

5. POTENTIAL FOR HUMAN EXPOSURE

5.1 OVERVIEW

Lead is dispersed throughout the environment primarily as the result of anthropogenic activities. Environmental fate processes may transform one lead compound to another; however, lead is not degraded and is still available for human exposure, even though the compounds containing it vary enormously.

The general population is exposed to lead in ambient air, in many foods, in drinking water, in soil, and in dust. Segments of the general population at highest risk of health effects from lead exposure are preschool-age children and pregnant women and their fetuses. Within these groups, relationships have been established between lead exposure and adverse health effects. Other segments of the general population at high risk include white males between 40 and 59 years of age and individuals living near sites where lead was produced or disposed.

Human exposure to lead above baseline levels is common. Baseline refers to the naturally-occurring level of lead in soil or dust that is not due to the influence of humans. Some of the more important lead exposures occur as a result of living in urban environments, particularly in areas near stationary emission sources (e.g., smelters); consumption of produce from family gardens; renovation of homes containing lead-based paint; pica (an abnormal eating habit in children); contact with interior lead paint dust; occupational exposure; secondary occupational exposure (e.g., families of workers using lead); smoking; and wine consumption. Higher than normal exposures may also occur to residents living in close proximity to National Priorities List (NPL) sites that contain elevated levels of lead. The highest and most prolonged lead exposures are found among workers in the lead smelting, refining, and manufacturing industries.

The primary source of lead in the environment has historically been anthropogenic emissions to the atmosphere. In 1984, combustion of leaded gasoline was responsible for approximately 90% of all anthropogenic lead emissions. EPA phased out the use of lead alkyls in gasoline, however, and by 1990, auto emissions accounted for only 33% of the annual lead emissions (EPA 1996h). Use of lead additives in motor fuels was totally banned after December 31, 1995 (EPA 1996f). The ban went into effect on February 2, 1996. Atmospheric deposition is the largest source of lead found in soils. Lead is transferred continuously between air, water, and soil by natural chemical and physical processes such as weathering, runoff, precipitation, dry deposition of dust, and stream/river flow; however, soil and sediments appear to be

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important sinks for lead. Lead particles are removed from the atmosphere primarily by wet and dry deposition. The average residence time in the atmosphere is 10 days. Over this time, long-distance transport, up to thousands of kilometers, may take place. Lead is extremely persistent in both water and soil. The speciation of lead in these media varies widely depending upon such factors as temperature, pH, and the presence of humic materials. Lead is largely associated with suspended solids and sediments in aquatic systems, and it occurs in relatively immobile forms in soil.

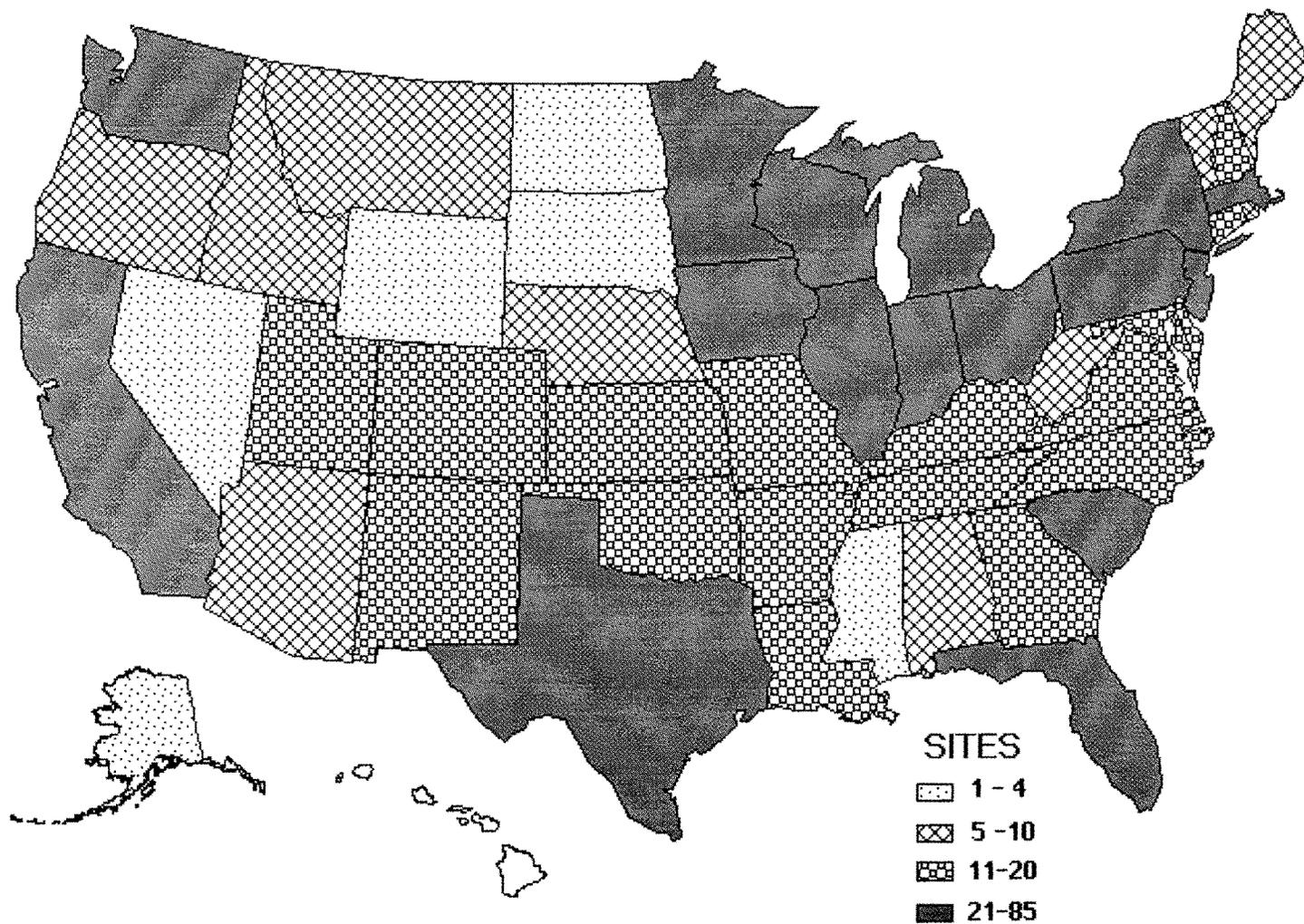
Lead has been identified in at least 1,026 of the 1,467 current or former EPA National Priorities List (NPL) hazardous wastes sites (HazDat 1998). However, the number of sites evaluated for lead is not known. The frequency of these sites within the United States can be seen in Figure 5-1. Of these sites, 1,017 are located in the United States, 1 is located in Guam (not shown), 1 is located in the Virgin Islands (not shown), and 7 are located in the Commonwealth of Puerto Rico (not shown).

5.2 RELEASES TO THE ENVIRONMENT

Lead is a naturally occurring element that has been found in the earth's crust, mostly as the sulfide galena, and in all compartments of the biosphere in various chemical forms. Although both natural and anthropogenic processes are responsible for the distribution of lead throughout the environment, anthropogenic releases of lead are predominant. Lead is regulated by several federal statutes and is a priority water pollutant and a hazardous air pollutant (see Chapter 7). Although combustion of leaded gasoline was once the primary source of anthropogenic atmospheric releases of lead, industrial releases to soil from nonferrous smelters, battery plants, chemical plants, and disturbance of older structures containing lead-based paints are now major contributors to total lead releases.

According to the Toxics Release Inventory, in 1996, a total of 16,938,957 pounds (7,683,382 kg) of lead was released to the environment from 1,494 large processing facilities (TRI96 1998). Table 5-1 lists amounts released from these facilities. In addition, an estimated 47,886 pounds (21,721 kg) were released by manufacturing and processing facilities to publicly owned treatment works (POTWs), and an estimated 350,783,734 pounds (159,112,825 kg) were transferred offsite (TRI96 1998). The TRI data should be used with caution because only certain types of facilities are required to report. This is not an exhaustive list.

Figure 5-1. Frequency of NPL Sites with Lead Contamination



Derived from HazDat 1998

Table 5-1. Releases to the Environment from Facilities That Manufacture or Process Lead

STATE ^b	NUMBER OF FACILITIES	Total of reported amounts released in pounds per year ^a							TOTAL ENVIRONMENT ^d
		AIR ^c	WATER	LAND	UNDERGROUND INJECTION	POTW TRANSFER	OFF-SITE WASTE TRANSFER		
AL	36	65,811	2,946	162,682	0	290	1,396,876	1,628,605	
AR	28	20,992	151	31,608	0	339	3,164,254	3,217,344	
AZ	15	50,091	0	3,161,245	0	103	7,483,002	10,694,441	
CA	77	12,975	1,449	19,534	0	506	22,124,851	22,159,315	
CO	8	3,307	1	21,400	0	262	956,475	981,445	
CT	28	1,272	126	2,961	0	21	602,596	606,976	
DE	4	3,176	3	0	0	859	5,180,089	5,184,127	
FL	10	7,170	29	0	0	259	7,135,984	7,143,442	
GA	38	24,366	933	78,755	0	230	16,693,844	16,798,128	
IA	14	14,725	463	75,505	0	307	11,441,116	11,532,116	
ID	3	140	0	0	0	73	325,555	325,768	
IL	99	215,426	2,165	825,038	0	3,494	13,220,136	14,266,259	
IN	92	42,695	6,331	252,632	0	3,038	43,425,409	43,730,105	
KS	25	9,242	129	334	26	210	36,021,657	36,031,598	
KY	35	18,572	486	34,010	0	1,581	8,519,181	8,573,830	
LA	14	12,509	858	141,507	0	354	12,306,959	12,462,187	
MA	34	26,288	265	0	0	257	494,906	521,716	
MD	7	277	3,221	1,600	0	93	141,564	146,755	
ME	1	4	0	0	0	0	4,476	4,480	
MI	58	30,696	588	140,721	0	2,835	2,186,206	2,361,046	
MN	18	20,125	10	0	0	847	4,217,763	4,238,745	
MO	41	578,521	1,011	4,209,789	0	2,567	18,807,358	23,599,246	
MS	31	8,073	1,216	66,631	0	551	602,724	679,195	
MT	2	43,547	1,053	3,696,200	0	5	211,193	3,951,998	
NC	35	9,058	1,265	7,456	0	628	6,175,024	6,193,431	
ND	1	370	0	8	0	0	407	785	
NE	16	27,710	2,111	3,766	0	649	4,624,654	4,658,890	
NH	11	3,483	10	255	0	42	503,346	507,136	
NJ	45	18,895	2,707	103,342	0	2,605	13,231,577	13,359,126	
NM	3	12,973	0	137,044	0	120	15,300	165,437	
NV	3	1,025	0	0	0	1	433,931	434,957	
NY	57	29,860	1,085	3,902	0	1,651	4,543,097	4,579,595	
OH	151	83,375	5,632	338,116	0	9,881	20,121,578	20,558,582	

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Table 5-1. Releases to the Environment from Facilities That Manufacture or Process Lead (continued)

Total of reported amounts released in pounds per year ^a									
STATE ^b	NUMBER OF FACILITIES	AIR ^c	WATER	LAND	UNDERGROUND INJECTION	POTW TRANSFER	OFF-SITE WASTE TRANSFER	TOTAL ENVIRONMENT ^d	
OK	16	2,476	109	204	408	108	1,529,271	1,532,576	
OR	8	1,149	30	0	0	62	5,171,205	5,172,446	
PA	95	131,991	6,604	81,490	0	2,920	19,412,751	19,635,756	
PR	4	2,735	1	1	0	32	135,582	138,351	
RI	14	923	62	0	0	43	291,972	293,000	
SC	30	25,388	950	45,352	0	1,691	8,622,651	8,696,032	
SD	2	10	5	0	0	10	34,400	34,425	
TN	46	20,123	1,883	5,589	0	3,801	2,666,922	2,698,318	
TX	89	101,176	4,464	580,600	360	2,145	34,812,211	35,500,956	
UT	13	20,277	641	873,943	0	292	1,364,131	2,259,284	
VA	24	5,046	459	43,229	0	348	719,947	769,029	
VT	5	38	5	346	0	250	976,762	977,401	
WA	13	5,320	250	590	0	49	620,089	626,298	
WI	44	9,952	290	0	0	1,406	7,786,748	7,798,396	
WV	9	5,560	10,657	0	0	71	246,004	262,292	
WY	2	5	0	0	0	0	80,000	80,005	

Source: TRI96 1998

^a Data in TRI are maximum amounts released by each facility

^b Post office state abbreviations used

^c The sum of fugitive and stack releases are included in releases to air by a given facility

^d The sum of all releases of the chemical to air, land, and water, and underground injection wells; and transfers off-site by a given facility

POTW = publicly owned treatment works

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Lead has been identified in a variety of environmental media (air, surface water, groundwater, leachate, soil, sediment, fish and game animals) collected at 1,026 of the 1,467 current and former NPL hazardous waste sites (HazDat 1998). Lead is the most frequently found metal at hazardous waste sites (Reed et al. 1995).

5.2.1 Air

According to the Toxics Release Inventory, in 1994, the estimated releases of lead of 1,728,918 pounds (784,224 kg) to air from 1,454 large processing facilities accounted for about 10.2% of the total environmental releases of lead (TRI96 1998). Table 5-1 lists amounts released from these facilities. The TRI data should be used with caution, however, since only certain types of facilities are required to report. This is not an exhaustive list.

Lead has been identified in air samples collected at 65 of the 1,026 NPL hazardous waste sites where it was detected in some environmental medium (HazDat 1998).

Of particular importance are emissions of lead to the atmosphere, which is the initial recipient for much of the lead released to the environment. Estimated atmospheric emissions of lead from anthropogenic point and nonpoint sources in the United States during 1989 (6 years before the total ban on lead in gasoline) were estimated to be 6,304 short tons (EPA 1996h). Stationary sources of lead, although found throughout the nation, tend to be concentrated near smelters, nonferrous foundries, and industrial operations dealing with lead-containing products. Lead may also be released in aerosol form from waste incinerators (Biswas et al. 1992). Natural emissions of lead to the atmosphere from volcanoes and windblown dust are believed to be of minor importance (EPA 1986a).

As indicated in Table 5-2, by 1988, transportation (i.e., automotive) emissions were no longer the largest source of lead emitted to the atmosphere. When such emissions were prevalent, more than 90% (mass basis) of automotive lead emissions from leaded gasoline were in the form of inorganic particulate matter (e.g., lead bromochloride [PbBrCl]) and <10% (mass basis) were in the form of organolead vapors (e.g., lead alkyls). In 1984 the average lead content of gasoline was 0.44 g lead/gallon (EPA 1986a); however, as of January 1986, the allowable lead content of leaded gasoline dropped to 0.1 g lead/gallon

Table 5-2. National Lead Emission Estimates, 1979–1989

Source category	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989
Transportation	94.6	59.4	46.9	46.9	40.8	34.7	15.5	3.5	3.0	2.6	2.2
Fuel combustion	4.9	3.9	2.8	1.7	0.6	0.5	0.5	0.5	0.5	0.5	0.5
Industrial processes	5.2	3.6	3.0	2.7	2.4	2.3	2.3	1.9	1.9	2.0	2.3
Solid waste	4.0	3.7	3.7	3.1	2.6	2.6	2.8	2.7	2.6	2.5	2.3
Total ^a	108.7	70.6	56.4	54.4	46.4	40.1	21.1	8.6	8.0	7.6	7.2

^aThe sums of sub-categories may not equal total because of rounding.

Source: Derived from EPA 1990c, 1991b

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(EPA 1985g), and as of February 2, 1996, a ban on addition of lead compounds to gasoline went into effect. Between January and June of 1990, the actual average lead concentration in leaded gasoline was 0.085 g lead/gallon, indicating consumption of approximately 230,000 kg of lead for the production of 2.74 billion gallons of leaded gasoline. During the same 6-month period, 49 billion gallons of unleaded gasoline were produced in the United States (EPA 1990b). In the early 1980s EPA allowed up to 0.05 g lead per gallon of unleaded gasoline (EPA 1982b). An analysis of unleaded gasolines conducted in the winter of 1991–1992 indicated that regular grade unleaded gasoline contained, on average, less than 0.0003 g lead/gallon (MVMA 1992). On February 2, 1996, addition of lead to any grade of gasoline intended for on-road transportation was banned in the United States.

