

6. POTENTIAL FOR HUMAN EXPOSURE

6.1 OVERVIEW

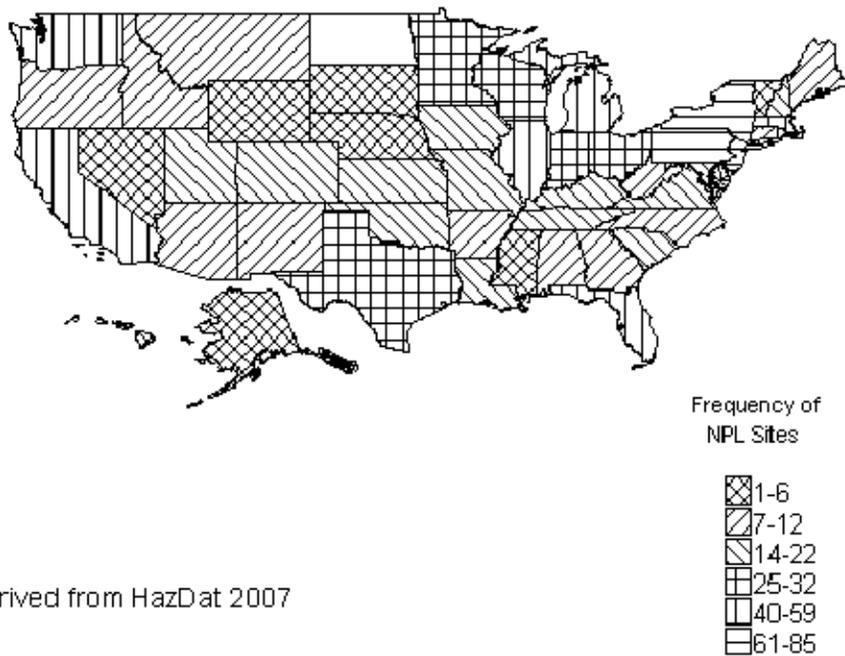
Cadmium has been identified in at least 1,014 of the 1,669 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2007). Cadmium compounds have been identified in at least 3 of the 1,669 hazardous waste sites. However, the number of sites evaluated for cadmium is not known. The frequency of these sites can be seen in [Figures 6-1](#) and [6-2](#). Of the 1,014 sites where cadmium has been identified, 1,005 are located within the United States, 6 are located in the Commonwealth of Puerto Rico (not shown), 2 are located in Guam, and 1 is located in the Virgin Islands. All sites where cadmium compounds were detected are located in the United States.

Cadmium occurs in the earth's crust at an abundance of 0.1–0.5 ppm and is commonly associated with zinc, lead, and copper ores. It is also a natural constituent of ocean water, with average levels between <5 and 110 ng/L; with higher levels reported near coastal areas and in marine phosphates and phosphorites (Morrow 2001). Natural emissions of cadmium to the environment can result from volcanic eruptions, forest fires, generation of sea salt aerosols, or other natural phenomena (EPA 1985a; Morrow 2001; Shevchenko et al. 2003). Cadmium is refined and consumed for use in batteries (83%), pigments (8%), coatings and platings (7%), stabilizers for plastics (1.2%), and nonferrous alloys, photovoltaic devices, and other (0.8%) (USGS 2008). Nonferrous metal mining and refining, manufacture and application of phosphate fertilizers, fossil fuel combustion, and waste incineration and disposal are the main anthropogenic sources of cadmium in the environment.

Cadmium can be released to the atmosphere through metal production activities, fossil fuel combustion, and waste incineration. The main cadmium compounds found in air are cadmium oxide, chloride, and sulfate, and these compounds are expected to undergo minimal transformation in the atmosphere (EPA 1980d). The major fate of cadmium in air is through transport and deposition. Cadmium can travel long distances in the atmosphere and then deposit (wet or dry) onto surface soils and water, which can result in elevated cadmium levels even in remote locations (Shevchenko et al. 2003). Results from the 2006 final report of EPA's Urban Air Toxic Monitoring program reported average daily cadmium levels of <0.01 $\mu\text{g}/\text{m}^3$ at several monitoring sites throughout the United States (EPA 2007). These sites include: Bountiful, Utah; Northbrook, Illinois; Austin, Texas; St. Louis, Missouri; Indianapolis, Indiana; and Birmingham, Alabama (EPA 2007). Atmospheric concentrations of cadmium are generally highest in the vicinity of cadmium-emitting industries (Elinder 1985a; Pirrone et al. 1996). Due to advances in pollution control technology, cadmium emissions to air are not expected to increase, even though

6. POTENTIAL FOR HUMAN EXPOSURE

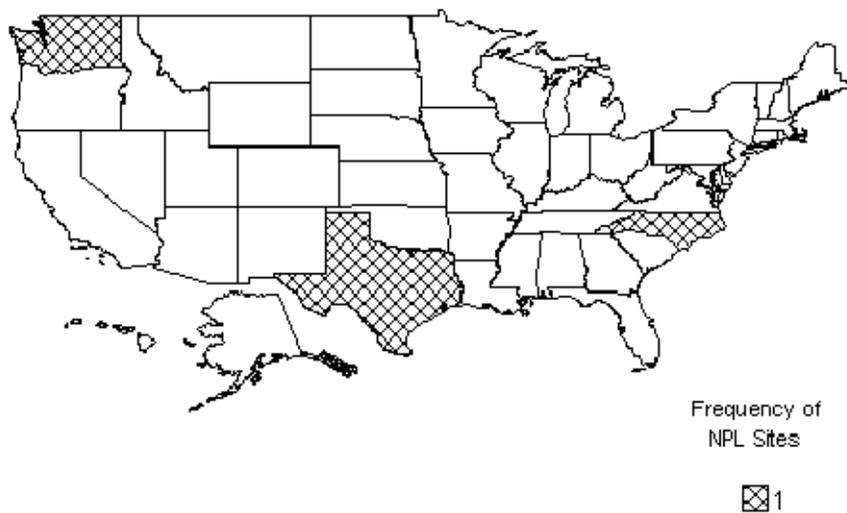
Figure 6-1. Frequency of NPL Sites with Cadmium Contamination



Derived from HazDat 2007

6. POTENTIAL FOR HUMAN EXPOSURE

Figure 6-2. Frequency of NPL Sites with Cadmium Compounds Contamination



Derived from HazDat 2007

6. POTENTIAL FOR HUMAN EXPOSURE

cadmium-emitting industries are expected to grow (Herron 2003; Morrow 2010; Schulte-Schrepping and Piscator 2002). Except for those who live near cadmium-emitting industries, inhalation of cadmium in the ambient air is not a major source of exposure.

The main sources of cadmium to soil include atmospheric deposition and direct application methods such as phosphate fertilizer use and sewage sludge disposal. Some phosphate fertilizers can contain up to 300 mg Cd/kg (Alloway and Steinnes 1999). Wet and dry deposition of cadmium from the atmosphere may also contribute sizable amounts of cadmium to soil in the areas surrounding sources of atmospheric emissions (EPA 1985a; Mielke et al. 1991). Cadmium's mobility in soil depends on several factors including the pH of the soil and the availability of organic matter. Generally, cadmium will bind strongly to organic matter and this will, for the most part, immobilize cadmium (Autier and White 2004). However, immobilized cadmium is available to plant life and can easily enter the food supply. Cadmium in soil tends to be more available when the soil pH is low (acidic) (Elinder 1992).

Water sources near cadmium-emitting industries, both with historic and current operations, have shown a marked elevation of cadmium in water sediments and aquatic organisms (Angelo et al. 2007; Arnason and Fletcher 2003; Brumbaugh et al. 2005; Mason et al. 2000; Paulson 1997). In surface water and groundwater, cadmium can exist as the hydrated ion or as ionic complexes with other inorganic or organic substances. While soluble forms may migrate in water, cadmium is relatively nonmobile in insoluble complexes or adsorbed to sediments. Cadmium is taken up and retained by aquatic and terrestrial plants and is concentrated in the liver and kidney of animals that eat the plants (Elinder 1985a).

For the U.S. population, cadmium exposure through the drinking water supply is of minor concern. EPA requires water suppliers to limit the cadmium concentration in water to <5 µg/L (EPA 2006a).

