), the difference in the Pb-B means for children near the smelter operation vs. those farther away was 9 µg/dl (20 µg/dl vs. 11 µg/dl, respectively). The average soil levels for the sites differed as much as 3,000 ppm. Calculations show that a 1 µg/dl rise occurred in the Pb-B level for each 330 ppm of soil; the corresponding relationship for lead in dust was essentially the same, 310 ppm.

Recent data of Bornschein et al. (1987b) and Clark et al. (1987) show that lead in soil and paint contributes to lead in dust; dust lead transmitted via children's hands to their mouths accounts for a significant fraction of Pb-B increases. Also, an increase of lead by 1,000 ppm raises Pb-B by 6.2 µg/dl.

The relationships of lead in soil and dust to Pb-B levels in various studies (see U.S. EPA, 1986a; Bornschein et al., 1987b) show a range of values, generally changing between 3 and 7 µg/dl for every 1,000 ppm change. Note that some of these studies derived Pb-B response data for Pb-B levels which are higher than those now judged as unacceptable. In the Baker et al. (1977) study, for example, lead levels in dust above 1,000 ppm caused a rise above 40 µg/dl. This observation implies that a lower Pb-B threshold for reference would have been associated with lead levels below 1,000 ppm.

A determination of the direct and indirect contributions of airborne lead to Pb-B, that is, the fraction from direct inhalation and that from fallout, has been noted in the lead isotope ratio study in Turin, Italy (Fachetti, 1985); results showed that 60% of the amount enters adult subjects via inhalation and 40% via indirect routes. In children, especially those living near

point sources of lead with heavily contaminated soil and dust, the fraction of atmospheric lead uptake through these indirect sources would presumably be much greater.

1. Estimation Strategies and Methods

The numbers of children exposed to lead in dust and soil cannot be separated, as noted above, from the numbers exposed to airborne lead (gasoline or stationary emissions) or leaded paint. Direct exposure to lead, i.e., airborne lead or leaded paint, also foretells simultaneous exposure to dust and soil; therefore, we should sum across estimates for these contributing sources for both potential and actual risk. This summing will lead to overestimates in the numbers of children, clearly making the totals upper bounds. One can determine a lower bound for the estimate by selecting the number of children exposed to the single, main contributor.

An alternative estimating process which avoids double counting, uses multimedia regression analysis. A regression approach, which separates the contributions of lead from various sources to children's blood lead levels, would yield a more precise estimate than the cumulative approach. A regression approach, however, would require (1) establishing a set of regression equations for urban and rural settings in different U.S. regions, (2) determining the prevalence of lead-contaminated dusts and soils in these regions, and (3) using the regression equations and the prevalence data to estimate the number of children exposed to dust and soil lead levels at concentrations large enough to cause adverse health effects.

The general type of regression equation needed simultaneously identifies the independent contributions of leaded paint, airborne lead, lead in dust, and lead in soil to children's blood lead levels. The form of this equation is

$$Y = A_o + C_1x_1 + C_2x_2 + C_3x_3 + C_4x_4$$

where Y = a child's blood lead level,

A_o = a constant,

C_1-$_4$ = the relative contributions of the independent sources of lead,

X_1 = the leaded paint level,

X_2 = the airborne lead level,
X_3 = the level of lead in dust,
X_4 = the lead level in soil, and is a random error term.

Unfortunately, no study to date has produced this type of general regression equation. Available equations either omit leaded paint (Angle et al., 1984; Charney et al., 1980; Walter et al., 1980; Yankel et al., 1977), airborne lead (Charney et al., 1980; Galke et al., 1975), or lead in dust (Galke et al., 1975; Yankel et al., 1977). Some of these omissions reflect regional differences. For example, leaded paint is not an important contributor to children's blood lead levels in the western U.S., and, therefore, it is absent from the multiple linear regression models for children in Idaho (Yankel et al., 1977) and Nebraska (Angle et al., 1984). Other environmental measurement omissions, however, reflect limitations in study design. To successfully use a regression approach for estimating the number of children exposed to hazardous levels of lead in dust and soil would require, at a minimum, an extensive effort in urban and rural areas in the four U.S. regions: West, Midwest, Northeast, and South. The purpose of this effort would be to test children's Pb-B levels and uniformly collect household-specific data on lead levels in paint, air, dust, and soil. These data would form the basis for constructing regional-specific regression equations to predict children's Pb-B levels in urban and rural areas.

A survey of a representative sample of dusts and soils from urban and rural areas of each region is necessary to establish the prevalence of lead-contaminated dusts and soils.

Using the regional- and urban- or rural-specific regression equations, one could determine "safe" lead levels in dust, i.e., levels which do not cause children's Pb-B levels to exceed 25 μg/dl (the present criteria level), or any such level in the future. This determination would be possible by using average lead levels in paint, air, and soil to solve the equation for a "safe" lead level in dust. Similarly, one could solve the equation for a "safe" lead level in soil by using average lead levels in paint, air, and dust.

Once "safe" dust- and soil-lead levels are determined for urban and rural areas in each region, an estimate of the number of children living with higher lead levels of dust and soil can be made. By definition, this estimate would be the number of children exposed to dust and soil lead levels at concentrations sufficient to cause adverse health effects.

2. Results

The numbers of children potentially exposed to lead in dust and soil, but without determining numbers of Pb-B elevations, are taken as the sum of totals exposed to primary contributors to dust and soil:

Paint lead in pre-1940 housing with highest lead content	5.9 Million children
Gasoline lead in 100 of the largest U.S. cities	5.6 Million children
Stationary Source Emissions	0.2 Million Children
Total	11.7 Million Children

As already noted, this total of 11.7 million is an overestimate of undetermined magnitude, since some fraction of children exposed to leaded paint have also been exposed to other sources.

Alternatively, one can select the largest number of children exposed to a single primary source of lead in dust and soil or leaded paint, and consider this number an underestimate. This method may be used because not all children exposed to leaded paint have contact with the other primary generators. This number is 5.9 million children. As an overall estimate, between 5.9 and 11.7 million children are potentially exposed to dust/soil lead.

