6. POTENTIAL FOR HUMAN EXPOSURE

6.1 OVERVIEW

Lead has been identified in at least 1,272 of the 1,684 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2006). However, the number of sites evaluated for lead is not known. The frequency of these sites can be seen in Figure 6-1. Of these sites, 1,258 are located within the United States, 2 are located in Guam, 10 are located in the Commonwealth of Puerto Rico, and 2 are located in the Virgin Islands (not shown).

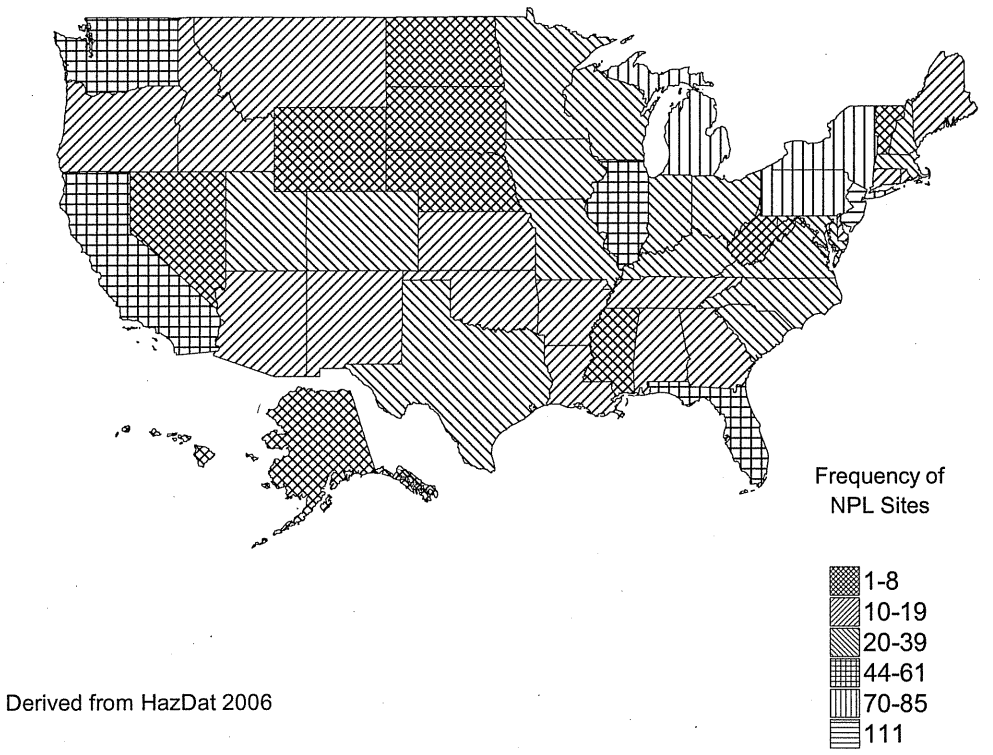
Lead is dispersed throughout the environment primarily as the result of anthropogenic activities. In the air, lead is in the form of particles and is removed by rain or gravitational settling. The solubility of lead compounds in water is a function of pH, hardness, salinity, and the presence of humic material. Solubility is highest in soft, acidic water. The sink for lead is the soil and sediment. Because it is strongly adsorbed to soil, it generally is retained in the upper layers of soil and does not leach appreciably into the subsoil and groundwater. Lead compounds may be transformed in the environment to other lead compounds; however, lead is an element and cannot be destroyed. Anthropogenic sources of lead include the mining and smelting of ore, manufacture of lead-containing products, combustion of coal and oil, and waste incineration. Many anthropogenic sources of lead, most notably leaded gasoline, lead-based paint, lead solder in food cans, lead-arsenate pesticides, and shot and sinkers, have been eliminated or strictly regulated due to lead's persistence and toxicity. Because lead does not degrade, these former uses leave their legacy as higher concentrations of lead in the environment.

Plants and animals may bioconcentrate lead, but lead is not biomagnified in the aquatic or terrestrial food chain.

The general population may be exposed to lead in ambient air, foods, drinking water, soil, and 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 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

Figure 6-1. Frequency of NPL Sites with Lead Contamination



exposures have occurred 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 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 gradually phased out the use of lead alkyls in gasoline, and by 1990, auto emissions accounted for only 33% of the annual lead emissions (EPA 1996b). Use of lead additives in motor fuels was totally banned after December 31, 1995 (EPA 1996a). 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 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.

6.2 RELEASES TO THE ENVIRONMENT

The Toxics Release Inventory (TRI) data should be used with caution because only certain types of facilities are required to report (EPA 2005i). This is not an exhaustive list. Manufacturers, processors, and users of lead and lead compounds are required to report information to the TRI only if they employ 10 or more full-time employees; if they operate in certain industrial sectors; and if their facility produces, imports, processes, or uses at least 100 pounds of lead, (exclusive of that contained in stainless steel, brass, or bronze alloys), or lead compounds in a calendar year. Prior to 2001, the threshold for reporting was much higher for persistent, bioaccumulative, toxic (PBT) chemicals such as lead. Facilities then had to report only when they manufactured, imported, or processed >25,000 pounds or used >10,000 pounds of lead or lead compounds in a calendar year. This higher threshold still applies to lead contained in

stainless steel, brass, or bronze alloys. The threshold for lead is determined using the weight of the metal, whereas the threshold for lead compounds is determined by the weight of the entire compound. Prior to 1998, only facilities classified within the SIC codes 20–39 (Manufacturing Industries) were required to report. After 1998, the industrial sector required to report was enlarged to include other industrial sectors, such as metal mining, coal mining, electrical utilities, and hazardous waste treatment (EPA 2001a).

While lead is a naturally-occurring chemical, it is rarely found in its elemental form. It occurs in the Earth's crust primarily as the mineral galena (PbS), and to a lesser extent as anglesite ($PbSO_4$) and cerussite ($PbCO_3$). Lead minerals are found in association with zinc, copper, and iron sulfides as well as gold, silver, bismuth, and antimony minerals. It also occurs as a trace element in coal, oil, and wood. Typical lead concentration in some ores and fuels are: copper ores, 11,000 ppm; lead and zinc ores, 24,000 ppm; gold ores, 6.60 ppm; bituminous coal, 3–111 ppm; crude oil, 0.31 ppm; No. 6 fuel oil, 1 ppm; and wood, 20 ppm (EPA 2001a).

Lead released from natural sources, such as volcanoes, windblown dust, and erosion, are minor compared with anthropogenic sources. Industrial sources of lead can result from the mining and smelting of lead ores, as well as other ores in which lead is a by-product or contaminant. In these processes, lead may be released to land, water, and air. Electrical utilities emit lead in flue gas from the burning of fuels, such as coal, in which lead is a contaminant. Because of the large quantities of fuel burned by these facilities, large amounts of lead can be released. For example, using the EPA emission factor for lignite coal, $4.2x10^{-4}$ pounds of lead/ton of coal, a boiler burning a million pounds of lignite coal will release 420 pounds of lead into the atmosphere (EPA 2001a). Many of the anthropogenic sources of lead have been eliminated or phased out because of lead's persistence, bioaccumulative nature, and toxicity. These include, most notably, lead in gasoline, lead-based paint, and lead-containing pesticides, and lead in ammunition and sinkers. Because lead does not degrade, these former uses leave their legacy as higher concentrations of lead in the environment.

According to the TRI, in 2004, a total of 12,112,037 pounds of lead were released to the environment from 4,347 reporting facilities (TRI04 2006). Another 4,767,316 pounds were transferred off-site. Table 6-1 lists amounts of lead released from these facilities grouped by state. In addition, a total of 405,285,570pounds of lead compounds were released to the environment from 4,294 reporting facilities and another 20,809,590 pounds were transferred off-site (TRI04 2006). Table 6-2 lists amounts of lead

Table 6-1. Releases to the Environment from Facilities that Produce, Process, or Use Lead[a]

	Reported amounts released in pounds per year[b]								
							Total release		
State[c]	RF[d]	Air[e]	Water[f]	UI[g]	Land[h]	Other[i]	On-site[j]	Off-site[k]	On- and off-site
AK	7	292	2	0	39,203	13	39,497	13	39,510
AL	115	11,377	625	39	1,857,435	904	1,800,604	69,778	1,870,382
AR	52	2,642	193	0	56,661	35,279	46,126	48,649	94,775
AZ	77	652	6,112	0	56,020	43,185	55,184	50,786	105,969
CA	290	2,208	1,669	359	35,634	47,985	31,094	56,761	87,855
CO	49	424	59	143	115,238	22,117	94,378	43,604	137,981
CT	69	286	36	0	138	11,454	311	11,603	11,914
DC	2	0	0	0	403	0	0	403	403
DE	3	3	1	0	0	0	4	0	4
FL	156	4,640	371	0	152,932	10,593	152,988	15,548	168,536
GA	123	3,786	150	0	78,219	37,659	76,673	43,140	119,813
GU	1	120	0	0	0	0	120	0	120
HI	10	784	1	1	125,691	1,681	126,477	1,682	128,159
IA	76	3,999	446	140	5,939	50,150	8,280	52,395	60,674
ID	20	580	29	0	4,397,661	831	4,398,246	855	4,399,101
IL	220	20,391	1,713	401	156,127	14,987	22,120	171,499	193,619
IN	191	6,973	1,411	176	784,642	825,635	19,525	1,599,312	1,618,837
KS	45	5,123	64	99	42,320	5,206	26,399	26,413	52,812
KY	85	13,914	377	1,250	84,887	101,249	91,819	109,859	201,678
LA	59	1,386	1,913	279	2,400	795	4,914	1,858	6,772
MA	103	722	34	0	3,200	12,226	2,188	13,994	16,182
MD	38	587	34	0	11,669	2,731	10,399	4,622	15,021
ME	18	516	159	0	1,386	15	1,363	714	2,077
MI	182	8,600	526	0	101,768	11,209	14,625	107,478	122,103
MN	97	629	519	0	3,058	800	630	4,376	5,006
MO	90	2,581	262	157	142,943	1,733	125,168	22,508	147,676
MS	57	1,986	393	27	44,196	449	43,659	3,392	47,050
MT	7	366	11	0	332,755	669	332,252	1,549	333,801
NC	141	2,197	563	0	223,107	8,242	212,004	22,104	234,108
ND	10	406	474	0	8,313	200	4,470	4,922	9,392
NE	54	6,788	70	0	5,155	5,535	7,692	9,855	17,547
NH	34	364	121	0	2,171	3,449	644	5,461	6,105
NJ	73	1,612	169	0	1,299	37,414	2,921	37,573	40,494
NM	17	90	0	0	32,544	3,413	32,634	3,413	36,047
NV	21	2,522	0	0	96,301	2,563	98,588	2,798	101,386
NY	169	4,296	7,241	1	26,877	38,864	18,677	58,602	77,279

Table 6-1. Releases to the Environment from Facilities that Produce, Process, or Use Lead[a]

		Reported amounts released in pounds per year[b]							
							Total release		
State[c]	RF[d]	Air[e]	Water[f]	UI[g]	Land[h]	Other[i]	On-site[j]	Off-site[k]	On- and off-site
OH	311	21,200	6,974	28,852	2,396,501	831,963	2,294,828	990,663	3,285,490
OK	59	6,250	25	162	84,903	712	55,964	36,088	92,052
OR	54	389	17	0	198,334	1,019	198,327	1,432	199,759
PA	229	18,426	2,534	0	44,891	336,150	49,233	352,768	402,001
PR	22	41	0	0	0	420	41	420	461
RI	25	58	46	0	0	2,176	70	2,209	2,280
SC	78	6,116	492	19,107	34,974	18,570	11,829	67,430	79,259
SD	17	105	26	0	11,103	1	11,208	28	11,236
TN	107	8,958	832	198	24,456	33,552	15,650	52,346	67,996
TX	232	19,573	2,036	133,133	339,613	39,359	453,503	80,211	533,714
UT	37	539	74	0	154,967	26,984	145,376	37,189	182,565
VA	91	6,437	1,152	0	385,614	26,809	32,753	387,259	420,012
VI	2	114	0	0	426	23	516	47	563
VT	6	14	18	0	0	2,658	14	2,676	2,691
WA	76	861	1,545	0	864,384	12,312	855,186	23,917	879,102
WI	178	10,688	1,015	0	79,703	32,974	11,181	113,200	124,380
WV	42	1,538	46	0	50,653	6,245	46,684	11,798	58,482
WY	10	66	0	0	27,052	0	27,003	115	27,118
Total	4,337	215,216	42,581	184,525	13,725,870	2,711,160	12,112,037	4,767,316	16,879,353

[a]The TRI data should be used with caution since only certain types of facilities are required to report. This is not an exhaustive list. Data are rounded to nearest whole number.
[b]Data in TRI are maximum amounts released by each facility.
[c]Post office state abbreviations are used.
[d]Number of reporting facilities.
[e]The sum of fugitive and point source releases are included in releases to air by a given facility.
[f]Surface water discharges, waste water treatment-(metals only), and publicly owned treatment works (POTWs) (metal and metal compounds).
[g]Class I wells, Class II-V wells, and underground injection.
[h]Resource Conservation and Recovery Act (RCRA) subtitle C landfills; other on-site landfills, land treatment, surface impoundments, other land disposal, other landfills.
[i]Storage only, solidification/stabilization (metals only), other off-site management, transfers to waste broker for disposal, unknown
[j]The sum of all releases of the chemical to air, land, water, and underground injection wells.
[k]Total amount of chemical transferred off-site, including to POTWs.