In the United States, the largest source of cadmium exposure for nonsmoking adults and children is through dietary intake (NTP 2005). Based on the mean cadmium daily intakes of males and females aged 6–60 years reported by Choudhury et al. (2001), age-weighted mean cadmium intakes of 0.35 µg/kg/day for males and 0.30 µg/kg/day for females were calculated for U.S. nonsmokers. In general, vegetables, particularly leafy vegetables such as lettuce (0.051 mg/kg) and spinach (0.124 mg/kg), have the highest concentrations of cadmium; the concentrations of cadmium in all vegetables ranged from 0.001 to 0.124 mg/kg (FDA 2010; Morrow 2001). Peanuts, soybeans, and sunflower seeds have naturally high levels of cadmium (Morrow 2001); the mean concentration of cadmium in legumes and nuts ranged from 0.001 to 0.054 mg/kg (FDA 2010). People who regularly consume shellfish and organ meats (liver and

6. POTENTIAL FOR HUMAN EXPOSURE

kidney) have an increased risk of cadmium exposure, as these organisms tend to accumulate cadmium (Elinder 1985a).

Tobacco leaves naturally accumulate cadmium (Morrow 2001). Cadmium levels in cigarettes vary greatly depending on the source of production. Cigarettes produced in Mexico were found to have the highest level of cadmium per cigarette (arithmetic mean [AM] \pm arithmetic standard deviation [ASD] = 2.03 $\mu\text{g}/\text{cigarette} \pm 0.33$), while cigarettes from India were found to have the lowest (arithmetic mean \pm arithmetic standard deviation = 0.35 $\mu\text{g}/\text{cigarette} \pm 0.09$). The arithmetic mean for the United States was 1.07 $\mu\text{g}/\text{cigarette} \pm 0.11$ (Watanabe et al. 1987). Tobacco contains approximately 0.5–2.0 μg cadmium per cigarette, and about 10% is inhaled when smoked (Morrow 2010). The geometric mean blood cadmium level for the heavy smoker subgroup in New York City was reported as 1.58 $\mu\text{g}/\text{L}$, compared to the geometric mean of 0.77 $\mu\text{g}/\text{L}$ for all New York City adults (McKelvey et al. 2007).

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 2005). This is not an exhaustive list. Manufacturing and processing facilities are required to report information to the TRI only if they employ 10 or more full-time employees; if their facility is included in Standard Industrial Classification (SIC) Codes 10 (except 1011, 1081, and 1094), 12 (except 1241), 20–39, 4911 (limited to facilities that combust coal and/or oil for the purpose of generating electricity for distribution in commerce), 4931 (limited to facilities that combust coal and/or oil for the purpose of generating electricity for distribution in commerce), 4939 (limited to facilities that combust coal and/or oil for the purpose of generating electricity for distribution in commerce), 4953 (limited to facilities regulated under RCRA Subtitle C, 42 U.S.C. section 6921 et seq.), 5169, 5171, and 7389 (limited S.C. section 6921 et seq.), 5169, 5171, and 7389 (limited to facilities primarily engaged in solvents recovery services on a contract or fee basis); and if their facility produces, imports, or processes $\geq 25,000$ pounds of any TRI chemical or otherwise uses $>10,000$ pounds of a TRI chemical in a calendar year (EPA 2005).

Additional releases of cadmium to the environment occur from natural sources and from processes such as combustion of fossil fuel, incineration of municipal or industrial wastes, or land application of sewage sludge or fertilizer (EPA 1985a). Quantitative information on releases of cadmium to specific environmental media is discussed below.

6. POTENTIAL FOR HUMAN EXPOSURE

6.2.1 Air

Estimated releases of 731 pounds (~0.3 metric tons) of cadmium to the atmosphere from 41 domestic manufacturing and processing facilities in 2009, accounted for about 0.18% of the estimated total environmental releases of cadmium from facilities required to report to the TRI (TRI09 2011). These releases are summarized in [Table 6-1](#). Estimated releases of 11,567 pounds (~5.2 metric tons) of cadmium compounds to the atmosphere from 85 domestic manufacturing and processing facilities in 2009, accounted for about 0.55% of the estimated total environmental releases of cadmium from facilities required to report to the TRI (TRI09 2011). These releases are summarized in [Table 6-2](#).

Cadmium is released to the atmosphere from both natural and anthropogenic sources. Cadmium is widely distributed in the earth's crust (EPA 1985a) with concentrations reported between 0.1 and 0.5 ppm and higher levels in sedimentary rocks (Morrow 2001). Consequently, cadmium may be released to the air from entrainment of dust particles, volcanic eruptions, forest fires, or other natural phenomena (EPA 1985a; Morrow 2001). Cadmium exists in ocean waters at average levels ranging from <5 to 110 ng/L and may transport to the atmosphere through natural processes like generation of sea-salt aerosols (Morrow 2001; Shevchenko et al. 2003). Increased cadmium levels in the air over the Russian Arctic have been detected during the summer and autumn seasons and are believed to be attributed to natural processes, while the levels detected during the winter and spring seasons were due to anthropogenic sources (Shevchenko et al. 2003).

However, industrial activities are the main sources of cadmium release to air (EPA 1985a), and emissions from anthropogenic sources have been found to exceed those of natural origin by an order of magnitude (IARC 1993). Major industrial sources of cadmium emissions include zinc, lead, copper, and cadmium smelting operations; coal and oil-fired boiler; other urban and industrial emissions; phosphate fertilizer manufacture; road dust; and municipal and sewage sludge incinerators (Alloway and Steinnes 1999; Morrow 2001). Emission of cadmium through nonferrous metal production in 1995 was highest in Asia with 1,176 tonnes and North America emitting 191 tonnes. Estimated emissions of cadmium from municipal waste and sewage sludge incineration in North America were 8 and 7 tonnes/year, respectively, in the mid-1990s (Pacyna and Pacyna 2001). Additional sources that contribute negligible amounts of cadmium are rubber tire wear, motor oil combustion, cement manufacturing, and fertilizer and fungicide application (Wilber et al. 1992). Average cadmium emission factors for combustion of coal and oil are about 0.1 and 0.05 g/ton, respectively. Cement production releases an estimated 0.01 g/ton cement and

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-1. Releases to the Environment from Facilities that Produce, Process, or Use Cadmium^a

State ^c	RF ^d	Air ^e	Reported amounts released in pounds per year ^b				Total release		
			Water ^f	UI ^g	Land ^h	Other ⁱ	On-site ^j	Off-site ^k	On- and off-site
WI	1	35	0	0	181	0	35	181	216
Total	41	731	338	142,567	253,065	6,059	357,597	45,164	402,760

^aThe 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.

^bData in TRI are maximum amounts released by each facility.

^cPost office state abbreviations are used.

^dNumber of reporting facilities.

^eThe sum of fugitive and point source releases are included in releases to air by a given facility.

^fSurface water discharges, waste water treatment-(metals only), and publicly owned treatment works (POTWs) (metal and metal compounds).

^gClass I wells, Class II-V wells, and underground injection.

^hResource Conservation and Recovery Act (RCRA) subtitle C landfills; other onsite landfills, land treatment, surface impoundments, other land disposal, other landfills.

ⁱStorage only, solidification/stabilization (metals only), other off-site management, transfers to waste broker for disposal, unknown

^jThe sum of all releases of the chemical to air, land, water, and underground injection wells.

^kTotal amount of chemical transferred off-site, including to POTWs.

RF = reporting facilities; UI = underground injection

Source: TRI09 2011 (Data are from 2009)

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-2. Releases to the Environment from Facilities that Produce, Process, or Use Cadmium Compounds^a

State ^c	RF ^d	Reported amounts released in pounds per year ^b							
		Air ^e	Water ^f	UI ^g	Land ^h	Other ⁱ	Total release		
							On-site ^j	Off-site ^k	On- and off-site
AK	3	129	3	0	61,176	0	61,308	0	61,308
AL	4	221	122	0	698,581	0	698,681	243	698,924
AR	1	0	0	0	0	131	0	131	131
AZ	3	611	5	0	63,392	0	63,753	255	64,008
CA	1	0	0	0	115	0	0	115	115
CO	1	1	0	0	0	0	1	0	1
CT	1	0	0	0	0	0	0	0	0
FL	1	0	0	0	0	0	0	0	0
GA	3	8	3	0	0	0	9	3	11
ID	2	6,409	6	0	386,455	0	392,870	0	392,870
IL	5	59	117	215	33,651	1,553	1,289	34,306	35,595
IN	3	76	6	0	13,361	41	82	13,402	13,484
LA	1	0	0	0	0	0	0	0	0
MA	3	25	251	0	0	1,021	25	1,272	1,297
MD	1	0	0	0	0	12,400	0	12,400	12,400
MI	1	0	5	0	250	0	0	255	255
MO	1	593	265	0	6,178	9	6,987	58	7,045
NC	1	0	0	0	0	0	0	0	0
NE	1	250	0	0	250	21,000	250	21,250	21,500
NJ	2	5	1	0	1,488	140	5	1,629	1,634
NV	4	40	0	0	165,471	0	165,506	5	165,511
NY	1	0	21	0	0	0	21	0	21
OH	10	37	102	0	4,264	15,604	1,471	18,536	20,007
OK	1	31	0	0	0	0	31	0	31
PA	8	646	326	0	2,596	9,442	721	12,289	13,010
SC	1	255	10	0	6,165	0	265	6,165	6,430
TN	5	1,997	597	0	192,927	428	195,516	433	195,949
TX	7	30	335	2,118	334,113	2,583	334,650	4,529	339,179
UT	4	130	133	0	54,954	107	55,217	107	55,324
WA	1	2	2	0	0	5	4	5	9
WI	3	10	27	0	1	6,623	10	6,651	6,661

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-2. Releases to the Environment from Facilities that Produce, Process, or Use Cadmium Compounds^a

State ^c	RF ^d	Reported amounts released in pounds per year ^b							Total release	
		Air ^e	Water ^f	UI ^g	Land ^h	Other ⁱ	On-site ^j	Off-site ^k	On- and off-site	
WV	1	2	7	0	1,018	0	518	509	1,027	
Total	85	11,567	2,346	2,333	2,026,406	71,087	1,979,190	134,549	2,113,739	

^aThe 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.