Estimates of children exposed to lead in dust and soil sufficient to elevate Pb-B levels to potentially toxic ranges cannot be readily obtained in any precise way. One can achieve totals at each Pb-B value, e.g., 15, 20, or 25 µg/dl, for the primary contributors--paint, gasoline, and stationary source emissions as given in earlier sections. A major difficulty for estimating such Pb-B elevations is finding a reliable method for apportioning of a given Pb-B value to either the primary contributor, e.g., paint, or to the receiving pathway, dust and soil.

F. NUMBERS OF CHILDREN EXPOSED TO LEAD IN DRINKING WATER

Studies in the United States and elsewhere have shown that drinking water is a potentially significant source of human lead exposure. Lead can

contaminate the water at three points: (1) the water source itself--rivers, reservoirs, and groundwater; (2) the distribution system from water supply to living units, i.e., water mains, and (3) the plumbing in the home, e.g., lead solder. Actually, contamination rarely occurs in water sources from service connection lines and goose necks (connectors for main street to house line), and little is associated with the distribution system. By far, most of the contamination comes from domestic plumbing and plumbing in such public buildings as elementary schools, day-care centers, kindergartens, etc. Specific sources are lead pipe service connections, lead-based solder in copper plumbing, and corrosive (lead-dissolving) water in the plumbing.

While lead in drinking water is usually considered a source in the child's home, a potentially significant exposure risk also exists in such public facilities as elementary schools, kindergartens, day-care centers, etc. One potentially important but little recognized problem in schools and other public facilities is lead contamination of drinking water obtained through taps, water fountains, and coolers. There are several reasons why water in schools could be a hazardous exposure source for young children:

1. Water-use patterns in schools (school periods, weekends, vacations) involve long standing times of water in these units, which permit leaching.

2. Both water cooler-fountains and building plumbing may have lead-soldered joints and other sites of leachable lead, such as lead-containing surfaces in cooling tanks or loose solder fragments in pipes.

3. Unlike the case with lead-containing plumbing in private residences, which affects only the occupants, a single lead-containing cooler-fountain could expose a large number of users.

These findings mean that young children can ingest lead from water at sites other than the home and that the numbers of children and other risk groups exposed to water lead may be expanded to the school-age group.

The magnitude of lead-contaminated drinking water as a public health problem can be seen in the situation that prevailed until recently for half the population of Scotland. The problem was traced to interactions between soft, plumbosolvent water from sources such as Loch Katrine, the city of Glasgow's water supply, and lead pipes and lead-lined tanks in the homes of residents.

Exposure was widespread, and prevalences for elevated Pb-B levels were high. The epidemiology of human lead exposure via drinking water is discussed in detail in EPA's lead criteria document (1986a, Chapter 7 and 11).

In the United States, interactions between drinking water and residential plumbing involve either lead connectors (goosenecks) and service lines (commonly used before 1920) entering the home or copper piping with lead-based solder in the joints--a form of solder that came into use about 1950. Some lead service lines and connectors may have been used after 1920, and lead service connections were occasionally installed until 1986.

Published reports, both in the United States and the United Kingdom, indicate that five major factors contribute to the problem of lead-contaminated drinking water: (1) the length of the time water is in contact with the plumbing--the first water drawn from a standing column of water that has been in contact with lead in plumbing can have a high concentration of lead; (2) water temperature--hot water from pipes containing lead or joined with lead solder lead has more lead than cold water; (3) age of the solder--copper plumbing with lead solder that is less than 5 years old causes higher levels of lead in water than copper plumbing with lead solder that is older; (4) significant lengths of solid lead pipes--water from long sections of lead pipes has high levels of lead; and (5) corrosive water source--all of the above conditions are intensified when the water source is corrosive. The most corrosive water is acidic, soft, or nonalkaline.

Many investigators, both in the United Kingdom and in this country (U.S. EPA, 1986a), have examined the quantitative relationships of Pb-B levels to lead in drinking water. These relationships, reflecting the complex interactions of lead with biological responses, are often described by cube-root and other mathematical functions. Worth et al. (1981) examined residents in Boston, MA, where lead plumbing is still relatively common. These workers found a clear association between lead levels in water and Pb-B levels in children under 6 years old. Children exposed to tap water with lead levels above the U.S. standard of 50 µg/l of water had elevated Pb-B levels, as a group, above 35 µg/dl. These differences were observed during a survey period before corrosion controls were implemented to reduce the lead levels in tap water significantly.

Toxicokinetically, lead in drinking water is probably absorbed more completely than lead in food or other media, especially when the water is drunk

between meals or on an empty stomach in the morning. For lead food intake by adults, 10 to 15% of the lead is absorbed, but for water, 35 to 50% or a higher percentage, is absorbed (U.S. EPA, 1986a). Viewed in the context of risk assessment for the case of adults, lead in water presents three to five times the risk for systemic exposure as does lead in food--given the same concentrations of lead. Relative absorption rates are generally higher in children, and the difference may result in rates from water well above the estimate of 50% in children for lead in food (see Chapter III).

1. Estimation Strategies and Methods

As noted earlier, there are three levels of exposure from lead in drinking water that can be defined for U.S. children: potential exposure; some actual exposure at a measurable but not necessarily toxic level; and actual exposure at high toxic risk levels.

In the first approach, estimates of the numbers of children at potential risk of exposure to unhealthy levels of lead from drinking water differ in precision. Several estimates are given below. The estimates of the Division of Housing and Demographic Analysis, Housing and Urban Development (HUD), also include inventories of older housing units (which, as stated earlier, tend to have lead pipe segments at service connections) and provide an index of the persistence of such units in the national housing inventory.

