56

Environ Res 50

Exposure

REVIEW

Determination of Numbers of Lead-Exposed American Children as a Function of Lead Source: Integrated Summary of a Report to the U.S. Congress on Childhood Lead Poisoning[1]

PAUL MUSHAK*,[2] AND ANNEMARIE F. CROCETTI†

*Department of Pathology, (Adjunct), University of North Carolina at Chapel Hill, North Carolina 27514, and †Department of Community Medicine, (Adjunct), New York Medical College, Valhalla, New York 10595

Received February 21, 1989

In 1986, the U.S. Congress [Section 118(f), Superfund Amendments and Reauthorization Act (SARA)] directed the Agency for Toxic Substances and Disease Registry to provide to it a quantitative assessment of the contributions of various sources of lead to childhood exposure. We provided both a quantitative response to the mandate and a critique of low-level lead sources for U.S. population segments. We also present here an integrated assessment of major and low-level lead sources. Significant sources of lead in childhood exposure include lead in paint, dust, soil, and drinking water. Approximately 6 million U.S. children <7 years old reside in the oldest housing, with highest exposure risk due to leaded paint. About 2 million in deteriorated units are at particularly high risk for exposure with ca. 1.2 million children in oldest, deteriorated housing estimated to have blood lead (PbB) levels above 15 μg/dl. Soil and dust lead are potential sources of exposure for 6–12 million children. Residential tap water lead is a measurable source for ca. 3.8 million children, of whom the U.S. EPA estimates ca. 240,000 have water-specific exposures at toxic levels. Leaded gasoline combustion mainly in past years has produced, and will continue to produce into the 1990s, significant numbers of exposed children with toxicologically elevated PbBs. For 1990, 1.25 million children will have their PbBs fall below 15 μg/dl. Food lead can cause significant exposure in certain cases.

INTRODUCTION

As an environmental pollutant and human toxicant, lead is distributed in many of those environmental media which also serve as human exposure sources and pathways. Adequate risk assessment, therefore, requires consideration of both source-specific lead exposures and their relative contributions to total lead exposure among high-risk population segments.

This paper, an integrated summary of a Congressional report, is organized into four major sections. The first provides a brief overview of the various elements

[1] Based on Chap. VI, "Examination of Numbers of Lead-Exposed U.S. Children by Lead Source" and Chap. VIII, "The Issue of Low-Level Lead Sources and Aggregate Lead Exposure of U.S. Children," contained in *The Nature and Extent of Childhood Lead Poisoning in Children in the United States, A Report to Congress*. Submitted July 1988 by the Agency for Toxic Substances and Disease Registry (ATSDR), U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA 30333.

[2] To whom correspondence should be addressed at: 811 Onslow St., Durham, NC 27705.

0013-9351/89 $3.00

that define the degrees of lead exposure and those variables of population behavior which modulate human exposures. The second section deals with the major U.S. sources of childhood lead exposure in quantitative terms and with regard to the relative reliability of the estimates for exposed children. Third, we present a brief discussion of the aggregate exposure impact of low-level lead sources, i.e., sources which by themselves are not deemed significant but which combine to produce elevated toxicity risk. Finally, an overview of the topic with some qualitative assessments of relative importance of sources is given.

A. GENERAL ISSUES

1. Types of Lead Exposure and Their Relative Utility in the Quantitation of Exposure Risk for Human Populations

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 directly by eating paint chips or gnawing painted surfaces often simultaneously contact dust from chalked or weathered paint as well as atmospheric lead fallout. 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 also difficult to quantify exposure once the important sources have been determined. Human lead exposure can be indexed either by external or internal means—that is, environmental or biological monitoring.

Environmental monitoring can serve to estimate external exposure risk in the population. However, such estimates are broad because they estimate the number of subjects at the lead source, regardless of the level of lead intake/uptake. Although these estimates produce the largest numbers, they are the least accurate when associating exposure with the actual risk of toxicity. In short, environmental monitoring defines "potential" exposure and adverse health risk.

As examples, leaded-paint exposure is estimated by counting the houses with high probabilities of leaded paint and proportionately distributing the number of children from U.S. census counts among them. Alternatively, actual U.S. census counts of children in U.S. housing stratified by age can be done.

A more accurate and precise exposure assessment can be achieved by biological monitoring, i.e., individuals are sufficiently exposed to a lead source as to cause change in some systemic measure, e.g., level of lead in whole blood (μg Pb/dl, PbB). This estimate is commonly determined by some empirical relationship, usually through use of regression equations, in which PbB is related to lead in such media as ambient air, dust/soil, and leaded paint in old housing (see, e.g., U.S. EPA, 1986a).

The most health-specific way to assess exposure for source-specific lead is to examine the extent to which a lead source produces sufficient elevation in an indicator, e.g., PbB, as to produce unacceptable risks for onset of adverse health effects.

On the basis of the above discussion and with reference to methodologies employed for exposure analyses in the Congressional report, up to three levels of

source-specific exposure assessments are presented in this paper: numbers of subjects estimated to be in contact with the lead source but without reference to level of lead intake or uptake, i.e., potential exposure; numbers of subjects so exposed as to cause some measurable rise in the level of the internal indicator, PbB; and numbers of subjects estimated to be sufficiently exposed as to have PbB levels associated with at least early adverse effects.

The number of levels of estimation differed across sources. In the case of tap water lead, three levels of estimation accuracy for health risk were achievable, while dust and soil estimates were restricted to the potential exposure range provided by dust/soil contributors, i.e., paint lead and atmospheric lead fallout.

2. Relationships of External to Internal Lead Exposure in Human Populations

Lead exposure populations invariably show a range of responses in a biological indicator, e.g., PbB, when the environmental source is uniform in concentration and this is due to a variety of host factors which can be broadly grouped as to those which are intrinsic, e.g., interindividual variations in such lead toxicokinetic parameters as absorption, distribution, and excretion/retention and those which are behavioral in nature, e.g., dietary habits (e.g., Mahaffey, 1988; U.S. EPA, 1986a).

