component of this effort and is important both for the numbers provided and for helping answer the obvious questions "Which children have the problem?" and "What can we start to do about it?"

From the key findings of Chapter V, we conclude that the total number of U.S. children exposed to lead at unacceptable levels (Pb-B >15 μg/dl), 2.4 million SMSA children or 17% of the SMSA child total, arise from many different socioeconomic and demographic strata.

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

The consequences of an estimating exercise, across strata, for exposure totals is simply that large numbers in a stratum's base population can have quite low prevalences for certain Pb-B levels and still yield numbers that are comparable to those obtained from high-risk strata that have smaller base populations of children but quite high prevalences of elevated Pb-B levels.

In Chapter V, a variety of estimating strategies were employed, and provide quite different numbers for exposure estimates. These differences were explained earlier in the summary. Furthermore, some of the totals complement each other, providing different views of the same total population of U.S. children. For example, examining the very detailed U.S. Census Bureau counts (not estimates) of children in the 318 SMSAs reported in terms of housing age and family income (Section C) produces the unexpected finding that more children in older housing (high paint-lead levels) were also in noncentral-city, nonpoverty families than were children associated with the typical risk groups. This observation corresponds to this report's projected Pb-B distributions in the nation's children.

These distributions in high-risk housing might account for why certain Pb-B prevalences in the otherwise lower-risk strata of U.S. SMSA children are as high as they are. In other words, distribution of the nation's children into high lead-exposure risk housing is uniform enough that all strata of such children, when examined by means of a national composite survey such as NHANES II, will produce significant prevalences. Clearly, however, other sources also

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)

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.

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

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.

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

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.

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.

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.

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.

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

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

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.

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

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

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.

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.

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

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.

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

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.

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 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

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

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.

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

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.