Next considered is an estimation of the numbers of children consuming waterborne lead at some elevated level. According to the analysis by EPA's Office of Policy Planning and Evaluation (U.S. EPA, 1986b), 42 million people in the United States may receive drinking water with lead levels that exceed the proposed EPA maximum contaminant level (MCL) for lead, 20 µg/l, at the tap. With this estimate, one can use Census Bureau data to calculate the number of children at risk for elevated Pb-B levels from water lead levels above 20 µg/l.

EPA's analysis of the extent of lead contamination of tap water uses data on water samples collected by the Culligan water softening company in a cooperative study with EPA. Laboratory analyses were performed by the Illinois Institute of Technology. These 772 grab samples taken at random times during the day were collected in 580 cities in 47 states. They indicate that 16% of the water from U.S. kitchen taps contain 20 µg/l of lead or more. In addition, newly installed plumbing is at particular risk of elevated lead levels because

there has not been sufficient time for a film of calcium carbonate to build up on the inside of the pipes, which produces a protective barrier between the water and the materials of the plumbing system. New homes were not included in the Culligan data. Therefore, EPA included the inhabitants of housing built within the past two years to be an additional subpopulation at risk of elevated lead levels in drinking water.

Since this national exposure analysis was completed in 1986, EPA has collected data on local conditions throughout the country. Data on lead leaching rates and contamination patterns in many places confirm that the occurrence of high levels of lead in drinking water is widespread, and may indicate that the national projections underestimate actual exposure (U.S. EPA, 1987c). A significant omission in the national estimate is the prevalence of high lead levels in drinking water in schools.

2. Results

Table VI-12 shows the most general estimates of numbers of U.S. children under 5 years old and those 5 to 13 years old tabulated by age of housing. Using Census Bureau data, U.S. EPA (1987a) estimates that 5.2 million children under 5 years old and 8.7 million children 5 to 13 years old are in older housing, some fraction of which would have old lead service connections for water supply.

The data tabulated in Table VI-12 are of the most general form. A more refined estimation of the numbers of children exposed to water in leaded plumbing is given in Table VI-13, which provides more relevant housing age categories for greatest risk, independent of the corrosivity of water, i.e., homes built before 1920 and the newest units built within the past 2 years. In Table VI-13, the most recent column of age/data is for 1983. In 1983, of the 21 million U.S. children under 6 years of age, 13% or 2.73 million lived in units that had lead water service connections. Similarly, 4% or 840,000 children lived in homes built within the last 2 years, which is the housing fraction having lead-soldered new copper plumbing. Table VI-14, a tabulation from EPA, is similar to that of Table VI-13, but expands the childhood age bands to age 13 and has a 1-year difference in the younger age band.

In Table VI-15, the tabulation shows the relative persistence of aging housing stock. Such data indicate the persistence of the lead service connection problem by virtue of persistence of older housing containing this exposure

TABLE VI-12. NUMBERS OF CHILDREN LIVING IN HOUSING CLASSIFIED BY HOUSING AGE[a]

Age of Housing (% Total)	<5 Years Old	5-13 Years Old	Total
Pre-1940 (29%)	5.2 M	8.7 M	13.9 M
1940 - 1949 (9%)	1.6 M	2.7 M	4.3 M
1950 - 1959 (16%)	2.8 M	4.8 M	7.6 M
1960 - 1969 (20%)	3.6 M	6.0 M	9.6 M
1970 - 1983 (27%)	4.6 M	7.8 M	12.4 M
Total	17.8 M	30.1 M	47.9 M

[a]Source: Statistical Abstracts, 1985: Table 27 (July 1, 1983) and Table 1315 (Fall, 1983).

TABLE VI-13. CHILDREN POTENTIALLY AT RISK FOR LEAD EXPOSURE BY HOUSEHOLD PLUMBING, BY AGE[a,b]

Age of Housing	Number of Children <6 Years 1973	1978	1983	Exposure Profile
Total (Number)	14 M	19 M	21 M	
In housing built:				
Pre-1920 (%)	13	13	13	Lead pipes (+ lead paint)
1920-1949 (%)	25	25	24	Iron pipes (+ lead paint)
1950-1984 (%)	54	55	59	Lead solder (+ lead paint)
Within past 2 years (%)	8	7	4	Fresh lead solder

[a]Source Totals: Special tabulations from 1973-1983 Annual Housing Surveys.

[b]Percentages from Special Report: Division of Housing Demographic Analysis, HUD, Communicated January 7, 1987.

source. For example, about 40% of the oldest, lead service connection (pre-1920) homes are still occupied. This may be an upper-bound estimate because old plumbing has been replaced in some of this old housing stock, but exact information for this is not available.

TABLE VI-14. ESTIMATED NUMBERS OF CHILDREN AT GREATEST RISK OF EXPOSURE TO LEAD IN HOUSEHOLD PLUMBING

New Housing		Population at Risk
8.8 million people in new housing with lead soldered piping[a]:		
(8.8 M)(7.6% of population less than 5 years old)	=	0.7 M
(8.8 M)(12.8% of population 5-13 years old)	=	1.1 M
Total number of children at risk in new housing	=	1.8 M
Old Housing[b,c]		
If one-third of housing units built before 1939 contain lead pipes,[d] then (0.33)(0.29) = 10% of housing have lead pipes.		
(0.10)(17.8 M children less than 5 years old)	=	1.8 M
(0.10)(30.1 M children 5-13 years old)	=	3.0 M
Total number of children at risk in old housing	=	4.8 M

[a]Source: Reducing Lead in Drinking Water: A Benefit Analysis (U.S. EPA, 1986b, based on 9.6 million in new homes and 92% of these homes with metal plumbing).

[b]Source: Derived from Statistical Abstracts, 1985; Table 27, and Table VI-12 of this report.

[c]This group is a subset of the category of children living in housing built before 1939.

[d]Source: David Moore, Office of Policy Development and Research, U.S. HUD, Submissions to ATSDR, January, 1987 and U.S. EPA.