In human populations analyzed as to their cumulative frequency distribution of blood lead concentrations, PbB values are distributed log-normally (see, e.g., Chap. 11, U.S. EPA, 1986a) with a skewing in the upper tail of the distribution. A public health consequence of this skewing is that this upper tail encompasses a larger fraction of the entire population than it would in a normal distribution. Consequently, when lead exposure populations number in the tens and even hundreds of millions, any seemingly small percentage in the upper distribution tail above a given PbB will translate to significant numbers of individuals.

In Fig. 1, the characteristics of an illustrative log-normal distribution with re-

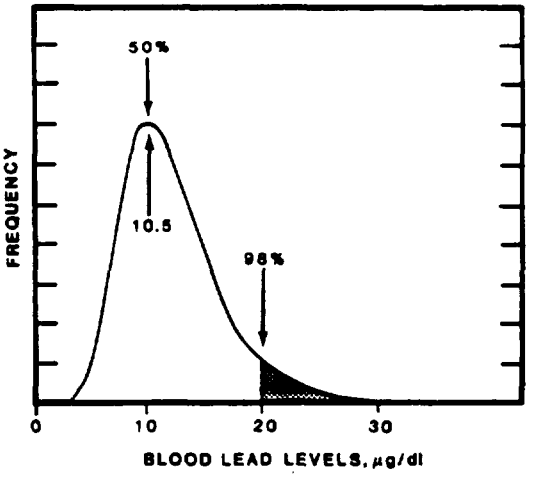

FIG. 1. Illustrative log-normal PbB distribution curve. Values originally from working paper for WHO (1987). Median = 10.5 µg/dl; 98% = 20.0 µg/dl; shaded area = 2%.

gard to actual calculations in a working paper prepared by the World Health Organization (WHO, 1987) for lead-exposed European adults are depicted. Available calculations done by the U.S. Environmental Protection Agency (U.S. EPA, Chap. 11, 1985, 1986a) using PbB data from the National Health and Nutrition Examination Survey (NHANES II, Annest and Mahaffey, 1984) indicate that children <7 years old have differing PbB distributions from adults.

A second but related feature of the distribution patterns of such lead exposure indices as PbB levels is that they better reflect risk for the entire population than do median or mean PbB levels. A rather moderate mean PbB in a huge population may disguise the significant risk assessment picture for those in the upper tail. In Fig. 1, the median level is ca. one-half that for the 2% of population at 20 μg/dl or above.

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

As discussed in detail by the U.S. EPA (Chap. 12, 1986a) and CEC/EPA (1989) and in critical studies and reviews cited in these documents, a number of socioeconomic/demographic variables can affect the systemic exposure to lead of such risk groups as very young children. Such factors affect the relative results that investigators find in relating such exposure indices as PbB to some measure of adverse effect.

To illustrate, 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 nor does not significantly contribute to an adverse effect. They simply imply that the interactions occur.

These factors do not diminish the consequences of low-level exposure in the overall lead problem nor should they distract 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 factors take on added meaning when we examine the claimed 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).

B. SOURCE-SPECIFIC LEAD EXPOSURE IN U.S. CHILDREN

1. Background Discussion

In this section there are estimated and discussed the numbers of U.S. children exposed or potentially exposed to six different lead sources/pathways: lead in paint, gasoline combustion, stationary source emissions, soil or dust lead, and lead in 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. Data of the latter type are contained in the report to Congress.

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.

2. Methods and Strategies for Source-Specific Lead Exposure Estimates

a. Estimates of numbers of children exposed to leaded paint. Estimates of U.S. children exposed to lead in paint are based on both potential exposures and estimates of the numbers of children predicted to have some elevated health risk because of paint-associated elevations in their PbB levels. Estimates of numbers of young children at potential exposure risk due to leaded paint include individuals living in all age-stratified and geographical region-stratified housing units and those units posing actual exposure risk because of deterioration: peeling paint, broken plaster, and other signs of deterioration. Data used include calculations by Pope (1986) and estimates of types and numbers of lead-painted dwellings from the *American Housing Survey of the U.S. Bureau of the Census, 1983* (U.S. Bureau of the Census, 1986). Estimates were based on a paint–lead exposure threshold level of 0.7 mg/cm^2 from the U.S. Centers for Disease Control (CDC, 1985).

Pope (1986) determined a child density factor of 0.287, i.e., children per dwelling unit, as obtained from the U.S. child population under 7 years old and the number of housing units in the Nation. In addition, national figures for housing yielded percentages of housing units having leaded paint as a function of age: pre-1940, 1940–1959, and 1960–1974. These housing age bands represent different prevalences for leaded-paint use. Data from the 1983 American Housing Survey provided, as well, percentages of total housing that these represent. Estimates were available of (1) the total number of children in homes with lead paint, (2) the numbers of children in homes both with leaded paint and meeting the indicated criteria for deterioration, (3) estimated numbers as both national best estimates and national upper bounds, and (4) numbers of children by four major regions: Northeast, Midwest, South, and West.

Direct nationwide estimates of the numbers of U.S. children having sufficient lead-paint exposure to produce measurable increases in PbB to toxic levels are not

available, per se. Such alternatives as the linking of PbB to leaded paint in national housing through published regressional analyses in individual epidemiological studies in lead-painted dwellings are not appropriate for purposes of this study. However, other alternate strategies were employed.

The first approach was to apply a prevalence rate of elevated PbB associated with leaded paint as derived by the U.S. Environmental Protection Agency (U.S. EPA, 1985) and based on a large study of PbB vs leaded paint presence in more than 80,000 living units in Chicago in 1978. This prevalence rate was then combined with Census Bureau data for numbers of U.S. children residing in deteriorated housing to provide an estimate number.

The second approach made use of projected prevalences (to 1984) of PbB levels based on NHANES II survey data for those socioeconomic/demographic strata where paint is likely to be the major, if not entire, source of these PbB levels, i.e., those encompassing low-income, innercity, major metropolitan black children. These particular PbB elevation rates were part of a comprehensive prevalence modeling analysis given in a separate part of the Congressional report.