Reduction trends for air emissions of lead have continued from the late 1970s through the 1980s for both point sources (from 2.9 $\mu\text{g}/\text{m}^3$ in 1979 to 0.4 $\mu\text{g}/\text{m}^3$ in 1988) and urban sites (from 0.8 $\mu\text{g}/\text{m}^3$ in 1979 to 0.1 $\mu\text{g}/\text{m}^3$ in 1988) (EPA 1990a). The large decrease for point sources resulted from the use of emission controls for industrial processes as well as automotive controls; the decrease for urban sites was primarily the result of the decreased use of leaded gasoline. In June 1990, unleaded gasoline comprised 94% of all gasoline produced compared with 91% in July 1989 (EPA 1990b).

Releases from lead-based paints are frequently confined to the area in the immediate vicinity of painted surfaces, and deterioration or removal of the paint can result in high localized concentrations of lead in indoor air (from sanding and sandblasting) and on exposed surfaces.

The largest volume of organolead vapors released to the atmosphere results from industrial processes; prior to its phaseout and ban, leaded gasoline containing tetraethyl lead as an anti-knock additive was also a major contributor. Tetraalkyl lead vapors are photoreactive, and their presence in local atmospheres is transitory. Halogenated lead compounds are formed during combustion by reaction of the tetraalkyl lead compounds with halogenated lead scavenger compounds. These halogenated lead compounds ultimately give rise to lead oxides and carbonates in the environment (EPA 1985b). Tetraalkyl lead compounds once contributed 5–10% of the total particulate lead present in the atmosphere. Organolead vapors were most likely to occur in occupational settings (e.g., gasoline transport and handling operations, gas stations, and parking garages) and high-traffic areas (Nielsen 1984).

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5.2.2 Water

Of the known aquatic releases of lead, the largest ones are from the steel and iron industries and lead production and processing operations (EPA 1982a). Urban runoff and atmospheric deposition are significant indirect sources of lead found in the aquatic environment. Lead reaching surface waters is sorbed to suspended solids and sediments (EPA 1982a).

Although aquatic releases from industrial facilities are expected to be small, lead may be present in significant levels in drinking water. In areas receiving acid rain (e.g., northeastern United States) the acidity of drinking water may increase; this increases the corrosivity of the water, which may, in turn, result in the leaching of lead from water systems, particularly from older systems during the first flush of water through the pipes (McDonald 1985). In addition, the grounding of household electrical systems to the plumbing can increase corrosion rates and the subsequent leaching of lead from the lead solder used for copper pipes. Areas where the pH of the water is less than 8.0 may have higher drinking water lead levels as well (Lee et al. 1989).

According to the Toxics Release Inventory, in 1996, the estimated releases of lead of 62,654 pounds (28,418 kg) to water from 1,454 large processing facilities accounted for about 0.4% of total environmental releases (TRI96 1998). Table 5-1 lists amounts released from these facilities. The TRI data should be used with caution, however, since only certain types of facilities are required to report. This is not an exhaustive list.

Lead has been identified in groundwater samples collected at 781 of the 1,026 NPL hazardous waste sites, in leachate samples collected at 146 of the 1,026 NPL hazardous waste sites, and in surface water samples collected at 458 of the 1,026 NPL hazardous waste sites where it was detected in some environmental medium (HazDat 1998).

5.2.3 Soil

Solid wastes that contain lead are produced primarily as a result of domestic ore production and ammunition use. Other sources include solder, weights and ballasts, bearing metals, and iron and steel production. These sources of lead-contaminated waste are concentrated primarily in landfills.

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According to the Toxics Release Inventory, in 1996, the estimated releases of lead of 15,147,385 pounds (6,870,738 kg) to land from 1,454 large processing facilities accounted for about 89.4% of total environmental releases (TRI96 1998). An additional 794 pounds (360 kg), constituting less than 0.005% of the total environmental releases, were released via underground injection (TRI96 1998). Also, some of the estimated 370,905,354 pounds (168,239,838 kg) of lead transferred off-site may be ultimately disposed of on land. It should be noted that TRI-reported releases to land include, but are not limited to, releases to soil. Table 5-1 lists amounts released from these facilities. The TRI data should be used with caution, however, since only certain types of facilities are required to report. This is not an exhaustive list.

Lead has been identified in soil samples collected at 675 of the 1,026 NPL hazardous waste sites, in sediment samples collected at 456 of the 1,026 NPL hazardous waste sites, and in soil-gas samples collected at 2 of the 1,026 NPL hazardous waste sites where it was detected in some environmental medium (HazDat 1998).

5.2.4 Paint

Flaking paint, paint chips, and weathered powdered paint, which are most commonly associated with deteriorated housing stock in urban areas, are major sources of lead exposure for young children residing in these houses, particularly for children with pica (the compulsive, habitual consumption of nonfood items) (Bornschein et al. 1986; EPA 1986a). Lead concentrations of 1–5 mg/cm² have been found in chips of lead-based paint (Billick and Gray 1978), suggesting that consumption of a single chip of paint would provide greater short-term exposure than any other source of lead (EPA 1986a). An estimated 40–50% of currently occupied housing in the United States may contain lead-based paint on exposed surfaces (Chisolm 1986).

In the late 1980s, the U.S. Department of Housing and Urban Development (HUD) conducted a national survey of lead-based paint in housing. The EPA subsequently sponsored a comprehensive technical report on the HUD-sponsored survey to provide estimates of the extent of lead-based paint in housing. In the EPA report, a home is considered to have lead-based paint if the measured lead concentration on any painted surface is 1.0 mg/cm² or greater. The EPA report estimates that 64 million (±7 million) homes, or 83% (±9%) of privately-owned housing units built before 1980, have lead-based paint somewhere in the building. Approximately 12 million (±5 million) of these homes are occupied by families with children under the age of 7 years. Approximately 49 million (±7 million) privately owned homes have lead-based

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paint in their interiors. By contrast, approximately 86% ($\pm 8\%$) of all pre-1980 public housing family units have lead-based paint somewhere in the building (EPA 1995c).

Damaged lead-based paint is associated with excessive dust lead levels. Approximately 14 million homes (19% of pre-1980 housing) have more than 5 square feet of damaged lead-based paint, and nearly half (47%) of those homes have excessive dust lead levels (EPA 1995c).

In the Cincinnati prospective lead study of public and private low- and moderate-income housing, the lead concentration ranges were: painted interior walls, 0.1–35 mg/cm²; interior home surface dust, 0.04–39 mg/m² and 72–16,200 µg/g; interior home dustfall, 0.0040–60 mg/m²/30 days; exterior dust scrapings, 20–108,000 µg/g; and dust on children's hands, 1–191 µg. The lead levels in older private deteriorating or dilapidated housing were higher than the levels in newer public and rehabilitated housing (Clark et al. 1985).

Releases from lead-based paints are frequently confined to the area in the immediate vicinity of painted surfaces, and deterioration or removal of the paint can result in high localized concentrations of lead in indoor air (from sanding and sandblasting) and on exposed surfaces. Disturbance of older structures containing lead-based paints is now a significant contributor to total lead releases.

The authors of a report of findings from the Third National Health and Nutrition Examination Survey (NHANES III), conducted in 1988 to 1991, comment that of the multiple sources of exposure, lead-based paint is the principal high-dose source of lead. Exposure occurs not only through the direct ingestion of flaking and chalking paint but also through the inhalation of dust and soil contaminated with paint (Brody et al. 1994). According to a study by the New York State Department of Health, renovation and remodeling activities that disturb lead-based paints in homes can produce significant amounts of lead dust, which can be inhaled or ingested (CDC 1997d).

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5.3 ENVIRONMENTAL FATE**5.3.1 Transport and Partitioning**

In the atmosphere, non-organic compounds of lead exist primarily in the particulate form. Upon release to the atmosphere, lead particles are dispersed and ultimately removed from the atmosphere by wet or dry deposition. Approximately 40–70% of the deposition of lead is by wet fallout; 20–60% of particulate lead once emitted from automobiles is deposited near the source. An important factor in determining the atmospheric transport of lead is particle size distribution. Large particles, particularly those with aerodynamic diameters of $>2 \mu\text{m}$, settle out of the atmosphere fairly rapidly and are deposited relatively close to emission sources (e.g., 25 m from the roadway for those size particles emitted in motor vehicle exhaust in the past); smaller particles may be transported thousands of kilometers. The dry deposition velocity for lead particles with aerodynamic diameters of $0.06\text{--}2.0 \mu\text{m}$ was estimated to range between 0.2 and 0.5 cm/second in a coniferous forest in Sweden, with an overall particle-size weighted dry deposition velocity of 0.41 cm/second (Lannefors et al. 1983). However, the use of an average net deposition velocity of 0.6 cm/second and an average atmospheric residence time of 10 days has been recommended by the National Academy of Sciences (NAS 1980). The amount of lead scavenged from the atmosphere by wet deposition varies widely; wet deposition can account for 40–70% of lead deposition depending on such factors as geographic location and amount of emissions in the area (Nielsen 1984). An annual scavenging ratio (concentration in precipitation, mg/L, to concentration in air, $\mu\text{g}/\text{m}^3$) of 0.18×10^{-6} has been calculated for lead, making it the lowest value among seven trace metals studied (iron, aluminum, manganese, copper, zinc, cadmium); this indicates that lead (which initially exists as fine particles in the atmosphere) is removed from the atmosphere by wet deposition relatively inefficiently. Wet deposition is more important than dry deposition for removing lead from the atmosphere; the ratio of wet to dry deposition was calculated to be 1.63, 1.99, and 2.50 for sites in southern, central, and northern Ontario, Canada, respectively (Chan et al. 1986). Lead particles from automobile emissions are quite small ($<0.1 \mu\text{m}$ in diameter) but can grow in size by coagulation (Chamberlain et al. 1979). Lead has been found in sediment cores of lakes in Ontario and Quebec, Canada, that were remote from any point sources of lead releases, indicating that long-range atmospheric transport was occurring (Evans and Rigler 1985).

The amount of lead that remains in solution in surface waters depends upon the pH of the water and the dissolved salt content. Equilibrium calculations show that at $\text{pH} > 5.4$, the total solubility of lead is approximately $30 \mu\text{g}/\text{L}$ in hard water and approximately $500 \mu\text{g}/\text{L}$ in soft water. Sulfate ions, if present in

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soft water, limit the lead concentration in solution through the formation of lead sulfate. Above pH 5.4, the lead carbonates, PbCO_3 and $\text{Pb}_2(\text{OH})_2\text{CO}_3$, limit the concentration. The carbonate concentration is in turn dependent upon the partial pressure of carbon dioxide, pH, and temperature (EPA 1986a). In most surface waters and groundwaters, the concentration of dissolved lead is low because the lead will form compounds with anions in the water such as hydroxides, carbonates, sulfates, and phosphates that have low water solubilities and will precipitate out of the water column (Mundell et al. 1989).

A significant fraction of lead carried by river water is expected to be in an undissolved form, which can consist of colloidal particles or larger undissolved particles of lead carbonate, lead oxide, lead hydroxide, or other lead compounds incorporated in other components of surface particulate matters from runoff. Lead may occur either as sorbed ions or surface coatings on sediment mineral particles, or it may be carried as a part of suspended living or nonliving organic matter in water. The ratio of lead in suspended solids to lead in dissolved form has been found to vary from 4:1 in rural streams to 27:1 in urban streams (Getz et al. 1977).

The fate of lead in soil is affected by the specific or exchange adsorption at mineral interfaces, the precipitation of sparingly soluble solid forms of the compound, and the formation of relatively stable organic-metal complexes or chelates with soil organic matter. These processes are dependent on such factors as soil pH, soil type, particle size, organic matter content of soil, the presence of inorganic colloids and iron oxides, cation exchange capacity (CEC), and the amount of lead in soil (NSF 1977; Reddy et al. 1995; Royer et al. 1992). Soil samples were extracted from the Powder River Basin in Wyoming to determine the relative distribution and chemical forms of lead and other metals in acidic environments (Reddy et al. 1995). As pH increased, the dissolved concentration of lead increased and then decreased. At near neutral pH, dissolved organic carbon-lead complexes were the predominant species in the soil water extracts. At low pH, the lead ionic form (Pb^{2+}) and ion pairs (e.g., PbSO_4^0) were predominant. It was also concluded that the availability and mobility of lead will increase in low pH environments due to the chemical form in which the metal is present in the soil solutions. The accumulation of lead in most soils is primarily a function of the rate of deposition from the atmosphere. Most lead is retained strongly in soil, and very little is transported into surface water or groundwater (EPA 1986a; NSF 1977). Clays, silts, iron and manganese oxides, and soil organic matter can bind metals electrostatically (cation exchange) as well as chemically (specific adsorption) (Reed et al. 1995). Lead is strongly sorbed to organic matter in soil, and although not subject to leaching, it may enter surface waters as a result of erosion of lead-containing soil particulates. Lead bromochloride, the primary form of lead emitted from motor vehicles which once burned

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leaded gasoline in the presence of organohalogen scavenger compounds, may be converted to the less soluble lead sulfate either by reactions in the atmosphere or by reactions at the soil surface. It has been determined that lead oxides, carbonates, oxycarbonates, sulfates, and oxysulfates become the most prominent constituents of aged automobile exhaust particles (i.e., those collected at locations more remote from traffic sources) (Ter Haar and Bayard 1971). Lead may also be immobilized by ion exchange with hydrous oxides or clays or by chelation with humic or fulvic acids in the soil (Olson and Skogerboe 1975). In soils with pH of 5 and with at least 5% organic matter content, atmospheric lead is retained in the upper 2–5 cm of undisturbed soil. Inorganic lead may be bound into crystalline matrices of rocks and remain essentially immobile; it can also be entrapped in the immobile water surrounding soil macro- and micropores (Reed et al. 1995). Lead complexes and precipitates in soil and their transformation depend on the soil type. In soil with a high organic matter content and a pH of 6–8, lead may form insoluble organic lead complexes; if the soil has less organic matter at the same pH, hydrous lead oxide complexes may form or lead may precipitate out with carbonate or phosphate ions. At a pH of 4–6, the organic lead complexes become soluble and leach out or may be taken up by plants (EPA 1986a). Entrainment or suspension of soil particles in moving air is another route of lead transport (EPA 1982f). This process may be important in contributing to the atmospheric burden of lead around some lead smelting facilities and NPL sites that contain elevated levels of lead in soil.

The downward movement of elemental lead and inorganic lead compounds from soil to groundwater by leaching is very slow under most natural conditions except for highly acidic situations (NSF 1977). The conditions that induce leaching are the presence of lead in soil at concentrations that either approach or exceed the cation exchange capacity (CEC) of the soil, the presence of materials in soil that are capable of forming soluble chelates with lead, and a decrease in the pH of the leaching solution (for example, acid rain) (NSF 1977). Partial favorable conditions for leaching may be present in some soils near lead smelting and NPL sites. Leaching of soluble lead from contaminated soils into groundwater may be minimized by the presence of lead carbonate in the soil and by maintaining a soil pH of 8–10 (Mundell et al. 1989). Tetraalkyl lead compounds, such as tetraethyl lead, are considered insoluble in water. In an aqueous media, tetraalkyl lead compounds are first degraded to their respective ionic trialkyl lead species and are eventually mineralized to inorganic lead (Pb^{2+}) by biological and chemical degradation processes (Ou et al. 1995). Tetraethyl lead can be transported through a soil column when it is present in a migrating plume of gasoline (Mansell et al. 1995).