^bData in TRI are maximum amounts released by each facility.

^cPost office state abbreviations are used.

^dNumber of reporting facilities.

^eThe sum of fugitive and point source releases are included in releases to air by a given facility.

^fSurface water discharges, waste water treatment-(metals only), and publicly owned treatment works (POTWs) (metal and metal compounds).

^gClass I wells, Class II-V wells, and underground injection.

^hResource Conservation and Recovery Act (RCRA) subtitle C landfills; other onsite landfills, land treatment, surface impoundments, other land disposal, other landfills.

ⁱStorage only, solidification/stabilization (metals only), other off-site management, transfers to waste broker for disposal, unknown

^jThe sum of all releases of the chemical to air, land, water, and underground injection wells.

^kTotal amount of chemical transferred off-site, including to POTWs.

RF = reporting facilities; UI = underground injection

Source: TRI09 2011 (Data are from 2009)

6. POTENTIAL FOR HUMAN EXPOSURE

pig iron and steel production releases an estimated 0.1 g/ton (Pacyna and Pacyna 2001). Atmospheric cadmium exists mainly in the forms of cadmium oxide and cadmium chloride (Morrow 2001).

Cadmium emissions have decreased dramatically since the 1960s as primary cadmium producers now use the electrolytic process and pollution control technologies such as agglomeration, electrostatic purification of gas exhaust, and exhaust filtration have been implemented (Herron 2003; Morrow 2001; Schulte-Schrepping and Piscator 2002). Anthropogenic cadmium emissions have decreased by over 90% in the last 50 years (Morrow 2010).

There is a potential for release of cadmium to air from hazardous waste sites. Cadmium has been detected in air samples collected at 50 of the 1,014 NPL hazardous waste sites where cadmium was detected in some environmental medium (HazDat 2007). Cadmium compounds were detected in air samples collected at one of three NPL hazardous waste sites where cadmium compounds were detected. The HazDat information used includes data from NPL sites only.

6.2.2 Water

Estimated releases of 338 pounds (~0.15 metric tons) of cadmium to surface water from 41 domestic manufacturing and processing facilities in 2009, accounted for about 0.084% of the estimated total environmental releases from facilities required to report to the TRI. This estimate includes releases to wastewater treatment and publicly owned treatment works (TRI09 2011). These releases are summarized in [Table 6-1](#). Estimated releases of 2,346 pounds (~1.1 metric tons) of cadmium compounds to surface water from 85 domestic manufacturing and processing facilities in 2009, accounted for about 0.11% of the estimated total environmental releases from facilities required to report to the TRI. This estimate includes releases to wastewater treatment and publicly owned treatment works (TRI09 2011). These releases are summarized in [Table 6-2](#).

Cadmium may be released to water by natural weathering processes, by discharge from industrial facilities or sewage treatment plants, atmospheric deposition, by leaching from landfills or soil, or phosphate fertilizers (EPA 1981a, 1985a; IJC 1989; Morrow 2001). Cadmium may also leach into drinking water supplies from pipes in the distribution system (Elinder 1985a). The average level of cadmium in ocean water has been reported between <5 and 110 ng/L, with higher levels reported near coastal areas and in marine phosphates and phosphorites (Morrow 2001).

6. POTENTIAL FOR HUMAN EXPOSURE

Smelting of nonferrous metal ores has been estimated to be the largest anthropogenic source of cadmium released into the aquatic environment. Cadmium contamination can result from entry into aquifers of mine drainage water, waste water, tailing pond overflow, and rainwater runoff from mine areas (IARC 1993). The upper Clark Fork River in Montana is contaminated with large amounts of cadmium from past mining activities between 1880 and 1972. While mining wastes are no longer released into the river, an estimated 14.5 million cubic meters of tailings have been incorporated into the river bed, floodplain, and reservoir sediments (Canfield et al. 1994). Other human sources include spent solutions from plating operations and phosphate fertilizers. Cadmium constitutes up to 35 mg/kg of phosphorous pentoxide, a component of phosphate-based fertilizers, in the United States (IARC 1993). Atmospheric fallout of cadmium to aquatic systems is another major source of cadmium to the environment (IARC 1993; Muntau and Baudo 1992).

A large proportion of the cadmium load in the aquatic environment is due to diffuse pollution originating from many different sources rather than from point sources. In the estuarine portion of the Hudson River, it has been found that more cadmium was released from agricultural and urban run-off than from industrial and municipal sewage treatment plants (Muntau and Baudo 1992). In an urban environment, there are also multiple sources of cadmium to waste water. In an urban waste water study conducted in the United Kingdom, cadmium was detected in the waste water originating from industrial, commercial, and private sectors, with the highest average cadmium concentration detected in the waste water of new (<5 years old) private housing (0.375 µg/L) (Rule et al. 2006).

There is also a potential for release of cadmium to water from hazardous waste sites. Cadmium has been detected in surface water samples collected at 354 of the 1,014 NPL hazardous waste sites, and in groundwater samples collected at 675 of the 1,014 NPL hazardous waste sites where cadmium has been detected in some environmental medium (HazDat 2007). The HazDat information used includes data from NPL sites only.

6.2.3 Soil

Estimated releases of 253,065 pounds (~115 metric tons) of cadmium to soils from 41 domestic manufacturing and processing facilities in 2009, accounted for about 63% of the estimated total environmental releases of cadmium from facilities required to report to the TRI (TRI09 2011). An additional 142,567 pounds (~65 metric tons), constituting about 35% of the total environmental emissions, were released via underground injection and to Class I wells, Class II-V wells (TRI09 2011).

6. POTENTIAL FOR HUMAN EXPOSURE

These releases are summarized in [Table 6-1](#). Estimated releases of 2,026,406 pounds (~919 metric tons) of cadmium compounds to soils from 85 domestic manufacturing and processing facilities in 2009, accounted for about 96% of the estimated total environmental releases from facilities required to report to the TRI. An additional 2,333 pounds (~1 metric tons), constituting about 0.1% of the total environmental emissions, were released via underground injection and to Class I wells, Class II-V wells (TRI09 2011). These releases are summarized in [Table 6-2](#).

Major sources of cadmium to soil include atmospheric emissions, direct application, and accidental or fugitive contamination. Direct application emissions refer to phosphate fertilizers, phosphogypsum and other byproduct gypsums (from the manufacture of phosphoric acid and phosphorite), sewage sludges, composted municipal solid waste, and residual ashes from wood, coal, or other types of combustion. Contamination sources include industrial site contamination, mine waste dumps, and corrosion of metal structures (Alloway and Steinnes 1999).

Approximately 61% of the 5.6 million dry tons of sewage sludge produced annually in the United States is landspread (NRC 2002). The EPA ceiling limit for the cadmium content of sludge applied to land is 85 mg/kg in sewage sludge, the cumulative pollution loading rate is 39 kg/ha, and the maximum annual cadmium loading of 1.9 kg-ha⁻¹·year⁻¹ (EPA 2011a). Estimated cadmium concentrations in sewage sludge range from 0.21 to >11.8 mg/kg (EPA 2009a). Sludges from treatment plants that serve cadmium industries (i.e., battery manufacturing) tend to have higher levels of cadmium (Alloway and Steinnes 1999).

Phosphate fertilizers are a major source of cadmium input to agricultural soils (EPA 1985a). The natural cadmium concentration in phosphates ranges from 3 to 100 µg/g (EPA 1985a; Singh 1994). Some may contain up to 300 mg Cd/kg (Alloway and Steinnes 1999). It is estimated that over 8 million tons of phosphate fertilizer were used in the United States in 2010 (USDA 2012). Any soil treated with these fertilizers will have a cadmium input, but exactly how much will vary (Alloway and Steinnes 1999). For example, continuous fertilization with a high rate of triple super-phosphate (1,175 kg P-ha⁻¹·year⁻¹) for a period of 36 years resulted in a 14-fold increase in cadmium content of surface soils (Singh 1994).