Although the 1978 Chicago study was confined to one metropolitan area, its large sampling size and focus on high-risk leaded-paint housing units increased its validity for broader estimation methods. As analyzed by the EPA (1985), these data allowed calculation of a PbB prevalence >30 μg/dl of 12.8% in 1978.

Projected prevalences of PbBs at criterion values of >15, >20, and >25 μg/dl and for those strata of children most exposed to leaded paint as indicated above were used. The rates for the corresponding criterion levels were 67.8, 30.8, and 10.6%. These were then combined with total numbers of children enumerated as indicated above.

b. Estimates of the numbers of children exposed to leaded gasoline. Examination of the total potential of direct (inhalation) and indirect (fallout) childhood exposure to lead in gasoline entails an approach focused on large urban/suburban areas of the United States, i.e., areas where airborne levels of lead from leaded gasoline combustion had been high enough to add a potentially significant fraction to total child lead body burden.

To determine potential exposure of children <7 years old, through a combination of inhalation and fallout contact, the number of individuals in the 100 largest U.S. cities were determined from U.S. Census Bureau 1984 estimates for total population and using 11% as that portion of the total U.S. population which is <7 years old.

Estimates of numbers of children having measurable elevations in their PbB values due to current/past use of leaded gasoline were not readily calculable or available, as such. As an alternative approach, we used estimates as generated by the U.S. EPA's Office of Policy Analysis (U.S. EPA, 1985) for those numbers of children whose PbB levels would decline below selected criterion values owing to leaded gasoline phase-down action in 1986 and projected to 1992.

The EPA employed logistic regression and other statistical analyses based on the NHANES II PbB database, which was gathered from 1976 to 1980. These regression analyses were applied to both black and white children using the rather broad age band of risk, children of 6 months–13 years old. Assuming a persisting

log-normal distribution of PbB levels with decreasing use of leaded gasoline, the EPA generated estimates of the mean and variance of transformed (normal) distribution for obtaining percentages of PbB values above specific ceilings, using the SAS/SURREGR computer program. Numbers of children falling below PbB values of 15, 20, and 25 μg/dl for each year, 1985 through 1990, were obtained. A more detailed statistical discussion appears in the original EPA document (U.S. EPA, 1985) and in Appendix G of the Congressional report.

c. Estimates of numbers of children exposed to lead from stationary sources. We employed two data sets dealing with modeling estimates of young children potentially exposed to lead emitted from U.S. stationary sources. One analysis, done for EPA's Office of Air Quality Planning and Standards (OAQPS) (GCA Corp., 1985), divided stationary sources into three categories: primary smelters, secondary smelters, and lead–acid battery plants. Total population estimates within each of these categories were then generated by use of different residential radii around the operations and reflecting lead dispersal patterns. For estimation of the fraction of children <7 years old in this population, a figure of 10.4% was used.

A second approach was that of the Lead Industries Association, Inc. (LIA) (TRC Environmental Consultants, Inc., 1986) which differs from the first-mentioned analysis with regard to numbers of units operating, quadrants examined, and radii around the operations. The LIA method did not appear to have taken into account lingering secondary child exposure from air lead fallout associated with now-closed facilities and based estimates on ambient airborne lead levels.

This approach would have within it an underestimation bias for aggregate exposure impact. The LIA study narrowed the radius of exposure population considerably, relative to the EPA/OAQPS modeling approach and considered lead movement only from dominant wind direction rather than including weighted adjustments for wind direction changes.

For estimates of actual stationary site lead exposures leading to elevated PbB values in children, use was made of reported rates of elevated PbB concentrations in surveys of primary and secondary smelter communities. PbB prevalence data for smelters in Montana (CDC, 1986a) and Idaho (CDC, 1986b) gave a range of 1–26% of smelter community children having both PbB levels ≥25 μg/dl and an erythrocyte protoporphyrin (EP) value ≥35 μg/dl whole blood. A recent examination of PbB levels in an area proximate to a secondary smelter in Dallas, Texas (City of Dallas, 1985) showed 4% of children with PbB values >20 μg/dl.

d. Estimates of numbers of children exposed to lead in dust and soil. The numbers of children exposed to lead in dust and soil cannot be separated from the numbers exposed to airborne lead or leaded paint. First, simultaneous exposures are occurring to all these media and second, paint and airborne lead are the principal contributors to dust and soil lead levels. One can use the latter relationships to estimate numbers of individuals with dust/soil exposures, i.e., a summing across the groups with exposures estimated as occurring from air and paint. Such a summation process, however, is likely to overestimate the numbers of subjects because of multiple exposures.

An alternative approach which avoids overcounting involves multimedia regression analysis. Regression analyses, however, require key empirical elements which do not exist currently for U.S. areas. These include regression equations (1) linking subject PbBs to area-specific dust/soil lead values at sites, (2) prevalences of lead-contaminated dusts and soils in different areas, and (3) estimation of numbers of children from (1) and (2). Such data, however, are not available in any systematic, comprehensive form.

Available data either omit leaded paint (e.g., Angle *et al.*, 1984; Charney *et al.*, 1980; Walter *et al.*, 1980) airborne lead (e.g., Galke *et al.*, 1975), or dust lead (e.g., Yankel *et al.*, 1977, provide incomplete data).

e. Estimates of numbers of children exposed to lead in drinking water. Three levels of drinking water exposure assessment were carried out in the Congressional report: potential water lead exposure of children, some actual exposure at a measurable but not necessarily toxic level, and actual exposure at relatively significant toxic risk levels.

Numbers of U.S. children potentially exposed to tap water lead were determined through two major data sets. The first, that of the Division of Housing Demographic Analysis, U.S. Department of Housing and Urban Development (HUD), was provided to ATSDR and the authors in a special report. The second involved combined estimates produced jointly by EPA's Office of Policy Analysis (U.S. EPA, 1986b) and HUD's Office of Policy Development and Research (provided as a special report to the authors).