TABLE VI-15. PERSISTENCE OF AGING HOUSING STOCK IN OCCUPIED U.S. HOUSING INVENTORY (MILLIONS)[a]

Cumulative Total Built	Census of						
	1920	1930	1940	1950	1960	1970	1980
Pre-1920	24		21	20(E)			9(E)
Pre-1930		30	30	29(E)	26(E)		
Pre-1940			35	34(E)	31	27	21
Pre-1950				43	39	36	30

[a]Tabulated values of Division of Housing and Demographic Analysis, HUD: Special Report of January 7, 1987.

(E) = Estimated by authors of HUD report.

Corrosivity of drinking water, i.e., softness, lower pH, etc., is an environmental factor that affects the presence of waterborne lead other than from lead in plumbing. U.S. EPA (1986b) has estimated that about 62 million U.S. people receive such water. If we assume that 11% of these individuals are children under 7 years of age, then about 6.8 million such children are in homes where corrosive water is liable to mobilize lead to some unquantifiable extent.

To estimate the numbers of children exposed to drinking water with sufficiently high lead levels to elevate Pb-B levels, we assumed that children receiving drinking water that exceeds 20 µg/l are at risk of some Pb-B elevation (U.S. EPA, 1986b). A total of 42 million people are estimated to receive water with lead having more than 20 µg/l of water. Census Bureau data indicate that 9% of the U.S. population are children under 6 years of age. Therefore, 3,780,000 children under 6 years of age are exposed to drinking water lead above 20 µg/dl.

In addition to exposure in the home, other sources of water lead exposure may exist during time spent by children in public facilities. Precise numbers of preschool and school age children who may be exposed or are exposed to lead in drinking water in schools, day care centers and other settings cannot now be accurately estimated, given that the necessary survey data are not available. However, exposure to lead in drinking water in these settings can be important. As noted earlier in this section, water use patterns in schools are different from those in homes and they favor the accumulation of leached lead after long lapses in use, for example, summer and holiday break periods and weekends. Such water standing in school plumbing may increase the risk of excessive lead exposure through ingestion of potable water.

With regard to the overall problem of lead in school drinking water, state-level education and health units in two states have reported results from school drinking water lead surveys within their jurisdictions. The Minnesota Department of Health (MDH) recently carried out two surveys of lead in drinking water in Minnesota schools. The first and smaller survey (Minnesota Department of Health, 1986) consisted of two phases. In Phase I, 31 schools in 30 Minnesota cities were surveyed, including 24 elementary schools or schools with elementary grades. Of the 67 water samples collected and analyzed, 17 (25%) exceeded the current EPA limit of 50 µg/l and 27 (40%) exceeded the proposed goal of 20 µg/l. These were "first-flush" samples

obtained after weekend or holiday periods. In Phase II, samples at five of the original sites were examined for both first-flush and post-flushing lead levels. First-flush levels ranged from 2.2 to 500 µg/l with a mean of 141 µg/l (median = 38 µg/l). After 1-minute flushing, the mean value was 9.5 µg/l (median = 6.7 µg/l).

The first survey was followed up with a much larger statewide survey, which has just been completed. Preliminary summary statistics provided by the MDH indicate that two first flush samplings, 30 days apart, were obtained from 104 (25%) of Minnesota's 414 school districts. These repeat samplings consisted of 1,157 and 1,051 water samples respectively. In the first data set, 74 samples (6.4%) exceeded the existing EPA limit of 50 µg/l and 166 samples (14.3%) exceeded the proposed 20 µg/l goal. For the second sampling, the corresponding exceedences were 85 (8.1%) and 150 (14.3%), respectively. In neither survey was the nature of the tap water drinking source specified, e.g., non-refrigerating fountain, electric cooler, or other sources.

In a similar type of survey, the Maryland Department of Health and Mental Hygiene (MDHMH) examined drinking water lead levels in three stages in the summer of 1986, as described in Maryland Department of Health and Mental Hygiene news releases (September 22, 1986 and September 30, 1986). These schools were characterized as buildings less than four years old or units where new plumbing was installed in the last four years. Lead leaching was such that flushed water lines began to accumulate the toxicant after two hours of standing without further use. In July 1986, a survey of 45 schools, including a number of schools in the city of Baltimore, showed that 20 of them (44%) had lead levels above the EPA limit of 50 µg/l. In September 1986, MDHMH reported that a second survey yielded 33 (30.5%) of 108 schools with lead levels exceeding the EPA limit of 50 µg/l. Further examination of MDHMH's summary data as provided in the releases indicates that water samples from 72 (67%) of these 108 schools were at or above the proposed EPA goal of 20 µg/l. In a third stage of the Maryland school survey, carried out in September 1986, 30 schools were examined for the first time, and two of them exceeded the 50 µg/l limit. However, examination of the MDHMH summary data indicates that 9 (30%) of the schools were at or above the proposed goal of 20 µg/l. From the available information it appears that the new lead soldering in school plumbing and/or lead contamination in fountains themselves are suspected sites of lead leaching into water.

Contamination of drinking water by lead in other public facilities has also been reported. Unpublished preliminary data on lead levels in water from drinking fountains/coolers at two U.S. Naval facilities have been made available to the U.S. EPA and ATSDR (see Table VI-16). Of the approximately 90 fountains/coolers sampled by the Navy contract laboratories, water lead levels above the current EPA 50 µg/l limit were found in about 35% of combined first-flow, 1-minute, and 2-minute draw samples. Of these fountains/coolers, over 60 were major brands; the remainder were not identified by brand. In this survey "first-flow" refers to the first water obtained from the source at various times during the day. Thus, such samples may not have been the first flush of water after overnight standing. Of 39 first-flow samples, 23 (58%) were at or above a proposed EPA goal of 20 µg/l, and 31 (72%) were at or exceeded the level of 10 µg/l. For the 1-minute draw samples, 26 (72%) were at or above 20 µg/l, while 30 (83%) were at or above 10 µg/l. While it is not fully clear how much of the lead in these samples was contributed by the fountains/coolers and how much by the buildings' plumbing, water from some fountains/coolers in the Navy survey contained additional lead above that expected based on water lead levels from plumbing lines or taps in the same building.