RF = reporting facilities; UI = underground injection

Source: TRI04 2006 (Data are from 2004)

Table 6-2. Releases to the Environment from Facilities that Produce, Process, or Use Lead Compounds[a]

		Reported amounts released in pounds per year[b]							
							Total release		
State[c]	RF[d]	Air[e]	Water[f]	UI[g]	Land[h]	Other[i]	On-site[j]	Off-site[k]	On- and off-site
AK	13	11,686	792	8,493,212	141,141,748	3	149,643,055	4,387	149,647,441
AL	108	30,437	10,300	1,001	1,709,923	786,593	1,450,722	1,087,531	2,538,253
AR	71	7,871	1,744	0	174,136	192,907	137,497	239,162	376,659
AZ	60	14,142	214	0	5,510,961	9,540	5,499,714	35,144	5,534,857
CA	276	9,530	1,462	123	4,989,235	124,884	3,760,943	1,364,291	5,125,234
CO	53	6,310	820	0	6,775,795	2,753	6,577,849	207,830	6,785,679
CT	72	1,842	50,808	0	55,894	46,837	2,552	152,828	155,380
DE	13	2,930	1,161	0	72,923	16,714	31,159	62,568	93,728
FA	110	23,635	2,624	0	505,968	21,557	444,097	109,687	553,784
FL	133	19,762	2,502	0	439,521	15,659	337,497	139,948	477,445
GU	3	4	1	0	4	0	10	0	10
HI	13	3,902	22	11	1,304	206	3,935	1,510	5,444
IA	62	19,705	1,919	3	250,083	104,373	54,916	321,167	376,084
ID	31	4,652	682	0	2,432,819	3,756	2,421,469	20,439	2,441,908
IL	223	27,715	6,780	1,139	2,252,474	166,469	1,752,530	702,047	2,454,577
IN	183	54,164	7,662	1,202	3,736,474	1,318,189	1,273,823	3,843,869	5,117,691
KS	54	11,010	309	0	104,267	83,899	100,975	98,510	199,485
KY	82	22,586	1,937	40	908,615	41,149	865,666	108,660	974,327
LA	76	16,143	26,110	0	1,104,894	3,061	948,072	202,136	1,150,209
MA	135	4,088	243	0	304,852	38,052	9,549	337,686	347,234
MD	38	4,202	1,873	9	261,495	56,339	235,657	88,260	323,918
ME	29	1,346	1,373	0	12,061	9,811	11,991	12,601	24,592
MI	136	23,248	9,161	80	711,353	94,813	296,369	542,284	838,653
MN	74	9,093	948	0	254,293	132,931	89,773	307,491	397,264
MO	111	181,782	10,114	0	28,889,783	3,098	27,669,492	1,415,285	29,084,777
MP	3	1	0	0	1	0	2	0	2
MS	59	14,205	1,624	254,800	112,794	3,928	330,719	56,634	387,352
MT	20	6,339	393	3,098	15,029,303	851	15,030,211	9,772	15,039,984
NC	154	19,778	2,287	13	452,288	365,461	422,797	417,030	839,828
ND	10	7,745	14	66	130,165	80	86,306	51,764	138,070
NE	28	4,918	135	0	52,093	32,663	52,489	37,321	89,810
NH	31	490	71	0	55,741	911	743	56,469	57,213
NJ	98	7,008	8,201	0	496,387	222,789	153,985	580,400	734,385
NM	22	1,252	881	0	596,749	17,894	583,007	33,768	616,776
NV	32	4,073	541	4	105,668,551	5,296	105,667,734	10,731	105,678,465
NY	129	16,840	9,737	0	744,058	168,013	687,725	250,922	938,648
OH	263	60,447	13,753	15,937	1,905,688	568,837	801,235	1,763,427	2,564,663
OK	47	54,622	363	401	290,227	73,571	338,813	80,372	419,184
OR	61	2,037	3,165	0	55,083	530	11,787	49,027	60,814

Table 6-2. Releases to the Environment from Facilities that Produce, Process, or Use Lead Compounds[a]

		Reported amounts released in pounds per year[b]							
							Total release		
State[c]	RF[d]	Air[e]	Water[f]	UI[g]	Land[h]	Other[i]	On-site[j]	Off-site[k]	On- and off-site
PA	246	69,995	6,769	987	3,479,169	1,229,509	1,714,804	3,071,625	4,786,428
PR	18	2,948	18	0	394	2,936	3,028	3,268	6,296
RI	24	38	67	1	3,701	805	44	4,568	4,612
SC	84	16,757	2,239	0	354,464	28,224	255,034	146,649	401,683
SD	17	1,936	762	0	1,446,132	386	1,446,330	2,884	1,449,215
TN	108	16,735	4,380	0	8,765,194	35,448	8,635,566	186,192	8,821,758
TX	236	45,373	5,779	1,089	2,492,627	54,544	2,015,842	583,571	2,599,413
UT	36	14,630	277	0	59,676,521	298,324	59,596,136	393,616	59,989,752
VA	98	14,524	4,397	558	381,233	35,396	267,225	168,883	436,108
VI	2	389	0	0	0	0	389	0	389
VT	10	25	50	0	2,712	7,136	41	9,882	9,923
WA	87	6,775	6,896	0	2,788,908	792,545	2,759,956	835,168	3,595,124
WI	136	11,702	2,165	0	389,469	56,300	110,108	349,527	459,636
WV	59	4,743	1,970	54	810,502	9,651	597,011	229,908	826,919
WY	17	5,336	20	0	112,415	309	97,189	20,891	118,080
Total	4,294	923,449	218,510	8,773,829	408,893,442	7,285,930	405,285,570	20,809,590	426,095,160

[a]The TRI data should be used with caution since only certain types of facilities are required to report. This is not an exhaustive list. Data are rounded to nearest whole number.
[b]Data in TRI are maximum amounts released by each facility.
[c]Post office state abbreviations are used.
[d]Number of reporting facilities.
[e]The sum of fugitive and point source releases are included in releases to air by a given facility.
[f]Surface water discharges, waste water treatment0(metals only), and publicly owned treatment works (POTWs) (metal and metal compounds).
[g]Class I wells, Class II0V wells, and underground injection.
[h]Resource Conservation and Recovery Act (RCRA) subtitle C landfills; other on0site landfills, land treatment, surface impoundments, other land disposal, other landfills.
[i]Storage only, solidification/stabilization (metals only), other off0site management, transfers to waste broker for disposal, unknown
[j]The sum of all releases of the chemical to air, land, water, and underground injection wells.
[k]Total amount of chemical transferred off0site, including to POTWs.

RF = reporting facilities; UI = underground injection

Source: TRI04 2006 (Data are from 2004)

compounds released from these facilities grouped by state. The TRI data should be used with caution because only certain types of facilities are required to report. This is not an exhaustive list.

Lead has been identified in a variety of environmental media (air, surface water, groundwater, soil, and sediment) collected at 1,272 of the 1,684 current and former NPL hazardous waste sites (HazDat 2006). Lead is the most frequently found metal at hazardous waste sites (Reed et al. 1995).

6.2.1 Air

According to the TRI, in 2004, a total of 215,216 pounds of lead were released to air from 4,337 reporting facilities (TRI04 2006). Table 6-1 lists amounts of lead released from these facilities grouped by state. In addition, a total of 923,449 pounds of lead compounds were released to air from 4,294 reporting facilities (TRI04 2006). Table 6-2 lists amounts of lead compounds released from these facilities grouped by state. Releases of lead and lead compounds to air constitute, respectively, 1.78 and 4 0.23% of all on-site releases. The TRI data should be used with caution because only certain types of facilities are required to report. This is not an exhaustive list.

Lead has been identified in air samples collected at 96 of the 1,272 NPL hazardous waste sites where it was detected in some environmental medium (HazDat 2006).

The emissions of lead and lead compounds to the atmosphere reported to TRI has declined from 2.8 million pounds in 1988 to about 1.1 million pounds in 2004 as new industries were added to TRI reporting requirements (TRI04 2006). In 2000, before the reporting thresholds were drastically reduced, air emissions were 1.5 million pounds. In the past, transportation, particularly automotive sources, were the major contributor to air emissions of lead. Today, industrial processes, especially metal processing, are the major sources of lead emissions to the atmosphere with the highest lead concentrations found around smelters and battery manufacturers (EPA 2003a). Based on emission estimates, EPA reports a 93% reduction in lead emissions to the atmosphere between 1982 and 2002 and a 5% reduction between 1993 and 2002. Air quality levels for lead, namely the maximum quarterly mean concentrations, declined 93% between 1983 and 2002 and 57% between 1993 and 2002. EPA estimated that 78% of emissions in 2001 were from industrial processes, 12% from transportation, and 10% from fuel combustion. It should be noted that aviation gasoline and racing fuels are not regulated for lead content and can use significant quantities of lead (EPA 2003a). Historical trends of lead emissions in the United States are provided in Table 6-3 (EPA 2007a).

Table 6-3. Historic Levels of Lead Emissions to the Atmosphere in the United States

Pounds of lead emitted annually								
1970	1975	1980	1985	1990	1995	2000	2005	2006
$4.4x10^8$	$3.2x10^8$	$1.5x10^8$	$4.6x10^7$	$1.0x10^7$	$8.0x10^6$	$4.0x10^6$	$6.0x10^6$	$4.0x10^6$

Source: EPA 2007a

EPA (2000) estimated lead emission between 1990 and 1993 from all sources, not just those covered by TRI, which is limited to certain industries. During this period, lead emissions were estimated to average 3,307 tons/year. The major contributors to these emissions were: metals processors (840 tons/year), chemical manufacturers (181 tons/year), other manufacturing operations (553 tons/year), waste disposal and recycling (270 tons/year), onroad (e.g., automobiles, trucks, buses, and motorcycles) mobile sources (418 tons/year), and nonroad (e.g., airplanes, boats, railway engines, lawnmowers, and off-road vehicles) mobile sources (778 tons/year).

A study that estimated the historical rate of atmospheric metal fluxes into Central Park Lake, New York City by analyzing sediment cores for levels of trace metals, indicated that lead fluxes were extremely high throughout the 20th century, reaching maximum values (>70 μg cm^{-2} year^{-1}) from the late 1930s to the early 1960. This occurred decades before the maximum emissions from the use of leaded gasoline (Chillrud et al. 1999). The trends closely resemble the history of solid waste incineration in the city. These results, and the widespread use of solid waste incineration during that time, suggest that this may have been the dominant source of lead in urban areas. The decline in the prevalence of small incinerators, increased recycling, and the decline in the use of lead in a variety of consumer and commercial products would indicate that atmospheric releases of lead from solid waste incineration is a much less important source of lead emissions today than it was in the past.

As indicated in Table 6-4, by 1988, transportation (i.e., automotive) emissions were no longer the dominant source of lead emitted to the atmosphere. When such emissions were prevalent, >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 (EPA 1985d). 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. In the early 1980s EPA allowed up to 0.05 g of lead in a gallon of unleaded gasoline (EPA 1982b).

In 1996, estimated mobile transportation source emissions of lead into air for the 48 contiguous states decreased from an average of 1,196 tons/year derived for 1990–1993 to 546.1 tons/year in 1996 (EPA 2000, 2001b). The estimates are based on data obtained from the 1996 National Toxics Inventory. The onroad estimate of 18.9 tons/year for 1996 was a dramatic decrease from the average estimate of

Table 6-4. National Lead Emission Estimates (in 103 Metric Tons/Year), 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

[a]The sums of categories may not equal the total because of rounding.

Source: derived from EPA 1991e

418 tons/year given for 1990–1993 (EPA 2000, 2001b). Likewise, nonroad emissions decreased from an average of 778 tons/year in 1990–1993 to 527.2 tons/year in 1996. These decreases were the result of the complete phase-out of leaded gasoline in 1996. Projected estimates of lead emissions in 2007 for onroad and nonroad sources were 22.0 and 585.2 tons/year, respectively. The major onroad lead emissions in 1996 were generated from light-duty gasoline vehicles (13.9 tons/year) and light-duty gasoline trucks (5.0 tons/year). The major generators of lead emissions in 1996 from nonroad sources were airports (526.1 tons/year).

Emissions of lead from electric utility steam generating plants totaled 71.37 tons/year in 1994 (EPA 1998b). The emissions varied depending on the fuel used in the electric generating facility; coal (62 tons/year), oil (8.9 tons/year), and natural gas (0.47 tons/year). It is projected that total lead emissions from electric steam generating plants will increase to 93.08 tons/year in 2010. This increase will be due to increased demand for electric power and an increased use of coal and natural gas as fuel sources to generate electricity. Lead emissions for coal, oil, and natural powered electric steam utilities are projected to be 87, 5.4, and 0.68 tons/year, respectively, in 2010.

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 by sanding or sandblasting can result in high localized concentrations of lead dust in both indoor and outdoor air.

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).

6.2.2 Water

According to the TRI, in 2004, a total of 42,581 pounds of lead were released to water from 4,337 reporting facilities (TRI04 2006). Table 6-1 lists amounts of lead released from these facilities

grouped by state. In addition, a total of 218,510 pounds of lead compounds were released to water from, 4,294 reporting facilities (TRI04 2006). Table 6-2 lists amounts of lead compounds released from these facilities grouped by state. Releases of lead and lead compounds to water constitute, respectively, 0.35 and 0.05 % of all on-site releases. The TRI data should be used with caution because only certain types of facilities are required to report. This is not an exhaustive list.

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).

Lead is released into surface water from lead shot and lead sinkers. A study of a shooting range in Southwestern Virginia found that the dissolved lead content of surface water ranged up to 473 ppb with the highest concentrations closest to the backstop (Craig et al. 1999). Upstream from the site the lead concentration was 0.5 ppb. In 1991, the U.S. Fish and Wildlife Service banned the use of lead shot when hunting waterfowl, such as geese or ducks, in order to avoid releasing lead directly to surface water.

Although aquatic releases of lead from industrial facilities are expected to be small with respect to emissions to land and air, 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. The age of a home or building and the type of plumbing installed will be a major factor regarding the levels of lead in drinking water (EPA 2005h). Lead-contaminated drinking water is most problematic in buildings and residences that are either very old or very new. It was not uncommon to use lead pipes for interior plumbing purposes at the start of the 20th century in the United States. Also, lead piping was often used for the service connections that join residences to public water supplies (this practice ended only recently in some localities). Plumbing installed before 1930 is most likely to contain lead pipes. In most new homes, copper pipes have replaced lead pipes and lead-free solder is used. However, lead-free means that solders and flux may not contain >0.2% lead, while pipes, pipe fittings, and well pumps may not contain >8% lead. New brass faucets and fittings can also leach lead, which is released directly into the water. Lead levels

decrease as the residence ages because as time passes, mineral deposits form a coating on the inside of the pipes, which insulates the water from the lead.

Lead has been identified in groundwater samples collected at 949 of the 1,272 NPL hazardous waste sites, and in surface water samples collected at 567 of the 1,272 NPL hazardous waste sites where it was detected in some environmental medium (HazDat 2006).

6.2.3 Soil

According to the TRI, in 2004, a total of 13,725,870 pounds of lead were released to the land, both on-site and off-site, by 4,337 reporting facilities (TRI04 2006). Table 6-1 lists amounts of lead released from these facilities grouped by state. In addition, a total of 408,893,442 pounds of lead compounds were released to land, both on-site and off-site, by 4,294 reporting facilities (TRI04 2006). Table 6-2 lists amounts of lead compounds released from these facilities grouped by state. In addition, 184,525 and 8,773,829 pounds of lead and lead compounds, respectively, were injected underground. Ninety-seven percent of lead compounds injected underground were by one facility, Kennecott Greens Creek Mining Co. in Juneau, Alaska. The TRI data should be used with caution because only certain types of facilities are required to report. This is not an exhaustive list.

While the majority of lead releases are to land, they constitute much lower exposure risks than releases to air and water. In 1997, before new industries were added to TRI, 95% of lead and lead compound releases to land reported to TRI were from the primary metals industrial sector, primarily metal smelters. In 2004, metal mining, coal mining, electrical utilities, and Resource Conservation and Recovery Act (RCRA)/solvent recoveries (hazardous waste facilities), as well as primary metals, are the industrial sectors contributing most heavily to releases to land. Many of these facilities with large releases, such as metal mines, are located in sparsely populated areas. Hazardous waste facilities are highly regulated. Most of the lead released to land becomes tightly bound and immobile.