In the first of these estimation processes, numbers of children <6 years old were enumerated for three time points—1973, 1978, and 1983—and percentages of the total numbers per time point were allocated to age-stratified housing stock having different types of interior/service plumbing. In the second method, numbers of children in two age strata, <5 years old and 5–13 years old, and residing in either new housing (having lead soldered copper plumbing joints) or houses built before 1939 (lead connectors and lead pipe in service lines) could be enumerated.

Estimation of numbers of U.S. children having tap water at levels expected to elevate PbB to some measurable amount employed EPA data (U.S. EPA, 1986b) showing that 42 million U.S. consumers of tap water from public systems will have lead levels above 20 μg/liter, a proposed EPA limit. From this total population, exposed children could be estimated using a proportion of 9% for subjects <6 years old as estimated from Census Bureau figures.

The U.S. EPA (U.S. EPA, 1986b) also carried out water lead-specific regression analyses to project the numbers of children estimated to exceed a PbB criterion value of >15 μg/dl owing to lead-contaminated tap water. Numbers of children having PbBs in the ranges 16 to 30, 31 to 50, and >50 μg/dl were also estimated.

In addition to tap water lead exposure in the home, other sources of tap water lead exist in public facilities used by children, especially kindergartens, day care centers, and elementary schools. Reliable estimates of the numbers of preschool and elementary school children exposed in these nonhome settings, while expected to be sizable, cannot be obtained on a national or even regional basis.

f. estimates of numbers of children exposed to lead in food. The number of persons potentially exposed to at least some level of lead in the U.S. food supply includes essentially the entire U.S. population because a centralized food production and distribution system serves all sections of the nation. For the numbers of children <6 years of age having some food lead intake/uptake, we calculated that the fraction of the 1985 estimated U.S. population in the indicated childhood age band was 9%.

To estimate the numbers of children having sufficient food lead exposure to raise PbB levels to those having high toxicity risk, the following assessment sequence was used:

(1) It could be calculated that a food lead contribution to total PbB should not exceed 10 μg/dl because of other source inputs to a total body burden expressed as a PbB value of 15–25 μg/dl.

(2) Use was made of an empirical relationship linking dietary lead to PbB, that of Ryu *et al.* (1983), in which PbB (diet) = 0.16 × daily dietary Pb.

(3) Combining of (1) and (2) was done to calculate a maximum diet intake/day of ca. 63 μg Pb.

(4) To estimate the total numbers of children exposed to dietary lead at levels at or above 63 μg/day, that percentile of children's dietary lead distributions corresponding to the limit figure of 63 μg was first determined. This required an appropriate adjustment for known declines in food lead content from the time of the reference food survey. Second, this percentile was multiplied by the number of total children in the United States in the indicated age band.

2. *Results and Discussion*

a. Estimates of numbers of children exposed to paint lead. According to the calculations of Pope (1986) and data from the 1983 American housing survey (U.S. Census Bureau, 1986) the percentages of age-stratified housing having paint lead >0.7 mg/cm^2 are pre-1940, 99%; 1940–1959, 70%, and 1959–1974, 20%. Combining these percentages with total age-stratified housing units gives corresponding lead-painted housing counts which are presented as best estimates and upper bounds in Table 1. Also presented in Table 1 are numbers of children allocated to the age-stratified housing using the factor of 0.287. As seen in Table 1, national best estimates indicate that there are approximately 42 million dwelling units in the U.S. housing stock which have leaded paint at levels exceeding the CDC threshold exposure value of 0.7 mg Pb/cm^2 (CDC, 1985). This figure is ca. 52% of all U.S. housing stock (U.S. Census Bureau, 1986).

Table 1 shows that about 12 million children <7 years old live in these age-stratified dwelling units. Note that half of this best estimate, ca. 6 million children, is attributable to the oldest housing with highest lead paint.

In considering the numbers of children who live in deteriorated houses that contain leaded paint, Pope (1986) classified the housing according to Census Bureau designations for unsound housing within the three house age groups. Table 2 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

TABLE 1
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 childen (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).

age and condition, along with the totals. Cumulatively, Table 2 shows the best national estimate and the national upper bound estimate of children under 7 years old living in unsound lead-painted housing to be 1,772,000 and 1,996,000, respectively.

From data of Pope (1986) one can provide estimated numbers of young children (<7 years old) living in deteriorated housing by the four major regions. By region, numbers of children living in housing which specifically have peeling leaded paint are as follows: 174,000, Northeast; 139,000, Midwest; 130,000, South; 77,000, West. As expected, the older developed areas, specifically urban areas in the Northeast and Midwest, have the highest and next highest figures. The West has

TABLE 2
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	1,772,000
		(6,965,000)[d]	(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.

the lowest figure, reflecting its status as the most recently developed area of the country.

In considering estimates of those children exposed to lead in paint who have elevated PbB levels because of this exposure, we first combined the numbers in Table 2 for children in unsound, lead-based painted housing with the 12.8% of children with >30 μg/dl PbB calculated by the EPA for all children residing in like units and who represent a large urban area. This approach gives an estimate of about 230,000 children. However, the sole available prevalence from the Chicago analyses is for a criterion PbB value, i.e., >30 μg/dl, which is now considered well above those concentrations currently associated with the onset of early effects in young children.

Next, the results of using prevalences of the selected PbB criterion values, 15, 20, and 25 μg/dl (using NHANES II projected prevalences), for strata that reflect children living in 100% deteriorated, high lead-based painted housing combined with base numbers of such children are tabulated in Table 3. The numbers in Table 3 are reasonable but may still be underestimates (see below).

The number of children in such housing having PbBs above 15 μg/dl is approximately 1.2 million, while the corresponding rounded-off figures for PbB limits of >20 and >25 μg/dl are 545,000 and 188,000, respectively.