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Plants and animals may bioconcentrate lead but biomagnification has not been detected. In general, the highest lead concentrations are found in aquatic and terrestrial organisms that live near lead mining, smelting, and refining facilities; storage battery recycling plants; areas affected by high automobile and truck traffic; sewage sludge and spoil disposal areas; sites where dredging has occurred; areas of heavy hunting (lead source from spent shot); and in urban and industrialized areas. Lead may be present on plant surfaces as a result of atmospheric deposition; its presence in internal plant tissues indicates biological uptake from the soil and leaf surfaces. Although the bioavailability of lead in soil to plants is limited because of the strong absorption of lead to soil organic matter, the bioavailability increases as the pH and the organic matter content of the soil are reduced. Lead is not biomagnified in aquatic or terrestrial food chains. It may contaminate terrestrial plants as a result of atmospheric deposition and uptake from soil, and animals as a result of inhalation of contaminated ambient air or ingestion of contaminated plants. Older organisms tend to contain the greatest body burdens of lead. In aquatic organisms, lead concentrations are usually highest in benthic organisms and algae, and lowest in upper trophic level predators (e.g., carnivorous fish). Exposure of a freshwater fish to several sublethal concentrations of lead for a period of 30 days showed significant accumulation of lead in the blood and tissues. The lead accumulation in tissues was found to increase with lead in water up to a concentration of 5 mg/L ($\mu\text{g/mL}$); at concentrations of 10 and 20 mg/L, the lead accumulation in the tissues, although indicating an increase, was not proportional to the lead concentration in water (Tulasi et al. 1992). High bioconcentration factors (BCFs) were determined in studies using oysters (6,600 for *Crassostrea virginica*), freshwater algae (92,000 for *Senenastrium capricornutum*) and rainbow trout (726 for *Salmo gairdneri*). However, most median BCF values for aquatic biota are significantly lower: 42 for fish, 536 for oysters, 500 for insects, 725 for algae, and 2,570 for mussels (Eisler 1988). Lead is toxic to all aquatic biota, and organisms higher on the food chain may experience lead poisoning as a result of eating lead-contaminated food. Organolead compounds, such as trialkyl and tetraalkyl lead compounds, are more toxic than inorganic forms and have been shown to bioconcentrate in aquatic organisms. Biomagnification of organolead compounds has not been shown and depuration is relatively rapid, with half-life values of 30–45 hours for rainbow trout exposed to tetramethyl lead. Tetraalkyl lead compounds are more toxic than trialkyl lead compounds, and ethyl forms are more toxic than methyl forms (Eisler 1988). Isolation of a *Pseudomonas aeruginosa* strain designated CHL004 which is able to remove lead from solidified media and soil has been reported (Vesper et al. 1996). The rate of uptake of lead nitrate by CHL004 was very rapid initially and then decreased greatly.

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Lead may be taken up in edible plants from the soil via the root system, by direct foliar uptake and translocation within the plant, and by surface deposition of particulate matter. The amount of lead in soil that is bioavailable to a vegetable plant depends on factors such as cation exchange capacity, pH, amount of organic matter present, soil moisture content, and the type of amendments added to the soil. Background agricultural soil lead concentrations for major growing areas of the United States have been determined (Holmgren et al. 1993).

Concentrations of lead (wet weight basis) in samples of eleven raw edible plants have been reported for growing areas in the United States that are uncontaminated by human activities other than normal agricultural practices (Wolnik et al. 1983a, 1983b). Results are as follows: plant (mean $\mu\text{g/g}$ wet weight); lettuce (0.013); peanut (0.010); potato (0.009); soybean (0.042); sweet corn (0.0033); wheat (0.037); field corn (0.022); onion (0.005); rice (0.007); spinach (0.045); tomato (0.002).

The influence of various combinations of soil amendments on lead uptake by soybeans was studied for a metal-contaminated alluvial soil (Pierzynski and Schwab 1993). Addition of limestone was found to be most effective in reducing the bioavailability of metals (including lead) as indicated by the reduction in labile soil metals, increased yields, and decreased soybean tissue metal content. Uptake of metals by lettuce and radishes grown in a loam soil spiked with cadmium chloride and lead nitrate (from 100 to 1000 mg/kg) was also studied (Nwosu et al. 1995). Results indicated that the mean uptake of lead by lettuce increased as the concentration of lead rose in the soil mixture. However, the uptake was small and this finding is inconsistent with other reports. Lead was not bioaccumulated by either plant regardless of soil lead concentrations. The response of kidney bean growth to the concentration and chemical form of lead in soils obtained near a zinc smelter in Japan has been studied (Xian 1989). It was found that the amount of lead in the total plant (approximately 35 to 80 μg) correlated strongly with the concentration of lead in the soil (0 to 240 mg/kg). The best relationship was found between the amount of metal uptake and the concentration of exchangeable and carbonate forms of lead in the soil.

Other factors such as absorption of lead from cooking water and cookware can influence the amount of lead in cooked vegetables. The degree to which lead is released from plant tissue once the vegetable or fruit is consumed also influences a person's uptake of lead.

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5.3.2 Transformation and Degradation**5.3.2.1 Air**

Information available regarding the chemistry of lead in air is limited. Before the ban on sales of leaded gasoline, lead particles were emitted to the atmosphere from automobiles as lead halides (mostly PbBrCl) and as double salts with ammonium halides (e.g., $2\text{PbBrCl} \cdot \text{NH}_4\text{Cl}$, $\text{Pb}_3[\text{PO}_4]_2$, and PbSO_4 [Biggins and Harrison 1979; Ter Haar and Bayard 1971]). After 18 hours, approximately 75% of the bromine and 30–40% of the chlorine disappeared, and lead carbonates, oxycarbonates and oxides were produced. These lead oxides are subject to further weathering to form additional carbonates and sulfates (Olson and Skogerboe 1975). Lead particles are emitted from mines and smelters primarily in the form of the lead-sulfur compounds, PbSO_4 , $\text{PbO} \cdot \text{PbSO}_4$, and PbS (EPA 1986a). In the atmosphere, lead exists primarily in the form of PbSO_4 and PbCO_3 . It is not completely clear how the chemical composition of lead changes during dispersion (EPA 1986a). Monitoring studies indicate that tetraalkyl lead, at one time present in both urban and rural air, may react with hydroxyl ions to form ionic trialkyl and dialkyl species that are more stable in the atmosphere. Urban air in England that is advected to rural areas may contain up to 5% of the total lead as alkyl lead; this percentage may increase to 20% for maritime air, with trialkyl lead being the predominant species (Hewitt and Harrison 1987).

Tetraalkyl lead compounds, once added to gasoline, are no longer present in significant quantities in the air. However, their degradation products are still present. Based on the vapor pressure of tetraethyl lead (0.26 mm Hg at 20 °C) and tetramethyl lead (26.0 mm Hg at 20 °C), these two compounds exist almost entirely in the vapor phase in the atmosphere (Eisenreich et al. 1981). When exposed to sunlight, they decompose rapidly to trialkyl and dialkyl lead compounds, and eventually to inorganic lead oxides by a combination of direct photolysis, reaction with hydroxyl radicals, and reaction with ozone. The half-life of tetraethyl lead in summer atmospheres is approximately 2 hours, and the half-life for tetramethyl lead is about 9 hours. In the winter, both compounds have half-lives of up to several days (DeJonghe and Adams 1986). Trialkyl compounds occur almost entirely in the vapor phase, and dialkyl compounds occur almost entirely in particulate form. Because of the relatively high water solubility of trialkyl and dialkyl lead compounds, washout in wet deposition was probably a major process for removing these compounds from air. In addition, the dialkyl lead compounds were significantly removed by dry deposition. Adsorption of tetraethyl and tetramethyl lead to atmospheric particles does not appear to be an important fate process (DeJonghe and Adams 1986; EPA 1985a).

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5.3.2.2 Water

The chemistry of lead in aqueous solution is highly complex because this element can be found in a multiplicity of forms. Lead has a tendency to form compounds of low solubility with the major anions found in natural waters. The amount of lead in surface waters is dependent on the pH and the dissolved salt content of the water. The dissolved salt content, in turn, is dependent on the pH and the partial pressure of CO₂ as well as the water temperature. In the environment, the divalent form (Pb²⁺) is the stable ionic species of lead. Hydroxide, carbonate, sulfide, and, more rarely, sulfate may act as solubility controls in precipitating lead from water. At a pH <5.4, lead sulfate limits the concentration of lead in solution, while at a pH >5.4, lead carbonates limit the lead concentrations (EPA 1979d). The relatively volatile organolead compound, tetramethyl lead, may form as a result of biological alkylation of organic and inorganic lead compounds by microorganisms in anaerobic lake sediments; however, if the water over the sediments is aerobic, volatilization of tetramethyl lead from the sediments is not considered to be important because the tetramethyl lead will be oxidized (EPA 1979d).

In water, tetraalkyl lead compounds are subject to photolysis and volatilization with the more volatile compounds being lost by evaporation. Degradation proceeds from trialkyl lead to dialkyl lead to inorganic lead. Tetraethyl lead is susceptible to photolytic decomposition in water. Triethyl and trimethyl lead are more water-soluble and therefore more persistent in the aquatic environment than tetraethyl or tetramethyl lead. The degradation of trialkyl lead compounds yields small amounts of dialkyl lead compounds. Removal of tetraalkyl lead compounds from seawater occurs at rates that provide half-lives measurable in days (DeJonghe and Adams 1986).

5.3.2.3 Sediment and Soil

Lead in its naturally-occurring mineral forms is a very minor component of many soils in the United States. Additional lead is added through processes such as wet and dry deposition from the atmosphere and via surface water flows. Now that lead in gasoline is banned, the major sources on the national level are industrial processes (58% of total estimated emissions in 1995) (EPA 1996h). Smelters in Pennsylvania, Missouri, and Nebraska are among the top 10 emitters. Lead particles emitted from mining operations and smelters are primarily in the form of lead-sulfur compounds PbSO₄, PbO•PbSO₄, and PbS (EPA 1986a). In the atmosphere, lead probably exists primarily in the form of PbSO₄ and PbCO₃ and impacts the soil in this form. Organic tetraalkyl lead compounds, once used extensively in motor fuel, are emitted from

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automobiles primarily in the form of lead bromochloride. The organolead compounds undergo photolysis and other reactions in the atmosphere to form lead carbonates, oxycarbonates, and oxides. Once these compounds encounter components of the soil, further reactions can occur to produce lead sulfate. Divalent lead ion also has a strong affinity for humic acids in soil and thus usually combines to form stable Pb-organic complexes.

Now that lead additives in motor fuels for highway use are banned, emissions of lead from this source have diminished to very low levels. However, the deposited organolead compounds and their transformation products are still in the soil. Limited data indicate that tetraethyl and tetramethyl lead are converted into water-soluble lead compounds in soil. Although tetraethyl and tetramethyl lead are not expected to leach significantly through soil, their highly water-soluble metabolites, the trialkyl lead oxides, may be subject to leaching (EPA 1985a). Recent laboratory studies have sought to explain how chemical degradation and biological metabolism of two ionic ethyl-lead species, triethyllead and diethyllead, occur in soil (Ou et al. 1995).

In a study of lead migration in forest soils in Vermont, Miller and Friedland (1994) used lead deposition time series and measurements of organic soil horizon lead content made in 1966, 1980, and 1990 to compute dynamic response times for lead storage in several types of soil. The authors concluded that maximum lead concentrations in organic soil occurred around 1980, with concentrations of about 85 $\mu\text{g/g}$ in soils of the northern hardwood forests of the study area and about 200 $\mu\text{g/g}$ in soils of the spruce-fir forests. The large surge of atmospheric lead deposited in these forests during the time when leaded gasoline was routinely used in motor vehicles is being redistributed in the soil profile rather than being retained in the organic horizon. Based on an analysis of lead transit times through mineral soil horizons, the pulse of lead may begin to be released to upland streams sometime in the middle of the next century (Miller and Friedland 1994).

Many plants commonly take up lead from soil, and lead will eventually be returned to soil when these plants decay unless they are harvested (to possibly enter the food chain) or removed (EPA 1986a).

5.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

Reliable evaluation of the potential for human exposure to lead depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. In reviewing data on lead

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levels monitored or estimated in the environment, it should also be noted that the amount of chemical identified analytically is not necessarily equivalent to the amount that is bioavailable.

5.4.1 Air

Lead levels in the ambient air have been monitored in a number of remote, urban, and nonurban areas of the United States and other countries (EPA 1986a). Atmospheric lead concentrations vary widely but usually decrease with vertical and horizontal distance from emission sources; they are generally 0.3–0.8 times lower indoors than outdoors, with an average ratio of 0.5. Levels of lead in ambient air range from $7.6 \times 10^{-5} \mu\text{g}/\text{m}^3$ in remote areas such as Antarctica (Maenhaut et al. 1979) to $>10 \mu\text{g}/\text{m}^3$ near stationary sources such as smelters, with an average annual concentration of below $1.0 \mu\text{g}/\text{m}^3$ for urban monitoring sites. Monitoring data from a composite of 147 sampling sites throughout the United States indicate that the maximum quarterly average lead levels in urban air were $0.36 \mu\text{g}/\text{m}^3$ during 1984 and $0.2\text{--}0.4 \mu\text{g}/\text{m}^3$ during 1986 (EPA 1988f, 1989h). Between 1979 and 1983, atmospheric lead concentrations in precipitation in Minnesota decreased from 29 to $4.3 \mu\text{g}/\text{L}$ at urban locations and from 5.7 to $1.5 \mu\text{g}/\text{L}$ at rural locations, indicating a reduction in lead emissions of more than 80%. This reduction resulted primarily from the decreased use of leaded gasoline (down 56%) and the use of more efficient emission controls on other sources (Eisenreich et al. 1986).

Since 1979, elemental concentrations of fine particles have been monitored in remote areas of the United States in networks operated for the National Park Service (NPS) and the EPA (Eldred and Cahill 1994). Lead at all sites decreased sharply through 1986, corresponding to the shift to unleaded gasoline, but has since leveled off at $1\text{--}2 \text{ ng}/\text{m}^3$ ($0.001\text{--}0.002 \mu\text{g}/\text{m}^3$), which is approximately 18% of the 1982 mean. The elevated lead concentrations (up to $5 \text{ ng}/\text{m}^3$) since 1986 at three of the twelve sites are probably associated with mining activity.

In the 1960s, the National Air Surveillance Network (NASN) was established to monitor ambient air quality levels of total particulate solids and trace metals, including lead, at sites in larger American cities. In 1981 some old sites were eliminated and new ones were added to give 139 urban sites for air monitoring purposes. In 1988, the average lead concentration for all 139 sites was $0.085 \mu\text{g}/\text{m}^3$, well below the National Ambient Air Quality Standard of $1.5 \mu\text{g}/\text{m}^3$, quarterly average concentration, that has been established for lead (EPA 1996h). In 1988, the average concentration of 18 point-source sites was $0.4 \mu\text{g}/\text{m}^3$, down from $2.9 \mu\text{g}/\text{m}^3$ in 1979, and the average concentration for urban sites was $0.1 \mu\text{g}/\text{m}^3$,

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down from 0.8 $\mu\text{g}/\text{m}^3$ in 1979 (EPA 1990c). This decrease was undoubtedly caused by decreased use of leaded gasolines in the period leading up to its total ban after December 1995. Composite urban air measurements of lead for 1989 and 1991 were 0.11 and 0.08 $\mu\text{g}/\text{m}^3$ (EPA 1996h). Although urban lead concentrations in air continue to decline, there are indications that the rate of decline has slowed. Between 1976 and 1995, ambient concentrations of lead in the United States declined by 97%. Between 1994 and 1995, national average lead concentrations remained unchanged at 0.04 $\mu\text{g}/\text{m}^3$ even though lead emissions declined 1% (EPA 1996h).

5.4.2 Water

Lead has been monitored in surface water, groundwater, and drinking water throughout the United States and other countries. The concentration of lead in surface water is highly variable depending upon sources of pollution, lead content of sediments, and characteristics of the system (pH, temperature, etc.). Levels of lead in surface water and groundwater throughout the United States typically range between 5 and 30 $\mu\text{g}/\text{L}$, although levels as high as 890 $\mu\text{g}/\text{L}$ have been measured (EPA 1986a). Mean levels of lead in surface water measured at 50,000 surface water stations throughout the United States are 3.9 $\mu\text{g}/\text{L}$ (based on 39,490 occurrences) (Eckel and Jacob 1988). Lead is estimated to be present in sea water at approximately 0.005 $\mu\text{g}/\text{L}$ (EPA 1982f). Lead concentrations in surface water are higher in urban areas than in rural areas (EPA 1982f).