TABLE VI-16. LEAD LEVELS IN WATER SAMPLES OBTAINED FROM WATER FOUNTAINS/COOLERS AT NAVAL FACILITIES IN MARYLAND[a]

Parameter	Lead in First Flow	Parameter	Lead in 1 or 2 Minute Flush
	N = 39		N = 90
High	570 µg/l	High	830 µg/l
Mean	101 µg/l	Mean	69 µg/l
Median	39 µg/l	Median	30 µg/l
Low	<5 µg/l[b]	Low	<5 µg/l[b]

[a]Source: Based on data obtained from U.S. Navy (1987).

[b]Analytical detection limit of 5 µg/l.

The above data from two separate states' surveys of schools and from Naval facilities in Maryland raise important questions about the potential for significant lead exposure from sources of potable water in public facilities. Systematic evaluation of this potential problem will be needed to determine its scope and any appropriate corrective measures.

Table VI-17 shows EPA's preliminary calculations of the relationship of tap-water lead in homes as a function of both pH and age of house. As expected, first-flush samples are more apt to have excessive lead concentrations than fully flushed collections. The more acid, i.e., more corrosive, water produces the greater lead contamination at values above 20 µg/l, the EPA proposed standard. Although the frequency of elevated lead samples decreases with the age of the home, there are still unacceptable first-flush percentages for corrosive and neutral waters, i.e., 51% and 14%, respectively, in houses 6 years and older. Equally important is the finding that in houses up to 2 years old that have corrosive water with a pH ≤6.4, 51% of the samples of fully flushed water contained more than 20 µg/l of lead.

TABLE VI-17. PERCENTAGE OF VARIABLY COLLECTED WATER SAMPLES EXCEEDING 20 µg/l OF LEAD AT DIFFERENT pH LEVELS AND BY AGE OF HOUSE[a]

Age of House	pH	Percent of Samples >20 µg/l	
		First Flush	Fully Flushed (2 min)
0-2 years	≤6.4	93	51
	7.0 - 7.4	83	5
	≥8.0	72	0
2-5 years	≤6.4	84	19
	7.0 - 7.4	28	7
	≥8.0	18	4
6+ years	≤6.4	51	4
	7.0 - 7.4	14	0
	≥8.0	13	3

[a]Source: U.S. EPA Office of Drinking Water (1987c); preliminary results from "Lead Solder Aging Study."

Estimations for the category of actual exposure to water lead sufficient to cause Pb-B levels with toxicity risk are those employed by U.S. EPA (1986b) in its examination of numbers of U.S. children who would be above certain criterion values because of drinking water lead levels above 20 µg/l. U.S. EPA (1986b) employed logistic regression analyses techniques analogous to those

employed for projections of children whose Pb-B levels would decline from specified Pb-B levels from phasedown of gasoline lead (U.S. EPA, 1985).

In this analysis of the benefits of reducing the lead standard for drinking water, U.S. EPA (1986b) estimated that 241,100 children had Pb-B levels above 15 μg/dl due to lead in their water as a result of the action of corrosive water on aged plumbing. Of these, EPA estimated that 100 children have Pb-B >50 μg/dl as a result of waterborne lead, 11,000 have Pb-B levels between 30 and 50 μg/dl, and 230,000 have Pb-B levels between 15 and 30 μg/dl.

G. NUMBERS OF CHILDREN EXPOSED TO LEAD IN FOOD

Dietary lead can account for a significant portion of the total body lead burden in populations not having sizable exposures to the sources already discussed. In many people, it can add enough to cause an elevation in Pb-B levels. Dietary lead intake is important because it is a source of exposure for the entire population. Since food is ingested in relatively large amounts, lead concentrations in food at the parts-per-billion (ppb) level correspond to intakes of microgram quantities. For example, an average food content of 50 ppb of lead yields an intake of 50 μg/day when 1 kg of food is eaten. These 50 μg will elevate the Pb-B level by a measurable amount, about 8 μg/dl, using one relationship cited in U.S. EPA (1986b).

Over the centuries, lead in food and beverages has provided much of the toxicologic record for lead poisoning, especially when lead was used in large amounts as an adulterant (see, for example, Wedeen, 1984). At present, lead enters food at production, harvesting, processing, and distribution steps (U.S. EPA, 1986a). In addition, beverages and other liquids can be contaminated by improperly glazed pottery (Klein et al., 1970) and various utensils.

We cannot determine how much of the total food lead is due to human activity without knowing the background level. Wolnik et al. (1983) gathered background or near-background levels in cereals, grains, vegetables, and several meats. When we relate the results from these studies to current food consumption surveys conducted by the FDA, we find that production and processing increase the lead content of food 2-fold to 12-fold. Sources of lead at the production stage include fallout onto plants, some lead uptake through the root system, and lead in forage and soil in areas where livestock graze. Lead enters in food processing mainly via lead-soldered cans.

In the late 1970s, the use of lead to solder the seams in cans began to be phased out, and consequently, the lead in canned food has significantly decreased. With infant foods, for example, lead levels in evaporated milk have declined, on the average, from 0.5 μg/g wet weight in the early 1970s to 0.07 μg/g in 1981. Similarly, lead levels in some juices have declined about 95%.

The daily dietary intake of lead in young children has been examined in several surveys. Beloian (1982, 1985) has proposed and used a food consumption model for evaluating daily contaminant intake. The elements of this model include the numbers of times specific foods are consumed in 14 days, lead content, and size of portions. Three age groups of children were examined: 0 to 5 months, 6 to 23 months, and 2 to 5 years. Lead levels in food groups were averaged over the period 1973-1978.

Table VI-18 shows data reported by Beloian (1982). They include the mean daily lead intakes and the distributions of lead intake by indicated percentiles, with food lead data for 1973-1978. Mean values are moderate, but at the higher percentiles the lead intakes are sizable, which emphasizes the importance of considering distribution phenomena. These numbers are based on older, higher food lead measures than may exist currently.