Numbers in Table 3 do not include any estimate of exposed children in old housing with high paint-lead levels but without deterioration. The totals in this case may be substantial. Other information in the Congressional report, dealing with age-stratified housing of children in Standard Metropolitan Statistical Areas (SMSAs), documents that a significant fraction of the oldest urban housing is not deteriorated. Consequently, the base populations used to obtain the figures in Table 3 should be viewed as probable lower bounds to the true count. Similarly, the stratum of the NHANES II survey selected as appropriate for assignment of these children may represent PbB prevalences which represent some mix of housing quality. The true projected PbB prevalences for present-day children in 100%

TABLE 3
ESTIMATED NUMBERS OF U.S. CHILDREN LIVING IN UNSOUND, LEAD-BASED PAINTED HOUSING ABOVE INDICATED PbB CRITERION VALUES[a,b]

Category	Housing age	Total children	Children with PbB (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	458,000	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 2.
[b] Selection of NHANES II stratum for use of specific prevalences is discussed in text. Prevalences are from Table V-1 (Report to Congress).

deteriorated, high lead-paint housing may therefore be somewhat higher. This factor would also contribute to underestimates.

On the other hand, the estimates in Table 3 may overlap. Units with peeling paint may also have been counted as having broken plaster, etc., in a number of instances.

b. Numbers of children exposed to lead in leaded gasoline. The estimated number of children <7 years old in the Nation's 100 largest cities in 1984 was 5.57 million, based on a total 1984 population estimate of 50.6 million for these cities by the U.S. Census Bureau and a proportion of about 11% for children in this age band.

The estimates of children 6 months–13 years old who will have PbB levels falling below selected criterion PbB values in the years 1985–1990, as reported by the EPA (1985) and due to phase-down of lead in gasoline, are depicted in Table 4. Fewer children are shown at higher PbB criterion values since the original numbers above the higher levels were smaller. Since the EPA used age-dependent PbB distributions in children in these analyses, it was not feasible to subdivide these estimates into narrow age bands that better accord with other source-specific estimates in this report.

As seen in Table 4, the estimated (rounded-off) number of children whose PbBs will decline below 15 μg/dl in 1990 is 1.25 million. Corresponding rounded numbers for 1988 and 1989 are 1.48 and 1.35 children, respectively.

c. Estimates of numbers of children exposed to lead from stationary sources. Table 5 presents the interim estimate data from EPA's OAQPS (GCA Corp., 1985) for the national total and the number of children (<7 years old) who are exposed to stationary emission sources for lead. Of the 230,000 children potentially exposed to lead by living near the three major categories of stationary sites, the bulk (80%) are potentially impacted by secondary lead smelters.

The corresponding estimate of exposed children in the LIA analyses (TRC Environmental Consultants, Inc., 1986) is 51,045, when the same radial distances for residence around stationary emission sources as used by EPA's modeling approach are factored into the analyses.

If one applies the range of prevalences for a PbB value ≥25 μg/dl for primary smelters, i.e., 1–26% (see above), to the estimate for the base child population around primary smelters given in Table 5, i.e., 21,000 subjects, then between 210

TABLE 4

ESTIMATED NUMBERS OF U.S. CHILDREN (THOUSANDS) FALLING BELOW INDICATED PbB (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	1726	1597	1476	1353	1252

[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.

TABLE 5
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 reexamination.

and 5500 children will have these blood lead concentrations. Similarly, combining the number of children impacted by secondary smelters in Table 5, 187,000, with the corresponding reported PbB prevalence for a PbB >20 μg/dl, 4%, gives an estimate of 7500 children having these elevated PbB concentrations.

d. Estimates of numbers of children exposed to dust and soil lead. The numbers of children potentially exposed to lead in dust and soil, i.e., without reference to actual PbB elevation rates, are taken as the sum of totals potentially exposed to the primary contributors to dust and soil:

5.9 Million children <7 years old due to highest lead in paint levels
5.6 Million children <7 years old due to leaded gasoline combustion and lead emissions in 100 largest U.S. cities
0.2 Million children <7 years old due to stationary site emissions: primary and secondary smelters, battery plants

This yields a total of 11.7 million children potentially exposed to lead in dust and soil, with a likelihood that some overestimating is present, since simultaneous exposures to paint lead, lead from combusted gasoline, and stationary lead emissions could occur and produce an upper bound. On the other hand, the lower bound to this estimate (below which potential exposure to lead in dust and soil would be unlikely) could be taken as the largest of the three primary contributor estimates, the 5.9 million children exposed to high lead paint. Overall, the potential impact would be in the range of 5.9 to 11.7 children <7 years old.

e. Estimates of numbers of children exposed to lead in drinking water. Tables 6 and 7 present differing analyses of the quantitative relationship of plumbing type/housing age to numbers of children allocated to these plumbing type/age-stratified housing groups. These tables address potential exposure of young children to drinking water. Table 6 provides information combining data from the 1973–1983 American Housing Surveys (see, e.g., U.S. Census Bureau, 1986) and housing age-stratified percentages of young children in housing in 1973, 1978, and 1983. The latter figures were kindly provided to the authors as a special report from HUD's Division of Housing Demographic Analysis.

Table 7 more precisely defines potential exposure using a complex array of data provided by EPA (U.S. EPA, 1986b), Tables of Statistical Abstracts, 1985 (U.S.

TABLE 6
CHILDREN POTENTIALLY AT RISK FOR LEAD EXPOSURE BY HOUSEHOLD PLUMBING, BY AGE[a,b]

Age of housing	Number of children < 6 years			Exposure profile
	1973	1978	1983	
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.

Census Bureau), and information furnished by HUD's Office of Policy Development and Research to the authors and to the EPA. This table focuses on those housing statistics reflecting oldest, high-lead plumbing and newest high-lead plumbing. In the former case, this referred to lead connectors and piping and in

TABLE 7
ESTIMATED NUMBERS OF CHILDREN AT GREATEST RISK OF EXPOSURE TO LEAD IN HOUSEHOLD PLUMBING

		Population at risk
New housing		
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.

the latter, new lead-soldered joints in copper plumbing having a high propensity for lead leaching.