Based on a survey of 900 public water supply systems, EPA (1988b) estimated that 99% of the 219 million people in the United States using public water supplies are exposed to drinking water with levels of lead <5 $\mu\text{g}/\text{L}$ and approximately 2 million people are served by drinking water with levels of lead greater than 5 $\mu\text{g}/\text{L}$. A survey of 580 cities in 47 states indicated that the national mean concentration of lead in drinking water was 29 $\mu\text{g}/\text{L}$ after a 30-second flushing period (EPA 1988f, 1989h); however, it was estimated that in 1988 the average lead content of drinking water was 17 $\mu\text{g}/\text{L}$ (Cohen 1988b). In 1986, the Safe Drinking Water Act Amendments banned the use of lead solder or flux containing more than 0.2% lead and the use of lead pipes or fittings that contained more than 8% lead (EPA 1988f, 1989h).

In a more recent Federal Register notice (EPA 1991d), EPA examined the occurrences of lead in source water and distributed water. By resampling at the entry point to the distribution system, few samples were found to contain lead at levels above 5 $\mu\text{g}/\text{L}$. EPA now estimates that approximately 600 groundwater systems may have water leaving the treatment plant with lead levels above 5 $\mu\text{g}/\text{L}$. Based on several data

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sets, it is estimated that less than 1% of the public water systems in the United States have water entering the distribution system with lead levels above 5 µg/L. These systems are estimated to serve less than 3% of the population that receives drinking water from public systems (EPA 1991d).

Lead levels ranging between 10 and 30 µg/L can be found in drinking water from households, schools, and office buildings as a result of plumbing corrosion and subsequent leaching of lead. The combination of corrosive water and lead pipes or lead-soldered joints in either the distribution system or individual houses can create localized zones of high lead concentrations that exceed 500 µg/L (EPA 1989f).

Quantitative data on the nationwide range of lead levels in drinking water drawn from the tap (which would include lead corrosion by-product) were insufficient to assign a national value at the time of the 1991 EPA publication. One set of data comprised of 782 samples taken in 58 cities in 47 states shows that the average lead level in tap water was 13 µg/L with 90% of the values below 33 µg/L (EPA 1991d).

According to EPA's National Compliance Report for calendar year 1996 (EPA 1998g), the vast majority of people in the nation received water from systems that had no reported violations of the maximum contaminant level and treatment technique requirements or significant monitoring and reporting requirements. Lead has a maximum permissible level of 15 µg/L delivered to any user of a public water system. Lead and copper are regulated in a treatment technique that requires systems to take tap water samples at sites with lead pipes or copper pipes that have lead solder and/or are served by lead service lines. The water system is required to take treatment steps if the action level (15 µg/L for lead) is exceeded in more than 10% of tap water samples. For calendar year 1996, nearly 6 million people in the United States were served by community water systems that reported maximum contaminant level and treatment technique violations of the Lead and Copper Rule (EPA 1998g).

A survey of 1,484 drinking water samples taken from various districts of the American Water Works Service Company showed that average lead levels in a 1-L first-draw sample for copper, galvanized, and plastic pipes were 9, 4.2, and 4.5 µg/L, respectively. These data show that even plumbing that did not use lead solder for copper pipes (e.g., plastic pipes) contained significant levels of lead, primarily from the brass faucet fixtures which are used in almost all plumbing. The brass fixtures may account for approximately one-third of the lead in the first-draw water (Lee et al. 1989). Lead levels are also known to increase when tap water is heated in boiling kettles that contain lead in their heating elements.

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Concentrations of lead in water at NPL sites can be at much higher levels. For example, in 1986, an NPL hazardous waste site was identified in Genesee County, Michigan, that contained a landfill and nine surface impoundments. The facility had accepted sludge and residual waste from a chemical warehouse as well as other hazardous wastes. Water samples taken from the impoundments had a maximum lead concentration of 25 mg/L (EPA 1986d).

5.4.3 Sediment and Soil

Sediments contain considerably higher levels of lead than corresponding surface waters. Concentrations of lead in river sediments have been estimated at about 23 mg/kg (EPA 1982f; Fitchko and Hutcheson 1975), and concentrations of lead in coastal sediments range from 1 mg/kg to 912 mg/kg with a mean value of 87 mg/kg (EPA 1982f; Nriagu 1978). Data from the STORET (1973–1979) database of Eastern and Midwestern river basins indicates maximum lead concentrations in river sediments of 440–1,000 mg/kg, and mean lead concentrations of 27–267 mg/kg (EPA 1980, 1982f). Surface sediment concentrations in Puget Sound ranged from 13 µg/g to 53 µg/g (Bloom and Crecelius 1987). An analysis of sediments taken from 10 lakes in Pennsylvania indicated that the elevated lead values were not derived from leaching of lead from the native rocks as a result of acid deposition, but rather originated from anthropogenic lead deposition (probably from automotive emissions) on the soil surface and subsequent runoff of soil particulates into the lake (Case et al. 1989).

The natural lead content of soil derived from crustal rock, mostly as galena (PbS), typically ranges from <10 to 30 µg/g soil. However, the concentration of lead in the top layers of soil varies widely due to deposition and accumulation of atmospheric particulates from anthropogenic sources. The concentration of soil lead generally decreases as distance from contaminating sources increases. The estimated lead levels in the upper layer of soil beside roadways are typically 30–2,000 µg/g higher than natural levels, although these levels drop exponentially up to 25 m from the roadway (EPA 1986a). Soil adjacent to a smelter in Missouri had lead levels in excess of 60,000 µg/g (Palmer and Kucera 1980). Soils adjacent to houses with exterior lead-based paints may have lead levels of >10,000 µg/g (EPA 1986a). As a result of lead reactions with the soil, extractable lead in surface soil samples (0–5 cm depth) from an agricultural area near a car battery manufacturing plant (taken at 0.3 km from the source) decreased from 117 µg/g

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to 1 $\mu\text{g/g}$ within 1 year after the plant stopped operating (Schalscha et al. 1987). Soil collected by scraping the top 2.5 cm of soil surface near homes and streetside in Louisiana and Minnesota contained median lead concentrations of greater than 840 $\mu\text{g/g}$ in New Orleans and 265 $\mu\text{g/g}$ in Minneapolis. In contrast, the small towns of Natchitoches, Louisiana, and Rochester, Minnesota, had soil lead concentrations of less than 50 $\mu\text{g/g}$ and 58 $\mu\text{g/g}$, respectively. These data suggest that lead-contaminated soil is a major source of lead exposure in urban areas (Mielke 1992).

Studies carried out in Maryland and Minnesota indicate that within large light-industrial urban settings such as Baltimore, the highest soil lead levels generally occur in inner-city areas, especially where high traffic flows have long prevailed (Mielke et al. 1983, 1985, 1989) and that the amount of lead in the soil is correlated with the size of the city (Mielke 1991). In 1981, soil lead levels in the Minneapolis/St. Paul inner-city area were 60 times higher (423 $\mu\text{g/g}$) than levels found in rural Minnesota (6.7 $\mu\text{g/g}$), with almost all the increase (95%) resulting from the combustion of leaded gasoline. A study conducted in Minneapolis, Minnesota, after the lead content of gasoline has been significantly reduced, found that median soil lead levels taken from the foundations of homes, in yards, and adjacent to the street were 700 $\mu\text{g/g}$, 210 $\mu\text{g/g}$, and 160 $\mu\text{g/g}$, respectively; median soil lead concentrations in comparable samples from the smaller city of Rochester, Minnesota, did not exceed 100 $\mu\text{g/g}$ at any location tested (Mielke et al. 1989). The Minneapolis data showed that average lead levels were elevated in soil samples taken from the foundations of homes, but that lead levels were low (<50 $\mu\text{g/g}$) in areas where children could be expected to play, such as parks that were located away from traffic but were higher in play areas around private residences. Soil samples taken from around the foundations of homes with painted exteriors had the highest lead levels (mean concentrations of 522 $\mu\text{g/g}$) but levels around homes composed of brick or stucco were significantly lower (mean concentration 158 $\mu\text{g/g}$) (Schmitt et al. 1988). Severely contaminated soils (levels #20,136 $\mu\text{g/g}$) were located near house foundations adjacent to private dwellings with exterior lead-based paint. Elevated soil lead concentrations were found in larger urban areas with 27, 26, 32, and 42% of the soil samples exceeding 300 $\mu\text{g/g}$ lead in Duluth, inner-city North Minneapolis, inner-city St. Paul, and inner-city South Minneapolis, respectively. Only 5% of the soil samples taken from the smaller urban areas of Rochester and St. Cloud, Minnesota, had lead levels in excess of 150 $\mu\text{g/g}$. It has been suggested that the higher lead levels associated with soils taken from around painted homes in the inner city are the result of greater atmospheric lead content, resulting from the burning of leaded gasoline in cars and the washdown of building surfaces to which the small lead particles adhere by rain (Mielke et al. 1989). A

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state-wide Minnesota study concluded that exterior lead-based paint was the major source of contamination in severely contaminated soils located near the foundations of private residences and that aerosol lead accounted for virtually all of the contamination found in soils removed from the influence of lead-based paint. Contamination due to lead-based paint was found to be highly concentrated over a limited area, while contamination due to aerosol lead was found to be less concentrated but more widespread (Minnesota Pollution Control Agency 1987).

In a study of associations between soil lead levels (PbSs) and childhood PbB levels in urban New Orleans and rural Lafourche Parish in Louisiana, childhood PbB levels appeared more closely associated with PbS than with age of housing. In the study, over 2,600 PbS and 6,000 PbB samples were paired by their median values and pre-1940 housing percentages for 172 census tracts. Census tracts with low median PbS were associated with new housing, but census tracts with high median PbS were split evenly between old and new housing. The same pattern was also observed for childhood PbB levels. High PbS was associated with high PbB, and low PbS was associated with low PbB. Risk factors for lead exposure were found to be low in Lafourche Parish, where there was no census tract in which median PbB was above 9 µg/dL and no indication of a statistical association between median PbB and either median PbS or age of housing (Mielke et al. 1997).

In the state of Maine, soil samples taken from areas of high risk (within 1–2 feet of a foundation of a building more than 30 years old) indicated that 37% of the samples had high lead concentrations (>1,000 µg/g). In 44% of the private dwellings, high lead levels were found in the soil adjacent to the foundation; high levels were found in only 10% of the public locations (playgrounds, parks, etc.). In addition, the largest percentage (54%) of highly contaminated soil was found surrounding homes built prior to 1950; homes built after 1978 did not have any lead contamination in the soil (Krueger and Duguay 1989).

In environmental health studies conducted near four NPL sites (plus a comparison area for each), ATSDR collected lead concentration data from both environmental media and human body fluids to estimate low-level exposure risk and to document the magnitude of human exposure to lead near those sites. Environmental samples collected at participants' homes included drinking water, yard soil, house dust, and house paint; body fluids collected from participants included venous blood and urine specimens. For the four sites, mean concentrations of lead in soil ranged from 317 to 529 mg/kg, and mean concentrations of lead in dust ranged from 206 to 469 mg/kg (ATSDR 1995).

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In 1972, household dust samples taken near nonferrous ore smelters in El Paso, Texas, which were known to emit 1,012 metric tons of lead per year, had lead levels of 22,191 $\mu\text{g/g}$ (geometric mean) and 973 $\mu\text{g/g}$ at distances from the smelter of 1.6 km and 6.4 km, respectively (Landrigan and Baker 1981).

Lead was measured in soil from a port facility where galena ore concentrate and smelter dross arriving by rail were offloaded, stored, and reloaded onto seagoing vessels from 1974 through 1985. The lead concentrations ranged from 1,900 to 183,000 mg/kg ($\mu\text{g/g}$) (Ruby et al. 1994).

In 1986, an NPL hazardous waste site that contained a landfill and nine surface impoundments was identified in Genesee County, Michigan. The facility had accepted sludge and residual waste from a chemical warehouse as well as other hazardous wastes. Lead was present in sludge samples taken from the impoundments at a maximum concentration of 11.6 mg/L , in sediment samples at a maximum concentration of 4,770 mg/kg dry weight, and in soil samples at 1,560 mg/kg (EPA 1986d). Thirty of 97 soil samples taken at a former foundry site in Dubuque, Iowa, which was on the NPL, had lead concentrations exceeding 5.0 mg/L as determined using the extraction procedure (EP) toxicity test (the maximum total lead concentration was 4,890 mg/kg). Most of the positive samples were from soil depths of less than 2.5 feet (Mundell et al. 1989).

5.4.4 Paint

Weathering of lead-based paint can contribute to the lead content of dust and soil. A 1974 study indicated that elevated PbB levels in children were most likely a result of ingesting lead-contaminated soil, and that the most likely source was lead-based paint rather than lead from automotive exhaust (Ter Haar and Aronow 1974). A state-wide Minnesota study concluded that exterior lead-based paint was the major source of contamination in severely contaminated soils located near the foundations of private residences (Minnesota Pollution Control Agency 1987). A soil lead study in Minneapolis, Minnesota, found that soil samples taken from around the foundations of homes with painted exteriors had a mean concentration of 522 $\mu\text{g/g}$ while soil samples taken from around the foundations of brick or stucco had a mean concentration of 158 $\mu\text{g/g}$ (Schmitt et al. 1988). Lead-based paint, removed from surfaces by burning (gas torch or hot air gun), scraping, or sanding have been found to result, at least temporarily, in higher levels of exposure for families residing in these homes.

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5.4.5 Other Sources

Lead has been detected in a variety of foods. Typical concentrations of lead in various foods are (EPA 1986a):

<u>Food group</u>	<u>Concentration ($\mu\text{g/g}$)</u>
Dairy products	0.003–0.083
Meat, fish, and poultry	0.002–0.159
Grain and cereal products	0.002–0.136
Vegetables	0.005–0.649
Fruit and fruit juices	0.005–0.223
Oils, fats, and shortenings	0.002–0.028
Sugar and adjuncts	0.006–0.073
Beverages	0.002–0.041 ($\mu\text{g/L}$)

Canning foods in lead-soldered cans may increase levels of lead 8–10-fold; however, the impact of canning appears to be decreasing as a result of a decrease in the use of lead-soldered cans. The use of three-piece lead-soldered cans ceased in 1991; however, older lead-soldered cans may still be present in some households. In 1974, for example, the lead level in evaporated milk in lead-soldered cans was 0.12 $\mu\text{g/g}$; in 1986, after these cans were phased out, the lead level in evaporated milk dropped to 0.006 $\mu\text{g/g}$ (Capar and Rigsby 1989). The lead content in canned foods dropped from an overall mean of 0.31 ppm in 1980 to 0.04 ppm in 1988 (NFPA 1992). A 1982 Canadian study found average lead concentrations in dairy milk of 0.00112 $\mu\text{g/g}$ and lead levels in various infant formulas that ranged from 0.0026 $\mu\text{g/g}$ for bottled water to 0.0737 $\mu\text{g/g}$ in infant formula powders (Dabeka and McKenzie 1987). Additional exposure to lead through dietary intake by people living in an urban environment is estimated to be approximately 28 $\mu\text{g/day}$ for adults and 91 $\mu\text{g/day}$ for children, all of which can be attributed to atmospheric lead (dust). Atmospheric lead may be added to food crops in the field or garden (through uptake from soil and from direct deposition onto crop surfaces), during transport to market, processing, and kitchen preparation (EPA 1986a).

The U.S. Fish and Wildlife Service reported on the concentration of metals in a total of 315 composite samples of whole fish sampled from 109 stations nationwide from late 1994 to early 1985. For lead, the geometric mean, maximum, and 85th percentile concentrations ($\mu\text{g/g}$ wet weight) were 0.11, 4.88, and 0.22. The mean concentration of lead was significantly lower than in the 1980–1981 survey. Lead concentrations in fish have declined steadily from 1976 to 1984, suggesting that reductions of leaded gasoline and controls

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on mining and industrial discharges have reduced lead in the aquatic environment (Schmitt and Brumbaugh 1990).