TABLE VI-18. DAILY MEAN DIETARY LEAD INTAKE BY PERCENTILES[a]

Age Group	Mean Intake (μg/day)	Percentiles			
		50	90	95	99
0-5 months	15	11	31	36	55
6-23 months	59	54	89	110	140
2-5 years	82	79	120	130	170

[a]Adapted from Beloian (1982). Based on averaged data from 1973-1978.

Bander et al. (1983) conducted a nationwide 7-day food consumption survey of 371 preschool children 0 to 5 years old, using food intake levels provided by the National Food Processors' Association in 1980. Table VI-19 shows the means of lead intake, the means related to kilocalories, and the means per unit mass of food as a function of age.

In the Beloian (1982) study, Table VI-18, the three age groups had mean intakes of 15, 59, and 82 µg/day. The corresponding lead intakes expressed as a function of body weight (kg) were 2.7, 6.1, and 5.6 µg/kg.

Bander et al. (1983) found that children had a mean intake of 62 µg/day lead on a subject basis and 22 µg/day on a caloric or food mass basis. Partitioned by age, the intake ranged from 49 µg/day for infants to 74 µg/day for 5-year-olds (Table VI-19). Because of the continual reduction in the lead content of foods, estimates of current dietary lead intake are smaller.

TABLE VI-19. MEAN DAILY DIETARY LEAD INTAKE IN PRESCHOOLERS CLASSIFIED AS TOTAL OR NORMALIZED DAILY INTAKE[a]

Age (years)	Total Mean (µg/day)	Mean/500 kcal	Mean/500 g Food
<1	49	17	16
1	55	24	21
2	56	22	20
3	65	22	22
4	65	22	21
5	74	20	22

[a]Adapted from Bander et al. (1983). Based on food lead measurements in 1980.

1. Estimation Strategies and Methods

The number of persons potentially exposed to some level of lead in food includes the entire U.S. population because a centralized food production and distribution system serves virtually all parts of the nation. Each food processing step adds to the amount of lead in food (U.S. EPA, 1986a). Therefore, all U.S. children under the age of 6 are at potential risk. We calculated the number for the 1985 estimated population, using the lowest projection method and found 9% of the total population to be under 6 years of age. This amounts to about 21 million children.

We used the following strategy to evaluate the number of children exposed to lead levels in food that are high enough to elevate the Pb-B so that it approaches some toxicity level:

(1) The Pb-B level associated with lead in food should not be more than 10 µg/dl, given other inputs such as from drinking water, contact with dust and soil, and even direct inhalation--all of which also contribute to

Pb-B and push the final level unacceptably close to 20 to 25 µg/dl, the lead toxicity risk levels identified by the CDC (1985) and WHO (1986).

(2) A relationship of lead in food to Pb-B in infants and toddlers derived from published data (Ryu et al. (1983) is Pb-B (µg/dl) = 0.16 x diet Pb/day (µg/day). From this relationship, a Pb-B less than or equal to 10 µg/dl from food requires a lead in food intake of less than or equal to 62.5 µg/day.

(3) The percentage of children who have lead intake at or above about 65 µg Pb/day should then be selected from those studies whose results can be applied to the nation as a whole.

Surveys by Beloian (1982) and Bander et al. (1983) were designed so that they can be applied nationally for the years when the lead levels in foods were measured. Given the centralized food system, any comprehensive U.S. survey of lead levels in food becomes a national survey.

The Beloian (1982) data, Table VI-18, show that the 95th percentiles of daily lead intake are 36, 110, and 130 µg/day for children aged 0 to 5 months, 6 to 23 months, and 2 to 5 years, respectively. If overall declines for average lead levels in food are assumed to be at least 50% from 1973-1978 to the present and across the entire distribution, these levels would now be 18, 55, and 65 µg/day. Uniform downward changes across the entire distribution are reasonable, assuming a centralized food supply and no major changes in general food intake habits from 1973 to the present. The last value, about 65 µg/day, is the upper limit of lead levels in food to produce Pb-B levels lower than 10 µg/dl. For the 6 to 23 months group, 55 µg/day is also close enough to the limit of concern for the 95th percentile. While we are not sure about the relative vulnerability of the very young (0 to 5 months old) to lead exposure, this youngest group has a very low intake and is not included in the analysis. This approach allows us to say that, at most, 5% of the children under 6 years of age, excluding children 0 to 5 months old, are at or approaching a dietary lead exposure that pushes their body burden close to that associated with early toxicity if they are also exposed to other typical lead sources.

In general, the Belonian percentiles agree with other data. Bander et al. (1983) noted that 8.9% of children had daily lead intakes of at least 100 µg/day. Most of these children were 4 to 5 years old. Considering that this study was conducted more recently and used food processors' data for 1980

analyses and the lower levels of lead at the 95th percentile for younger children, the Bander et al. (1983) results are consistent with those of Beloian (1982).

Uncertainties are inherent in these approaches for estimating the numbers of children who are exposed to lead in food to a degree sufficient to cause measurable elevations in Pb-B levels. First, the percentage of decline in food lead from 1973-1978 to more recent periods is difficult to determine. We cannot specifically compare data gathered before 1981-1982 with more recent survey findings. The lead content of certain categories of foods has been markedly reduced (see Chapter IX), but changes in the overall dietary intake of lead are difficult to measure. Second, the direct/indirect air lead contributions are difficult to measure. Third, the criterion Pb-B value selected for calculating background levels is not a precise measure. Finally, selecting a single value for lead intake in food about a given percentile affects the margin of toxicological safety. If the current value is significantly below about 63 µg/day at the 95th percentile of the distribution then the resulting Pb-B will be lower, and the other levels must be adjusted accordingly.

2. Results

The number of children under 6 years is estimated to be 21,405,000 (World Alamanac, 1987); this number is based on the Census Bureau projections for 1985 by the lowest of three methods. Since we exclude infants 0 to 5 months old from our calculations, the base population is reduced to about 19,474,000.