Lead-containing material from home and commercial use may be sent to municipal landfills. It is important to note that land is the ultimate repository for lead, and lead released to air and water ultimately is deposited in soil or sediment. For example, lead released to the air from leaded gasoline or in stack gas from smelters and power plants will settle on soil, sediment, foliage, or other surfaces. The heaviest contamination occurs near the highway, in the case of leaded gasoline, or near the facility, in the case of a power plant or smelter.

Lead has been identified in soil samples collected at 901 of the 1,272 NPL hazardous waste sites, and in sediment samples collected at 605 of the 1,272 NPL hazardous waste sites where it was detected in some environmental medium (HazDat 2006).

6.2.4 Paint

Although the sale of residential lead-based paint was banned in the United States in 1978, flaking paint, paint chips, and weathered powdered paint, which are most commonly associated with deteriorated housing stock in urban areas, remain major sources of lead exposure for young children residing in these houses, particularly for children afflicted with pica (the compulsive, habitual consumption of nonfood items) (Bornschein et al. 1986; EPA 1986a). Lead concentrations of 1–5 mg/cm^2 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 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/cm^2. 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 paint in their interiors. By contrast, approximately 86% (±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 >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/cm^2; interior home surface dust, 0.04–

39 mg/m^2 and 72–16,200 µg/g; interior home dustfall, 0.0040–60 mg/m^2/30 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 dust in air (from sanding and sandblasting) and on exposed surfaces. A study was conducted in New Orleans where power sanding is a common practice during repainting old houses and median, 90^{th} percentile, and maximum lead concentrations in 31 study houses were 35, 126, and 257 mg/g, respectively (Mielke et al. 2001). Lead concentrations in dust and soil samples from one study of a house where the paint chips contained about 90 mg Pb/g were very high. If the house had been sanded down to bare wood, 7.4 kg of lead would have been released to the environment. 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 1988–1991, 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).

6.3 ENVIRONMENTAL FATE

6.3.1 Transport and Partitioning

In the atmosphere, non-organic compounds of lead exist primarily in the particulate form. The median particle distribution for lead emissions from smelters is 1.5 µm with 86% of the particle sizes under 10 µm (Corrin and Natusch 1977). The smallest lead-containing particulate matter (<1 µm) is associated with high-temperature combustion processes. 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 µm, 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 0.06–2.0 μm 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, $\mu g/m^3$) 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). While lead particles from automobile emissions are quite small (<0.1 μm in diameter), they may coagulate, resulting in larger particulates (Chamberlain et al. 1979). Lead has been found in sediment cores of lakes in Ontario and Quebec, Canada far from any point sources of lead releases, suggesting that long-range atmospheric transport was occurring (Evans and Rigler 1985). However, the results reported by Allen-Gil et al. (1997) do not support the contention of long-range transport of lead from smelters in the Arctic, based on lead concentrations in sediments obtained from Arctic lakes in the United States. In fact, data summarized by Berndtsson (1993) indicate that local sources dominate the deposition of lead; lead is primarily deposited <10 kilometers from emission sources.

The amount of soluble lead in surface waters depends upon the pH of the water and the dissolved salt content. Equilibrium calculations show that at pH >5.4, the total solubility of lead is approximately 30 μg/L in hard water and approximately 500 μg/L in soft water. Sulfate ions, if present in soft water, limit the lead concentration in solution through the formation of lead sulfate. Above pH 5.4, the lead carbonates, $PbCO_3$ and $Pb_2(OH)_2CO_3$, limit the amount of soluble lead. The carbonate concentration is in turn dependent upon the partial pressure of carbon dioxide, pH, and temperature (EPA 1986a).

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 (NSF 1977).

The fate of lead in soil is affected by the 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). Soil samples were extracted from the Powder River Basin in Wyoming to determine the relative distribution and speciation of lead and other metals in acidic environments (Reddy et al. 1995). At near neutral pH, organic carbon-lead complexes were the predominant species in the soil water extracts. At low pH, dissolved lead in ionic form (Pb^{2+}) and ion pairs (e.g., $PbSO_4$) were the predominant species. It was concluded that the mobility of lead will increase in environments having low pH due to the enhanced solubility of lead under acidic conditions. 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 through runoff to surface water or leaching to groundwater except under acidic conditions (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 leaded gasoline in the presence of organohalogen scavenger compounds, are converted to the less soluble lead sulfate either by reactions in the atmosphere or by reactions at the soil surface, thus limiting it's mobility in soil. 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 $\geq$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 occur in water entrapped in soil macro- and micropores (Reed et al. 1995). Lead complexes and precipitates in soil. In soil with 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 1982c). 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 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 (e.g., acid rain) (NSF 1977). Favorable conditions for leaching may be present in some soils near lead smelting and NPL sites. Tetraalkyl lead compounds, such as tetraethyl lead, are insoluble in water and would not be expected to leach in soil. However, they can be transported through a soil column when it is present in a migrating plume of gasoline (USAF 1995). In 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).

Plants and animals may bioconcentrate lead, but biomagnification is not expected. In general, the highest lead concentrations are found in aquatic and terrestrial organisms with habitats 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 and fishing (lead from spent shot or sinkers); 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 adsorption of lead to soil organic matter, the bioavailability increases as the pH and the organic matter content of the soil are reduced. Plants grown in lead-contaminated soils were shown to accumulate low levels of lead in the edible portions of the plant from adherence of dusts and translocation into the tissues (Finster et al. 2004). Thirty-two different types of fruits or vegetables were grown in urban gardens with soils containing high lead levels (27–4,580 mg/kg). Samples were harvested and washed with either water or detergents and analyzed for lead content. Only one fruiting vegetable among 52 samples contained lead levels greater than the detection limit of 10 μg/g

in the edible portion. However, 39% of the leafy vegetables and herbs had lead levels >10 μg/g in the edible shoot portion following washing of the vegetables with detergent and water (Finster et al. 2004).

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 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).

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 1,000 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 low 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 35–80 μg) correlated strongly with the concentration of lead in the soil (0–240 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.

Uptake of lead in animals may occur as a result of inhalation of contaminated ambient air or ingestion of contaminated plants. However, lead is not biomagnified in aquatic or terrestrial food chains. 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 fresh-water 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 (μg/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*), fresh-water algae (92,000 for

Senenastrum 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 up in 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 found to occur. 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.

6.3.2 Transformation and Degradation

6.3.2.1 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 automobile exhaust as lead halides (mostly PbBrCl) and as double salts with ammonium halides (e.g., $2PbBrCl \cdot NH_4Cl$, $Pb_3[PO_4]_2$, 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 was released, 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 elemental lead and lead-sulfur compounds, $PbSO_4$, $PbO \cdot PbSO_4$, and PbS (Corrin and Natusch 1977; EPA 1986a; Spear et al. 1998). The lead emitted from the combustion of waste oil was found to be in the form of $PbCl_2$, PbO, and elemental lead (Pb^0) (Nerin et al. 1999). 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).

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 25 °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 reactions with hydroxyl radicals during summer is approximately 5.7 hours, based on a rate constant of $6.8x10^{-11}$ cm^3/molecule - sec (Nielsen et al. 1991). The half-life for tetramethyl lead is about 65 hours based on a rate constant of $5.9x10^{-12}$ cm^3/molecule - sec. In the winter, both compounds have half-lives of up to several days since the concentration of atmospheric hydroxyl radicals is lower than in summer months (DeJonghe and Adams 1986). Trialkyl compounds occur almost entirely in the vapor phase and have life-times in air that are 3 times longer than for the corresponding tetraalkyl compounds (Hewitt and Harrison 1986, 1987). 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 would be the major process for removing these compounds from air. Dialkyl lead compounds would be removed from the air 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). Monitoring studies in England indicate that urban air advected to rural areas may contain up to 5% of 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).

6.3.2.2 Water

The chemistry of lead in aqueous solution is highly complex because this element can be found in multiple forms. Lead has a tendency to form compounds of low solubility with the major anions found in natural waters. The amount of lead dissolved in surface waters is dependent on the pH and the dissolved salt content of the water. The maximum solubility of lead in hard water is about 30 µg/L at pH>5.4 and the maximum solubility of lead in soft water is approximately 500 µg/L at pH>5.4 (EPA 1977). In the environment, the divalent form (Pb^{2+}) 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 pH<5.4, the formation of lead sulfate limits the concentration of soluble lead in water, while at pH>5.4, the formation of lead carbonates limits the amount of soluble lead (EPA 1979). 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 1979).

The speciation of lead was found to differ in fresh water and seawater. In fresh water, lead may partially exist as the divalent cation (Pb^{2+}) at pHs below 7.5, but complexes with dissolved carbonate to form insoluble $PbCO_3$ under alkaline conditions (Long and Angino 1977). Even small amounts of carbonate ions formed in the dissolution of atmospheric CO_2 are sufficient to keep lead concentrations in rivers at the 500 µg/L solubility limit (EPA 1979). Lead chloride and lead carbonate are the primary complexes formed in seawater (Long and Angino 1977). The speciation of lead in water is also dependent on the presence of other ligands in water. Lead is known to form strong complexes with humic acid and other organic matter (Denaix et al. 2001; Gao et al. 1999; Guibaud et al. 2003). Lead-organic matter complexes are stable to a pH of 3 with the affinity increasing with increasing pH, but decreasing with increased water hardness (EPA 1979). In seawater, there is the presence of lead complexed to Fe-Mn oxides, which is due to the content of these oxides in seawater (Elbaz-Poulichet et al. 1984). Sorption of lead to polar particulate matter in freshwater and estuarine environments is an important process for the removal of lead from these surface waters. The adsorption of lead to organic matter, clay and mineral surfaces, and coprecipitation and/or sorption by hydrous iron and manganese oxides increases with increasing pH (EPA 1979).

In water, tetraalkyl lead compounds, such as tetraethyl lead and tetramethyl lead, are subject to photolysis and volatilization. Degradation proceeds from trialkyl species to dialkyl species, and eventually to inorganic lead oxides. Removal of tetraalkyl lead compounds from seawater occurs at rates that provide half-lives measurable in days (DeJonghe and Adams 1986). Some of the degradation products include trialkyl lead carbonates, hydroxides, and halides. These products are more persistent than the original tetraalkyl lead compounds.

6.3.2.3 Sediment and Soil

Lead in its naturally-occurring mineral forms is a very minor component of many soils in the United States. Additional sources of lead are incorporated to soils from atmospheric wet and dry deposition. Since the ban on leaded gasoline, the major source of lead emissions to the environment arise from industrial processes (EPA 1996b). 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 $PbSO_4$, $PbO{\cdot}PbSO_4$, and PbS (EPA 1986a). In the atmosphere, lead most likely exists primarily as $PbSO_4$ and $PbCO_3$ and is deposited onto soil as lead sulfates and lead carbonates. Organic tetraalkyl lead compounds, once used extensively in motor fuel, are emitted from automobiles primarily in the form of lead bromochloride, which is ultimately transformed to lead sulfate. The organolead

compounds also 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, resulting in a complex variety of lead compounds. The speciation of lead in soils is dependent upon the properties of the soil. In a calcareous soil, PbSO4 and PbCO3 were shown to account for <5% of the total lead content, whereas in road side dust, $PbSO_4$, elemental lead, Pb_3O_4, $PbO{\cdot}PbSO_4$, and $2PbCO_3{\cdot}Pb(OH)_2$ were present in significant quantities (Chaney et al. 1988). It was also reported that after adding 3,000–4,000 mg/kg of lead in the form of PbSO4, subsequent extractions revealed that the lead sulfate was rapidly transformed to other lead compounds in the soil (Chaney et al. 1988).

Nearly all forms of lead that are released to soil from anthropogenic sources, such as $PbSO_4$, $PbCO_3$, PbS, $Pb(OH)_2$, $PbCrO_4$, and PbClBr, are transformed by chemical and biotic processes to adsorbed forms in soil (Chaney et al. 1988). The transformation process involves the formation of lead complexes with binding sites on clay minerals, humic acid and other organic matter, and hydrous iron oxides (Chaney et al. 1988; Chuan et al. 1996; Sauve et al. 1997). The ability of soils to bind lead is dependent on soil pH and the cation exchange capacity of the soil components (e.g., hydrous iron oxides on clay and organic matter) (Chaney et al. 1988; EPA 1986a). Only a small fraction (0.1–1%) of lead appears to remain water-soluble in soil (Khan and Frankland 1983). The solubility of lead in soil is dependent on pH, being sparingly soluble at pH 8 and becoming more soluble as the pH approaches 5 (Chuan et al. 1996). Between pH 5 and 3.3, large increases in lead solubility in soil are observed. These changes in lead solubility appear to correlate with the pH-dependent adsorption and dissolution of Fe-Mn oxyhydroxides. In addition to pH, other factors that influence lead solubility in soil are total lead content and the concentrations of phosphate and carbonate in soils (Bradley and Cox 1988; Ge et al. 2000; Pardo et al. 1990; Sauve et al. 1997).

Since the ban on the use of leaded gasoline, atmospheric lead deposition to soil has decreased considerably. However, the deposited organolead compounds and their transformation products remain in the soil. Limited data indicate that tetraethyl and tetramethyl lead are converted into water-soluble lead compounds in soil through microbial metabolism (Ou et al. 1994). Using an Arredondo fine sand from Florida (92% sand, 7% silt, 1% clay, 11.8 g/kg organic carbon, pH 5.5), tetraethyl lead was shown to degrade sequentially to monoionic triethyl lead, diionic diethyl lead, and eventually Pb^{+2} (Ou et al. 1994). Experiments were conducted using non sterilized and autoclaved soil samples. The presence of monoionic triethyl lead and diionic diethyl lead was generally lower in the autoclaved samples, suggesting that both abiotic and biotic mechanisms are responsible for the degradation of tetraethyl lead. At the end of a 28-day incubation period, no tetraethyl lead was present in the soil; however, there were

significant quantities of monoionic triethyl lead and diionic diethyl lead, which suggest that the degradation products are more persistent than the original species. Although tetraethyl and tetramethyl lead are not expected to leach significantly through soil, their more water-soluble metabolites may be subject to leaching (EPA 1985a).

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 μg/g in soils of the northern hardwood forests of the study area and about 200 μg/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). However, Wang et al. (1995) observed that lead migration in forest soils is slowed considerably due to a decrease in solubility when lead moves from the soil surface horizon to streams. Their results suggest that lead is effectively trapped in the subsurface soil horizons, which may greatly reduce its release to streams.

Lead content in plants is largely the result of atmospheric deposition. This is due to the strong retention of particulate matter on plant surfaces that is difficult to remove through washing (EPA 1977). Some plants are capable of taking up lead from soil through their root systems, although this uptake does not appear to be appreciable (IARC 1980; Nwosu et al. 1995). The distribution of lead in plants is mainly in the roots and much less in the stems or leaves (Deng et al. 2004; Nan and Cheng 2001). Eventually, the lead will be returned to soil when these plants decay unless they are harvested (to possibly enter the food chain) or removed (EPA 1986a).

6.4 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. Concentrations of lead in unpolluted atmospheres and in pristine surface waters are often so low as to be near the limits of current analytical methods. In reviewing data on lead 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. The analytical methods available for monitoring lead in a variety of environmental media are detailed in Chapter 7.