In order to reduce lead exposure from consumption of lead-contaminated fish and shellfish, consumption advisories are issued by states recommending that individuals restrict their consumption of specific fish and shellfish species from certain waterbodies where lead concentrations in fish and shellfish tissues exceed the human health level of concern. This level of concern is set by individual state agencies and used to issue advisories recommending no consumption, or restricted consumption, of contaminated fish and shellfish from certain waterbody types (e.g., lakes and/or rivers). In 1995, the EPA Office of Water issued guidance to states on sampling and analysis procedures to use in assessing the health risks from consuming locally caught fish and shellfish. The risk assessment method proposed by EPA was specifically designed to assist states in developing fish consumption advisories for recreational and subsistence fishers (EPA 1995b). These two groups within the general population consume larger quantities of fish and shellfish than the general population and frequently fish the same waterbodies routinely. Because of this, these populations are at greater risk of exposure to lead and other chemical contaminants if the waters they fish are contaminated. In 1997, 10 advisories restricting the consumption of lead-contaminated fish and shellfish were in effect in 5 states (2 in Missouri, 4 in Ohio, 1 in Louisiana, 1 in Tennessee (rescinded), and 1 in Hawaii) and 1 territory (1 in American Samoa) (EPA 1998).

Elevated levels of lead in the blood of cattle grazing near a lead smelter have been reported, although no implications regarding lead in beef were made. The mean lead levels for the herd were highest near the smelter and decreased with distance. Ingestion of soil along with the forage was thought to be a large source of additional metal (Neuman and Dollhopf 1992). Evidence has also been shown for transfer of lead to milk and edible tissue in cattle poisoned by licking the remains of storage batteries burned and left in a pasture (Oskarsson et al. 1992). Levels of lead in muscle of acutely sick cows which were slaughtered ranged from 0.23 to 0.5 mg/kg (wet weight basis). Normal lead levels in bovine meat from Swedish farms are <0.005 mg/kg. For eight cows that were less exposed, levels of lead in milk taken 2 weeks after the exposure were 0.08 ± 0.04 mg/kg. The highest lead level found in the milk of eight cows studied for 18 weeks was 0.22 mg/kg. Lead in most milk samples decreased to values <0.03 mg/kg 6 weeks after exposure. Two affected cows delivered a calf at 35 and 38 weeks after the exposure. There was a high lead level in the blood of the cows at the time of delivery, which suggests mobilization of lead in connection with the latter stages of gestation and delivery. Lead levels in colostrum were increased as compared to

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mature milk samples taken 18 weeks after exposure. The concentration of lead in milk produced after delivery decreased rapidly with time and was almost down to the limit of detection in mature milk.

Many non-Western folk remedies used to treat diarrhea or other ailments may contain substantial amounts of lead. Examples of these include: Alarcon, Ghasard, Alkohol, Greta, Azarcon, Liga, Bali Goli, Pay-loo-ah, Coral, and Rueda. In addition, an adult case of lead poisoning was recently attributed to an Asian remedy for menstrual cramps known as Koo Sar. The pills contained lead at levels as high as 12 ppm (CDC 1998). The source of the lead was thought to be in the red dye used to color the pills.

Tamarindo jellied fruit candy from Mexico, and lozeena, a bright orange powder from Iraq used to color rice and meat, have been implicated in lead poisoning (CDC 1998). The lozeena, containing 7.8–8.9% lead, was purchased in Iraq and brought into the United States. Tamarindo candy and jam products, restricted from importation into the United States since 1993, were purchased by a woman visiting her family in Mexico. Although no product was available for testing, several commercial retail lots of tamarindo and tejocote jellied fruit candy were embargoed by the state of California in 1993 because of high lead levels. The fruit candies were packaged in stoneware or ceramic jars. The lead-based glazing applied to the jars appeared to have been the major source of the lead, although some of the fruits from plastic-lined jars also contained substantial amounts of lead.

Lead may leach from lead crystal decanters and glasses into the liquids they contain. Port wine that contained an initial concentration of 89 $\mu\text{g/L}$ lead was stored for 4 months in crystal decanters containing up to 32% lead oxide. At the end of 4 months lead concentrations in the port were 5,331, 3,061, and 2,162 $\mu\text{g/L}$ in decanters containing 32%, 32%, and 24% lead oxide, respectively. Lead was also found to elute from lead crystal wine glasses within minutes. Mean lead concentrations in wine contained in 12 glasses rose from 33 $\mu\text{g/L}$ initially to 68, 81, 92, and 99 $\mu\text{g/L}$ after 1, 2, 3, and 4 hours, respectively (Graziano and Blum 1991).

Lead is also present in tobacco at concentrations of approximately 2.5–12.2 $\mu\text{g/cigarette}$, of which approximately 2–6% may actually be inhaled by the smoker (WHO 1977).

Hair dyes and some cosmetics may contain lead compounds (Cohen and Roe 1991). Hair dyes formulated with lead acetate may have lead concentrations 3 to 10 times the allowable concentration in paint. Measured lead concentrations of 2,300 to 6,000 μg of lead per gram of product have been reported (Mielke

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et al. 1997b). Lead acetate is soluble in water and easily transferred to hands and other surfaces during and following application of a hair dye product. Measurements of 150–700 μg of lead on each hand following application have been reported (Mielke et al. 1997b). In addition to transfer of lead to the hand-to-mouth pathway of the person applying the product, lead is transferred to any other surface (comb, hair dryer, outside of product container, counter top, etc.) that comes into contact with the product. It is also on the hair it is applied to and the hands applying it. Objects coming into contact with hair dyed with a lead-containing product also become contaminated. A dry hand passed through dry hair dyed with a lead-containing product in cream form has been shown to pick up about 786 μg of lead. A dry hand passed through dry hair dyed using foam or liquid lead-containing hair dye products picked up less lead: 69 $\mu\text{g}/\text{hand}$ for foam products and 73 $\mu\text{g}/\text{hand}$ for liquid products (Mielke et al. 1997b).

Cases of lead poisoning have been related to less common sources of exposure. Illicit "moonshine" whiskey made in stills composed of lead-soldered parts (e.g., truck radiators) may contain high levels of lead. Detectable levels of lead with a maximum concentration of 5.3 mg/L were found in 7 of 12 samples of Georgia moonshine whiskey (Gerhardt et al. 1980). So-called recreational drug users who "sniff" leaded gasoline vapors are also at risk of reaction to organolead compounds as well as the hydrocarbon components of gasoline (Edminster and Bayer 1985). Use of lead ammunition may result in exposure to lead dust generated during gun or rifle discharge at levels up to 1,000 $\mu\text{g}/\text{m}^3$ (EPA 1985c), from lead pellets ingested or imbedded in animals that are used as food sources, and from lead pellets imbedded in humans from shooting incidents (Johnson and Mason 1984).

Lead poisoning has been caused by ingestion of a Chinese herbal medicine to which metallic lead was added to increase its weight and sales price (Wu et al. 1996). Lead contaminants also are present in some calcium supplements. Fourteen of 25 brands tested had lead ingestion rates greater than the provisional total tolerable daily intake of 6 μg . The highest found was 25.1 μg per day based on a calcium dosage of 1,000 mg, an amount commonly ingested by children (Bourgoin et al. 1993). A lead poisoning hazard for young children exists in imported vinyl miniblinds that have had lead added to stabilize the plastic. Over time, the plastic deteriorates to produce lead dust that can be ingested when the blinds are touched by children who then put their hands in their mouths (CPSC 1996). The U.S. Consumer Product Safety Commission (CPSC) has requested that manufacturers change the manufacturing process to eliminate the lead. As a consequence, vinyl miniblinds should now be lead-free. The CPSC recommends that consumers with young children remove old vinyl miniblinds from their homes and replace them with new miniblinds made without added lead or with alternative window coverings.

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5.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

Exposure of the general population to lead is most likely to occur through the ingestion of contaminated food and drinking water, and by the inhalation of lead particulates in ambient air. Direct inhalation of lead accounts for only a small part of the total human exposure; however, lead that is adsorbed to soil may be inhaled as dust and reentrainment of lead-contaminated dust is common. Fruits, vegetables, and grains may contain levels of lead in excess of background levels as a result of plant uptake of lead from soils and direct deposition of lead onto plant surfaces (EPA 1986a). Between 1979 and 1989, lead-soldered food cans were virtually eliminated as a source of lead contamination of canned food. The CDC has concluded that the most common source of lead exposure for children (Section 5.6) is lead-based paint that has deteriorated into paint chips and lead dusts and that the most common sources of lead exposure for adults are occupational (CDC 1997b).

Those who use recreational shooting ranges may be exposed to lead and soluble lead compounds, such as carbonates and sulfates, in soil. Surface lead concentrations at a range in Michigan were 10 to 100 times greater than background level of 25 mg/kg; mobilization of lead appeared to be occurring and may present a threat to ground and surface waters (Murray et al. 1997).

Exposure may also result from engaging in hobbies that use lead. For example, molten lead can be used in casting ammunition and making fishing weights or toy soldiers; leaded solder is used in making stained glass; leaded glazes and frits are used in making pottery; artists' paints may contain lead; lead compounds are used as coloring agents in glassblowing; and lead may be present in platinum printing and screen printing materials (Grabo 1997).

In 1982–1983, the baseline value for daily intake of lead by inhalation in a nonurban environment was estimated to be 0.5 $\mu\text{g}/\text{day}$ for a 2-year-old child, 1.0 $\mu\text{g}/\text{day}$ for an adult working indoors, and 2.0 $\mu\text{g}/\text{day}$ for adults working outdoors; these figures are based on an average atmospheric lead concentration of 0.1 $\mu\text{g}/\text{m}^3$ and an indoor/outdoor lead concentration ratio of 0.5. In an urban environment, the indoor/outdoor ratio was assumed to be approximately 0.8, giving a lead exposure estimate of 1.0 $\mu\text{g}/\text{m}^3$ for adults assuming a 2-hour/day exposure to an outside lead concentration of 0.75 $\mu\text{g}/\text{m}^3$, a 20-hour/day exposure to an indoor lead concentration of 0.6 $\mu\text{g}/\text{m}^3$, a 2-hour/day exposure to 5 $\mu\text{g}/\text{m}^3$ in high traffic, and an average daily intake of air by an adult of 20 m^3 . These estimates indicate that urban and nonurban residents inhaled approximately the same amount of lead dust (EPA 1986a). Drastic reductions in the lead content of

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gasoline since 1986 have resulted in a 64% decrease in lead emissions to the atmosphere (see Section 5.4.1).

In 1991 the composite average concentration of lead in air at EPA National Air Monitoring Systems sites was $0.053 \mu\text{g}/\text{m}^3$, the same as the “all sites” average. The average lead concentration at point-source oriented sites was $0.7 \mu\text{g}/\text{m}^3$; the average urban site concentration in 1991 was $0.1 \mu\text{g}/\text{m}^3$ (EPA 1992b). For 1991 data, if the indoor/outdoor ratio is again assumed to be 0.8 for urban atmospheres and the 2-hour exposure in high traffic of $5 \mu\text{g}/\text{m}^3$ is replaced by $0.1 \mu\text{g}/\text{m}^3$, then the average intake by an adult can be calculated as 20 hours at $0.08 \mu\text{g}/\text{m}^3$ and 4 hours at $0.1 \mu\text{g}/\text{m}^3$ for a weighted average intake of $0.083 \mu\text{g}/\text{m}^3$, or $2 \mu\text{g}/\text{day}$. This exposure is significantly lower than the $1.0 \mu\text{g}/\text{m}^3$ estimated to be inhaled in an urban setting in 1982–1983 and is comparable to what an adult breathed in a rural setting in 1982–1983.

Between 1979 and 1989 there was a virtual elimination of the use of lead-soldered food cans, with a concomitant drop in lead levels in food. Average daily intakes of lead for adults, based on an analysis of 27 market basket samples taken nationwide for a 1980–1982 Total Diet Study, were as follows (Gartrell et al. 1986b):

<u>Food group</u>	<u>Average adult intake ($\mu\text{g}/\text{day}$)</u>
Dairy products	4.54
Meat, fish, and poultry	4.09
Grain and cereal products	9.84
Potatoes	1.39
Leafy vegetables	0.94
Legume vegetables	9.18
Root vegetables	1.39
Garden fruits	4.44
Fruits	10.00
Oils and fats	1.23
Sugar and adjuncts	2.34
Beverages	6.86
Total lead intake	56.50

This value is only slightly higher than the estimated lead intake of $54 \mu\text{g}/\text{day}$ found in a Canadian 24-hour duplicate diet study conducted during 1981. The average lead content of the 10 food groups used in the Canadian study ranged from $0.088 \mu\text{g}/\text{g}$ for drinking water to $0.654 \mu\text{g}/\text{g}$ for cheese (Dabeka et al. 1987).

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Based on data from the FDA's Total Diet Food Studies, baseline values for average daily intake of lead by consumption of food, water, and beverages are presented in Table 5-3. The estimates of lead intake presented in Table 5-3 are based on measurements of lead in foods prepared for consumption and on consumption patterns for those foods (or food groups) from dietary surveys in which survey participant data were grouped by age and sex. The Total Diet Food Studies conducted between 1982 and 1988 determined daily intakes of a variety of pesticides, industrial chemicals, and elements for eight age and sex groups. In 1984, lead residues were found in 193 of the 201 foods analyzed. A comparison of daily intakes of lead by age group (6 months, 2 years, and adult) showed that lead intakes dropped by approximately 50% for each group between 1980 and 1984 (Gunderson 1988) and continued to decrease through 1990 for all age and sex groups (Bolger et al. 1991; FDA 1992b). Data from the 1990–1991 Total Diet Survey indicate that dietary lead intake now ranges from 1.8 to 4.2 $\mu\text{g}/\text{day}$ for all age groups combined, primarily as a result of reduced lead solder in cans and the phase-out of leaded gasoline. Further reductions in lead exposure will be more difficult to identify and achieve (Bolger et al. 1991, 1996).

Plastic food wrappers may be printed with pigments that contain lead chromates. Plastic wrappers used for 14 different national brands of bread collected in New Jersey contained a mean concentration of 26 mg of lead for a bag size of 2,000 cm^2 . A survey of 106 homemakers who buy such breads indicated that 39% of them reused the bags and 16% of the respondents turned the bags inside out to reuse them, suggesting that the potential exists for lead leaching from the paint into the stored food (Weisel et al. 1991).

Another source of dietary lead is the use of inadequately glazed or heavily worn earthenware vessels for food storage and cooking. Due to the number of incidences of lead poisoning that have resulted from the use of earthenware vessels, the FDA has established action levels of 0.5 $\mu\text{g}/\text{mL}$ lead for pitchers to 5.0 $\mu\text{g}/\text{mL}$ for cups and mugs soaked for 24 hours in a 4% acetic acid solution (FDA 1992a). However, inadequately glazed pottery manufactured in other countries continues to pose a significant health hazard. Likewise, homemade or craft pottery and porcelain-glazed vessels have been found to release large quantities of lead, particularly if the glaze is chipped, cracked, or improperly applied. In addition, glaze on vessels that are washed repeatedly may deteriorate, and a vessel that previously met FDA standards may become unsafe (CDC 1985; EPA 1986a).

Blood lead levels measured as a part of the National Health and Nutrition Examination Surveys (NHANES) revealed that between 1976 and 1991, the mean PbB levels of the U.S. population aged from 1 to 74 years dropped 78%, from 12.8 to 2.8 $\mu\text{g}/\text{dL}$. The prevalence of PbB levels $\geq 10 \mu\text{g}/\text{dL}$ also decreased sharply from 77.8% to 4.3%. The major cause of the observed decline in PbB levels is most likely

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Table 5-3. Daily Average Intake of Lead (μg lead/day)

Age	Sex	1980	1982	1984	1986	1988	1990
6–11 months	Male/female	≈34	20	16.7	10	5	3.8
2 years	Male	≈45	25.1	23.0	12.8	5.0	4.3
	Female	No data					
14–16 years	Female	No data	No data	28.7	15.2	6.1	6.1
14–16 years	Male	No data	No data	40.9	21.8	8.2	8.5
25–30 years	Female	No data	32.0	28.7	14.8	7.9	6.7
25–30 years	Male	84	45.2	40.9	21.2	10.0	8.5
60–65 years	Female	No data	No data	30.4	15.6	No data	2.2
60–65 years	Male	No data	No data	37.6	19.1	No data	8.1

Source: Derived from Bolger et al. 1991; FDA 1992b; Gunderson 1988

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the removal of 99.8% of lead from gasoline and the removal of lead from soldered cans (Pirkle et al. 1994). PbB levels were consistently higher for younger children than for older children, for older adults than for younger adults, for males than for females, for blacks than for whites, and for central-city residents than for non-central-city residents. PbB levels also correlated with low income, low educational attainment, and residence in the Northeast region of the United States. Data from Phase 2 of NHANES III (conducted during October 1991 to September 1994) indicate that PbB levels in the U.S. population aged ≥ 1 year continued to decrease and that PbB levels among children aged 1–5 years were more likely to be elevated among those who were poor, non-Hispanic black, living in large metropolitan areas, or living in older housing (CDC 1997b). During 1991–1994, the overall geometric mean PbB of the population aged ≥ 1 year was 2.3 $\mu\text{g}/\text{dL}$. Among those aged 1–5 years, approximately 4.4% had PbB levels of 10 $\mu\text{g}/\text{dL}$, representing an estimated 930,000 children with levels high enough to be of concern (CDC 1997b).