Of this estimated number, a maximum 5%, or 973,700 children under 6 years old, receive enough lead from food alone to constitute a potentially unacceptable lead burden, as indicated by Pb-B levels. If the increase in Pb-B from food is lower than the one selected here (i.e., less than 10 µg/dl), then the numbers of children at risk would be lower for a given daily lead intake. On the other hand, the reference Pb-B level of 20 µg/dl selected for the estimate is not the lowest criterion level for the purpose.

H. SUMMARY AND OVERVIEW

In this chapter, estimates of the numbers of lead-exposed young children, arranged by the source of exposure, are derived and described. These estimates

define different degrees of exposure, both within an exposure category and across exposure categories. Overall, the data sets used for these source-based analyses vary highly in their precision and accuracy, in their assessment and definition of actual versus potential exposure, in their representativeness for childhood exposure nationwide, and, finally, in the relationship between the degree of exposure and some toxicity risk.

With regard to the accuracy and precision of numbers relative to source of lead, it is not possible to measure the level of estimation error for each of the source categories. For example, in some cases we deal with actual counts of individuals, while in other cases we are confined to combining various elements of an estimation analysis, each having variable and relatively undefined precision. Some estimates are also judged to be upper or lower bounds for actual values.

In a number of the estimation procedures, available data for the prevalence of elevated Pb-B levels among children are derived from either regional analyses or reasonably matched strata of children from NHANES II, and their representativeness for the actual census of source-exposed children must be carefully appraised.

Finally, the various source-based estimates of actual exposure to lead will differ as to how closely measures such as blood lead can be related to what we term some toxicity risk. In some cases, a Pb-B criterion value associated with presently understood toxicity risk is used, while in others we can only say that a blood lead elevation is predicted to occur at some undefined upper value. It should be noted that definitions of toxicity in terms of Pb-B values have been declining. For this reason, multiple Pb-B values are used where calculable. For purposes of summary and discussion, each source-based estimation analysis is treated separately.

1. Paint Lead as an Exposure Source

We have presented three levels of estimates for young children exposed to lead in paint. The first two analyses involve actual U.S. Census and housing counts and can be taken as reasonably accurate enumerations of children who are at least potentially, if not actually, exposed to lead in paint.

The most general approach was to determine U.S. Census enumerations of young children (less than 7 years old) and then compile which portion of those

children will be in residential units having leaded paint meeting a minimal definition of exposure: greater than or equal to 0.7 mg/cm^2. In so doing, we determined that the best available data for this analysis provided a "national best estimate" of about 12 million children potentially exposed to lead in paint, with about 13.6 million children for the upper bound. Of these, about 5.9 million children are in pre-1940 residential units. In this case, we did not address leaded paint in the presence of those factors that might additionally enhance childhood lead paint exposure, for example, deterioration indexed by peeling leaded paint, lead-painted broken plaster, etc. Such indices of deterioration are not required before leaded paint exposure becomes a problem. For example, children can readily gnaw and chew on lead-painted woodwork such as window sills, even if the painted surfaces are in good repair.

The next level estimated involved enumeration of children determined to be residing in unsound/substandard housing. Here we would expect that the probability for lead exposure sufficient to elevate blood lead to some level would be much higher than it would be through a simple housing count, where undeteriorated and deteriorated housing is combined. Using figures from one study, the best national and upper-bound estimates for the numbers of children in such lead-painted, unsound residential units are about 1.8 and 2.0 million children, respectively. These figures are probably an overestimate since units having one characteristic of disrepair would also have others, yielding double counting in at least some cases.

These figures complement the numbers of children presented as census data enumerations by SMSA presented in Chapter V, Section C. For example, the total census count of children in the 318 SMSAs who live in residential units built before 1950 amounts to about 4.4 million (Table V-20). From Table VI-3, the number of children (national best estimate) in all pre-1940 housing is approximately 5.9 million. Taking into account all factors of differences in the two analyses, approximately 75 to 80% of all children in older U.S. housing having leaded paint are found in urban areas.

The numbers of children in unsound housing tabulated in this chapter do not include those children of families who are now moving back to older housing in cities and rehabilitating such housing as part of the phenomenon of gentrification. This number may be sizable but it is not possible to estimate such a subset of the urban child population.

To examine the number of children estimated to have actual lead exposure sufficient to cause Pb-B elevation, we have employed prevalences of different Pb-B criterion values for children in inner-city housing and derived from either a survey of Chicago housing (Pb-B >30 μg/dl) or NHANES II projected prevalences at Pb-B levels >15, >20, and >25 μg/dl. The Chicago-based prevalence of 12.8% applied to children in deteriorated housing yields a total of approximately 230,000 children with Pb-Bs above 30 μg/dl. The numbers of children in such housing with Pb-B levels above 15, 20, and 25 μg/dl were, respectively, about 1.20 million (15 μg/dl), 0.55 million (20 μg/dl), and 0.19 million (25 μg/dl). Although we recognize that leaded paint in elementary schools, kindergartens, etc., will pose potential risk for young children, we cannot as yet quantify this segment of the leaded paint problem. Similarly, leaded paint inputs to dusts and soils around public facilities are not readily quantifiable.

2. Gasoline Lead as an Exposure Source

Estimating the numbers of young children with potential exposure to lead from combustion of leaded gasoline is not a straightforward process. In this report, an estimate was based on the numbers of children living in the largest U.S. urban areas where vehicular traffic is expected to figure significantly in childhood exposure. For the largest 100 U.S. cities and for children less than 7 years of age, this figure is approximately 5.6 million children.