6.4.1 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. Lead levels in ambient air range from $7.6x10^{-5}$ μg/m^3 in remote areas such as Antarctica (Maenhaut et al. 1979) to >10 μg/m^3 near stationary sources such as smelters. Due to decreases in lead emissions to the atmosphere from automobiles, the level of lead in air has declined significantly over the past 3 decades. Monitoring data from a composite of 147 sampling sites throughout the United States indicated that the maximum quarterly average lead levels in urban air were 0.36 μg/m^3 during 1984 and 0.2–0.4 μg/m^3 during 1986 (EPA 1986a, 1989e). Between 1979 and 1983, lead concentrations in precipitation in Minnesota decreased from 29 to 4.3 μg/L at urban locations and from 5.7 to 1.5 μg/L at rural locations, indicating a reduction in lead emissions of >80%. This reduction resulted primarily from the decreased use of leaded gasoline and the use of more efficient emission controls on stationary lead 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 1–2 ng/m^3 (0.001–0.002 μg/m^3), which is approximately 18% of the 1982 mean. The elevated lead concentrations (up to 5 ng/m^3) since 1986 at 3 of the 12 sites are thought to be 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 μg/m^3, well below the National Ambient Air Quality Standard of 1.5 μg/m^3, quarterly average concentration, that has been established for lead (EPA 1996b). Data from the EPA National Air Quality Monitoring Program indicated that the 2002 average air quality concentration for lead is about 94% lower than in the early 1980s, with a mean atmospheric concentration below 0.05 μg/m^3 in 2002 (EPA 2005k). In 1988, the

average concentration of 18-point-source sites was 0.4 μg/m^3, down from 2.9 μg/m^3 in 1979, and the average concentration for urban sites was 0.1 μg/m^3, down from 0.8 μg/m^3 in 1979 (EPA 1990). This decrease was undoubtedly caused by decreased use of leaded gasoline in the period leading up to its total ban after December 1995. Composite urban air concentrations of lead for 1989 and 1991 were 0.11 and 0.08 μg/m^3 (EPA 1996b). Although lead concentration in urban air continues 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 μg/m^3 even though lead emissions declined 1% (EPA 1996b).

Concentrations of lead in ambient air that result from emission, both mobile and stationary, have been estimated to average 0.0058 μg/m^3 in 1996, while the concentration of lead attributed to mobile sources alone was 0.0035 μg/m^3 (EPA 2001a).

Lead concentrations in air and dust in the indoor environment were measured in residential homes as part of the National Human Exposure Assessment Survey (NHEXAS) in EPA Region V (Indiana, Illinois, Michigan, Minnesota, Ohio, and Wisconsin). Mean (±1 standard deviation [SD]) and median concentrations of lead in indoor air from 213 residences were 15.2 ng/m^3 (37.6 ng/m^3) and 6.17 ng/m^3, respectively, with a maximum value of 293.5 ng/m^3 (Bonanno et al. 2001). The median lead concentration in outdoor air was 8.84 ng/m^3 (Clayton et al. 2002). Lead concentrations were higher in households where one or more residents smoked indoors (mean concentration of 21.8 ng/m^3) as compared to households with nonsmoking residents (mean concentration of 7.79 ng/m^3) (Bonanno et al. 2001). In dust collected from the living areas of 238 residences, the mean (±1 SD) and median lead concentrations were 467.4 μg/g (2,100 μg/g) and 131.6 μg/g, respectively, with a maximum value of 30,578 μg/g. Dust samples collected from window sills had mean (±1 SD) and median lead concentrations of 987 μg/g (2,723 μg/g) and 207.5 μg/g, respectively, with a maximum value of 21,120 μg/g. For both indoor air and dust measurements, higher concentrations of lead were correlated with dilapidated and suburban homes.

In another analysis of the NHEXAS EPA Region V data, Pellizzari et al. (1999) looked at potential differences in lead concentrations in indoor air and personal air exposures between minorities (e.g., Hispanics and African-Americans) and nonminorities (e.g., Caucasian). Some differences were noted in the mean (±1 SD) lead concentrations between minorities of 57 ng/m^3 (±24 ng/m^3) and nonminorities of 22 ng/m^3 (±3.4 ng/m^3) in personal air exposures, although the differences were not significant (p=0.147). Similarly, differences were noted between minorities (26±12 ng/m^3) and nonminorities (13±2.6 ng/m^3) in indoor air, although these too were not significantly different (p=0.266). When the age of the home was

considered in the analysis, it was found that lead concentrations were significantly (p=0.036) higher in homes built before 1940 than in homes built between 1960 and 1979, with mean (±1 SD) values of 46 ng/m^3 (±1.6 ng/m^3) and 13 ng/m^3 (±2.1 ng/m^3), respectively. The lead concentrations measured in indoor air in homes built before 1940 were not significantly different from mean (±1 SD) lead concentrations of 22 ng/m^3 (±5.1 ng/m^3) and 23 ng/m^3 (±5.1 ng/m^3) measured in indoor air in homes built between 1940 and 1959 and between 1980 and 1995, respectively.

6.4.2 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 µg/L, although levels as high as 890 µg/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 µg/L (based on 39,490 occurrences) (Eckel and Jacob 1988). The median lead level in natural river water is 5 µg/L, with a range of 0.6–120 µg/L (Bowen 1966). Lead levels in seawater are estimated as 0.005 µg/L (EPA 1982c). Lead concentrations in surface water are higher in urban areas than in rural areas (EPA 1982c). Using the EPA Storage and Retrieval (STORET) database, from January 1, 2005 to May 16, 2005, lead had been detected in surface water in Washington, Utah at concentrations of 20.5 and 142 µg/L and surface water from Salt Lake City, Utah at 7.75 µg/L (EPA 2005j). Lead was not detected above the detection limits in 224 other surface water samples obtained from various locations in Utah and Iowa over the sampling period (EPA 2005j).

Urban storm water runoff is an important source of lead entering receiving waterways. Lead is found in building material (brick, concrete, painted and unpainted wood, roofing, and vinyl), and automotive sources (brakes, used oil), which contribute to runoff (Davis et al. 2001). The largest contributing sources were siding and roofing.

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 µg/L and approximately 2 million people are served by drinking water with levels of lead >5 µg/L. A survey of 580 cities in 47 states indicated that the national mean concentration of lead in drinking water was 29 µg/L after a 30-second flushing period (EPA 1986a, 1989e); however, it was

estimated that in 1988 the average lead content of drinking water decreased to 17 µg/L (Cohen 1988). In 1986, the Safe Drinking Water Act Amendments banned the use of lead solder or flux containing >0.2% lead and the use of lead pipes or fittings that contained >8% lead (EPA 1986a, 1989e).

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 µg/L. EPA now estimates that approximately 600 groundwater systems may have water leaving the treatment plant with lead levels above 5 µg/L. Based on several data sets, it is estimated that <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 <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 1989d).

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). In the NHEXAS study that was conducted during 1995–1996, lead concentrations were measured in tap drinking water (flushed for 15 minutes) taken from 82 homes in Arizona (O'Rourke et al. 1999), 441–444 homes in EPA Region V (Thomas et al. 1999), and 381 homes in Maryland (Ryan et al. 2000). Median lead concentrations of 0.4, 0.37, and 0.33 µg/L were determined in the Arizona, EPA Region V, and Maryland regional studies, respectively. Mean values (±1 SD) of 0.84 µg/L (±1.8 µg/L) and 1.08 µg/L (±2.01 µg/L) were calculated for the EPA Region V and Maryland studies, respectively, and are much lower than the mean concentrations of lead in drinking water determined in previous EPA estimates.

According to EPA's National Compliance Report for calendar year 1998 (EPA 1999), 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 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 >10% of tap water samples.

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. Lead concentrations in tap water were found to vary depending on the age of nine homes in New Jersey. In homes built in the 1980s, median lead concentrations in the first-draw sample were higher (17.9 µg/L) than in first-draw samples (1.86 µg/L) taken from homes built in the 1970s (Murphy and Hall 2000). Leaching of lead from kitchen plumbing fixtures was given as the reason for the high lead concentrations in the first-draw samples. An additional water draw (>2 L) found decreased lead concentrations in tap water for all homes. However, the median concentration of lead in samples taken from homes built in the 1980s was higher (2.45 µg/L) than in samples taken from 1970s homes (0.14 µg/L). The lead concentrations in these higher volume samples are attributed to lead leaching from solder joints in basement piping and the water meter on the public water service line that may be more prevalent in the more recently built homes.

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 1986b).

6.4.3 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 1982c; Fitchko and Hutchinson 1975), and concentrations of lead in coastal sediments range from 1 to 912 mg/kg with a mean value of 87 mg/kg (EPA 1982c; 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 1982c). More current data obtained from the EPA STORET database (from January 1, 2004 to May 16, 2005), showed that lead has been detected in sediment samples from Honolulu, Hawaii (0.75–6.2 mg/kg), various locations of South Carolina (<1–21 mg/kg), Dade County, Florida (4.7–17.9 mg/kg), and various locations in Tennessee (6–50 mg/kg) (EPA 2005k). Surface sediment concentrations in Puget Sound ranged from 13 to 53 mg/kg (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). Local sources of lead releases can also contribute significantly to lead content in sediments (Gale et al. 2004). For example, lead concentrations in sediments located near mines and or sites containing mine tailings in the old lead belt of Missouri were greatly elevated, 10,550–12,400 mg/kg sediment (dry weight) compared to unaffected sediments (72–400 mg/kg dry weight) (Gale et al. 2002).

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 to 1 µg/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 >840 µg/g in New Orleans and 265 µg/g in Minneapolis. In contrast, the small towns of Natchitoches, Louisiana, and Rochester, Minnesota, had soil lead concentrations of <50 and 58 µg/g, respectively. These data suggest that lead-contaminated soil is a major source of lead exposure in urban areas (Mielke 1993). As would be expected, soils in elementary school properties were also found to have the same pattern of lead levels as the soils in the surrounding residences. Lead concentrations in soils collected from inner-city schools in New Orleans were higher (median

concentration of 96.5 μg/g) than soils collected from mid-city (30.0 μg/g) and outer-city (16.4 μg/g) elementary schools (Higgs et al. 1999).

Studies conducted in Maryland and Minnesota indicate that within large, light-industrial, urban settings such as Baltimore, the highest soil lead levels generally occur near inner-city areas, especially where high traffic flows have long prevailed (Mielke et al. 1983, 1984/1985, 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 μg/g) than levels found in rural Minnesota (6.7 μg/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 had been significantly reduced, found that median soil lead levels taken from the foundations of homes, in yards, and adjacent to the street were 700, 210, and 160 μg/g, respectively; median soil lead concentrations in comparable samples from the smaller city of Rochester, Minnesota, did not exceed 100 μg/g at any location tested (Mielke et al. 1989). The Minneapolis data suggested that average lead levels were elevated in soil samples taken from the foundations of homes, but that lead levels were low (<50 μg/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 μg/g), but levels around homes composed of brick or stucco were significantly lower (mean concentration 158 μg/g) (Schmitt et al. 1988). Severely contaminated soils (levels as high as 20,136 μg/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 μg/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 >150 μg/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 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” (MPCA 1987).

Lead was analyzed in dust wipes and soil samples from 67 public housing projects containing 487 dwelling units across the United States (Succop et al. 2001). A total of 5,906 dust wipes and 1,222 soil samples were included in the data set. The median soil levels were 194 ppm near the foundation, 177 ppm near the walkways, and 145 ppm elsewhere in the yard. The maximum level, 3,900 ppm, was found in a foundation sample. Median dust lead loading from kitchens, living rooms, and two children's bedrooms were 151, 936, and 8,560 μg m^{-2} for floor window sills and window troughs, respectively. Thirteen percent of the floor samples and 30% of the window sill samples from the rooms exceeded the HUD Interim Dust Lead Standards of 431 and 2,690 μg m^{-2} for floor and window sill samples, respectively.

Blood lead levels (PbBs) in children have been shown to correlate with lead concentration in soils in urban areas. In a study of children in New Orleans, Mielke et al. (1999) found that those living in areas classified as high (median soil lead concentrations >310 μg/kg) and low (median soil lead concentrations <310 μg/kg) metal census tract regions correlated well with median PbB above and below 9 μg/dL, respectively. In an analysis of data collected in an ATSDR study of children living near four NPL sites, it was concluded that a PbB of 5.99 μg/dL could be predicted for children exposed to soil lead levels of 500 mg/kg (Lewin et al. 1999). However, there was a high degree of uncertainty and variability associated with the predicted correlation between blood and soil lead levels, suggesting the contribution of other factors to PbB, such as lead levels in household dust, interior paint, and drinking water.

In a study of associations between soil lead levels and childhood blood lead levels (PbBs) in urban New Orleans and rural Lafourche Parish in Louisiana, childhood PbBs appeared more closely associated with soil lead levels than with age of housing. In the study, over 2,600 lead-containing soil 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 lead-containing soil levels were associated with new housing, but census tracts with high median lead-containing soil levels were split evenly between old and new housing. The same pattern was also observed for childhood PbBs. High lead-containing soil levels were associated with high PbB, and low lead-containing soil levels were 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 >9 μg/dL and no indication of a statistical association between median PbB and either median lead levels in soil or age of housing (Mielke et al. 1997a).

In the state of Maine, soil samples taken from areas where the risk of lead contamination was considered high (within 1–2 feet of a foundation of a building >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). Environmental health studies conducted near four NPL sites measured mean concentrations of lead in soil ranging from 317 to 529 mg/kg, and mean concentrations of lead in dust ranging from 206 to 469 mg/kg (Agency for Toxic Substances and Disease Registry 1995).

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/year, had lead levels of 22,191 μg/g (geometric mean) and 973 μg/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 (μg/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 1986b).

6.4.4 Paint

Weathering of lead-based paint can contribute to the lead content of dust and soil. A 1974 study indicated that elevated PbBs 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 (MPCA 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 μg/g while soil samples taken from around the foundations of brick or stucco had a mean concentration of 158 μg/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.

6.4.5 Other Sources

Concentrations of lead (wet weight basis) in samples of 11 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 μg/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); and tomato (0.002).

Lead has been detected in a variety of foods. Lead may be introduced into food through uptake from soil into plants or atmospheric deposition onto plant surfaces, during transport to market, processing, and kitchen preparation (EPA 1986a). In the FDA Total Diet Study (TDS) 1991–1996, food was purchased 4 times/year from each of four geographic regions of the United States and a market basket consisting of about 260 foods from three representative cities within the geographical region analyzed for different elements, including lead (Capar and Cunningham 2000). Lead was below the limit of quantitation in all TDS food in the following food categories: milk and cheese; eggs; meat, poultry, and fish; legumes and nuts; grain and cereal products; vegetables; mixed dishes and meals; desserts; snacks; fats and dressings; and infant and junior foods. Only five products had quantifiable concentrations of lead, namely: canned peaches (0.032 mg/kg), canned pineapple (0.013 mg/kg), canned fruit cocktail (0.031 mg/kg), sweet cucumber pickles (0.036 mg/kg), and dry table wine (0.023 mg/kg). Typical concentrations of lead in various foods are shown in Table 6-5 for the TDS 1991–1996. Results of a previous FDA TDS in which samples were collected in 27 cities between October 1980 and March 1983 are shown for comparison (Gartrell et al. 1986a).