Wet and dry deposition of cadmium from the atmosphere may also contribute sizable amounts of cadmium to soil in the areas surrounding sources of atmospheric emissions, such as incinerators and vehicular traffic, which may release cadmium from burned fuel and tire wear (EPA 1985a; Mielke et al. 1991). High-temperature sources, such as smelters and incinerators, release small particles that are ideal

6. POTENTIAL FOR HUMAN EXPOSURE

for long-range atmospheric transport. Also, vapors emitted from high temperature processes will preferentially condense onto smaller particles, thus making vapor emissions available for transport (Steinnes and Friedland 2006). Aerosols containing cadmium can be carried very long distances in the atmosphere before being deposited to soils. In the soils in southern Norway, most of the cadmium and other heavy metals that are deposited from the atmosphere originate from other parts of Europe (Alloway and Steinnes 1999). Long-range atmospheric deposition is more evident in organic-rich soils as they have a tendency to concentrate heavy metals (Steinnes and Friedland 2006).

There is also a potential for release of cadmium to soil from hazardous waste sites. Cadmium has been detected in soil samples collected at 606 of the 1,014 NPL hazardous waste sites and in sediment samples collected at 392 of the 1,014 NPL hazardous waste sites where cadmium has been detected in some environmental medium (HazDat 2007). The HazDat information used includes data from NPL sites only.

6.3 ENVIRONMENTAL FATE

6.3.1 Transport and Partitioning

Cadmium is expected to partition primarily to soil (80–90%) when released to the environment. Although particulate and vapor cadmium may be released to the air, the net flux to soil will be positive as cadmium will eventually deposit onto soils (Morrow 2001; Wilber et al. 1992).

Cadmium and cadmium compounds have negligible vapor pressures (see [Table 4-2](#)) but can be released to the environment by emissions from municipal waste incinerators, nonferrous metal production, and other high-temperature processes (Morrow 2001). Cadmium emitted to the atmosphere from combustion processes condense onto very small particulates that are in the respirable range (<10 μm) and are subject to long-range transport (Steinnes and Friedland 2006; Wilber et al. 1992). These cadmium pollutants may be transported from a hundred to a few thousand kilometers and have a typical atmospheric residence time of about 1–10 days before deposition occurs (EPA 1980d). Larger cadmium-containing particles from smelters and other pollutant sources are also removed from the atmosphere by gravitational settling, with substantial deposition in areas downwind of the pollutant source. Cadmium-containing particulates may dissolve in atmospheric water droplets and be removed from air by wet deposition.

Cadmium is more mobile in aquatic environments than most other heavy metals (e.g., lead). In most natural surface waters, the affinities of complexing ligands for cadmium generally follow the order of humic acids > CO_3^{2-} > $\text{OH}^- \geq \text{Cl}^- \geq \text{SO}_4^{2-}$ (EPA 1979). In unpolluted natural waters, most cadmium

6. POTENTIAL FOR HUMAN EXPOSURE

transported in the water column will exist in the dissolved state as the hydrated ion $\text{Cd}(\text{H}_2\text{O})_6^{2+}$. Minor amounts of cadmium are transported with the coarse particulates, and only a small fraction is transported with the colloids. In unpolluted waters, cadmium can be removed from solution by exchange of cadmium for calcium in the lattice structure of carbonate minerals (EPA 1979). In polluted or organic-rich waters, adsorption of cadmium by humic substances and other organic complexing agents plays a dominant role in transport, partitioning, and remobilization of cadmium (EPA 1979). Cadmium concentration in water is inversely related to the pH and the concentration of organic material in the water (EPA 1979). Because cadmium exists only in the +2 oxidation state in water, aqueous cadmium is not strongly influenced by the oxidizing or reducing potential of the water. However, under reducing conditions, cadmium may form cadmium sulfide, which is poorly soluble and tends to precipitate (EPA 1983c; McComish and Ong 1988). Free (ionic) cadmium seems to be the toxic form and becomes much more prevalent at low salinity (Sprague 1986). Cadmium has a relatively long residence time in aquatic systems. In Lake Michigan, a mean residence time of 4–10 years was calculated for cadmium compared to 22 years calculated for mercury (Wester et al. 1992).

Precipitation and sorption to mineral surfaces, hydrous metal oxides, and organic materials are the most important processes for removal of cadmium to bed sediments. Humic acid is the major component of sediment responsible for adsorption. Sorption increases as the pH increases (EPA 1979). Sediment bacteria may also assist in the partitioning of cadmium from water to sediments (Burke and Pfister 1988). Both cadmium-sensitive and cadmium-resistant bacteria reduced the cadmium concentration in the water column from 1 ppm to between 0.2 and 0.6 ppm, with a corresponding increase in cadmium concentration in the sediments in the simulated environment (Burke and Pfister 1988). Studies indicate that concentrations of cadmium in sediments are at least one order of magnitude higher than in the overlying water (EPA 1979). The mode of sorption of cadmium to sediments is important in determining its disposition to remobilize. Cadmium associated with carbonate minerals, precipitated as stable solid compounds or co-precipitated with hydrous iron oxides, is less likely to be mobilized by resuspension of sediments or biological activity. Cadmium that is adsorbed to mineral surfaces such as clay, or to organic materials, is more easily bioaccumulated or released in the dissolved state when the sediment is disturbed (EPA 1979). Cadmium may redissolve from sediments under varying ambient conditions of pH, salinity, and redox potential (DOI 1985; EPA 1979; Feijtel et al. 1988; Muntau and Baudo 1992). Cadmium is not known to form volatile compounds in the aquatic environment, so partitioning from water to the atmosphere does not occur (EPA 1979).

6. POTENTIAL FOR HUMAN EXPOSURE

Debusk et al. (1996) studied the retention and compartmentalization of lead and cadmium in wetland microcosms. Differences between measured concentrations in inflow and outflow samples indicated that approximately half of the added cadmium was retained in the wetland microcosms. Experiments showed that nearly all trace metals were present in the sediments as sulfides, limiting their bioavailability and toxicity. The results of their analyses and a lack of noticeable biological effects suggested that in wetlands containing organic sediments, the sediment chemistry dominates cycling of the trace metals.

In soils, pH, oxidation-reduction reactions, and formation of complexes are important factors affecting the mobility of cadmium (Bermond and Bourgeois 1992; Herrero and Martin 1993). Cadmium can participate in exchange reactions on the negatively charged surface of clay minerals. In acid soils, the reaction is reversible. However, adsorption increases with pH and may become irreversible (Herrero and Martin 1993). Cadmium also may precipitate as insoluble cadmium compounds, or form complexes or chelates by interaction with organic matter. Available data suggest that organic matter is more effective than inorganic constituents in keeping cadmium unavailable (McBride 1995). Examples of cadmium compounds found in soil are $\text{Cd}_3(\text{PO}_4)_2$, CdCO_3 , and $\text{Cd}(\text{OH})_2$ (Herrero and Martin 1993). These compounds are formed as the pH rises. It has been found that about 90% of cadmium in soils remains in the top 15 cm (Anonymous 1994).

The mobility and plant availability of cadmium in wetland soils are substantially different from upland soils. Cadmium tends to be retained more strongly in wetland soils and is more available to plants under upland conditions (Gambrell 1994). Debusk et al. (1996) compared heavy metal uptake by cattails and duckweed wetland microcosms and found that duckweed, on a whole-plant basis, accumulates cadmium more effectively than cattail does. The potential cadmium removal rate for duckweed is 2–4 mg $\text{Cd}/\text{m}^2/\text{day}$.

Cadmium in soils may leach into water, especially under acidic conditions (Elinder 1985a; EPA 1979). Roy et al. (1993) demonstrated that chlorine complexation in the leachate of ash from a municipal solid waste incinerator can result in a decrease in cadmium sorption by two common clays, kaolinite and illite. They also found that cationic competitive sorption enhances mobility in soils. Cadmium-containing soil particles may also be entrained into the air or eroded into water, resulting in dispersion of cadmium into these media (EPA 1985a). Contamination of soil by cadmium is of concern because the cadmium is taken up efficiently by plants and, therefore, enters the food chain for humans and other animals. A low soil pH, which is becoming prevalent in many areas of the world due to acid rain, increases the uptake of cadmium by plants (Elinder 1992).