To examine numbers of children having exposure to gasoline lead at various Pb-B criterion values, this report relied on data from EPA's Office of Policy Analysis. The EPA report derived numbers of children predicted to fall below indicated Pb-B values with the phasedown of lead in gasoline, and projections were extended to years beyond 1987. For 1987, for example, 563,000 children up to 13 years old will have Pb-B declines to below 20 μg/dl. The corresponding number for a Pb-B level of 15 μg/dl was approximately 1.6 million. Please note that in this case the age interval for defining children extends to 13 years of age. It is not possible to adjust these figures to the age interval up to 5 or 6 years employed in most cases, since Pb-B distributions are child age-dependent and do not permit a simple linear fractionation of these figures.

Estimates provided by EPA do not reveal specific Pb-B values for the children in the analysis, but rather shifts in Pb-B levels sufficient to cause

declines below Pb-B criterion values are shown. With data displayed in this manner, the effect of gasoline lead reduction is focused on relative toxicity risk, that is, the Pb-B criterion values.

3. Lead From Stationary Sites as an Exposure Source

The numbers of children exposed to lead from stationary source emissions have not been well catalogued and related to potential exposure risk. One study found that approximately 230,000 children are in the exposure zones associated with operations such as smelters, refineries, acid-lead battery plants, etc.

A range of estimates for the number of children actually exposed to lead from such sources was also presented where prevalences were restricted to results from several specific smelters. A likely national best estimate could not be derived from the limited data. For primary smelter operations, prevalences range from 1 to 26% (Pb-B ≧25 μg/dl and ≧35 μg/dl EP). The corresponding figure for secondary smelters, based on one survey, is 4% (Pb-B >20 μg/dl). The corresponding numbers for primary operation communities range from 210 to around 5,500 children. With secondary smelters, the number is approximately 7,500 children.

4. Lead in Dust and Soils as an Exposure Source

To estimate the number of young children exposed to lead in dust and soil, we combined the numbers of children exposed to the primary generators of this source category, i.e., lead in paint and lead from stationary/mobile (gasoline) lead sources. As described in the report, the upper limit of young children potentially exposed in this category is approximately 11.7 million and the lower limit is 5.9 million. The number of children actually exposed to dust/soil lead levels sufficient to cause elevated Pb-B levels that are also distinguishable from elevations due to paint, etc., cannot be easily determined by the various methods for obtaining estimates.

5. Lead in Drinking Water as an Exposure Source

One estimation method shows that approximately 1.8 million children less than 5 years old and 3.0 million children 5 to 13 years old have potential risk

for lead exposure from parts of old residential plumbing. For new homes with new and leachable plumbing lead solder, the corresponding numbers are calculated as 0.7 million and 1.1 million, respectively.

Next, the estimate for numbers of children having drinking water lead exposure sufficient to cause some Pb-B elevation but not necessarily to toxic levels is given. As derived from EPA's statistics, 20% of public drinking water supplies exceed the proposed Maximum Contaminant Level (MCL) of 20 µg/l, which eventually yields a total of about 3.8 million children. These children will have Pb-B elevations calculated as being at or above 3 to 6 µg/dl, based on the relationship between ingested lead and Pb-B [Pb-B = 0.16 x µg Pb/day (from water)] with daily intake of 1 or 2 liters of water at or above 20 µg/l. At this time, we estimate that 241,000 of these 3.8 million children would have a Pb-B level above 15 µg/dl, with 230,000 between 15 and 30 µg/dl, 11,000 over 30 µg/dl, and 100 over 50 µg/dl.

6. Lead in Food as an Exposure Source

When we examine this category of source-specific lead exposure in young children, we not only have a large affected population base for potential exposure, but one that is the focus of much activity to reduce the lead in this medium.

With respect to potential lead exposure from food at even some modest level of contact, virtually all young children will ingest some measurable amount of lead: that is 21 million children. The children estimated to have a lead exposure risk sufficient to cause some rise in Pb-B is approximately 5% of this base population, about 1 million children. This number is based on both lead levels in foods measured in the 1970s and on those levels adjusted for declines in more recent times; therefore, it may be an overestimate to some extent.

7. Ranking of Lead-Exposed Children by Source

It is not possible to rank rigidly the numbers of lead-exposed children from each lead source, a difficulty that applies to both potential and predicted actual lead exposure. For health assessment, it is more useful to consider the overall impact of each category and to rank qualitatively their specific characteristics. The reasons for this are related to the nature of the

different estimation approaches, the precision of the estimation process, and the definitions of potential and actual lead exposure and the size of the age intervals employed in source-specific estimating.

When specifically estimating actual lead exposure, any rigid ranking of children exposed by source can be misinterpreted with underestimates of particular concern. A good example of this is the data for children exposed to lead in paint at levels that elevate Pb-B to the toxic range where at least two factors would militate in the calculation of a low estimate.

We can provide the following general findings and conclusions about lead sources for childhood exposure and in utero exposure.

- In terms of both quantitative impact and persistence of the hazard, as well as dispersal of the source into the population, leaded paint has been and remains a major source for childhood exposure and intoxication.
- Following close to leaded paint as a troublesome and persistent lead source is dust/soil lead, dispersed over huge areas of the nation.
- Drinking water lead is now recognized as a potentially significant exposure source in both the home and in schools and other public facilities; a particular hazard is electric water coolers, as documented in the section on drinking water.
- From examining the above lead sources, they are all related, collectively as multi-source exposure in old housing and particularly old housing in varying stages of disrepair.
- Gasoline lead is declining significantly as a major lead source, particularly since the 1970s when it was adding about 40 to 50% to total Pb-B levels in the U.S. population.
- Stationary sources provide a very limited, though potentially high, source of lead exposure.
- Lead in food is declining in importance as a general exposure source, but daily intakes for recent years are still enough to add measurable amounts to total Pb-B levels of children.
- Time did not permit the detailed quantification of fetal exposure, via pregnant women as the surrogate risk group, in terms of source-specific exposure.
- With pregnant women, lead from food and water would be the main contributors to Pb-B levels above those considered "safe" for fetal protection.

- Since food and water lead primarily produce the projected Pb-B levels and total exposure counts for the four pregnancy categories in Chapter VII, we can estimate the joint contribution of food plus water to produce these numbers.