Other factors such as absorption of lead from cooking water and cookware can influence the amount of lead in cooked vegetables. Ceramic dishes may contain lead in their glazes, and lead in glass has been shown to leach into wine. The degree to which lead is released from food once it is consumed also influences a person's uptake of lead.

A survey conducted in five Canadian cities during 1986–1988 in which food was purchased from retail stores and analyzed for lead in composite samples (n=756), determined the lead levels in 11 food categories as well as the average dietary intake of different population groups (Dabeka and McKenzie

Table 6-5. Lead Levels in Various Food Categories

	Mean concentration (µg/g)	
Food category	TDS 1980–1982a	TDS 1991–1996b
Dairy products	0.006	
Milk and cheese		<0.02–<0.05
Eggs		<0.03
Meat, fish, and poultry	0.016	nd–<0.05
Grain and cereal products	0.023	nd–<0.05
Vegetables	0.010–0.041	nd–<0.05
Legumes and nuts	0.124	nd–<0.05
Fruits	0.046–0.060	nd–0.032
Mixed dishes and meals		nd–<0.04
Desserts		nd–<0.04
Snacks		<0.05
Oils, fats, shortenings, and dressings	0.017	nd–<0.04
Sugar, adjuncts, condiments, and sweeteners	0.028	<0.03–0.036
Infant and junior foods		nd–<0.04
Beverages	0.010	nd–0.0.023

[a]Gartrell et al. 1986b
[b]Capar and Cunningham 2000

nd = not detectable (<0.008 µg/g); TDS = Total Dietary Study

1995). Results of this study are found in Table 6-6. The lead level in all of the foods ranged from <0.4 to 523.4 ng/g with a mean of 23.2 ng/g for all food categories. The highest mean Pb levels were found in canned luncheon meats (163 ng/g), canned beans (158 ng/g), canned citrus fruit (126 ng/g), and canned peaches (133 ng/g). In canned foods, mean Pb levels decreased from 73.6 ng/g in 1985 to 46 ng/g in 1988, at which time it was estimated that 97–99% of Canadian canned foods were in lead-free cans. 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 μg/g; in 1986, after these cans were phased out, the lead level in evaporated milk dropped to 0.006 μg/g (Capar and Rigsby 1989). A survey conducted in five Canadian cities during 1986–1988 in which food was purchased from retail stores and analyzed for lead in composite samples (n=756), determined the lead levels in 11 food categories as well as the average dietary intake of different population groups (Dabeka and McKenzie 1995). Results of this study are found in Table 6-6. The lead level in all of the foods ranged from <0.4 to 523.4 ng/g with a mean of 23.2 ng/g for all food categories. The highest mean Pb levels were found in canned luncheon meats (163 ng/g), canned beans (158 ng/g), canned citrus fruit (126 ng/g), and canned peaches (133 ng/g). In canned foods, mean Pb levels decreased from 73.6 ng/g in 1985 to 46 ng/g in 1988, at which time it was estimated that 97–99% of Canadian canned foods were in lead-free cans. 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 μg/g; in 1986, after these cans were phased out, the lead level in evaporated milk dropped to 0.006 μg/g (Capar and Rigsby 1989).

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 1995. For lead, the geometric mean, maximum, and 85th percentile concentrations (μg/g wet weight) were 0.11, 4.88, and 0.22, respectively. 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 on mining and industrial discharges have reduced lead in the aquatic environment (Schmitt and Brumbaugh 1990).

Table 6-6. Lead Levels in Canadian Foods 1986–1988

Food category	Number of samples	Concentration (ng/g)		
		Mean	Median	Maximum
Milk and milk products	64	7.7	3.9	44.7
Meat and poultry	89	20.2	8.2	523.2
Fish	28	19.3	13.7	72.8
Soups	20	15.5	8.7	48.7
Bakery goods and cereals	120	13.7	10.5	66.4
Vegetables	190	24.4	8.7	331.7
Fruits and fruit juices	127	44.4	15.9	372.7
Fats and oils	15	9.6	<8.8	19.7
Sugar and candies	35	18.3	10.3	111.6
Beverages	35	9.9	<3.1	88.8
Miscellaneous	33	41.7	23.4	178.9
All categories	756	23.2	9.2	523.4

Source: Dabeka and McKenzie 1995

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 2007, 8 advisories restricting the consumption of lead-contaminated fish and shellfish were in effect in 5 states (Hawaii, Idaho, Washington, Kansas, and Missouri) and 1 territory (American Samoa) (EPA 2007b).

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 that 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 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.

The FDA investigated the prevalence and concentration of lead in a variety of dietary supplements with an emphasis on botanical-based products (Dolan et al. 2003). The concentration of lead in the 95 major product components tested was <20–48,600 µg/kg and the median concentration was 403 µg/kg. Levels of lead found in 11 products would result in exposures that exceed the tolerable lead intakes for children and women of child-bearing age, particularly pregnant women, 6 and 25 µg Pb/day. Of the 136 brands of nutritional supplements containing calcium (calcium supplements, mineral-vitamin supplements, antacids, and baby formulas) purchased in California in 1996, two-thirds failed to meet the 1999 California criteria for acceptable lead levels in consumer products, >1.5 µg lead/g calcium (Scelfo and Flegal 2000). The lowest levels were found in infant formulas and antacids, which all contained either synthesized or refined calcium. Lead concentrations were undetectable (<0.02 µg/g) in all infant formulas tested. Of the natural calcium supplements, none of the dolomite brands (n=5), five of the oyster shell brands (n=26), and half of the bonemeal brands (n=9) met the 1999 California criteria, while two dolomite brands and one oyster shell brand exceeded the federal limit, 7.5 µg Pb/g calcium.

Many non-Western folk remedies used to treat diarrhea or other ailments may contain substantial amounts of lead. Examples of these include: Alarcon, Ghasard, Alkohl, 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. Lead was the most common heavy metal contaminant/adulterant found in samples (n=54) of Asian traditional remedies available at health food stores and Asian groceries in Florida, New York, and New Jersey (Garvey et al. 2001). Sixty percent of the remedies tested would give a daily dose of lead in excess of 300 mg when taken according to labeling instructions. 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). Ayurveda is a traditional form of medicine practiced in India and other South Asian countries; the medications used often contain herbs, minerals, metals, or animal products and are made in standardized and nonstandardized formulations (CDC 2004). During 2000–2003, 12 cases of lead poisoning among adults were reported in five states due to the use of ayurveda medications obtained from ayurvedic physicians (CDC 2004).

Because lead concentrations in urban soil can be very high, a pilot study was conducted in an urban neighborhood in Chicago in order to gauge the levels of lead in an array of fruits, vegetables, and herbs (Finster et al. 2004). The soil lead concentrations where the plants were sampled varied from 27 to 4,580 ppm (median 800 ppm, geometric mean 639 ppm). Detectable lead levels in the edible fruit,

vegetables, and herbs sampled ranged from 11 to 81 ppm. Only one fruiting vegetable (cucumber 81 ppm) among the 52 sampled had detectable levels of lead in the edible portion. However, 12 of the 31 leafy vegetables and herbs sampled contained lead in the edible shoot part of the plant (range, 11–60 ppm). The lead concentrations in the four samples of root vegetables ranged from 10 to 21 ppm. No significant correlation was found between the lead concentrations in the edible portion of plant and the soil lead level.

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 μg/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 μg/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 μg/L initially to 68, 81, 92, and 99 μg/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 μg/cigarette, of which approximately 2–6% may actually be inhaled by the smoker (WHO 1977). This lead may have been due to the use of lead arsenate pesticides or lead-containing vehicle exhaust contaminating the tobacco plants. While no recent data were found on the concentration of lead in tobacco, higher levels of lead in indoor air and PbBs are associated with households with smokers (Bonanno et al. 2001; Mannino et al. 2003).

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

(Mielke 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 μg/hand for foam products and 73 μg/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). Of the 115 suspected moonshine samples seized by local law enforcement between 1995 and 2001 and analyzed by the Bureau of Alcohol, Tobacco, and Firearms, 33 samples (28.7%) contained lead levels >300 μg/dL. The median and maximum levels were 44.0 and 53,200 μg/dL, respectively (Parramore et al. 2001).

Use of lead ammunition may result in exposure to lead dust generated during gun or rifle discharge at levels up to 1,000 $\mu g/m^3$ (EPA 1985c), from lead pellets ingested by or imbedded in animals that are used as food sources, and from lead pellets or fragments imbedded in humans from shooting incidents (Burger et al. 1998; Johnson and Mason 1984; Raymond et al. 2002). Exposures to airborne lead dust from firearm discharge in indoor shooting ranges has been shown to result in increases in blood lead concentration that are 1.5–2 times higher than preexposure concentrations (Greenberg and Hamilton 1999; Gulson et al. 2002). However, the use of copper-jacketed bullets, nonlead primers, and well-ventilated indoor firing ranges lessen the impact of airborne lead on blood lead levels (Gulson et al. 2002).

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.

Inexpensive metallic jewelry items specifically intended for children and teenagers have been shown to contain varying levels of lead (Maas et al. 2005). A total of 311 chemical assays conducted using 285 jewelry items purchased in 20 different stores in California revealed that a considerable amount of lead was added to the items, presumably to increase their weight or to impart some type of metallic coating to the surface of the item. The mean weight percentage of lead for all 311 assays was 30.6%. Of the 311 samples tested, 169 contained at least 3% lead by weight in at least one portion of the jewelry piece and 123 of the samples were found to contain >50% lead by weight (Maas et al. 2005). In addition, 62 pieces of the purchased jewelry were tested for surface levels of lead that could potentially be transferred dermally through the routine handling of these pieces. Using standard laboratory wipes, the surface of the jewelry pieces were wiped for a total of 20 seconds and subsequently analyzed for lead content. Mean lead levels in the wipes ranged from 0.06 to 541.97 μg. The authors characterized the potential lead exposure from these dermal transfer experiments as either low exposure (<1 μg of lead transferred to the laboratory wipe), moderate exposure (1–10 μg of lead transferred to the laboratory wipe), high exposure (10–50 μg of lead transferred to the laboratory wipe), and very high exposure (>50 μg of lead transferred to the laboratory wipe). Approximately 35% of the 62 pieces tested were characterized as having low exposure, 48% were characterized as moderate exposure, 11% were characterized as high exposure, and 5% were characterized as very high exposure (Maas et al. 2005).

6.5 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 6.6) 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 soil concentrations of lead at a range in Michigan were 10–100 times greater than background level of 25 mg/kg; mobilization of lead appeared to be occurring and may present a threat to groundwater and surface water (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 μg/day for a 2-year-old child, 1.0 μg/day for an adult working indoors, and 2.0 μg/day for adults working outdoors; these figures are based on an average atmospheric lead concentration of 0.1 $\mu g/m^3$ and an indoor/outdoor lead concentration ratio of 0.5. In an urban environment, the indoor/outdoor lead concentration ratio was assumed to be approximately 0.8, yielding an estimated lead exposure of 1.0 $\mu g/m^3$ for adults. This estimate assumed a 2-hour/day exposure to an outdoor lead concentration of 0.75 $\mu g/m^3$, a 20-hour/day exposure to an indoor lead concentration of 0.6 $\mu g/m^3$, a 2-hour/day exposure to 5 $\mu g/m^3$ 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 gasoline since 1986 have resulted in a 64% decrease in lead emissions to the atmosphere (see Section 6.4.1).

Using the EPA National Air Quality Monitoring System, the average maximum 24-hour atmospheric lead concentration in the United States was 0.84 $\mu g/m^3$ in 2004 (EPA 2005k). There were two maximum 24-hour monitoring values measured in 2004 in which the 10 $\mu g/m^3$ level was exceeded (11.76 and 11.53 $\mu g/m^3$ in Muncie, Indiana). All other atmospheric lead levels measured throughout the rest of the United States were <10 $\mu g/m^3$ threshold in 2004.

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. The contribution of various food categories to the 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, are shown in Table 6-7 (Gartrell et al. 1986b). This value is only slightly higher

Table 6-7. Contribution of Various Food Categories to the Average Daily Intake (AVDI) of Lead in Adults (1980–1982)

Food category	AVDI (µg/day)
Dairy products	4.54
Meat, fish, and poultry	4.09
Grain and cereal products	9.84
Potatoes	1.39
Leafy vegetables	0.94
Leafy legumes	9.18
Root vegetables	1.39
Garden fruit	4.44
Fruits	10.00
Oils, fats, and shortenings	1.23
Sugar and adjuncts	2.34
Beverages	6.86
Total	56.50

Source: Gartrell et al. 1986b

than the estimated lead intake of 54 µg/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 µg/g for drinking water to 0.654 µg/g for cheese (Dabeka et al. 1987).

Based on data from the FDA's Total Diet Food Studies (Bolger et al. 1991; Gunderson 1988), dietary values for average daily intake of lead by different population groups from 1980 to 1990 have been estimated (Table 6-8). The estimates of lead intake presented in Table 6-8 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). Data from the 1990–1991 Total Diet Survey indicate that dietary lead intake ranged from 1.8 to 4.2 µg/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). The daily dietary intake of lead estimated from the 1986–1988 Canadian Survey was 24 µg/day for all ages, male and female (Dabeka and McKenzie 1995). The highest contribution among 11 food categories to Pb intake was beverages (20.9%) and bakery goods and cereals (20.6%). The FDA Total Diet Survey (TDS) 1991–1996 tested 18 market baskets consisting of about 260 foods collected from three cities (representing standard metropolitan statistical areas) within four different geographical regions (Capar and Cunningham 2000). Mean and median Pb concentrations in all foods were 0.005 and 0 mg/kg, respectively. These results are similar to those in previous TDS 1982–1988 and 1990–1991 surveys except those in canned foods. The lower Pb concentrations in canned foods for TDS 1991–1996 is attributed to the reduction and ultimate ban in 1995 on the use of lead-soldered food cans in the United States.