As seen in Table 6, data for 1983 indicate that 13% of the 21 million U.S. children <6 years old, or 2.73 million, have at least potential exposure to tap water lead owing to lead connectors/lead piping in pre-1920 residences. Similarly, a total of 63% of these children, or 13.23 million, reside in dwellings with lead-soldered plumbing. Of these, 0.84 million are in the newest homes, homes <2 years old.

Table 7 shows that a total of 1.8 million children up to 13 years of age are in new housing with the highest risk of lead—soldered joint leaching of lead—and 0.7 million of these children are <5 years old. In the oldest housing apt to have lead-based connectors and service-line piping, 4.8 million individuals are similarly affected and, of these, the younger group number 1.8 million.

In the aggregate, 2.5 million children <5 years old have the highest potential risk from lead plumbing in U.S. housing.

The U.S. EPA (1986b) has calculated that 42 million U.S. residents on public drinking water systems had tap water levels above 20 μg/liter. Of this number, one can estimate that 3.78 million are children <6 years of age. Consumption of 1–2 liters of tap water >20 μg Pb/liter by these children gives a daily total of >20–40 μg Pb/day. Using the Ryu *et al.* (1983) multiplier of 0.16 (see methods section), PbB will rise >3.2–6.4 μg/dl.

With respect to water-associated elevations in PbB to levels >15 μg/dl, i.e., above early effects onset, the EPA (1986b) used regression analysis techniques (see methods section) to calculate that 241,100 children <6 years old had PbB concentrations >15 μg/dl. Of this tally, 230,000 had PbBs between 15 and 30 μg/dl and 11,000 had levels between 30 and 50 μg/dl. The remainder, 100 children, are estimated to have PbBs >50 μg/dl due to tap water lead.

As noted in the methods section, it is not possible at this time to provide national estimates for children exposed to tap water lead in schools, kindergartens, etc. However, a number of community and state-level efforts are now underway to explore the scope of the problem. In public buildings, tap water lead exposure occurs from lead-containing connectors, service pipes, and lead-soldered building core plumbing as well as lead fittings and components in water fountains/coolers and, in some cases, brass fixtures with relatively high lead content.

Illustrative of the problems of lead exposure that can be associated with water emerging from fountains/coolers are data tabulated in the Congressional report showing results provided to the authors and the U.S. EPA for tap water analyses at U.S. Naval facilities in Maryland (Gardels, 1989; U.S. Navy, 1987). In addition, school tap water supplies are being systematically examined throughout the United States, largely in response to provisions of new Federal legislation, the *Lead Contamination Control Act of 1988* (P.L. 100-242). Also included in the Congressional document are summary data from school drinking water surveys in several states.

f. Estimates of numbers of children exposed to lead in food. The number of children <6 years old for the most current period having some potential food lead

exposure was estimated, using Census Bureau estimates for 1985 at the lowest fertility rate (World Almanac, 1987), to be 21.41 million (rounding off). If one adjusts this estimate to exclude the youngest infant group, 0–5 months, owing to the nature of the available dietary data, the figure is 19.47 million. These are the numbers having some finite dietary lead exposure.

Of this estimated number, we have calculated that a maximum 5% or 973,000 children <6 years old would have PbB increases approaching 10 μg/dl owing to lead intake/uptake from the diet. This required using dietary lead distribution data of Beloian (1982) for the period 1973–1978 and a downward adjustment of 50% of these lead levels at the various percentiles to account for known lead declines in dietary components ingested by children. Since the fraction of annual domestic can production which is lead free continues to decline and atmospheric fallout from air lead is declining further, the present exposure number is also lower to some extent.

C. LOW-LEVEL LEAD SOURCES AND AGGREGATE EXPOSURE/EFFECT RISK

This report emphasized analyses of traditional sources, where lead levels were expected to be quite high, i.e., high enough to produce PbBs which would have produced relatively high risk of lead intoxication, acutely, subchronically, or chronically.

However, since lead is a multimedia pollutant and toxicant, it can provide a collectively significant toxicity risk even when source-specific lead inputs are, by themselves, quite modest. There are two reasons for this.

First, lead from all sources is systemically integrated to provide a single, toxicologically active body and target tissue burden as reflected in indicators of such risk, e.g., PbBs or chelatable lead (Mushak, 1989; WHO, 1987; Chap. 10, U.S. EPA, 1986a). Second, increasingly lower levels of lead exposure are being recognized as posing toxicity risks to young children and the fetus that are of concern and this translates to risk from low-level as well as traditionally high lead sources.

To illustrate, consider that a PbB level of 10–15 μg/dl in preschool children is to be avoided in order to minimize subtle adverse effect risk (Needleman and Bellinger, 1989; Davis and Svendsgaard, 1987; Mushak *et al.*, in press). If lead exposure in this child population is occurring through, say, four common but low-level lead sources, each contributing 4 μg/dl to a measured Pb-B of 16 μg/dl (which is above the indicated threshold), how is source-specific exposure to be ranked for significance and how can one apportion, i.e., disaggregate, the individual contributions?

Several elements of low-level lead exposure especially require consideration by regulatory agencies and other risk managers. Reference has already been made to quantitating contributions from a given low-lead medium to total PbB. There is also the question of feasible quantitation (at a national level) of numbers of children and other risk groups exposed to sources of low lead exposures and the relative ease of quantitating changes in these exposure estimates.

The U.S. EPA's OAQPS has approached this problem in several ways (U.S. EPA, 1989a, b). The most encompassing strategy entails development and refine-

ment of a risk population, age-specific uptake/biokinetic model. In this comprehensive modeling approach, based on the early model of Kneip *et al.* (1983) with further refinements to provide an integrated metabolic model for lead in humans of all ages (Harley and Kneip, 1985), one can estimate geometric mean PbB levels in children in a given community provided that key environmental lead inputs to systemic exposure are accurately known. With tandem use of geometric means and a geometric standard deviation (GSD) appropriate to the population, PbB distributions can be also estimated for site-specific numbers of subjects at greatest exposure risk.