Information on occupational exposure to lead is obtained primarily from the National Occupational Exposure Survey (NOES) and industry surveys of workers. While occupational exposure is widespread, environmental monitoring data on levels of exposure in many occupations are not available. OSHA has established a permissible exposure limit (PEL) for lead of 50 $\mu\text{g}/\text{m}^3$ for workplace air (OSHA 1991). NIOSH has estimated that more than 1 million American workers were occupationally exposed to inorganic lead in more than 100 occupations (NIOSH 1977a, 1978a). According to NOES, conducted by NIOSH between 1980 and 1983, an estimated 25,169 employees were exposed to tetraethyl lead (not used in gasoline since December 31, 1995); approximately 57,000 employees were exposed to various lead oxides mostly in non-ferrous foundries, lead smelters, and battery plants; 3,902 employees were exposed to lead chloride; and 576,579 employees were exposed to some other form of lead in the workplace in 1980 (NIOSH 1990). Workers who operate and maintain solid waste incinerators are also exposed to air lead levels as high as 2,500 $\mu\text{g}/\text{m}^3$ (Malkin 1992).

Potentially high levels of lead may occur in the following industries: lead smelting and refining industries, battery manufacturing plants, steel welding or cutting operations, construction, rubber products and plastics industries, printing industries, firing ranges, radiator repair shops and other industries requiring flame soldering of lead solder, and gas stations (EPA 1986a; Feldman 1978; Goldman et al. 1987; NIOSH 1978a). In these work areas, the major routes of lead exposure are inhalation and ingestion of lead-bearing dusts and fumes. In the smelting and refining of lead, mean concentrations of lead in air can reach 4,470 $\mu\text{g}/\text{m}^3$; in the manufacture of storage batteries, mean airborne concentrations of lead from 50 to 5,400 $\mu\text{g}/\text{m}^3$ have been recorded; and in the breathing zone of welders of structural steel, an average lead concentration of 1,200 $\mu\text{g}/\text{m}^3$ has been found (Fu and Boffeta 1995). Evaluations by NIOSH from 1979 to 1990 in radiator repair shops found that 68% of the workers sampled had airborne lead exposures exceeding the OSHA

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standard of 0.05 mg/m³ (Tharr 1993). Also, past studies of PbB levels of 56 radiator shop mechanics in the Boston area revealed that 80% had PbB levels greater than 30 µg/dL and 16 had PbB levels exceeding 50 µg/dL (Tharr 1993).

Studies have been conducted to determine exposure of firearm instructors to lead at outdoor firing ranges when either nonjacketed (pure lead) or jacketed (copper-coated) bullets were used. Instructors are likely to have higher exposure than shooters because they spend more time at the range. In studies at an outdoor range in Virginia, the mean breathing zone lead level when nonjacketed bullets were fired was 67.1 µg/m³ for one instructor and 211.1 µg/m³ for another (Tripathi et al. 1991). When jacketed bullets were used, breathing zone levels decreased to 8.7 µg/m³ or less. PbB levels of the instructors did not exceed the OSHA return standard of 1.93 µmol/L (40 µg/dL) or removal standard of 2.4 µmol/L (50 µg/dL) in either case. When shooters fired conventional lead bullets, their mean exposures to airborne lead were 128 µg/m³ in the personal breathing zone and 68 µg/m³ in the general area. When totally copper-jacketed lead bullets were fired, the mean breathing zone and general area air sample concentrations were 9.53 and 5.80 µg/m³, respectively (Tripathi et al. 1990). At an outdoor uncovered range in Los Angeles, instructors who spent an average of 15 to 20 hours per week behind the firing line were found to be exposed to breathing zone lead concentrations of 460 and 510 µg/m³ measured as 3-hour, time-weighted averages. The PbB of one instructor reached 3.38 µmol/L (70 µg/dL). After reassignment to other duties, repeat testing indicated his PbB had dropped to 1.35 µmol/L (28 µg/dL) (Goldberg et al. 1991).

In 1991, NIOSH conducted a survey of the Federal Bureau of Investigations (FBI) Firearms Training Unit firing ranges and related facilities to determine occupational lead exposures among FBI and Drug Enforcement Agency (DEA) firing range personnel (NIOSH 1996). Sixty-one personal breathing-zone and 30 area samples for airborne lead were collected. Exposures ranged up to 51.7 µg/m³ (mean 12.4 µg/m³), 2.7 µg/m³ (mean 0.6 µg/m³), and 4.5 µg/m³ (mean 0.6 µg/m³) for range instructors, technicians, and gunsmiths, respectively. Exposure of custodians ranged from non-detectable to 220 µg/m³ during short-term cleaning of a large indoor range. Carpet dust sampling of dormitory rooms of students who practiced at the firing ranges revealed statistically significant ($p < 0.0005$) higher dust-lead concentrations when compared to non-student dormitories (dust-lead concentration range of 116 to 546 µg/g with a geometric mean of 214 µg/g in the student's rooms versus a dust-lead concentration range of 50 to 188 µg/g with a geometric mean of 65 µg/g for the non-student rooms). This suggested that the students were contaminating their living quarters with lead.

Field surveys of three radiator repair shops in the Cincinnati area revealed that local exhaust ventilation (LEV) systems are effective in controlling airborne lead levels. The highest concentration of airborne lead

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measured during a brief period of continuous soldering in a shop equipped with an LEV was only $7.1 \mu\text{g}/\text{m}^3$. In a shop where no LEV was used, the 13 personal samples averaged $209 \mu\text{g}/\text{m}^3$ with a maximum of $810 \mu\text{g}/\text{m}^3$ measured for a 56-minute sample worn while tearing down and resoldering a single radiator (Tharr 1993).

Airborne dusts settle onto food, water, clothing, and other objects, and may subsequently be transferred to the mouth. A more recent study suggests that lead, applied to the skin as lead acetate or lead nitrate, was rapidly absorbed through the skin and was detected in sweat, blood, and urine within 6 hours of application (Stauber et al. 1994). In this study, 4.4 mg of lead was applied to the skin under a covered wax/plastic patch on the forearms of human subjects; of the applied dose, 1.3 mg of lead was not recovered from skin washings. The amount that actually remained in (or on) the skin and the mass balance of the fate of this lead was not determined; it may have been dermally absorbed or eliminated from the skin by exfoliation of epidermal cells. Thus, while this study provides evidence for dermal absorption of lead, it did not quantify the fraction of applied dose that was absorbed. The quantitative significance of the dermal absorption pathway as a contributor to lead body burden remains uncertain.

In these occupational areas, good housekeeping and good ventilation have a significant impact on the extent of worker exposure. Workers who were (or are) involved in the production of gasoline additives, tetraethyl lead and tetramethyl lead (now banned from highway use in the United States) are exposed to both inorganic lead and alkyl lead. The major potential hazard to these workers appears to be from dermal exposure since alkyl leads may be absorbed through the skin (Bress and Bidanset 1991; EPA 1986a). Others who may be occupationally exposed to lead are artists and crafts persons who may be exposed to lead used in paints, ceramic glazes, and lead solder for sculpture and stained glass (Fischbein et al. 1992; Hart 1987) and welders where lead concentrations in the welding fumes generated by gas metal arc welding of carbon steel ranged from 1.0 to $17.6 \mu\text{g}/\text{m}^3$, well below the established PEL for the workplace (Larson et al. 1989). A study conducted at two lead battery factories in Taiwan revealed a high correlation between ambient air concentration of lead and PbB levels in workers; improvement of hygienic practices proved to be more effective at lowering PbB levels than reducing the ambient air lead concentration (Lai et al. 1997).

Lead exposure is frequently monitored by biological testing (e.g., determination of urinary lead levels, PbB levels, urinary coproporphyrin levels, or δ -aminolevulinic acid [ALA] levels) rather than monitoring the workplace environment for lead concentrations (EPA 1986a; NIOSH 1978a). A recent employer survey of California industries that use lead indicated that 229,434 employees were potentially exposed to lead in the workplace; of these workers, 59,142 (25%) had received routine biological monitoring (i.e., determination of PbB levels), and only 24,491 (10%) were in positions where environmental monitoring (workplace air

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lead levels) had ever been conducted. In addition, approximately 12% of the potentially exposed individuals were in the construction industry, which has only recently required air or blood monitoring (OSHA 1993; Rudolph et al. 1990).

Workers in an electronic components plant that makes ceramic-coated capacitors and resistors using leaded glass for the ceramic coating were found to be exposed to ambient lead levels ranging from 61 to 1,700 $\mu\text{g}/\text{m}^3$, and to have PbB levels ranging from 16 to 135 $\mu\text{g}/\text{dL}$. Approximately 30% of the workforce was found to be on medical leave as a result of their PbB levels exceeding 40 $\mu\text{g}/\text{dL}$. An analysis of PbB levels among family members of the exposed workers gave mean levels of 10.2 $\mu\text{g}/\text{dL}$ compared with 6.2 $\mu\text{g}/\text{dL}$ for families of nonexposed workers, indicating possible secondary occupational exposure from workers to their families (Kaye et al. 1987).

5.6 EXPOSURES OF CHILDREN

This section focuses on exposures from conception to maturity at 18 years in humans and briefly considers potential pre-conception exposure to germ cells. Differences from adults in susceptibility to hazardous substances are discussed in Section 2.6, Children's Susceptibility.

Children are not small adults. A child's exposure may differ from an adult's exposure in many ways. Children drink more fluids, eat more food, and breathe more air per kilogram of body weight, and have a larger skin surface in proportion to their body volume. A child's diet often differs from that of adults. The developing human's source of nutrition changes with age: from placental nourishment to breast milk or formula to the diet of older children who eat more of certain types of foods than adults. A child's behavior and lifestyle also influence exposure. Children crawl on the floor; they put things in their mouths; they may ingest inappropriate things such as dirt or paint chips; they spend more time outdoors. Children also are closer to the ground, and they do not have the judgement of adults in avoiding hazards (NRC 1993).

The American Academy of Pediatrics (AAP) (1998) has concluded that although monitoring data demonstrate a decline in the prevalence of PbB levels, lead remains a common, preventable, environmental health threat. The AAP supports the CDC guidelines endorsing universal screening in certain areas and targeted screening for children at high risk (CDC 1997c). Many children continue to be at risk for ingestion of lead-based paint and of soil and dust contaminated through the deterioration of lead-based paint and the residues from combustion of leaded gasoline. A 1974 study indicated that elevated PbB levels in children were most likely a result of ingesting lead-contaminated soil, and that the most likely source was lead-based paint rather than lead from automotive exhaust (Ter Haar and Aronow 1974). However, subsequent data

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have shown that children with the highest PbB levels live in areas with high traffic flow where lead particles in the air may fall directly to the soil or adhere to the outer surfaces of building and wash to the soil with rain (Mielke et al. 1989). Studies of children in Minnesota showed that PbB levels in children were correlated with soil lead levels, which were highest in inner-city areas; soil lead levels and blood lead levels were not correlated with the age of housing, although the presence of lead-based paint or lead abatement procedures may be of significance for individual children (Mielke et al. 1989). The CDC has concluded that the most common source of lead exposure for children is lead-based paint that has deteriorated into paint chips and lead dusts (CDC 1997b).

FDA estimated that in 1990, toddlers (2-year-olds) received 16% of their total lead exposure from food (5 µg/day), 1% from soil, 7% from water, and 75% from dust. EPA estimated that in 1990 lead intake from U.S. drinking water would be 11.9 µg/day for a 6-year-old child and 7.5 µg/day for an infant less than 1 year old (Cohen 1988b). A study of lead in the diet of Canadian infants found an average intake by children 0–1 years of age to be 16.5 µg/day when both food and water ingestion were considered (Dabeka and McKenzie 1988).

Lead intoxication has been observed in children, but rarely in adults, in residential settings (Sedman 1989). The geometric mean blood lead level (GM PbB) for children has dropped dramatically since the late 1970's. Results of the CDC NHANES II and NHANES III, Phases I and II, study of blood lead levels for children aged 1–5 are summarized below (CDC 1997b, 1997d).

	NHANES II	NHANES III Phase I	NHANES III Phase II
<u>Children Aged 1–5 Years</u>	<u>1976–1980</u>	<u>1988–1991</u>	<u>1991–1994</u>
GM PbB	15.0 µg/dL	3.6 µg/dL	2.7 µg/dL
PbB ≥ 10 µg/dL	88.2%	8.9%	4.4%

The NHANES II and NHANES III, Phase I, results showed that from 1976 to 1991, PbB levels were consistently higher for younger children than for older children (Pirkle et al. 1994). In general, PbB levels also correlated with low income, low educational attainment, and residence in the Northeast region of the United States. Data from Phase 2 of NHANES III (conducted during October 1991 to September 1994) indicate that PbB levels in the U.S. population aged 1 year continued to decrease and that PbB levels among children aged 1–5 years were more likely to be elevated among those who were poor, non-Hispanic black, living in large metropolitan areas, or living in older housing (with potential exposure to lead from

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lead-based paint) (CDC 1997b). During 1991–1994, the overall geometric mean PbB of the population aged 1 year was 2.3 $\mu\text{g}/\text{dL}$. Among those aged 1–5 years, approximately 4.4% had PbB levels $\geq 10 \mu\text{g}/\text{dL}$, representing an estimated 930,000 children in the general population with levels high enough to be of concern (CDC 1997b). In addition, 1.3% of children aged 1–5 years had PbB levels $\geq 15 \mu\text{g}/\text{dL}$, and 0.4% had PbB levels $\geq 20 \mu\text{g}/\text{dL}$. For the NHANES III, Phase II data, the GM PbB levels were higher for children aged 1–2 years (3.1 $\mu\text{g}/\text{dL}$) than for children aged 3–5 years (2.5 $\mu\text{g}/\text{dL}$). (CDC 1997b).

In 1982–1983, the baseline value for daily intake of lead by inhalation in a nonurban environment was estimated to be 0.5 $\mu\text{g}/\text{day}$ for a 2-year-old child. The baseline value was based on an average atmospheric lead concentration of 0.1 $\mu\text{g}/\text{m}^3$ and an indoor/outdoor lead concentration ratio of 0.5. In an urban environment, the indoor/outdoor ratio was assumed to be approximately 0.8 (EPA 1986a). Drastic reductions in the lead content of gasoline since 1986 have resulted in a 64% decrease in lead emissions to the atmosphere (see Section 5.4.1).