More recent data on lead intakes from the U.S. diet come from the results of the NHEXAS studies. Mean and median dietary intakes of lead for study participants in the EPA Region V study were calculated to be 0.25 and 0.10 µg/kg body weight/day, respectively, or 17.5 and 7.0 µg/day for a 70-kg adult, respectively (Thomas et al. 1999). These results were obtained from measurement of concentrations in water and dietary samples. The median dietary lead intake for the Region V population agrees with the 0.09–0.10 µg/kg body weight/day calculated in the FDA TDS (1986–1991) (Gunderson 1995), but is

Table 6-8. Daily Average Intake of Lead (µg Lead/Day)

Age	Sex	1980[a]	1982[a]	1984[a]	1986[a]	1988[a]	1990–1991[b]
6–11 Months	Male/female	≈34	20	16.7	10	5	1.82
2 Years	Male	≈45	25.1	23.0	12.8	5.0	1.87
	Female	No data	No data	No data	No data	No data	No data
14–16 Years	Female	No data	No data	28.7	15.2	6.1	2.63
	Male	No data	No data	40.9	21.8	8.2	3.24
25–30 Years	Female	No data	32.0	28.7	14.8	7.9	3.28
	Male	84	45.2	40.9	21.2	10.0	4.17
60–65 Years	Female	No data	No data	30.4	15.6	No data	3.05
	Male	No data	No data	37.6	19.1	No data	3.46

[a]Bolger et al. 1991
[b]Bolger et al. 1996

substantially lower than the average in the 1986–1988 Canadian study of 0.4 µg/kg body weight/day (calculated from an average intake of 24 µg/day for a 60-kg person (Dabeka and McKenzie 1995). Higher mean daily intakes of lead of 8.14 mg/day were reported from the NHEXAS Maryland study (Ryan et al. 2001). This intake was determined from the consumption levels determined in the NHEXAS Maryland study and the concentration in food from the 1997 ATSDR Toxicological Profile for Lead, 11.0 ppb. This intake would be much lower if the more recent levels of lead in food as reported in Capar and Cunningham (2000) were used. The mean and median concentrations of lead in combined solids and liquids in the EPA Region V study were 4.5 and 3.1 µg/kg, respectively. The mean (median) Pb intakes from dietary, water, and inhalation routes were 10.9 (7.3), 1.7 (0.66), and 0.333 (0.156) µg/day, respectively. While water lead contributed significantly to dietary intake, dietary intake was greater than that calculated for intake from home tap water. Mean and median flushed tap water from Region V homes contained 0.84 and 0.33 µg/L, while standing tap water contained 3.9 and 1.9 µg/L of lead, respectively.

In another approach to determining daily lead intake within subpopulations in the United States, Moschandreas et al. (2002) used the Dietary Exposure Potential Model (DEPM) and data obtained from Combined National Residue Database (CNRD) to estimate dietary lead intake based on food consumption patterns in 19 subpopulation groups. The food items used in the model are based on 11 food groups consisting of approximately 800 exposure core foods that represent 6,500 common food items. The results of their model (Table 6-9) yielded an average dietary lead intake in the U.S. population of 1.009 µg/kg body weight/day, or 70.6 µg/day for a 70-kg adult. Of the various subpopulation groups, nonnursing infants and children ages 1–6 years had much higher lead intakes/kg body weight than the general population, 3.117 and 1.952 µg/kg body weight, respectively.

The NHEXAS Arizona study evaluated exposure to lead for a study population from multiple media and pathways (O'Rourke et al. 1999). The concentrations of lead in the various media sampled are presented in Table 6-10 and the estimated total human exposure to the study population and various subpopulations is shown in Table 6-11. The daily total lead intake to the study population from all media ranged from 11 to 107 µg/day, with a mean of 36 µg/day. This compares with a range of 15–312 µg/day reported by the World Health Organization (WHO 1995).

Moonshine consumption was strongly associated with elevated PbBs (Morgan and Parramore 2001). A 2000 study found a median PbB of 11 µg/dL among 35 moonshine consumers versus 2.5 µg/dL in 68 randomly-selected nonmoonshine consumers. Gulson et al. (2001b) studied the contribution of lead

Table 6-9. Dietary Exposure Estimates of U.S. Populations to Lead Based on the Dietary Exposure Potential Model (DEPM)

Subpopulation	Lead intake (µg/kg body weight/day)
U.S. population	1.009
Age/gender	
Nonnursing infants	3.117
Children 1–6	1.952
Children 7–12	1.164
Females 13–19	0.824
Females 20+	0.920
Females 55+	0.946
Males 13–19	0.890
Males 20+	0.895
Males 55+	0.918
Ethnicity	
Hispanic	1.177
Non-Hispanic white	1.095
Non-Hispanic black	0.797
Non-Hispanic other	0.871
Geographic region[a]	
North central	0.611
Northeast	0.968
Southern	0.966
Western	1.133
Family income[b]	
Poverty 0–130%	1.094
Poverty 131%+	0.986

[a]The regional classification is as defined by the U.S. Department of Agriculture, and is based upon U.S. Census Bureau regions.
[b]Annual household income as a percentage of the Poverty Index

Source: Moschandreas et al. 2002

Table 6-10. Lead Concentrations for Various Media From the NHEXAS Arizona Study

Media	Number of samples	Percent BDL	Lead concentration—percentile			
			Units	50th	75th	90th
Air—indoors	119	100	ng/m^3	BDL	BDL	BDL
Air—outdoors	116	100	ng/m^3	BDL	BDL	BDL
Dust	135	86	µg/g	BDL	BDL	131.0
Soil	139	85	µg/g	BDL	BDL	118.1
Food	159	0.6	µg/kg	6.4	9.2	16.1
Beverage	154	29	µg/kg	1.9	4.1	7.1
Drinking water consumed	73	51	µg/L	BDL	0.4	2.0
Tap water consumed	82	1	µg/L	0.4	0.9	1.3

BDL = below detection limit

Source: O'Rourke et al. 1999

Table 6-11. Total Lead Exposure of Subject Population From the NHEXAS Arizona Study

Exposure population	Number of subjects	Lead intake (µg/day)		
		Mean	Median	Range
All subjects	176	36	31	11–107
Adult male (>18 years of age)	55	42	37	16–107
Adult female (>18 years of age)	86	35	30	11–96
Children (<18 years of age)	35	27	25	15–45
Hispanic	54	40	34	14–107
Non-Hispanic	119	34	29	11–-96

Source: O'Rourke et al. 1999

from calcium supplements to blood lead in 21 adults divided into three treatment groups over a 6-month period. One treatment group received a complex calcium supplement (carbonate/phosphate/citrate), another group received calcium carbonate, and the last, the control group, received no supplement. The isotopic composition of the supplements differed from that of the subject's blood allowing the investigators to estimate the contribution of the supplements to PbBs. While the changes from baseline to treatment in isotopic composition were significant in the treatment groups, there was no discernable increase in PbB concentration during the study. The change in isotopic contribution, however, indicates that there is a limited input of lead from the diet into the blood in adults. These results are consistent in other investigations that showed minimal gastrointestinal absorption of lead in the presence of calcium in adults.

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 µg/mL lead for pitchers to 5.0 µg/mL for cups and mugs soaked for 24 hours in a 4% acetic acid solution (FDA 1992). 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 NHANES revealed that between 1976 and 1991, the mean PbBs of the U.S. population aged from 1 to 74 years dropped 78%, from 12.8 to 2.8 µg/dL. The prevalence of PbBs ≥10 µg/dL also decreased sharply from 77.8 to 4.3%. The major cause of the observed decline in PbBs is most likely the removal of 99.8% of lead from gasoline and the removal of lead from soldered cans (Pirkle et al. 1994). PbBs 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 noncentral-city residents. PbBs also correlated with low income, low educational attainment, and residence in the Northeast region of the United States. Data analyses of the PbBs from NHANES surveys 1991–1994 and 1999–2002 are provided in Table 6-12 (CDC 2005a). Geometric means as well as 95% confidence intervals were calculated, and the results were organized by age, race/ethnicity, and sex. For 1999–2002, the overall prevelance of elevated PbBs (≥10 μg/dL) was 0.7%, down from 2.2% in the 1991–1994 survey. Children aged 1–5 years had the highest prevelance, 1.6%, of all age groups for levels ≥10 μg/dL in the 1999–2002 survey. This percentage is down from 4.4% in the 1991–1994 NHANES survey (CDC 2005a). Approximately 310,000 children in this age group were at risk of exposure to harmful levels of lead. The largest decline (72%) in elevated PbB in the two surveys, from 11.2 to 3.1%, was among non-Hispanic black children aged 1–5 years. In 2000, the year that had been targeted for the elimination of PbBs, >25 μg/dL in children aged 6 months–5 years, a total of 8,723 children had been identified with PbBs ≥25 μg/dL. Lead surveillance data collected by states between 1997 and 2001 also show a decline in the number of children aged 1–5 years with PbBs ≥10 μg/dL from 130,512 in 1997 to 74,887 in 2001 (Meyer et al. 2003).

The Adult Blood Lead Epidemiology and Surveillance (ABLES) program, which tracks cases of adult (aged ≥16 years) elevated PbBs from workplace exposure, reported updated results from 25 participating states for the period 1998–2001 (Roscoe et al. 2002). During that period, the prevalence of adults with PbBs ≥25 μg/dL was 13.4 per 100,000 employed adults, compared with 15.2 per 100,000 for 1994–1997. For adults with blood lead levels ≥40 μg/dL, the prevalence rate was 2.9 per 100,000 during 1998–2001, compared with 3.9 per 100,000 for 1994–1997. ABLES surveillance data from 2004 tracked the blood lead levels of females of childbearing age (16–44 years) in 37 different states (CDC 2007). The results indicated that 0.06 per 100,000 had PbBs ≥40 μg/dL, 0.7 per 100,000 had PbBs ≥25 μg/dL, 3.9 per 100,000 had PbBs ≥10 μg/dL, and 10.9 per 100,000 had PbBs ≥5 μg/dL (CDC 2007).

A 1992 survey of lead in blood of 492 Inuit adults from the Arctic region of Quebec, Canada resulted in geometric mean lead concentrations of 0.42 μmol/L, with a range of 0.04–2.28 μmol/L. Analysis of variance revealed that smoking, age, and consumption of waterfowl were associated with elevated lead levels (Dewailly et al. 2001). A Swedish study was aimed at characterizing PbBs in 176 men and 248 women, 49–92 years of age (Baecklund et al. 1999). Blood lead levels ranged from 5.6 to 150 μg Pb/L (median 27 μg Pb/L) and were higher in men than in women (median 30 versus 24 μg Pb/L). In both men and women, PbBs decreased between 50 and 70 years of age, which was thought to be a result of decreased energy intake. In women, PbBs peaked at 50–55 years of age, which is probably related to

Table 6-12. Geometric Mean Blood Lead Levels (µg/dL) and the 95th Percentile Confidence Interval, by Race/Ethnicity, Sex, and Age

Sex/age (years)	Number in sample	All racial/ethnic groups	White, non-Hispanic	Black, non-Hispanic	Mexican American
NHANES 1991–1994 geometric mean (95% confidence interval)					
			Both sexes		
≥1	13,472	2.3 (2.1–2.4)	2.2 (2.0–2.3)	2.8 (2.5–3.0)	2.4 (2.3–2.6)
1–5	2,392	2.7 (2.5–3.0)	2.3 (2.1–2.6)	4.3 (3.6–5.0)	3.1 (2.7–3.5)
6–19	2,960	1.7 (1.5–1.8)	1.5 (1.4–1.7)	2.3 (2.1–2.6)	2.0 (1.8–2.1)
20–59	5,596	2.2 (2.1–2.3)	2.1 (2.0–2.2)	2.6 (2.4–2.8)	2.5 (2.4–2.6)
≥60	2,524	3.4 (3.2–3.5)	3.3 (3.2–3.4)	4.3 (3.7–4.9)	3.1 (2.7–3.6)
			Males		
≥1	6,204	2.8 (2.6–2.9)	2.6 (2.5–2.8)	3.6 (3.3–4.0)	3.1 (2.9–3.3)
1–5	1,211	2.8 (2.5–3.1)	2.3 (2.1–2.6)	4.7 (3.9–5.5)	3.3 (2.9–3.6)
6–19	1,443	1.9 (1.7–2.1)	1.7 (1.5–1.9)	2.7 (2.4–3.1)	2.3 (2.0–2.5)
20–59	2,365	2.9 (2.7–3.1)	2.7 (2.5–3.0)	3.6 (3.2–3.9)	3.4 (3.2–3.6)
≥60	1,185	4.2 (4.0–4.4)	4.0 (3.8–4.2)	6.3 (5.4–7.1)	4.1 (3.5–4.8)
			Female		
≥1	7,268	1.9 (1.8–2.0)	1.8 (1.7–1.9)	2.2 (2.0–2.4)	1.9 (1.8–2.1)
1–5	1,181	2.7 (2.4–2.9)	2.3 (2.0–2.6)	4.0 (3.2–4.8)	2.9 (2.4–3.4)
6–19	1,517	1.5 (1.3–1.7)	1.4 (1.2–1.6)	2.0 (1.7–2.2)	1.7 (1.5–1.9)
20–59	3,231	1.7 (1.6–1.8)	1.6 (1.5–1.7)	1.9 (1.8–2.1)	1.8 (1.7–1.9)
≥60	1,339	2.9 (2.7–3.0)	2.8 (2.7–3.0)	3.3 (2.8–3.8)	2.5 (2.1–2.9)
NHANES 1999–2002 geometric mean (95% confidence interval)					
			Both sexes		
≥1	16,825	1.6 (1.5–1.6)	1.5 (1.5–1.6)	1.8 (1.7–1.9)	1.6 (1.6–1.7)
1–5	1,160	1.9 (1.8–2.1)	1.8 (1.6–2.0)	2.8 (2.5–3.1)	1.9 (1.8–2.0)
6–19	6,283	1.1 (1.1–1.2)	1.1 (1.0–1.1)	1.5 (1.4–1.6)	1.3 (1.2–1.4)
20–59	5,876	1.5 (1.5–1.6)	1.5 (1.4–1.5)	1.7 (1.6–1.8)	1.8 (1.6–1.9)
≥60	3,056	2.2 (2.1–2.3)	2.2 (2.1–2.3)	2.7 (2.5–2.8)	2.1 (1.9–2.3)
			Males		
≥1	8,202	1.9 (1.8–2.0)	1.9 (1.8–1.9)	2.1 (1.4–1.6)	2.0 (1.9–2.2)
1–5	846	1.9 (1.8–2.1)	1.8 (1.6–2.0)	2.8 (2.5–3.2)	2.0 (1.8–2.1)
6–19	3,158	1.3 (1.3–1.4)	1.2 (1.1–1.3)	1.7 (1.5–1.8)	1.5 (1.4–1.6)
20–59	2,689	2.0 (1.9–2.0)	1.9 (1.8–2.0)	2.1 (2.0–2.3)	2.3 (2.2–2.5)
≥60	1,509	2.7 (2.6–2.8)	2.6 (2.5–2.7)	3.4 (3.1–3.6)	2.6 (2.3–2.8)
			Female		
≥1	8,623	1.3 (1.3–1.3)	1.3 (1.2–1.3)	1.5 (1.4–1.6)	1.3 (1.2–1.4)
1–5	764	1.9 (1.8–2.1)	1.8 (1.5–2.1)	2.8 (2.5–3.2)	1.8 (1.7–2.0)
6–19	3,125	1.0 (0.9–1.0)	0.9 (0.8–1.0)	1.3 (1.2–1.5)	1.1 (1.0–1.2)
20–59	3,187	1.2 (1.2–1.2)	1.2 (1.1–1.2)	1.4 (1.3–1.5)	1.3 (1.2–1.4)
≥60	1,547	1.9 (1.8–2.0)	1.9 (1.8–2.0)	2.3 (2.1–2.4)	1.8 (1.6–2.0)

Source: CDC 2005a

postmenopausal bone mineralization. Increases in PbBs after age 70 was thought to be a result from higher lead exposure in the past for this group. Nash et al. (2004) reported median adjusted PbBs that were 25–30% higher than for premenopausal women (2.0 μg/dL). Users of hormone replacement therapy had significant lower median PbBs. Lead stored in the bones of women is released into the blood during post menopausal bone mineral resorption.