6. POTENTIAL FOR HUMAN EXPOSURE

Aquatic and terrestrial organisms bioaccumulate cadmium (Handy 1992a, 1992b; Kuroshima 1992; Naqvi and Howell 1993; Roseman et al. 1994; Suresh et al. 1993). Cadmium concentrates in freshwater and marine animals to concentrations hundreds to thousands of times higher than in the water (EPA 1979). Reported bioconcentration factors (BCFs) range from <200 to 18,000 for invertebrates (van Hattum et al. 1989), from 3 to 4,190 for fresh water aquatic organisms (ASTER 1995), and from 5 to 3,160 for saltwater aquatic organisms (ASTER 1994). Bioconcentration in fish depends on the pH and the humus content of the water (John et al. 1987). Because of their high ability to accumulate metals, some aquatic plants have been suggested for use in pollution control. For example, it has been suggested that the rapidly-growing water hyacinth (*Eichhornia crassipes*) could be used to remove cadmium from domestic and industrial effluents (Ding et al. 1994; Muntau and Baudo 1992).

The data indicate that cadmium bioaccumulates in all levels of the food chain. Cadmium accumulation has been reported in grasses and food crops, and in earthworms, poultry, cattle, horses, and wildlife (Alloway et al. 1990; Beyer et al. 1987; Gochfeld and Burger 1982; Kalac et al. 1996; Munshower 1977; Ornes and Sajwan 1993; Rutzke et al. 1993; Sileo and Beyer 1985; Vos et al. 1990). The metal burden of a crop depends on uptake by the root system, direct foliar uptake and translocation within the plant, and surface deposition of particulate matter (Nwosu et al. 1995). In general, cadmium accumulates in the leaves of plants and, therefore, is more of a risk in leafy vegetables grown in contaminated soil than in seed or root crops (Alloway et al. 1990). He and Singh (1994) report that, for plants grown in the same soil, accumulation of cadmium decreased in this order: leafy vegetables, root vegetables, and grain crops. Alloway et al. (1990) also demonstrated that uptake of cadmium decreased in this order: lettuces, cabbages, radishes, and carrots. Nwosu et al. (1995) investigated the uptake of cadmium and lead in lettuce and radish grown in loam soil spiked with known mixtures of CdCl_2 and $\text{Pb}(\text{NO}_3)_2$. They found that the mean uptake of cadmium by lettuce and radish increased as the concentrations of cadmium and lead in the soil increased. Their results supported previous findings that cadmium is absorbed by passive diffusion and translocated freely in the soil. The observed decline in cadmium uptake by lettuce at 400 mg/kg could be attributed to saturation of the active binding sites on the plant root system or by early toxicological responses of the plant root. The study also supported earlier findings that radish did not accumulate as much cadmium as lettuce.

Some studies have concluded that soil pH is the major factor influencing plant uptake of cadmium from soils (Smith 1994). Amending soil with lime raises the pH, increasing cadmium adsorption to the soil and reducing bioavailability (He and Singh 1994; Thornton 1992). One study found that in peeled potato

6. POTENTIAL FOR HUMAN EXPOSURE

tubers, potato peelings, oat straw, and ryegrass, cadmium concentrations generally decreased as simple linear functions of increasing soil pH over the range of pH values measured (pH 3.9–7.6) (Smith 1994). Soil type also affects uptake of cadmium by plants. For soils with the same total cadmium content, cadmium has been found to be more soluble and more plant-available in sandy soil than in clay soil (He and Singh 1994). Similarly, cadmium mobility and bioavailability are higher in noncalcareous than in calcareous soils (Thornton 1992). Oxidation-reduction potential may also have a large effect on soil-to-plant cadmium transport. The absorption of cadmium paddy rice is significantly affected by the oxidation-reduction potential of the soil. The oxidation-reduction potential of rice paddy soils shifts drastically compared to upland soils due to submerging and draining techniques. Cadmium to rice ratios (cadmium concentration in brown rice/cadmium concentration in soil) were the smallest when the rice was grown under submerged conditions during the whole growth period. The ratios were the largest when the soil (coarse Toyama soil) was drained after the tillering stage. This is due to changes in cadmium solubility. Under flooded conditions, cadmium sulfide formation increases, and thus, cadmium solubility decreases (Iimura 1981).

Since cadmium accumulates largely in the liver and kidneys of vertebrates and not in the muscle tissue (Harrison and Klaverkamp 1990; Sileo and Beyer 1985; Vos et al. 1990), and intestinal absorption of cadmium is low, biomagnification through the food chain may not be significant (Sprague 1986). In a study of marine organisms from the Tyrrhenian Sea, no evidence of cadmium biomagnification was found along pelagic or benthic food webs (Bargagli 1993). Although some data indicate increased cadmium concentrations in animals at the top of the food chain, comparisons among animals at different trophic levels are difficult, and the data available on biomagnification are not conclusive (Beyer 1986; Gochfeld and Burger 1982). Nevertheless, uptake of cadmium from soil by feed crops may result in high levels of cadmium in beef and poultry (especially in the liver and kidneys). This accumulation of cadmium in the food chain has important implications for human exposure to cadmium, whether or not significant biomagnification occurs.

Boularbah et al. (1992) isolated six cadmium-resistant bacterial strains from a soil receiving dredged sediments and containing 50 mg Cd/kg. The isolates tolerated higher cadmium concentrations than the control strain and accumulated cadmium at concentrations ranging from 0 to 100 mg/L. One of the isolates, *Bacillus brevis*, was found to be the most resistant to cadmium, with the ability to accumulate up to 70 mg Cd/g of cells dry weight, and may have some use in reclamation of metal-contaminated soils.

6. POTENTIAL FOR HUMAN EXPOSURE

6.3.2 Transformation and Degradation**6.3.2.1 Air**

Little information is available on the atmospheric reaction of cadmium (EPA 1980d). The common cadmium compounds found in air (oxide, sulfate, chloride) are stable and not subject to photochemical reactions (EPA 1980d). Cadmium sulfide may photolyze to cadmium sulfate in aqueous aerosols (Konig et al. 1992). Transformation of cadmium among types of compounds in the atmosphere is mainly by dissolution in water or dilute acids (EPA 1980d).

6.3.2.2 Water

In fresh water, cadmium is present primarily as the cadmium(+2) ion and $\text{Cd}(\text{OH})_2$ and CdCO_3 complexes, although at high concentrations of organic material, more than half may occur in organic complexes (McComish and Ong 1988). Some cadmium compounds, such as cadmium sulfide, cadmium carbonate, and cadmium oxide, are practically insoluble in water. However, water-insoluble compounds can be changed to water-soluble salts by interaction with acids or light and oxygen. For example, aqueous suspensions of cadmium sulfide can gradually photooxidize to soluble cadmium (IARC 1993). Cadmium complexation with chloride ion increases with salinity until, in normal seawater, cadmium exists almost entirely as chloride species (CdCl^+ , CdCl_2 , CdCl_3^-) with a minor portion as Cd^{2+} . In reducing environments, cadmium precipitates as cadmium sulfide in the presence of sulfide ions (McComish and Ong 1988). Photolysis is not an important mechanism in the aquatic fate of cadmium compounds (EPA 1983c), nor is biological methylation likely to occur (EPA 1979).

6.3.2.3 Sediment and Soil

Transformation processes for cadmium in soil are mediated by sorption from and desorption to water, and include precipitation, dissolution, complexation, and ion exchange (McComish and Ong 1988). Important factors affecting transformation in soil include the cation exchange capacity, pH, and content of clay minerals, carbonate minerals, oxides, organic matter, and oxygen (McComish and Ong 1988).

6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

Reliable evaluation of the potential for human exposure to cadmium depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. Concentrations of cadmium in unpolluted atmospheres and in pristine surface waters are often so low as to be near the limits

6. POTENTIAL FOR HUMAN EXPOSURE

of current analytical methods. In reviewing data on cadmium 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 cadmium in a variety of environmental media are detailed in Chapter 7.

6.4.1 Air

Cadmium levels in ambient air generally range from 0.1 to 5 ng/m³ in rural areas, 2–15 ng/m³ in urban areas, and 15–150 ng/m³ in industrialized areas. Remote areas can contain lower levels of cadmium (Morrow 2001). Cadmium can undergo long-range atmospheric transport and deposition causing cadmium contamination in areas with no local cadmium inputs. Smoking can greatly affect indoor air concentrations of cadmium. In nonsmoking environments, there is little difference between indoor and outdoor air quality (Morrow 2001). Monitoring studies conducted for EPA's 2006 Final Report for the Urban Air Toxics Monitoring Program detected cadmium in ambient air at several monitoring sites throughout the United States. At all detection sites in Bountiful, Utah; Northbrook, Illinois; Austin, Texas; St. Louis, Missouri; Indianapolis, Indiana; and Birmingham, Alabama average daily cadmium levels in ambient air were <0.01 µg/m³. In Bountiful, Utah average daily cadmium levels were reported as 0.0008 µg/m³ (EPA 2007).