The lead content of dusts can be a significant source of exposure, especially for young children. Baseline estimates of potential human exposure to dusts, including intake due to normal hand-to-mouth activity, are 0.2 g/day for children 1–6 years old versus 0.1 g/day for adults when both indoor and outdoor ingestion of soil including dust is considered (EPA 1989c). For children who engage in pica behavior, the ingestion rate of soil can be as high as 5 g/day. Although ingestion of lead-containing paint may lead to elevated PbB levels in young children, the major source of moderately elevated PbB levels (30–80 $\mu\text{g}/\text{dL}$) in inner city children is mostly likely to be contaminated household dust and subsequent hand contamination and repetitive mouthing (Charney et al. 1980). Weathering of lead-based paint can contribute to the lead content of dust and soil. Lead levels of indoor dust and outdoor soil were found to be strongly predictive of PbB levels in over 200 urban and suburban infants followed from birth to 2 years of age; however, the PbB levels were not correlated with indoor air or tap water lead levels, nor the size of nearby roadways. Indoor dust lead levels and soil lead levels in the homes of children with high PbB levels ($>8.8 \mu\text{g}/\text{dL}$) were 72 $\mu\text{g}/\text{wipe}$ (window sill dust) and 1,011 $\mu\text{g}/\text{g}$, respectively; children with low PbB levels ($<3.7 \mu\text{g}/\text{dL}$) were exposed to 22 $\mu\text{g}/\text{wipe}$ and 380 $\mu\text{g}/\text{g}$, respectively. In addition, 79% of the homes of children with high PbB levels had been renovated, while only 56% of the homes of children with low PbB levels had been renovated, suggesting that renovating the interior of homes previously painted with leaded paint may increase, at least temporarily, a child's exposure to lead dust (Rabinowitz et al. 1985).

Lanphear et al. (1996a, 1996b, 1997, 1998b) studied factors affecting PbB levels in urban children and found the following independent predictors of children's PbB levels: dust lead loading in homes, African-American race/ethnicity, soil lead levels, ingestion of soil or dirt, lead content and condition of painted

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surfaces, and water lead levels (Lanphear et al. 1996a). Differences in housing conditions and exposures to lead-containing house dust appear to contribute to the racial differences in urban children's PbB levels. In addition, white children were more likely to put soil in their mouths (outdoor exposure) and suck their fingers, and African-American children were more likely to put their mouths on window sills (indoor exposure) and to use a bottle. Exterior lead exposures were more significant for white children, and interior lead exposures were more significant for African-American children (Lanphear et al. 1996b). Mouthing behaviors are an important mechanism of lead exposure among urban children (Lanphear et al. 1997). Community characteristics such as residence within a city, proportion African Americans, lower housing value, housing built before 1950, higher population density, higher rates of poverty, lower percent of high school graduates, and lower rates of owner-occupied housing have been used to identify children with elevated blood levels (Lanphear et al. 1998b). An analysis of children's PbB levels and multiple measures of lead concentrations in household dust, water, soil, and paint has been used to predict the effect of changing concentrations of lead in environmental media on children's PbB levels. An increase in dust lead loading from background to 200 $\mu\text{g}/\text{ft}^2$ was estimated to produce an increase of 23.3% in the percentage of children estimated to have a PbB level $>10 \mu\text{g}/\text{dL}$; an increase in water lead concentration from background to 15 $\mu\text{g}/\text{L}$ was estimated to produce an increase of 13.7% in the percentage of children estimated to have a PbB level $>10 \mu\text{g}/\text{dL}$; and an increase in soil lead concentration from background to 400 $\mu\text{g}/\text{g}$ was estimated to produce an increase of 11.6% in the percentage of children estimated to have a PbB level $>10 \mu\text{g}/\text{dL}$ (Lanphear et al. 1998a)

Outdoor lead dust was found to be a more potent contaminant of children's hands than indoor lead dust at day care centers in New Orleans; boys, in general, had higher hand lead levels than girls. The conclusions were based on lead analysis of hand wipe samples taken before and after children played outdoors at four different day care centers (a private inner-city site, a private outer-city site, a public inner-city site, and a public outer-city site). The private inner-city site had a severely contaminated outdoor play area with measured soil lead concentrations ranging from 287 to 1,878 mg/kg . The outdoor play area at the public inner-city site, where children exhibited the lowest hand lead measurements of any site in the study, had been completely paved over with concrete or rubberized asphalt and had well-maintained equipment (Viverette et al. 1996).

In addition to the ingestion of hand soil/dust through normal hand-to-mouth activity, some children engage in pica behavior (consumption of non-food items), which can put them at increased risk through ingestion of large amounts of soil contaminated with lead. It has been estimated that an average child may ingest between 20 and 50 mg of soil per day and that a pica child may ingest 5,000 mg or more of soil per day (LaGoy 1987; Mielke et al. 1989). If the soil contains 100 $\mu\text{g}/\text{g}$ of lead, an average child may be exposed to

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5 µg of lead per day from this source alone (Mielke et al. 1989), and a pica child may be exposed to more than 100 times that amount. At the EPA's *Soil Screening Guidance* concentration of 400 mg Pb/kg soil, a 13 kg child who consumes 5 g of soil during a pica episode would have a dose from soil of 0.2 mg Pb/kg of body weight, which is 10 times the nonlethal toxic dose (Calabrese et al. 1997b; Stuik 1974). Yard soil containing lead concentrations >500 mg/kg has been associated with a mean PbB of 10 µg/dL in children 6 to 71 months of age in a multi-site study (ATSDR 1995).

Fetuses are at even greater risk. As discussed in Section 2.8, lead can readily cross the placenta; therefore, exposure of women to lead during pregnancy results in uptake by the fetus. Furthermore, since the physiological stress of pregnancy may result in mobilization of lead from maternal bone, fetal uptake of lead can occur from a mother who was exposed to lead before pregnancy, even if no lead exposure occurs during pregnancy. Prenatal exposure may be related to postnatal mental retardation, impaired postnatal neurobehavioral development, and reduced birth weight and gestational age (EPA 1986a).

Maternal PbB levels during pregnancy were significantly higher for a group of 1,428 immigrant women (geometric mean 2.3 µg/dL) than for a group of 504 non-immigrant women (geometric mean 1.9 µg/dL) in a study conducted at a medical center in South Central Los Angeles, one of the most economically depressed regions in California. Immigrant PbB levels were strongly dependent on time elapsed since immigration to the United States, with PbB levels being highest in those women who had immigrated most recently. Elevated PbB levels in immigrant women were also associated with engagement in pica and with low dietary calcium during pregnancy (Rothenberg et al. 1999b).

Lead concentrations in maternal and umbilical cord blood have been reported by Greek researchers for 50 parturient women at delivery. Twenty-five of the women lived in industrial areas with high air pollution, and twenty-five lived in agricultural areas with low air pollution. The mean lead concentrations (expressed as mean ± standard deviation) for the women living in areas with high air pollution were 37.2±4.7 µg/L in maternal blood and 20±3.4 µg/L in umbilical cord blood (correlation coefficient, $r = 0.57$). The mean lead concentrations for the women living in areas with low air pollution were 20.5±5.6 µg/L in maternal blood and 12.9±3.6 µg/L in umbilical cord blood (correlation coefficient, $r = 0.70$). The authors conclude that the placenta demonstrates a dynamic protective function that is amplified when maternal PbB levels are raised (Vasilios et al. 1997).

Concentrations of lead in umbilical cord blood of two groups of women giving birth in a Boston Hospital in 1980 and 1990 have also been reported. Mean lead concentration of umbilical cord blood was 6.56±3.19 µg/dL for the 1980 group and 1.19±1.32 µg/dL for the 1990 group (Hu et al. 1996).

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In a study of blood samples collected from 113 mothers of 23 different nationalities and from their neonates (cord blood), mean maternal PbB levels were 14.9 ± 2.14 $\mu\text{g/dL}$ (range, 6.6–27.8 $\mu\text{g/dL}$) and mean cord PbB levels were 13 ± 2.5 $\mu\text{g/dL}$ (range, 6.0–30 $\mu\text{g/dL}$). Sixteen percent of mothers and nearly 10% of cord blood samples had PbB levels >20 $\mu\text{g/dL}$ (Al Khayat et al. 1997b).

Improper removal of lead from housing known to contain lead-based paint can significantly increase lead levels in dust, thus causing lead toxicity in children living in the home during the lead-removal process. Four such cases have been documented (Amitai et al. 1987). In January 1995, the New York State Department of Health identified 320 children in 258 households in New York State (excluding New York City) with PbB levels ≥ 20 $\mu\text{g/dL}$ that were considered to be attributable to residential renovation and remodeling (CDC 1997d). PbB levels in children have been found to increase during the summer months when children play outdoors and soil dust is more common. After interior and exterior lead dust cleanup procedures were instituted around areas known to have high soil lead levels, the PbB levels dropped in 52% of the children (Mielke et al. 1992). Authors of a study of PbB levels in children in Toronto, Canada, before and after abatement of lead-contaminated soil and house dust found they could neither strongly support nor refute beneficial effects of abatement. The failure to reach a definite conclusion from the results of the study, which included data from 12 cross-sectional blood-screening surveys that were conducted over an 8-year period, was due in part to a low response rate (32–75%) to questionnaires used to determine behavioral, household, lifestyle, neighborhood, and environmental factors relating to study participants (Langlois et al. 1996).

EPA conducted the Urban Soil Lead Abatement Demonstration Project (USLADP), also known as the “Three City Lead Study,” in Boston, Baltimore, and Cincinnati (EPA 1996i). The purpose was to determine whether abatement of lead in soil could reduce PbB levels of inner-city children. No significant evidence was found that soil abatement had any direct impact on children’s PbB levels in either the Baltimore or Cincinnati studies. In the Boston study, however, a mean soil lead reduction of 1,856 ppm resulted in a mean decline of 1.28 $\mu\text{g/dL}$ PbB at 11 months post-abatement (Weitzman et al. 1993). Phase II extended the study to 2 years and included soil abatement of the two comparison areas from Phase I (Aschengrau et al. 1994). Combined results from Phase I and II suggested a higher impact of soil remediation on PbB levels (2.2 to 2.7 $\mu\text{g/dL}$). EPA reanalyzed the data from the USLADP in an integrated report (EPA 1996i). They concluded that when soil is a significant source of lead in the child’s environment, under certain conditions, the abatement of that soil will result in a reduction in exposure and consequently, PbB level. Crump (1997) criticized the Boston data, including EPA’s integrated report, for poor selection of statistical methods, failure to adequately examine confounding variables, selective interpretation of results, and lack of control group in phase II of the study. Regardless, his reevaluation of the data, based on randomization

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analysis, resulted in a significant, yet modest effect of soil abatement (1.37 µg/dL) consistent with the conclusions of Weitzman et al. (1993) (1.28 µg/dL). Clearly, the results of the USLADP suggest that a number of factors are important in determining the influence of soil remediation on PbB levels in children. These include the site-specific exposure scenario, the magnitude of the remediation, and the magnitude of additional sources of lead exposure.

A study by Davis et al. (1992, 1994) used electron microprobe analysis of soil and waste rock from Butte, Montana, to help explain the low PbB levels observed in young children living in that mining community. They hypothesized that, if soils were ingested, the lead bioavailability would be constrained by alteration and encapsulation of the lead-bearing minerals of the Butte ore body (galena, anglesite, cerussite, and plumbojarosite), which would limit the available lead-bearing surface area. Kinetic limitations relative to the residence time of soil in the gastrointestinal tract also affect the bioavailability of lead (Ruby et al. 1992). The inherent chemical properties of soil-lead adsorption sites may reduce the bioavailability of soil-lead compared to soluble lead salts and lead compounds ingested without soil (Freeman et al. 1992). It has been shown that lead in impacted unleaded and leaded automobile exhaust particulate matter is readily leachable, but lead in paint may not be as leachable (Que Hee 1994). Thus, the differential availability may cause differential lead bioaccessibility and hence bioavailability. The extent of absorption of lead into the tissues of young Sprague-Dawley rats has been determined (Freeman et al. 1992). The animals were fed various concentrations of lead-contaminated mining waste soil mixed with a purified diet for 30 days. The overall percentage bioavailability values, based on lead acetate as the standard, were: 20% based on blood data; 9% based on bone data; and 8% based on liver data. These low bioavailabilities agree favorably with the low blood levels (average 3.5 µg/dL) found in children in Butte, Montana (Freeman et al. 1992). EPA (1989c) uses 0.2 g/day as a typical soil ingestion rate (including both dirt and dust) for children 1–6 years of age.

Trace metals, including lead, have been detected in human breast milk, so breast-feeding could deliver lead to an infant. Levels of lead in human milk vary considerably depending on the mother's exposure and occupation. For example, levels of lead in the milk of a mother who had worked in a battery factory for the first 6 months of pregnancy varied from 63 to 4 µg/L in samples taken soon after the birth of the child up to 32 weeks later. These concentrations were similar to those in control samples even though the PbB of the mother was about 3 times higher than that of the control subject. The pharmacokinetic model for lead may be complex since more than 90% of the lead body burden is stored in bone tissue and lead is strongly bound to hemoglobin, which may impede its partition to milk (Wolff 1983). On the other hand, an analysis of 210 human milk samples taken across Canada showed a mean lead level of 1.01 µg/L (1.04 ng/g; range, <0.05–15.8 ng/g). Women who resided in homes that were more than 30 years old, lived in high-traffic

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areas for more than 5 years, or had drunk 3 or more cups of coffee in the preceding 24 hours prior to taking the milk sample, had higher lead levels. The increased lead levels resulting from coffee drinking were thought to be the result of mobilization by the coffee of the lead stored in tissues and bone (Dabeka et al. 1988). In a paper by Abadin et al. (1997b), results of several additional studies of lead in human milk are summarized and discussed from a public health perspective. Among other citations, the median lead in milk concentrations from 41 volunteers in Sweden was 2 µg/L (Larsson et al. 1981); the mean value for urban residents of Germany in 1983 was 9.1 µg/L (Sternowsky and Wessolowski 1985); and the concentration in 3-day postpartum milk samples from 114 women in Malaysia averaged 47.8 µg/L (Ong et al. 1985).

Gulson et al. (1998) used measured lead isotope ratios ($^{207}\text{Pb}/^{206}\text{Pb}$ and $^{206}\text{Pb}/^{204}\text{Pb}$) in mothers' breast milk and in infants' blood to establish that, for the first 60-90 days postpartum, the contribution from breast milk to blood lead in the infants varied from 36% to 80%. Maternal bone and diet appear to be the major sources of lead in breast milk. Mean lead concentration (\pm standard deviation) in breast milk for participants in the study was 0.73 ± 0.70 µg/kg.

In a review of data on occupational chemicals that may contaminate breast milk (Byczkowski et al. 1994), it is stated that lead may be excreted in milk in amounts lethal to the infant and that the metal may be mobilized from bone stores to milk during the lactation period. Even when the concentration of lead in mother's milk is low, the absorption of metals into the systemic circulation of infants is generally high when they are on a milk diet. To better understand the sensitivity of the nursing infant to chemicals, epidemiological studies, chemical monitoring, and model development and application are needed.

Lead has also been reported in home-prepared reconstituted infant formula. Two of forty samples collected in a Boston-area study had lead concentrations >15 µg/L. In both cases, the reconstituted formula had been prepared using cold tap water run for 5 to 30 seconds, drawn from the plumbing of houses >20 years old. Three preparation practices for infant formula should be avoided: (1) excessive water boiling, (2) use of lead-containing vessels, and (3) morning (first-draw) water (Baum and Shannon 1997). Gulson et al. (1997a) measured lead in household water throughout the day when the plumbing system of an unoccupied test house was not flushed. Water concentration data ranged from 119 µg/L for the initial (first-draw) sample to 35–52 µg/L for hourly samples to 1.7 µg/L for a fully flushed sample. The water concentration data were used in the EPA's Integrated Exposure Uptake and BioKinetic (IEUBK) Model for Lead in Children to predict PbB levels in infants drinking water (or formula reconstituted using water) drawn from the same tap. Predicted PbB levels in infants only exceeded 10 µg/L when 100% of the water consumed contained 100 µg Pb/L (Gulson et al. 1997a).

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Lead-containing ceramic ware used in food preparation has also been associated with childhood lead exposure in children of Hispanic ethnicity in San Diego County, California. One study (Gersberg et al. 1997) used the IEUBK to determine that dietary lead exposure from beans prepared in Mexican ceramic bean pots may account for a major fraction of blood lead burden in children whose families use such ceramic ware.

Workers occupationally exposed to lead apparently carry lead home on clothing, bodies, or tools. PbB levels of children in households of occupationally exposed workers were almost twice those of children in neighboring homes whose parents were not occupationally exposed to lead (median ranges were 10–14 and 5–8 µg/dL, respectively) (Grandjean and Bach 1986). Young children (<6 years old) of workers exposed to high levels of lead in workplace air at an electronic components plant (61–1,700 µg lead/m³ ambient concentrations) had significantly elevated PbB levels (13.4 µg/dL) compared with children from the same locale whose parents did not work in the electronics plant (7.1 µg/dL) (Kaye et al. 1987). Exposures of lead workers' families have been identified in nearly 30 different industries and occupations. Industries in which exposure of family members has been reported most often include lead smelting, battery manufacturing and recycling, radiator repair, electrical components manufacturing, pottery and ceramics, and stained glass making (NIOSH 1995). Children of lead-exposed construction workers may also be at increased risk (Whelan et al. 1997).