Mourning doves and other game birds consume lead pellets from hunting fields for grit. Recreational and subsistence hunters and their families who consume large amounts of these birds may ingest lead from this source (Burger et al. 1998).

Lead is a component of tobacco and tobacco smoke, and smokers often have higher lead blood levels than nonsmokers (Bonanno et al. 2001; Mannino et al. 2003). Using data from the NHEXAS EPA Region V study, PbB levels in smokers and nonsmokers were analyzed and a correlation between tobacco smoke and exposure levels was observed (Bonanno et al. 2001). The mean PbBs in smokers, nonsmokers exposed to environmental tobacco smoke (ETS), and nonsmokers without ETS were 2.85, 2.06, and 1.81 μg/dL, respectively (Bonanno et al. 2001).

Table 6-13 provides geometric means and selected percentiles of lead levels in the urine in segments of the U.S. population (CDC 2003, 2005b). These data will continue to be updated as new information becomes available.

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 $\mu g/m^3$ for workplace air (OSHA 2005d; Tripathi and Llewellyn 1990). NIOSH has estimated that >1 million American workers were occupationally exposed to inorganic lead in >100 occupations (NIOSH 1978a). According to NOES, conducted by NIOSH between 1980 and 1983, an estimated 25,169 workers 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 nonferrous foundries, lead smelters, and battery plants; 3,902 workers were exposed to lead chloride; and 576,579 workers 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 $\mu g/m^3$ (Malkin et al. 1992).

Table 6-13. Geometric Mean and Selected Percentile Urine Concentrations (µg/L) of Lead in the U.S. Population From 1999 to 2002

Group and survey years	Geometric mean	Percentile				
		50th	75th	90th	95th	Sample size
Age 6 and older						
1999–2000	0.766	0.800	1.30	2.10	2.90	2,465
2001–2002	0.677	0.600	1.20	2.00	2.60	2,690
6–11 Years						
1999–2000	1.07	1.00	1.50	2.40	3.40	340
2001–2002	0.753	0.800	1.20	2.00	2.60	368
12–19 Years						
1999–2000	0.659	0.600	1.10	1.70	2.20	719
2001–2002	0.564	0.600	0.900	1.50	1.90	762
20 Years and older						
1999–2000	0.752	0.700	1.40	2.10	2.90	1,406
2001–2002	0.688	0.700	1.20	1.90	2.80	1,560
Males						
1999–2000	0.923	0.900	1.60	2.40	3.40	1,227
2001–2002	0.808	0.700	1.30	2.40	3.20	1,335
Females						
1999–2000	0.642	0.600	1.20	1.90	2.40	1,238
2001–2002	0.573	0.500	1.00	1.50	2.20	1,335
Mexican Americans						
1999–2000	1.02	1.00	1.70	2.80	4.10	884
2001–2002	0.833	0.80	1.50	2.40	3.20	683
Non-Hispanic blacks						
1999–2000	1.11	1.10	1.90	2.90	4.20	568
2001–2002	0.940	0.900	1.50	2.60	3.70	667
Non-Hispanic whites						
1999–2000	0.695	0.700	1.30	1.90	2.60	882
2001–2002	0.610	0.600	1.00	1.80	2.40	1,132

Source: CDC 2003, 2005b

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 (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 μg/m^3; in the manufacture of storage batteries, mean airborne concentrations of lead from 50 to 5,400 μg/m^3 have been recorded; and in the breathing zone of welders of structural steel, an average lead concentration of 1,200 μg/m^3 has been found (Fu and Boffetta 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 standard of 0.05 mg/m^3 (Tharr 1993). Also, past studies of PbBs of 56 radiator shop mechanics in the Boston area revealed that 80% had PbBs >30 μg/dL and 16 had PbBs 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/m^3 for one instructor and 211.1 μg/m^3 for another (Tripathi and Llewellyn 1990). When jacketed bullets were used, breathing zone levels decreased to 8.7 μg/m^3 or less. PbBs 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/m^3 in the personal breathing zone and 68 μg/m^3 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/m^3, respectively (Tripathi and Llewellyn 1990). At an outdoor uncovered range in Los Angeles, instructors who spent an average of 15–20 hours/week behind the firing line were found to be exposed to breathing zone lead concentrations of 460 and 510 μg/m^3 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/m^3 (mean, 12.4 μg/m^3), 2.7 μg/m^3 (mean, 0.6 μg/m^3), and 4.5 μg/m^3 (mean, 0.6 μg/m^3) for range instructors, technicians, and gunsmiths, respectively. Exposure of custodians ranged from nondetectable to 220 μg/m^3 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 nonstudent dormitories (dust-lead concentration range of 116–546 μg/g with a geometric mean of 214 μg/g in the student's rooms versus a dust-lead concentration range of 50–188 μg/g with a geometric mean of 65 μg/g for the nonstudent 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 measured during a brief period of continuous soldering in a shop equipped with an LEV was only 7.1 μg/m^3. In a shop where no LEV was used, the 13 personal samples averaged 209 μg/m^3 with a maximum of 810 μg/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 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 μg/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 PbBs in workers; improvement of hygienic practices proved to be more effective at lowering PbBs 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, PbBs, urinary coproporphyrin levels, or δ-aminolevulinic acid [ALA] levels) rather than monitoring the workplace environment for lead concentrations (EPA 1986a; NIOSH 1978a). An 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 PbBs), and only 24,491 (10%) were in positions where environmental monitoring (workplace air lead levels) had ever been conducted. In addition, approximately 12% of the potentially exposed individuals were in the construction industry (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 μg/m^3, and to have PbBs ranging from 16 to 135 μg/dL. Approximately 30% of the workforce was found to be on medical leave as a result of their PbBs exceeding 40 μg/dL. An analysis of PbBs among family members of the exposed workers gave revealed levels of 10.2 μg/dL compared with 6.2 μg/dL for families of nonexposed workers, indicating possible secondary occupational exposure from workers to their families (Kaye et al. 1987).

Data from the NHANES III was used to compile statistics regarding the PbBs in U.S. workers (Yassin et al. 2004). The greatest levels tended to occur in mechanical and construction trades, while the lowest levels were observed for workers involved in professional labor categories such as managerial positions and health care professionals. Lead levels increased with age, decreased with education level, and male workers had a much higher geometric mean blood level, 3.3 μg/dL, than female workers, 1.8 μg/dL. Tables 6-14 and 6-15 summarize the results from these data for different industries and occupations. Okun et al. (2004) evaluated trends in occupational lead exposure in U.S. industries following the establishment of the general industrial lead standard in 1978 and the construction lead standard in 1993. They used data collected by OSHA under their compliance and consultation programs. On the basis of

Table 6-14. Median, Range, and Weighted Geometric Mean Blood Lead Levels in U.S. Workers, Ages 18–64 in 1988–1994

Occupation	Number of workers	µg/dL		
		Median	Range	WGM (GSD)
Vehicle mechanics	169	5.10	0.70–28.10	4.80 (3.88)
Food service workers	700	2.30	0.70–27.00	2.00 (2.69)
Management, professional, technical and sales	4,768	2.20	0.70–39.40	2.13 (4.05)
Personal service workers	1,130	2.90	0.70–25.90	2.48 (4.52)
Agricultural workers	498	3.80	0.70–23.40	2.76 (4.02)
Production workers: machine operators, material movers, etc.	1,876	3.30	0.70–52.90	2.88 (4.24)
Laborers other than construction	137	4.70	0.70–21.80	3.47 (3.36)
Transportation workers	530	3.85	0.70–22.30	3.49 (5.10)
Mechanics other than vehicles	227	4.10	0.70–16.60	3.50 (4.91)
Construction trades people	470	4.30	0.70–16.90	3.66 (4.64)
Construction workers	122	4.70	1.20–36.00	4.44 (7.84)
Health service workers	499	2.00	0.70–22.40	1.76 (2.24)
All	11,126	2.80	0.70–52.90	2.42 (6.93)

GSD = geometric standard deviation; WGM = weighted geometric mean

Source: Yassin et al. 2004

Table 6-15. Median, Range, and Weighted Geometric Mean Blood Lead Levels in U.S. Workers, Ages 18–64 by Industrial Categories[a]

Industry	Number of workers	Blood lead (µg/dL)		
		Median	Range	WGM (GSD)
Repair services (SIC 75–76)	188	4.80	0.70–28.10	4.54 (5.05)
Wholesale and retail trade (SIC 50–59)	2,229	2.50	0.70–39.40	2.25 (3.38)
Finance, insurance, and real estate (SIC 60–65, 67)	1,117	2.40	0.70–28.70	2.30 (2.74)
Agriculture (SIC 01–02, 07–08)	493	3.80	0.70–23.40	2.68 (4.09)
Transportation and utility (SIC 40–49)	764	3.10	0.70–22.30	2.58 (3.49)
Manufacturing (SIC 20–32, 34–39)	2,008	3.10	0.70–41.80	2.66 (4.51)
Metal (SIC 33)	188	3.80	0.70–52.90	3.50 (2.91)
Construction (SIC 15–17)	671	4.40	0.70–36.00	3.68 (5.66)
Mining (SIC 10, 12–14	41	3.90	1.10–12.90	4.66 (6.23)
Services (SIC 770, 72–73, 78–79, 80–84, 86–89, 91–97)	3,449	2.30	0.70–23.70	2.05 (4.39)
All	11,148	2.80	0.70–52.90	2.42 (6.93)

[a]Working population aged 18–64: U.S. Third National Health and Nutrition Examination Survey, 1988–1994

GSD = standard deviation of geometric mean, SIC = standard industrial code; WGM = weighted geometric mean

Source: Yassin et al. 2004

these data, there has been a decline in occupational lead exposures for general industry facilities since 1979. The median exposure level for these facilities declined 5–10-fold. With the exception of retail trade, these declines were for the major industry divisions and the majority of four-digit SIC codes including some high risk industries. A decline was not observed in the construction industry, but in this case, the data are only for a limited number of years.

6.6 EXPOSURES OF CHILDREN

This section focuses on exposures from conception to maturity at 18 years in humans. Differences from adults in susceptibility to hazardous substances are discussed in Section 3.7, 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, 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, put things in their mouths, sometimes eat inappropriate things (such as dirt or paint chips), and spend more time outdoors. Children also are closer to the ground, and they do not use the judgment of adults to avoid hazards (NRC 1993).

The American Academy of Pediatrics (AAP) (1998) has concluded that although monitoring data demonstrate a decline in the prevalence of PbBs, 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 PbBs 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 have shown that children with the highest PbBs 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 PbBs in children were correlated with soil lead levels, which were highest in inner-city areas; soil lead levels and PbBs 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).

The results of successive NHANES monitoring studies suggest that from 1976 to 2002, PbBs have declined, but were consistently higher for younger children than for older children (CDC 1997b, 1997d, 2003, 2005a, 2005b; Pirkle et al. 1994). In general, PbBs also correlated with low income, low educational attainment, and residence in the Northeast region of the United States. Data from Phase II of NHANES III (conducted during October 1991 to September 1994) and the most recent data (1999–2002) indicate that PbBs in the U.S. population aged ≥1 year continued to decrease and that PbBs 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 lead-based paint) (CDC 1997b, 2003. 2005a, 2005b; Pirkle et al. 1998). During 1991–1994, the overall geometric mean PbB of the population aged ≥1 year was 2.3 μg/dL. Among those aged 1–5 years, approximately 4.4% had PbBs ≥10 μg/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 PbBs ≥15 μg/dL and 0.4% had PbBs ≥20 μg/dL. For the NHANES III Phase II data, the geometric mean PbBs were higher for children aged 1–2 years (3.1 μg/dL) than for children aged 3–5 years (2.5 μg/dL) (CDC 1997b). For the most recent 1999–2002 NHANES sample, the geometric mean PbB for children ≥1 year was 1.6 μg/dL and among those aged 1–5 years, approximately 1.6% had PbBs ≥10 μg/dL (CDC 2005a). These data have been summarized in Table 6-12.

The U.S. Navy instituted a pediatric lead surveillance program in 1995 because of public health concerns over pediatric PbBs (Bohnker et al. 2003). The database contained 38,502 samples from 1995 to 2001 with 1.6% containing levels ≥10 μg/dL. Samples were obtained at the time of the 12-month well-child visit. Results were similar to those for the NHANES survey.

Fetuses are at even greater risk. As discussed in Section 3.5, 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 PbBs 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 PbBs were strongly dependent on time elapsed since immigration to the United States, with PbBs being highest in those women who had immigrated most recently. Elevated PbBs in immigrant women were also associated with pica and with low dietary calcium during pregnancy (Rothenberg et al. 1999a, 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 25 lived in agricultural areas with low air pollution. The mean lead concentrations (expressed as mean±SD) for the women living in areas with high air pollution were 3.72±0.47 µg/dL in maternal blood and 2.0±0.34 µg/dL in umbilical cord blood (correlation coefficient, r=0.57). The mean lead concentrations for the women living in areas with low air pollution were 2.05±0.56 µg/dL in maternal blood and 1.29±0.36 µg/dL in umbilical cord blood (correlation coefficient, r=0.70). The authors concluded that the placenta demonstrates a dynamic protective function that is amplified when maternal PbBs 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 concentrations of umbilical cord blood were 6.56±3.19 µg/dL for the 1980 group and 1.19±1.32 µg/dL for the 1990 group (Hu et al. 1996b).

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

Malcoe et al. (2002) assessed lead sources and their effect on blood lead in rural Native American and white children living in a former mining region. Blood samples, residential environmental samples (soil, dust, paint, water) and caregiver interviews (hand-mouth behaviors, socioeconomic conditions) were obtained from a representative sample of 245 children ages 1–6. There were no ethnic differences in the results. However poor children were especially vulnerable. Regression analysis showed that mean floor dust lead loading >10.1 µg/ft^2 and yard soil lead >165.3 mg/kg were independently associated with blood lead levels ≥10 µg/dL.

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 <1 year old (Cohen 1988). 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 for children has dropped dramatically since the late 1970s. Results summarizing the CDC NHANES II and NHANES III, Phases I and II, study of blood lead levels for children aged 1–5 years are provided in Table 6-16 (CDC 1997b, 1997d, 2005a).

In 1982–1983, the baseline value for daily intake of lead by inhalation in a nonurban environment was estimated to be 0.5 μg/day for a 2-year-old child. The baseline value was based on an average atmospheric lead concentration of 0.1 $\mu g/m^3$ 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 6.4.1).