Emission rates of cadmium from solid waste incinerators have been found to range from 20 to 2,000 µg/m³ from the stacks of traditional incinerators and from 10 to 40 µg/m³ from advanced incinerators. Advances in pollution control and increased government regulations have resulted in decreased cadmium emissions to the environment (EPA 1990a; Herron 2003; Morrow 2001; Schulte-Schrepping and Piscator 2002). Although there may be an increase in fossil fuel combustion and waste incineration, it does not appear likely that overall cadmium emissions to air will increase substantially in the United States.

Cadmium levels in aerosols over Russian Arctic seas were measured in order to understand the magnitude of long-range atmospheric deposition. Ten-year average monthly mean concentrations ranged from 0.002 to 0.080 ng/m³ in Franz Josef Land and from 0.0026 to 0.048 ng/m³ in Sevemaya Zemlya. The highest concentrations were reported in the spring season and the lowest concentrations reported in the autumn for both sampling sites. During the winter and spring months, it was estimated that >50% of the average air pollutant concentrations in the Russian Arctic are due to atmospheric pollution. The

6. POTENTIAL FOR HUMAN EXPOSURE

anthropogenic sources of cadmium to the Russian Arctic are the industrial areas of Northern Europe, Kola Peninsula, and the Urals and Norilsk regions (Shevchenko et al. 2003).

Atmospheric concentrations of cadmium are generally highest in the vicinity of cadmium-emitting industries such as smelters, municipal incinerators, or fossil fuel combustion facilities (Elinder 1985a; Pirrone et al. 1996). The mean annual concentration of airborne cadmium in an area about 1 km from a zinc smelter in Colorado was $0.023 \mu\text{g}/\text{m}^3$ ($2.3 \times 10^{-5} \text{ mg}/\text{m}^3$) (IARC 1993). Sweet et al. (1993) conducted a study of airborne inhalable particulate matter (PM-10) over a 2-year period in two urban/industrial areas (southeast Chicago and East St. Louis) and one rural area in Illinois. There was a significant difference between the cadmium levels in the urban areas and the cadmium levels in the rural area. Cadmium concentrations in the East St. Louis area were 5–10 times higher, with a range of <4 to $115 \text{ ng}/\text{m}^3$ (average $15[24] \text{ ng}/\text{m}^3$) for fine particles and a range of <4–97 ng/m^3 (average $10[18] \text{ ng}/\text{m}^3$) for coarse particles. In the Kikinda region of Serbia and Montenegro, where metal processing and construction industries are located, a mean annual atmospheric deposit of $36.0 \mu\text{g}/\text{m}^2$ per day was reported in 1995. A period of decreased industrial production, which decreased atmospheric cadmium deposits by 93%, resulted in 17% cadmium reduction in cattle feed and 13% in milk (Vidovic et al. 2005). Moss studies conducted by Hasselbach et al. (2005) in the area of the Red Dog Mine in Alaska reported cadmium levels $>24 \text{ mg}/\text{kg}$ dry weight in moss adjacent to the ore haul road. Ore dust containing heavy metals escapes from the ore trucks on the haul road and can be deposited in the nearby area (Hasselbach et al. 2005).

Annual average concentrations of atmospheric cadmium over three Great Lakes reflect the influence of industrialization and urbanization; Lake Erie's levels of $0.6 \text{ ng}/\text{m}^3$ were higher than fine particle concentrations of $0.2 \text{ ng}/\text{m}^3$ over Lake Michigan and $<0.2 \text{ ng}/\text{m}^3$ over Lake Superior (Sweet et al. 1998). In the Lake Michigan Urban Air Toxics Study of dry deposition of metals, the flux of cadmium on the south side of Chicago was reported at about $0.01 \text{ mg}/\text{m}^2/\text{day}$ and levels in rural Michigan and over Lake Michigan were far lower (Holsen et al. 1993).

6.4.2 Water

The average level of cadmium in ocean water has been reported between <5 and $110 \text{ ng}/\text{L}$, with higher levels reported near coastal areas and in marine phosphates and phosphorites (Morrow 2001).

6. POTENTIAL FOR HUMAN EXPOSURE

Thornton (1992) reports that waters from the vicinity of cadmium-bearing mineral deposits may have cadmium concentrations of $\geq 1,000$ $\mu\text{g/L}$. The cadmium concentration of natural surface water and groundwater is usually < 1 $\mu\text{g/L}$ (Elinder 1985a, 1992). EPA requires water suppliers to limit the cadmium concentration in drinking water to < 5 $\mu\text{g/L}$ (EPA 2006a).

Groundwater in New Jersey has an estimated median level of 1 $\mu\text{g Cd/L}$ with a high level of 405 $\mu\text{g/L}$. In a survey of groundwater surrounding waste sites, a concentration of 6,000 $\mu\text{g Cd/L}$ was found (NTP 1994). The National Urban Runoff Program measured cadmium concentrations in urban storm water runoff; concentrations ranged from 0.1 to 14 $\mu\text{g/L}$ in 55% of samples that were positive for cadmium (Cole et al. 1984). Cadmium in highway run-off has been detected at levels of 0.0–0.06 mg/L (0.0–60 $\mu\text{g/L}$).

In the estuarine portion of the Hudson River, more cadmium was released from agricultural and urban run-off than from industrial and municipal sewage treatment plants (Muntau and Baudo 1992). In an urban environment, there are also multiple sources of cadmium to waste water, based on an urban waste water study conducted in the United Kingdom. Cadmium was detected in the waste water originating from industrial, commercial, and private sectors, with the highest average cadmium concentration detected in the foul water of new (< 5 years old) private housing (0.375 $\mu\text{g/L}$) (Rule et al. 2006). Cadmium was detected in the contaminated groundwater plume near in the Moon Creek watershed in the Couer D'Alene Mining District of Idaho at concentrations of ≤ 0.077 mg/L . The cadmium was transported to the creek with the plume where it was subsequently diluted (Paulson 1997). In the Spring River Basin of Kansas, Missouri, and Oklahoma, part of the Tri-State Mining District, cadmium was detected in surface waters at concentrations ranging from < 1.0 to 24 $\mu\text{g/L}$ (peak flow) and from < 1.0 to 75.0 $\mu\text{g/L}$ (base flow). It was detected in the sediment of the sampling sites at concentrations ranging from 0.62 to 300 $\mu\text{g/g}$ dry weight in the < 250 μm sediment fraction and from 0.89 to 180 $\mu\text{g/g}$ dry weight in the < 63 μm fraction (Angelo et al. 2007).

6.4.3 Sediment and Soil

Cadmium concentrations in soils not contaminated by anthropogenic sources range from 0.06 to 1.1 mg/kg , with a minimum of 0.01 mg/kg and a maximum of 2.7 mg/kg (Alloway and Steinnes 1999). Cadmium content in marine sediments ranges from 0.1 to 1.0 $\mu\text{g/g}$ (ppm) in the Atlantic and Pacific oceans (Thornton 1992). Average cadmium concentration in agricultural soils of remote locations was reported as 0.27 mg/kg (Holmgren et al. 1993). Soils with parent materials such as black shale (cadmium

6. POTENTIAL FOR HUMAN EXPOSURE

content up to 24 mg/kg) may have higher concentrations of natural cadmium. Since the U.S. mandatory limit of cadmium in sewage sludge is <20 mg/kg, soils receiving sewage sludge should not have heightened cadmium levels (Alloway and Steinnes 1999). Topsoil concentrations are often more than twice as high as subsoil levels as the result of atmospheric fallout and contamination (Pierce et al. 1982). Cadmium will partition mostly to soil and sediment when released to the environment. Atmospheric deposition is a major source of surface soil contamination, which allows cadmium to be introduced into the food supply (Alloway and Steinnes 1999; Morrow 2001).

Markedly elevated levels may occur in topsoils near sources of contamination. Moss studies conducted by Hasselbach et al. (2005) in the area of the Red Dog Mine in Alaska reported cadmium levels >24 mg/kg dry weight in moss (n=151), as a measure atmospheric deposition onto soil surfaces, within 10 m of the ore haul road. Ore dust containing heavy metals escapes from the ore trucks during loading and unloading at the mine and port site settles on the surfaces of the trucks, which blow off the trucks during transport on the haul road and deposited in the nearby area. The mean cadmium concentrations in moss and subsurface soil throughout the entire study were 1.86 and 0.27 mg/kg dry weight, respectively. Cadmium concentrations in moss and subsurface soil were 0.08–24.30 and 0.07–0.75 mg/kg dry weight. There did not appear to be a connection between the elevated subsurface cadmium levels and the local geochemistry. Geospatial analysis showed that areas as far as 12 km north of the haul road may be affected by mining emission depositions (Hasselbach et al. 2005). In the vicinity of a smelter in Helena, Montana, average soil values were 72 ppm within 1 km and 1.4 ppm between 18 and 60 km (EPA 1981a). Total cadmium concentrations in soil samples taken from a Superfund site in southeast Kansas ranged from 15 to 86 mg/kg (ppm). In the same study, soil samples were extracted with diethylenetriamine-pentaacetic acid (DPTA) to approximate the plant-available metal concentrations. Extractable cadmium concentrations ranged from 0.6 to 10 mg/kg (ppm) (Abdel-Saheb et al. 1994). Soil cadmium levels in five Minnesota cities were highest in areas with the most vehicular traffic (>2 ppm in about 10% of inner-city samples) and also showed a pattern consistent with past deposition from a sewage-sludge incinerator (Mielke et al. 1991). Cadmium levels >750 mg/kg have been found in sites polluted by nonferrous metal mining and smelting have been reported (Alloway and Steinnes 1999).