Children may be exposed to lead because of activities associated with certain hobbies and artistic activities practiced by adults in the home. Some of the more obvious hobbies and activities involving use of lead-containing materials (casting, stained glass, pottery, painting, glassblowing, screenprinting) are discussed in Section 5.5. Activities involving use of lead-containing materials should always be done in an area well-ventilated with outdoor air and should never be done with children in the same room or in close proximity.

Children may be exposed to lead from other hobby or recreational activities that are not as obviously dangerous. For example, two case studies (one in North Carolina and one in Arizona) of lead poisoning in children from homes in which environmental surveys indicated no identifiable lead hazards have been reported. More extensive investigations revealed that both children had been observed on several occasions with pool cue chalk in their mouths. Subsequent chemical analysis of 23 different types of pool cue chalk identified three types as having lead concentrations in excess of 7,000 mg/kg (Miller et al. 1996). Accidental or intentional ingestion of folk remedies containing lead (discussed in Section 5.5) represents another source for potential lead-poisoning in children. Acute lead encephalopathy in early infancy has been reported in a Middle Eastern study for 14 infants following the use of *Bint al Thahab*, a traditional medicine containing 91% lead monoxide, and for 5 infants following application of lead-containing

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kohl/surma, a preparation used as eye makeup (Al Khayat et al. 1997a). Hair dyes formulated with lead acetate represent a potential source for lead-poisoning both by accidental ingestion and by hand-to-mouth activity following contact with lead-contaminated surfaces, including dyed hair of adults (Mielke et al. 1997b).

5.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

In addition to workers exposed to lead in the workplace, several other population groups at risk for potential exposure to high levels of lead can be identified: preschool-age children and fetuses (see Section 5.6), white males between 40 and 59 years of age (EPA 1986d), and those persons (“sniffers”) who purposely inhale leaded gasoline vapors. Individuals living near sites where lead was produced or sites where lead was disposed, and individuals living near one of the 1,026 NPL hazardous waste sites where lead has been detected in some environmental media (EPA 1986d; HazDat 1998) also may be at risk for exposure to high levels of lead.

General population exposure is most likely to occur through the ingestion of food and water that are contaminated with lead; however, some individuals and families may be exposed to additional sources of lead in their homes. This is particularly true of older homes that may contain lead-based paint. In an attempt to reduce the amount of exposure due to deteriorating leaded paint, the paint is commonly removed from homes by burning (gas torch or hot air gun), scraping, or sanding. These activities have been found to result, at least temporarily, in higher levels of exposure for families residing in these homes. In addition, those individuals involved in the paint removal process (i.e., do-it-yourself renovators and professionals who remove lead) can be exposed to such excessive levels that lead poisoning may occur (Chisolm 1986; Feldman 1978; Fischbein et al. 1981; Rabinowitz et al. 1985).

Special populations at risk of high exposure to tetraethyl lead include workers at hazardous waste sites and those involved in the manufacture and dispensing of tetraethyl lead (Bress and Bidanset 1991). Recreational drug “sniffers” of leaded gasoline are also at risk (Edminster and Bayer 1985).

Populations living near any of the 1,026 NPL sites that were identified as having lead present in the environmental media may be at risk for exposure to high levels of lead (HazDat 1998). However, the available data are insufficient to allow characterization of the sizes of these populations or intake levels of lead to which they may be exposed.

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5.8 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of lead is available. Where adequate information is not available, ATSDR, in conjunction with NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of lead.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce or eliminate the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

5.8.1 Identification of Data Needs

Physical and Chemical Properties. The physical and chemical properties of lead and its compounds are sufficiently well defined to allow an estimation of the environmental fate of lead to be made (Howe 1981; HSDB 1996; Lide 1996; Merck 1989; Sax 1984; Sax and Lewis 1987). Availabilities of the various forms need to be modeled and the connectivities to bioaccessibilities and bioavailabilities determined.

Production, Import/Export, Use, and Release and Disposal. Lead is produced and imported for widespread use in the United States. Therefore, the potential for human exposure in the workplace, the home, the environment, and at waste sites may be substantial.

Lead is produced from both primary (i.e., mined ore) and secondary (i.e., scrap metal and wastes) sources, and is imported by the United States. In 1997, production from primary and secondary sources was 343,000 metric tons and 1.1 million metric tons, respectively (Smith 1998), and imports reached 265,000 metric tons (Larrabee 1998; Smith 1998). Approximately 1.6 million metric tons of lead were consumed in the United States in 1997 (Smith 1998). Of lead used in 1997, 86.9% was used for storage batteries, 7.8% was used in metal products, and 5.3% was used in miscellaneous applications (Smith 1998). Because of the adverse health effects associated with exposure to lead, its use in paints, ceramic products, gasoline additives (now banned), and solder has declined dramatically in recent years. In 1997, exports of

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lead metal totaled 37,400 metric tons, and exports of lead waste and scraps totaled 88,400 metric tons (Larrabee 1998; Smith 1998).

Although certain uses of lead preclude recycling (e.g., use as a gasoline additive), lead has a higher recycling rate than any other metal (Larrabee 1998). An estimated 90–95% of the lead consumed in the United States is considered to be recyclable. In the United States, 77.1% of the lead requirements were satisfied by recycled lead products (mostly lead-acid batteries) in 1996. This compares to 69.5% in 1990 and 55.2% in 1980 (Larrabee 1997, 1998).

Industrial wastes, as well as consumer products, containing lead are disposed of in municipal and hazardous waste landfills. Current information on the amounts being disposed of is needed to evaluate the potential for exposure to lead.

The federal government regulates the release and disposal of lead. EPA has established national ambient air quality standards for lead. Under the Safe Drinking Water Act, EPA limits the level of lead in drinking water. Industrial emissions are regulated by the Clean Water Act. Lead and certain of its compounds are designated hazardous substances; CERCLA requires that the person in charge of a vessel or facility notify the National Response Center immediately when there is a release of a hazardous substance in an amount equal to or greater than the reportable quantity for that substance. Such data should be useful in determining potential for exposure and relating it to health effects.

According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit chemical release and off-site transfer information to the EPA. The Toxics Release Inventory (TRI), which contains this information for 1996 became available in May of 1998. This database will be updated yearly and should provide a list of industrial production facilities and emissions.

Environmental Fate. Lead released to the atmosphere partitions to surface water, soil, and sediment (EPA 1986a; Getz et al. 1977; Mundell et al. 1989; NAS 1980; Nielsen 1984; NSF 1977). Lead is transported in the atmosphere and in surface water. Organolead compounds are transformed in the atmosphere by photodegradation (DeJonghe and Adams 1986); however, the atmospheric transformation of inorganic lead compounds is not completely understood (EPA 1986a). Organolead compounds are transformed in surface waters by hydrolysis and photolysis (EPA 1979d). Inorganic lead compounds may be strongly sorbed to organic matter in soils and sediments (EPA 1986a). Lead is a naturally occurring element and is extremely persistent in the environment. Additional information on the atmospheric

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transformations of organic and inorganic lead compounds in the atmosphere would provide a basis for determining the lead compounds to which humans are most likely to be exposed. Modeling the availability of lead compounds needs to be done.

Bioavailability from Environmental Media. Available pharmacokinetic data indicate that lead is absorbed by humans following inhalation of particulate lead in ambient air and ingestion of contaminated foods, drinking water, and soil (Chamberlain et al. 1978; EPA 1986a; Morrow et al. 1980). In addition, children may ingest paint chips that contain lead. The bioavailability of lead from soil or dust on the hand after mouthing activity needs to be modeled. Absorption following dermal exposure is much more limited, although absorption of organolead compounds through the skin occurs (Kehoe and Thamann 1931; Laug and Kunze 1948; Moore et al. 1980).

Food Chain Bioaccumulation. Lead is bioaccumulated by terrestrial and aquatic plants and animals (Eisler 1988). However, lead is not biomagnified in terrestrial or aquatic food chains (Eisler 1988). No additional information is needed.

Exposure Levels in Environmental Media. Environmental monitoring data are available for lead in ambient air, indoor air, surface water, groundwater, drinking water, sediments, soils, and foodstuffs (Eckel and Jacob 1988; EPA 1982f, 1986a, 1988b, 1988f, 1989f, 1989h, 1990c; Lee et al. 1989; Maenhaut et al. 1979; Mielke 1992; Mielke et al. 1983, 1985, 1989); however, these data are not current and additional monitoring data on lead levels in all environmental media, particularly data gathered after EPA lowered and eventually banned the lead content of gasoline, would be helpful in determining current exposure levels. Estimates of human intake from inhalation of ambient air and ingestion of contaminated foods and drinking water are available (Dabeka et al. 1987; EPA 1986a, 1991d; Gartrell et al. 1986b; Gunderson 1988). Additional information on the concentrations of lead compounds in environmental media, particularly at hazardous waste sites, and an estimate of human intake would be helpful in establishing human exposure to lead. Absorption of lead through the skin may be a significant exposure pathway (Stauber et al. 1994) and may be deserving of further study.

Reliable monitoring data for the levels of lead in contaminated media at hazardous waste sites are needed so that the information obtained on levels of lead in the environment can be used in combination with the known body burdens of lead to assess the potential risk of adverse health effects in populations living in the vicinity of hazardous waste sites.

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Exposure Levels in Humans. Lead can be measured in human blood, hair, perspiration, teeth, bones, feces, and urine (Aguilera de Benzo et al. 1989; Batuman et al. 1989; Blakley and Archer 1982; Blakley et al. 1982; Christoffersson et al. 1986; Delves and Campbell 1988; Ellen and Van Loon 1990; Exon et al. 1979; Hu et al. 1989, 1990, 1991; Jason and Kellogg 1981; Manton and Cook 1984; NIOSH 1977a, 1977d, 1977e, 1977f, 1977g, 1977h; Que Hee and Boyle 1988; Que Hee et al. 1985a; Wielopolski et al. 1986). The most common method of assessing human exposure involves measurement of lead in blood (PbB) (Aguilera et al. 1989; Delves and Campbell 1988; Manton and Cook 1984; NIOSH 1977a, 1977d, 1977e, 1977f, 1977g, 1977h; Que Hee et al. 1985a). PbB levels have been correlated with ambient air exposure levels and dust, and dietary intake levels (Rabinowitz et al. 1985). Additional information on the biological monitoring of populations living in the vicinity of hazardous waste sites would be helpful in estimating exposure of these populations to lead compounds. The relationships between the major biological monitoring media should be determined. Alkyl lead compounds can be measured in exhaled breath and the diethyllead metabolite of tetraethyl lead can be measured in urine. This information is necessary for assessing the need to conduct health studies on these populations.

Exposures of Children. Estimates are available for intake by children through ingestion of contaminated soils, dust, paint chips (EPA 1989c), and breast milk (Wolff 1983). However, some of these estimates are not current or well understood. To better understand the sensitivity of the nursing infant to chemicals such as lead, epidemiological studies, chemical monitoring, and model development and application are needed (Byczkowski et al. 1994). The bioavailability of lead from soil or dust on the hand after mouthing activity needs to be modeled.

Exposure Registries. No exposure registries for lead were located. This substance is not currently one of the compounds for which a subregistry has been established in the National Exposure Registry. The substance will be considered in the future when chemical selection is made for subregistries to be established. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to exposure to this substance.

5.8.2 Ongoing Studies

Ongoing studies regarding potential for environmental exposure to lead were reported in the Federal Research in Progress File (FEDRIP 19968) database. Table 5-4 presents a summary of these studies.

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Table 5-4. Ongoing Studies on Potential Environmental Exposure to Lead

Investigator	Affiliation	Research description
K.M. Franken, W.L. Cornell	Bureau of Mines Rolla Research Center, Rolla, MO	Developing methods for treating lead-bearing wastes to render them non-hazardous
J.L. Gardea-Torresda	University of Texas at El Paso	Developing methods for recovery of toxic heavy metals from contaminated water supplies using plants
K.B. Kidd, F.E. Dierberg	Tpi, Inc., Albuquerque, NM; <i>Aqua Chem Analyses, Inc., Palm Bay, FL</i>	Investigating the use of plants for <i>remediation of wastes contaminated with lead</i>
R.L. Chaney	Beltsville Agricultural Research Center, Beltsville, MD	Evaluating the use of composts for reduction of the bioavailability and phytoavailability of lead and other heavy metals in contaminated soils
D. Heil, A. Hansen	New Mexico State University, Las Cruces, NM	Evaluating the use of solvent extraction as a technique for the remediation of soils contaminated with lead
R. Wang, A.N. Clarke	Wamax, Inc., Bellevue, WA; Eckenenfelder, Inc., Nashville, TN	Developing new lead fixation technologies
D. Leppert	Teague Mineral Products, Salem, OR	Evaluating the naturally occurring mineral clinoptilolite for removal of lead from drinking water
W.F. Bleam	University of Wisconsin, Madison, WI	Determining the soil conditions where lead binds to soils
J.H. Graziano	Columbia University, New York, NY	Conducting a study to determine the bioavailability of lead in soils for representative sites in the U.S.
J.J. Hassett	University of Illinois, Urbana, IL	Determining the relationships between soil lead concentrations and soil characteristics to develop management strategies for reducing bioavailability of lead in soil that would serve as alternatives to removal and disposal of lead-contaminated soils
J.W. Odor	Auburn University, Auburn, AL	Determining the occurrence, accumulation, and plant bioavailability of lead and other metals in acid ultisols.
L.M. Schuman	University of Georgia, Griffin, GA	Investigating the equilibrium of lead and other metals in soils and the effects on water quality

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Table 5-4. Ongoing Studies on Potential Environmental Exposure to Lead (continued)

Investigator	Affiliation	Research description
J.H. Peverly	Cornell University, Ithaca, NY	Characterizing trace element chemistry in soil and waste to predict plant uptake and movement of trace elements in soils
G.A. O'Connor, Q. Ma	University of Florida, Gainesville, FL	
G.M. Pierzynski, T.J. Logan	Kansas State University, Manhattan, KS; Ohio State University, Columbus, OH	Chemistry and bioavailability of waste constituents in soils
J.H. Peverly, J.L. Hutson	Cornell University, Ithaca, NY	Determining the fate and movement of nutrients and metals in representative plant/soil systems amended with sewage sludge, composts, and other wastes
C.D. Pepper	Boston University	Performing research to determine why high blood levels continue to occur in bridge construction workers despite government regulations and industry recommendations
H.J. Simpson	Mount Sinai School of Medicine	Conducting a study to identify previously unrecognized urban sources of environmental lead using dated environmental samples
P.J. Landrigan	Mount Sinai School of Medicine	Heading a collaborative, multidisciplinary, Superfund Hazardous Substances Basic Research Program to study the current urban sources, environmental distribution, and toxic effects on human health of lead
J.W. Gillett	Cornell University, Ithaca, NY	Conducting Superfund basic research on the bioavailability and impact of hazardous substances in human health and ecological risk as well as remediation of sites containing heavy metals such as lead
T.E. Ford	Harvard School of Public Health, Boston, MA	Evaluating the interrelationships between the microbial and metal pollutants in the New Bedford Harbor area, an EPA-designated Superfund Site
M.H. Conklin	University of Arizona, Tucson, AZ	Investigating the transport of trace metals, including lead, in a polluted aquifer in Pinal Creek, AZ, a state Superfund site

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Table 5-4. Ongoing Studies on Potential Environmental Exposure to Lead (continued)

Investigator	Affiliation	Research description
K. Belitz	Dartmouth College, Hanover, NH	Evaluating the importance of subsurface physical and chemical heterogeneity on the transport of metals, including lead, in geologically complex materials
A.J. Friedland	Dartmouth College, Hanover, NH	Identifying sources and determining mobility of lead in soil, groundwater, and vegetation

Source: FEDRIP 1998