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 PbBs in young children, the major source of moderately elevated PbBs (30–80 μg/dL) in inner city children is most 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 PbBs in over 200 urban and suburban infants followed from birth to 2 years of age; however, the PbBs 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 PbBs (>8.8 μg/dL) were 72 μg/wipe (window sill dust) and 1,011 μg/g, respectively; children with low PbBs (<3.7 μg/dL) were exposed to 22 μg/wipe and 380 μg/g, respectively. In addition, 79% of the homes of children with high PbBs had

Table 6-16. Blood Levels of Lead in Children (1–5 Years) in 1976–2002

Children (1–5 Years)	NHANES			
	1976–1980	1988–1991	1991–1994	1999–2002
Geometric mean (µg/dL)	15.0	3.6	2.7	1.9
Blood lead ≥10 µg/dL	88.2%	8.9%	4.4%	1.6%

NHANES = National Health and Nutrition Examination Survey

Sources: CDC 1997b, 1997d, 2005a; Pirkle 1994

been renovated, while only 56% of the homes of children with low PbBs 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). Regular use of dust control methods (e.g., wet mopping of floors, damp-sponging of horizontal surfaces, high-efficiency vacuum cleaner) has been shown in some, although not all, cases to reduce indoor dust, lead dust, and blood lead levels in some, although not all, older homes containing leaded paints (Lanphear et al. 2000b; Rhoads et al. 1999). Decreases of between 17 and 43% in blood lead concentrations were observed in children where regular dust control methods had been used to reduce indoor levels of lead (Rhoads et al. 1999).

Lanphear and Roghmann (1997) and Lanphear et al. (1996a, 1996b, 1998b) studied factors affecting PbBs in urban children and found the following independent predictors of children's PbBs: dust lead loading in homes, African-American race/ethnicity, soil lead levels, ingestion of soil or dirt, lead content and condition of painted 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 PbBs. 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 and Roghmann 1997). Community characteristics such as residence within a city, proportion of 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 PbBs 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 PbBs. An increase in dust lead loading from background to 200 μg/ft^2 was estimated to produce an increase of 23.3% in the percentage of children estimated to have a PbB >10 μg/dL; an increase in water lead concentration from background to 15 μg/L was estimated to produce an increase of 13.7% in the percentage of children estimated to have a PbB level >10 μg/dL; and an increase in soil lead concentration from background to 400 μg/g was estimated to produce an increase of 11.6% in the percentage of children estimated to have a PbB level >10 μg/dL (Lanphear et al. 1998a).

Outdoor lead dust was found to be a more potent contaminant of children's hands than indoor 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).

EPA conducted the Urban Soil Lead Abatement Demonstration Project (USLADP), also known as the "Three City Lead Study," in Boston, Baltimore, and Cincinnati (EPA 1996c). The purpose was to determine whether abatement of lead in soil could reduce PbBs of inner-city children. No significant evidence was found that soil abatement had any direct impact on children's PbBs 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 μg/dL PbB at 11 months postabatement (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 PbBs (2.2–2.7 μg/dL). EPA reanalyzed the data from the USLADP in an integrated report (EPA 1996c). 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 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 PbBs in children. These include the site-specific exposure scenario, the magnitude of the remediation, and the magnitude of additional sources of lead exposure.

Authors of a study of PbBs in children in Toronto, Canada, before and after abatement of lead-contaminated soil and house dust found that 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).

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 PbBs 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.

Seasonal variations in PbBs in children have been observed in a number of studies. Mean PbBs in the State of New York have been shown to increase by 15–30% in the late summer as compared to mean values obtained during late winter/early spring (Haley and Talbot 2004). Blood lead measurements taken from children aged 0–6 years in Syracuse, New York over a 48-month period beginning in January 1992, showed a regular yearly periodicity in blood lead concentrations, which peaked in the late summer (Johnson and Bretsch 2002). These seasonal variations in PbBs have been attributed to ingestion of lead in soil. Indeed, the work of Johnson and Bretsch (2002), which looked at the relationship between PbBs measured in children and soil lead concentrations within small regional grids (600 m by 600 m) laid out over the City of Syracuse, New York, showed a correlation between the geometric mean PbBs and the median soil lead concentrations (r^2>0.65).

In addition to the ingestion of hand soil/dust through normal hand-to-mouth activity, some children engage in pica behavior (consumption of nonfood 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/day and that a pica child may ingest 5,000 mg or more of soil/day (LaGoy 1987; Mielke et al. 1989). If the soil contains 100 µg/g of lead, an average child may be exposed to 5 µg of lead/day from this source alone (Mielke et al. 1989), and a pica child may be exposed to >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 ≥10 µg/dL in children 6–71 months of age in a multi-site study (Agency for Toxic Substances and Disease Registry 1995).

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 PbBs ≥20 µg/dL that were considered to be attributable to residential renovation and remodeling (CDC 1997d).

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 4 to 63 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 >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. Women who resided in homes that were >30 years old, lived in high-traffic areas for >5 years, or had drunk three 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. (1998a, 2001c) used measured lead isotope ratios ($^{207}Pb/^{206}Pb$ and $^{206}Pb/^{204}Pb$) 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%. Lead release during maternal bone loss and maternal diet appear to be the major sources of lead in breast milk fed infants. Other sources of lead, such as air, soil, and dust are considered to contribute minimally to lead concentrations in infant blood. Mean lead concentration (±SD) in breast milk for participants in the study was 0.73±0.70 µg/kg.

Sowers et al. (2002b) examined the relationship between lead concentrations in breast milk, maternal blood lead concentration, and maternal bone loss in 15 mothers who breast-fed compared to 30 mothers who bottle-fed commercial formula. The data showed a modest correlation ($p<0.07$) between maternal blood lead and breast milk concentrations at 1–2 months postpartum. However, a stronger correlation ($p<0.001$) was observed between the mean extent of bone loss (5.6%) and lead concentrations in breast milk in women who breast-fed between 1.5 and 6 months postpartum.

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 40 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 5–30 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 PbBs in infants drinking water (or formula reconstituted using water) drawn from the same tap. Predicted PbBs in infants only exceeded 10 µg/L when 100% of the water consumed contained 100 µg Pb/L (Gulson et al. 1997a).

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. PbBs 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/m^3 ambient concentrations) had significantly elevated PbBs (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). Based upon data collected from 1987–1994, children aged 1–5 years (n=139) of workers whose occupation resulted in lead exposure had a geometric mean PbB of 9.3 µg/dL as compared to a U.S. population geometric mean of 3.6 µg/dL (Roscoe et al. 1999). Of this group, 52% of the children had PbBs ≥10 µg/dL compared to 8.9% of the U.S. population and 21% had PbBs ≥20 µg/dL compared to 1.1% of the U.S. population (Roscoe et al. 1999). 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 6.5. 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. Recent data by Maas et al. (2005) indicate that high levels of lead are prevalent in inexpensive cosmetic jewelry that is sold to the general public at retail stores (see Section 6.4.5).

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 6.4.5) 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 *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).

Children may be exposed to lead through the inhalation of second-hand smoke. Mannino et al. (2003) employed data from the NHANES III and analyzed PbBs of children aged 4–16 who were exposed to high, low, and intermediate levels of second-hand smoke. Serum levels of the nicotine biomarker cotinine were used to classify the children into one of the three second-hand smoke exposure categories. The geometric mean PbBs were 1.5, 1.9, and 2.6 μg/dL for children with low (≤0.050–0.104 ng/mL), intermediate (0.105–0.562 ng/mL), and high (0.563–14.9 ng/mL) serum cotinine levels, respectively (Mannino et al. 2003).

6.7 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 6.6), individuals living near sites where lead was produced or sites where lead was disposed, and individuals living near one of the 1,272 NPL hazardous waste sites where lead has been detected in some

environmental media (EPA 1986b; HazDat 2006; Murgueytio et al. 1998) also may be at risk for exposure to high levels of lead. Since lead is often detected in tobacco and tobacco smoke, persons who use chewing tobacco or smoke, may have higher PbB levels than persons that do not use these products (Bonanno et al. 2001).

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; 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). Populations living near any of the 1,272 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 2006). 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.

6.8 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 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.

6.8.1 Identification of Data Needs

Physical and Chemical Properties. The physical and chemical properties of lead and its compounds are sufficiently characterized to permit an estimation of the environmental fate of lead to be made (Howe 1981; Lide 1996; Budavari et al. 1989; Sax 1984; Sax and Lewis 1987). Availabilities of the various forms need to be modeled and the connectivities to bioaccessabilities and bioavailabilities determined.

Production, Import/Export, Use, Release, and Disposal. According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit substance release and off-site transfer information to the EPA. The TRI, which contains this information for 2004, became available in May of 2006. This database is updated yearly and should provide a list of industrial production facilities and emissions.

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 lead metal totaled 37,400 metric tons, and exports of lead waste and scraps totaled 88,400 metric tons (Larrabee 1998; Smith 1998). Exports of lead in ore and concentrates and lead materials, excluding scrap, rose from 93,500 and 103,000 metric tons in 1999 to 253,000 and 123,000 metric tons, respectively, in 2003. In 2003, 92,800 metric tons of lead scrap were exported (USGS 2003).

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.

Environmental Fate. Lead released to the atmosphere partitions to surface water, soil, and sediment (EPA 1986a; 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 1979). Inorganic lead compounds are strongly adsorbed to minerals and organic matter in soils and sediments (Chaney et al. 1988; Chuan et al. 1996; EPA 1986a; Gerritse et al. 1981; Sauve et al. 1997). Some work has been conducted to assess the speciation of lead in air, water, and soil (Chaney et al. 1988; Corrin and Natusch 1977; EPA 1986a; Long and Angino 1977; Nerin et al. 1999; Spear et al. 1998). Lead is a naturally occurring element and is extremely persistent in the environment. Additional information on the atmospheric 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. Additional data regarding the chemical speciation and the transformation pathways of lead in soils and water with varying properties such as pH, oxygen content and salinity are necessary to fully understand the environmental fate of lead in soils and water.

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 (MPCA 1987). 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). Dermal absorption models of lead would be useful in modeling total exposure pathways of lead.

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 1982c, 1986a, 1988b, 1989d, 1989e, 1990; Lee et al. 1989; Maenhaut et al. 1979; Mielke 1993; Mielke et al. 1983, 1984/1985, 1989). More current data (1995–1996) on lead in ambient and indoor air, drinking water, and foodstuffs for residents in Arizona, EPA Region V (Illinois, Indiana, Michigan, Minnesota, Ohio, and Wisconsin), and Maryland are available through the NHEXAS (Bonanno et al. 2001; Clayton et al. 2002; O'Rourke et al. 1999; Pellizzari et al. 1999; Ryan et al. 2000; Thomas et al. 1999). 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. Lead has been found in tobacco and tobacco smoke and higher levels of lead have been detected in indoor air of the homes of smokers when compared to non smokers (Bonanno et al. 2001; Mannino et al. 2003). It is unclear whether the source of this lead is from plant uptake, atmospheric deposition of lead compounds to the surface of tobacco plants, or from tobacco plants being grown in soils that had previously been treated with arsenate pesticides. A study to determine the source of this lead in tobacco is needed in order to help reduce the risk of lead exposure to smokers and those that may inhale second hand smoke.

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 burden of lead to assess the potential risk of adverse health effects in populations living in the vicinity of hazardous waste sites.

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 1977b, 1977c; 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 de Benzo et al. 1989; Delves and Campbell 1988; Manton and Cook 1984; NIOSH 1977b, 1977c; Que Hee et al. 1985a). PbBs have been correlated with ambient air exposure levels and dust, and dietary intake levels (Rabinowitz et al. 1985). In their critical evaluation of reports of historic occupational aerosol exposure to lead, Vincent and Werner (2003) recommended that exposure measurements be made using sampling techniques and strategies that relate to the health effects underlying the need for exposure assessment. Additionally, sufficient detail must be included so that the quality and value of the data can be judged. This is necessary so the data can be pooled for broad hazard surveillance purposes. 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. The most recent NHANES Report, containing data from 1999 to 2002 and released in 2005, contains blood lead levels for the U.S. population (CDC 2005a, 2005b). The data pertaining to lead levels in the U.S. population are summarized in Tables 6-12 and 6-13. 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. Lead levels in blood (CDC 2005a, 2005b) and urine

(CDC 2003, 2005b) of children are available from the NHANES monitoring data, and have been summarized in Tables 6-12 and 6-13.

Child health data needs relating to susceptibility are discussed in Section 3.12.2, Identification of Data Needs: Children's Susceptibility.

Exposure Registries. No exposure registries for lead were located. This substance is not currently one of the compounds for which a sub-registry has been established in the National Exposure Registry. The substance will be considered in the future when chemical selection is made for sub-registries 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.

6.8.2 Ongoing Studies

The Federal Research in Progress (FEDRIP 2005) database provides additional information obtainable from a few ongoing studies that may fill in some of the data needs identified in Section 6.8.1. These studies are summarized in Table 6-17.

Table 6-17. Ongoing Research Regarding the Environmental Fate and Exposure of Humans to Lead

Investigator	Affiliation	Research description	Sponsor
Blum CB	Columbia University, New York, New York	The estimation of bioavailability to lead and arsenic from soil currently use assumptions based on bioavailability data from animal or *in vitro* models. Using the technique of stable Pb isotope dilution, a method was developed for estimating soil Pb bioavailability in humans. This model examines changes in the ratio of ^{206}Pb to ^{207}Pb in blood, following the ingestion of trace quantities of Pb-contaminated soils.	National Institute of Environmental Health Sciences
Cochran JK and Veron A	SUNY at Stony Brook, Stony Brook, New York	This three-year award for United States-France collaboration in environmental geochemistry involves State University of New York at Stony Brook and the Centre Europeen de Recherche et d'Enseignement de Geosciences in Marseilles, France. The investigators will determine the history of input rates and sources of stable lead to coastal areas.	National Science Foundation
Basta NK and Lower SK	Ohio State University, Columbus, Ohio	The goals of this project are to: (1) determine the ability of chemical speciation methods that measure heavy metal bioavailability; (2) estimate ecotoxicity of contaminated soil; (3) determine the effect of soil chemical properties on chemical speciation and heavy metal bioavailability in contaminated soil and the ability of soil chemical properties to define ecotoxicity categories in development of ecological soil screening levels; (4) determine the ability of diammonium phosphate to reduce bioavailable chemical species of heavy metal contaminants in soil.	Department of Agriculture
Spraks DL	University of Delaware, Newark, Delaware	The goals of this project are to (1) determine the effect of reaction conditions and residence time on sorption/release of important metals/metalloids (Cu, Cd, Cr, Ni, Pb, As) on soil components and Delaware soils; and (2) ascertain metal/metalloid reaction mechanisms on soil components/soils using molecular level spectroscopic (e.g., x-ray absorption fine structure [XAFS] and microscopic [atomic force microscopy (AFS]) techniques. Metal/metalloid sorption studies will be examined as a function of residence time, pH, and total metal loading on soil components/soils, using a pH-stat batch method.	Department of Agriculture

Source: FEDRIP 2005