These approaches are, however, site-specific in their estimating ability and are not readily applicable to the purposes of this report.

D. OVERVIEW

In this paper, and in the Congressional report on which it is based, national estimates and enumerations of U.S. children exposed to lead in diverse sources have been provided.

Enumerations primarily were drawn from U.S. Census Bureau figures for population segment and housing counts of various statistical type. Estimates, commonly using enumerated data in their derivation, included such analyses as projected and extended PbB prevalence rates for actual exposure to toxic ranges or use of calculated elevations in lead-contaminated media to calculate PbB changes.

A number of these sources involve large numbers of affected children, both with regard to potential exposed population and estimates for exposures causing actual elevations in body lead burden. We have estimated, using Census Bureau enumerations combined with reasonably derived prevalence projections for high-risk PbB levels, that 1.2 million young children have sufficient paint lead-based exposure to raise their PbB levels above 15 μg/dl. While there is some overcounting in this subset, there are a number of other factors which indicate this is an underestimate by a considerable amount (see above).

Enough lead exposure occurs through lead in tap water that 3.8 million young children are estimated to have sufficient intake/uptake of water lead to predictably increase PbBs. By separate estimate, ca. 240,000 children have elevations in PbB to toxic ranges due to tap water lead.

As seen in Table 4, sizable numbers of children had, and are projected to continue to have into the 1990s, enough leaded gasoline exposure via direct and indirect (fallout) routes to cause declines in PbBs below indicated cutoffs.

Lead in dust and soil is known to cause elevated PbBs in preschool children who engage in such normal exploratory behavior as mouthing activity, based on a large number of epidemiological studies (see, e.g., Chap. 11, U.S. EPA, 1986a; CDC, 1985; Duggan and Inskip, 1985; Brunekreef, 1984). We therefore would assume that any national estimate of that which is well documented in many site-specific studies would produce large numbers of actual exposures. We cannot, however, easily sort out such numbers from simultaneous exposures via other sources.

While the broad range in methods of analysis, the differing degrees of precision of estimation across sources, and the often indefinable bounds to the accuracy of

the original data sets analyzed would complicate any rigid ranking of sources as to importance, there are clear qualitative differences in U.S. lead exposures which are source-specific.

On the basis of the results of these analyses, the principal sources of lead exposure for U.S. children are leaded paint, lead in dust and soil, and lead in drinking water. These are principally residence-based exposures. There are also recognized but presently unquantified further exposures to lead in these sources at public sites, e.g., kindergartens, elementary and higher schools, and playground/common areas.

Two traditionally significant sources of lead exposure in children, lead in food and lead in gasoline, are judged to be declining in significance compared to past years. Even so, these continue to have residual impacts on childhood lead exposure.

Phase-down action by the EPA (50 FR 30791, July 29, 1985), in place since the beginning of 1986, has reduced further inputs of lead into air from leaded gasoline combustion. However, the contamination legacy of 60 years of leaded gasoline contribution to soils, dusts, aquatic sediments, etc., persists. Highly dispersed dusts and lead in soils are particularly refractory sources of exposure for preschool children. This lingering contribution from leaded gasoline is also a factor in the large numbers of children continuing to register PbB declines after initiation of phase-down action, as shown in Table 4.

Food lead levels continue to decline, from phasing out domestic production of lead-seamed food containers and reduced input of lead to food crops due to lower atmospheric fallout rates (see, e.g., Chap. 7, U.S. EPA, 1986a). A lingering problem is in the form of lead-seamed containers for imported foods. The latter source is of particular concern for the diets of ethnic, urbanized low-income groups where imported canned food use might be relatively high at the same time that there is elevated exposure from the other urban sources.

ACKNOWLEDGMENTS

The authors thank Dr. Barry L. Johnson, Associate Administrator, ATSDR, and Agency coordinator for the report to Congress, for his support and encouragement. Many thanks also go to Drs. Lester D. Grant and J. Michael Davis of the U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office, Research Triangle Park, North Carolina, both for their scientific comments and logistical support in the preparation of the Congressional document. Furthermore, special thanks are due to Vandy Bradow, EPA/ECAO, for overseeing the physical preparation and editing of the document and much appreciation is extended to EPA support staff, especially Miriam Gattis and Lorrie Godley. Finally, we thank the many peer reviewers of the Congressional report.

REFERENCES

Angle, C. R., Marcus, A., Cheng, I.-H., and McIntire, M. S. (1984). Omaha childhood blood lead and environmental lead: A linear total exposure model. *Environ. Res.* 35, 160–170.

Annest, J. L., and Mahaffey, K. R. (1984). "Blood Lead Levels for Persons Ages 6 Months–74 Years, United States, 1976–1980. U.S. Department of Health and Human Services." DHHS Publication No. 84-1683. National Health and Nutrition Examination Survey Series II, No. 233.

Beloian, A. (1982). Use of a food consumption model to estimate human contaminant intake. *Environ. Monit. Assess.* 2, 115–127.

Brunekreef, B. D. (1984). The relationship between air lead and blood lead in children: A critical review. *Sci. Total Environ.* 38, 79–123.

Centers for Disease Control (1985). "Preventing Lead Poisoning in Young Children: A Statement by the Centers for Disease Control, January." Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA, No. 99-2230.

Centers for Disease Control (1986a). "East Helena, Montana, Child Lead Study, Summer, 1983: Final Report of Center for Environmental Health/Centers for Disease Control." Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA, July.

Centers for Disease Control (1986b). "Final report: Kellogg Revisited—1983. Childhood Blood Lead and Environmental Status Report." Panhandle District Health Department, Idaho Department of Health and Welfare, Center for Environmental Health/Center for Disease Control, Public Health Service, United States Environmental Protection Agency, July.

Charney, E., Sayre, J., and Coulter, M. (1980). Increased lead absorption in inner city children: Where does the lead come from? *Pediatrics* 65, 226–231.