In the Spring River Basin of Kansas, Missouri, and Oklahoma, part of the Tri-State Mining District, cadmium was detected in surface waters at concentrations of <1.0–24 µg/L (peak flow) and <1.0–75.0 µg/L (base flow). Cadmium was detected in the sediment of the sampling sites at concentrations ranging from 0.62 to 300 µg/g dry weight in the <250 µm sediment fraction and from 0.89 to 180 µg/g dry weight in the <63 µm fraction (Angelo et al. 2007). A study conducted in 1999 at the Patroon Creek

6. POTENTIAL FOR HUMAN EXPOSURE

Reservoir in Albany County, New York sampled sediment cores for heavy metals, including cadmium. The watershed includes two industrial sites: one in operation from 1955 to present and the other operating from 1958 to 1984. Sediment samples in the interval of 0–1.68 m showed an average cadmium concentration of 1.69 mg/kg. This concentration is comparable to other stream and reservoir sediments impacted by industrial pollution (Arnason and Fletcher 2003). Sediments of the Sawmill River in Yonkers, New York contained the highest cadmium levels (6.9 mg/kg) in the Hudson River Basin during a sampling study conducted between 1992 and 1995 (USGS 1998b).

Surficial sediments collected from 18 locations in three major tributaries to Newark Bay, New Jersey, had a mean cadmium concentration of 10 ± 6 mg/kg (ppm) dry weight (Bonnievie et al. 1994). The highest cadmium concentrations were found in the Ironbound section of the Passaic River, a heavily industrialized area (29 mg/kg and 14 mg/kg), and in the Arthur Kill on the northwest side of Prall's Island (15 mg/kg). An investigation of metals distribution in sediments along the Hudson River estuary revealed that cadmium concentrations in suspension were higher than in the bottom sediments by a factor of 30 (Gibbs 1994).

Soils derived from dredged material in confined disposal facilities in the Great Lakes Region had cadmium concentrations (dry weight) of <1.9–32 ppm (Beyer and Stafford 1993). In an analytical survey of sewage sludges from 16 large cities in the United States, cadmium concentrations ranged from 2.72 to 242 ppm (dry weight). Besides the sample with a cadmium concentration of 242 ppm, all other sludges had cadmium contents ≤ 14.7 ppm (Gutenmann et al. 1994).

6.4.4 Other Environmental Media

Cadmium levels in food can vary greatly depending on the type of food, agricultural and cultivating practices, and amount atmospheric deposition and other anthropogenic contamination. In general, leafy vegetables, such as lettuce and spinach, and staples, such as potatoes and grains, contain relatively high values of cadmium. Peanuts, soybeans, and sunflower seeds have naturally high levels of cadmium. Meat and fish contain lower amounts of cadmium, with the exception of animal organ meats, such as kidney and liver, as these organs concentrate cadmium (Morrow 2001).

As part of the U.S. Food and Drug Administration (FDA) Total Diet Study, average concentrations of cadmium in 14 food groups were analyzed from samples collected in 56 American cities. Cadmium was found in nearly all samples at varying concentrations. In general, the eggs, milk and cheese, condiments

6. POTENTIAL FOR HUMAN EXPOSURE

and sweeteners, beverages, and fats and dressings groups contained low concentrations of cadmium (≤ 0.004 mg/kg) (FDA 2010). Food items that contained high levels of cadmium were dry roasted peanuts (0.054 mg/kg), shredded wheat cereal (0.050 mg/kg), boiled spinach (0.124 mg/kg), iceberg lettuce (0.051 mg/kg), leaf lettuce (0.064 mg/kg), and potato chips (0.057 mg/kg) (FDA 2010). [Table 6-3](#) summarizes the data from this study.

Watanabe et al. (1996) measured the cadmium content in rice samples from various areas in the world during the period from 1990 to 1995. Twenty-nine samples collected in the United States had a geometric mean of 7.43 ng Cd/g, with a standard deviation of 2.11. Shellfish, liver, and kidney meats have higher concentrations than other fish or meat (up to 1 ppm) (Elinder 1985a; IARC 1993; Schmitt and Brumbaugh 1990). Particularly high concentrations of cadmium of 2–30 mg/kg (ppm) fresh weight have been found in the edible brown meat of marine shellfish (Elinder 1992). Cadmium concentrations up to 8 $\mu\text{g/g}$ in oysters and 3 $\mu\text{g/g}$ in salmon flesh have been reported (IARC 1993). Sprague (1986) reviewed tissue concentrations of cadmium for marine mollusks and crustaceans. They found that drills, a type of sea snail, were higher in cadmium (average, 26 $\mu\text{g/g}$ dry weight) than almost all other mollusks, although scallops and whelks also tended to be high. Oysters from polluted areas averaged 18 $\mu\text{g/g}$ dry weight, which was significantly higher than oysters from clean areas (average concentration 1.4 $\mu\text{g/g}$ dry weight). Clams were relatively low in cadmium (average, 0.5–1.0 $\mu\text{g/g}$ dry weight). The average concentration of cadmium in clams from polluted areas was only 2.7 $\mu\text{g/g}$ dry weight, but this was significantly higher than levels in clams from clean areas. In Fiscal Year (FY) 1985/1986, the FDA conducted a survey of cadmium, lead, and other elements in fresh clams and oysters collected from U.S. coastal areas used for shellfish production (Capar and Yess 1996). Average cadmium levels (wet weight) were 0.09 ± 0.06 mg/kg (ppm) ($n=75$) in hardshell clams, 0.05 ± 0.04 mg/kg ($n=59$) in softshell clams, 0.51 ± 0.31 mg/kg ($n=104$) in Eastern oysters, and 1.1 ± 0.6 mg/kg ($n=40$) in Pacific oysters. In FY91, FDA analyzed 5 samples of domestic clams and 24 samples of domestic oysters (collected from both coasts) for cadmium and found average concentrations of 0.06 and 0.62 mg/kg, respectively (Capar and Yess 1996). Although no conclusions can be drawn in light of the small numbers of FY91 samples, these results do not appear to be appreciably different from those of the FY85/86 survey..

Cadmium is accumulated mainly in the hepatopancreas (digestive gland) of the crab, and cadmium levels as high as 30–50 ppm have been detected in this edible part of the animal. Cadmium levels as high as 10 ppm also have been measured in some species of wild-growing edible mushrooms (Lind et al. 1995). Lind et al. (1995) conducted a feeding study in mice to determine the bioavailability of cadmium from crab hepatopancreas and mushroom in relation to organic cadmium. The cadmium accumulation in the

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-3. Mean Concentrations of Cadmium for FDA's Total Diet Study Market Baskets 2006-1 through 2008-4

Food product	Mean concentration range (mg/kg)
Milk and cheese	Not detected–0.002
Eggs	Not detected–0.0003
Meat, poultry, and fish	Not detected–0.069
Legumes and nuts	0.001–0.054
Grain products	0.0001–0.028
Fruit	Not detected–0.015
Vegetables	0.001–0.124
Mixed dishes and meals	0.003–0.021
Desserts	Not detected–0.028
Condiments and sweeteners	0.001–0.0002
Fats and dressings	Not detected–0.004
Beverages	Not detected–0.001
Infant and junior foods	0.0002–0.026

Source: FDA 2010

6. POTENTIAL FOR HUMAN EXPOSURE

liver and kidney of the mice was used as an estimate of the intestinal absorption. The group that was fed crab accumulated less cadmium in the liver and kidney than the groups fed mushrooms or inorganic cadmium salt. They concluded from the results of the study that cadmium from boiled crab has a lower bioavailability for absorption in the gastrointestinal tract of mice than inorganic cadmium and cadmium from dried mushrooms. Almost all (99%) of the cadmium in the boiled crab hepatopancreas was associated with insoluble ligands, probably denatured protein. In fresh crab hepatopancreas, most of the cadmium is in a soluble form bound to metallothionein (Lind et al. 1995).