City of Dallas Department of Health (1985)." "Blood Lead Screening Program: East Oak Cliff and West Dallas. Final Report: Fall."

Commission of the European Communities/U.S. Environmental Protection Agency (1989)." Lead Exposure and Child Development: An International Assessment" (M. A. Smith, L. D. Grant, and I. Sors, Eds.). Kluwer, Dordrecht, The Netherlands, pp. 515.

Davis, J. M., and Svendsgaard, D. J. (1987). Low-level lead exposure and child development. *Nature (London)* 329, 297–300.

Duggan, M. J., and Inskip, M. J. (1985). Childhood exposure to lead in surface dust and soil: A community health problem. *Public Health Rev.* 13, 1–54.

Galke, W. A., Hammer, D. I., Keil, J. E., and Lawrence, S. W. (1975). Environmental determinants of lead burdens in children. *In* "International Conference on Heavy Metals in the Environment" (T. C. Hutchinson, S. Lestein, A. L. Page, F. Van Loon, and T. Davey, Eds.), Vol. 3, October, pp. 53–74. Toronto, Ontario, Canada.

Gardels, M. C. (1989). Laboratory study of lead leaching from drinking water coolers. *J. Am. Water Works Assoc.* 81, 73 (Abstract 28–3)

GCA Corp. (1985). "Estimated Numbers of Children Residing Near Lead Point Sources." Office of Air Quality Planning and Standards, Ambient Standards Branch, Research Triangle Park, NC. Contract No. 68-02-3804.

Harley, N. H., and Kneip, T. H. (1985). "An Integrated Metabolic Model for Lead in Humans of All Ages: Final Report." EPA Contract No. B 44899, New York University, New York.

Kneip, T. J., Mallon, R. P., and Harley, N. H. (1983). Biokinetic modeling for mammalian lead metabolism. *Neurotoxicology* 4, 189–192.

Mahaffey, K. R. (1988). Factors affecting biological responses to chemicals. *In* "Mechanisms of Cell Injury: Implications for Human Health" (B. A. Fowler, Ed.), pp. 315–331. Wiley, New York.

Mushak, P. (1989). Biological monitoring of lead exposure in children: Overview of selected biokinetic and toxicological issues. *In* "Lead Exposure and Child Development: An International Assessment" (M. A. Smith, L. D. Grant, and A. I. Sors, Eds.), pp. 129–145. Commission of the European Communities and the United States, Kluwer, Dordrecht, The Netherlands.

Mushak, P., Davis, J. M., Crocetti, A. F., and Grant, L. D. Prenatal and postnatal effects of low-level lead exposure: Integrated summary of a report to the U.S. Congress on childhood lead poisoning. *Environ. Res.*, 50, 11–36.

Pope, A. (1986). "Exposure of Children to Lead-Based Paints." EPA Contract No. 68-02-4329, Strategies and Air Standards Division, U.S. Environmental Protection Agency, Research Triangle Park, NC.

Rabinowitz, M., Leviton, A., and Bellinger, D. C. (1985). Home refinishing: Lead paint and infant blood lead levels. *Amer. J. Public Health* 75, 403–404.

Ryu, J. E., Ziegler, E. E., Nelson, S. E., and Fomen, S. J. (1983). Dietary intake of lead and blood lead concentration in early infancy. *Amer. J. Dis. Child.* 137, 886–891.

TRC Environmental Consultants, Inc. (1986). "Exposure to Airborne Lead from Stationary Sources: An Evaluation of Proposed National Ambient Air Quality Standards for Lead." Project No. 3220-551, TRC Environmental Consultants, Inc., Wethersfield, CT.

U.S. Bureau of the Census (1986). "American Housing Survey, 1983. Part B: Indicators of Housing and Neighborhood Quality by Financial Characteristics." U.S. Department of Commerce, Washington, DC.

U.S. Environmental Protection Agency (1985). "Costs and Benefits of Reducing Lead in Gasoline: Final Regulatory Impact Analysis." EPA Report No. EPA-230/05-85-006. Office of Policy, Planning and Evaluation (J. Schwartz, H. Pitcher, R. Levin, B. Ostro, and A. L. Nichols, Eds.). Washington, DC.

U.S. Environmental Protection Agency (1986a). "Air Quality Criteria for Lead." EPA Report No. EPA-600/8-83/028aF-dF. 4 Vol. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. Available from: NTIS, Springfield, VA; PB87-142378.

U.S. Environmental Protection Agency (1986b). "Reducing Lead in Drinking Water: A Benefit Analysis." EPA Report No. EPA-230-09-86-019. Office of Policy Planning and Evaluation. Washington, DC.

U.S. Environmental Protection Agency (1989a). "Review of the National Ambient Air Quality Standards for Lead: Assessment of Scientific and Technical Information." OAQPS Draft Staff Paper, March, 1989, Office of Air quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC.

U.S. Environmental Protection Agency (1989b). "Review of the National Ambient Air Quality Standards for Lead: Exposure Methodology and Validation." OAQPS Draft Staff Report, February, 1989, Office of Air Quality Planning and Standards, Ambient Standards Branch, Research Triangle Park, NC.

U.S. Navy (1987). "Data on Results of Analyses of Potable Tap Water Samples." Report on the Safety Office, David Taylor Naval Ship Research and Development Center, Annapolis, MD.

Walter, S. D., Yanke, A. J., and von Lindern, I. H. (1980). Age-specific risk factors for lead absorption in children. *Arch. Environ. Health* 35, 53–58.

World Almanac and Book of Facts (1987). "United States Population," pp. 217–247, Pharos/Scripps Howard; New York.

World Health Organization (1987). "Air Quality Guidelines for Europe. European Series No. 23, Lead," Chap. 23, pp. 242–261. Regional Office for Europe, Copenhagen.

Yankel, A. J., von Lindern, I. H., and Walter, S. D. (1977). The Silver Valley lead study: The relationship between childhood blood lead levels and environmental exposure. *J. Air Pollut. Control Assoc.* 27, 763–767.