Significant concentrations of cadmium have been observed in fish living in stormwater ponds in Florida, especially in the redear sunfish, a bottom feeder (Campbell 1994). The mean cadmium concentration in redear sunfish living in stormwater ponds was 1.64 mg/kg wet weight compared to 0.198 mg/kg for redear sunfish living in control ponds. Similarly, the mean cadmium concentration in largemouth bass living in stormwater ponds was 3.16 mg/kg wet weight compared to 0.241 mg/kg for largemouth bass living in control ponds. Red drum, flounder, and seatrout collected from South Carolina estuaries during the period 1990–1993 had consistently low cadmium levels throughout the sampling area and with respect to species (Mathews 1994). The mean concentration for all fillets and whole fish was 86.2 ppb wet weight, with 70.7% (n=164) of the samples having <25 ppb.

Cadmium and other heavy metals were detected in several of the freshwater invertebrates and fish of two Maryland streams. Due to their remote location and lack of source inputs, it is believed that the cadmium contamination was a result of long-range atmospheric deposition. Samples were taken from the Herrington Creek tributary (HCRT) and Blacklick Run (BLK) during October 1997, April 1998, and July 1998. Cadmium concentrations in the trout of BLK ranged from about 37 to 90 ng/g wet, with the older specimens having the higher cadmium concentrations. Cadmium concentrations in crayfish ranged from about 40 to 160 ng/g wet in BLK, with the younger specimens containing the highest levels of cadmium. Crayfish in HCRT ranged from 45 to 155 ng/g, with the highest levels in the middle age group. In crayfish, cadmium strongly accumulates in the gills, while the kidney accumulates cadmium in trout (Mason et al. 2000).

Cadmium concentrations in the fish of the mining-contaminated waters of Oklahoma were reported by Brumbaugh et al. (2005). This area was part of the Tri-State Mining District that was extensively mined for lead and zinc from the mid-1800s to the 1950s, and contains nonremediated sites. Blood and carcass cadmium concentrations differed between species and sites, but were generally greatest in carp. Carcass cadmium in catfish were relatively low, with <0.1 µg/g dry weight in 34 of 36 samples.

6. POTENTIAL FOR HUMAN EXPOSURE

Cadmium concentrations of ≥ 0.5 ppm have been found in rice grown in cadmium-polluted areas of Japan (Nogawa et al. 1989) and China (Shiwen et al. 1990). Tobacco also concentrates cadmium from the soil, and cadmium content of cigarettes typically ranges from 1 to 2 $\mu\text{g}/\text{cigarette}$ (Elinder 1985a, 1992).

Some food crops, including confectionery sunflowers, have a propensity to take up cadmium from the soil in which they are grown and deposit it in the kernels. In a study to determine the cadmium burden of persons who report regular consumption of sunflower kernels, Reeves and Vanderpool (1997) analyzed 19 different lots of sunflower kernels from the 1995 crop grown in the northern Great Plains region of North Dakota and Minnesota. They found a range of 0.33–0.67 $\mu\text{g Cd/g}$, with a mean \pm standard deviation of 0.48 ± 0.11 $\mu\text{g/g}$ fresh weight. The study showed that high intakes of sunflower kernels increased the intake of cadmium. However, the amount of cadmium in whole blood or in red blood cells was not affected by cadmium intake. The authors pointed out that an increased intake of sunflowers will increase not only the cadmium intake, but also the intake of copper and phytate. In turn, this could reduce the availability of cadmium from this food source.

DOI (1985) examined the concentrations of cadmium in a variety of aquatic and terrestrial flora and fauna and identified six trends: (1) in general, marine biota contained significantly higher cadmium residues than their freshwater or terrestrial counterparts; (2) cadmium tends to concentrate in the viscera of vertebrates, especially in the liver and kidneys; (3) cadmium concentrations are higher in older organisms than in younger ones, especially in carnivores and marine vertebrates; (4) higher concentrations for individuals of a single species collected at various locations are almost always associated with proximity to industrial/urban areas or point-source discharges of cadmium-containing wastes; (5) background levels of cadmium in crops and other plants are generally < 1.0 mg/kg (ppm); and (6) cadmium concentrations in biota are dependent upon the species analyzed, the season of collection, ambient cadmium levels, and the sex of the organism.

During a study monitoring cadmium levels in 331 cigarette packs from over 20 areas around the world it was found that the mean cadmium level per cigarette was 1.15 $\mu\text{g}/\text{cigarette} \pm 0.43$ (AM \pm ASD) or 1.06 $\mu\text{g}/\text{cigarette} \pm 1.539$ (geometric mean [GM] \pm geometric standard deviation [GSD]). Cigarettes from Mexico had the highest mean level of cadmium with an AM \pm ASD of 2.03 $\mu\text{g}/\text{cigarette} \pm 0.33$ or a GM \pm GSD of 2.00 $\mu\text{g}/\text{cigarette} \pm 1.190$. Cigarettes from India had the lowest mean levels of cadmium with an AM \pm ASD of 0.35 $\mu\text{g}/\text{cigarette} \pm 0.09$ or a GM \pm GSD of 0.34 $\mu\text{g}/\text{cigarette} \pm 1.284$. The arithmetic

6. POTENTIAL FOR HUMAN EXPOSURE

mean for the United States was 1.07 $\mu\text{g}/\text{cigarette} \pm 0.11$ and the GM \pm GSD was 1.06 $\mu\text{g}/\text{cigarette} \pm 1.115$ (Watanabe et al. 1987).

The cadmium content of coals varies widely; concentrations of 0.01–180 $\mu\text{g}/\text{g}$ (ppm) have been reported for the United States (Thornton 1992; Wilber et al. 1992).

6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

The general population may be exposed to cadmium through ingestion of food and drinking water, inhalation of particulates from ambient air or tobacco smoke, or ingestion of contaminated soil or dust. For nonsmokers, food is the major source of cadmium exposure (NTP 2005). Inhalation of cigarette smoke is the major source of cadmium exposure for smokers (CDC 2005). Cadmium is introduced to the food chain through agricultural soils, which may naturally contain cadmium, or from anthropogenic sources such as atmospheric deposition or direct application methods such as phosphate fertilizer application and municipal waste composting (Alloway and Steinnes 1999; Morrow 2001). Cadmium-plated utensils and galvanized equipment used in food processing and preparation; enamel and pottery glazes with cadmium-based pigments; and stabilizers used in food-contact plastics are also sources of food contamination (Galal-Gorchev 1993). Cadmium levels in soils are not a direct indicator of the level of cadmium in the food supply, with the exception of extreme contamination, as other factors such as the type of crop and farming methods are important (Morrow 2001).

Based on food intake rates and food-cadmium concentrations, the estimated geometric mean daily intake of cadmium for the U.S. population is 18.9 $\mu\text{g}/\text{day}$, down from an estimated 30 $\mu\text{g}/\text{day}$ in the 1980s (Choudhury et al. 2001; Gartrell et al. 1986). Based on the mean cadmium daily intakes for males and females aged 6–60 years reported by Choudhury et al. (2001), age-weighted mean cadmium intakes of 0.35 $\mu\text{g}/\text{kg}/\text{day}$ for males and 0.30 $\mu\text{g}/\text{kg}/\text{day}$ for females were calculated for U.S. nonsmokers.

In the Fourth National Report on Human Exposures to Environmental Chemicals reported by the CDC (2011) results from the National Health and Nutrition Examination Survey (NHANES) 1999–2008 were reported. Cadmium levels in blood (see [Table 6-4](#)), urine (creatinine corrected) (see [Table 6-5](#)), and urine (see [Table 6-6](#)) was evaluated for a variety of age groups and ethnicities. Blood cadmium reflects both recent and cumulative exposures and urinary cadmium reflects cadmium exposure and the concentration of cadmium in the kidneys.

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-4. Geometric Mean and Selected Percentile Blood Concentrations (µg/L) of Cadmium in the U.S. Population from 1999 to 2008

Group	Survey years	Geometric mean ^a (95% CI)	Selected percentiles (95% CI)				Sample size
			50 th	75 th	90 th	95 th	
Total, age 1 and older	1999–2000	0.412 (0.378–0.449)	0.300 (0.300–0.400)	0.600 (0.500–0.600)	1.00 (0.900–1.00)	1.30 (1.20–1.40)	7,970
	2001–2002	Not calculated	0.300 (<LOD–0.300)	0.400 (0.400–0.500)	0.900 (0.900–1.10)	1.30 (1.20–1.60)	8,945,
	2003–2004	0.304 (0.289–0.320)	0.300 (0.300–0.300)	0.500 (0.500–0.600)	1.10 (1.00–1.20)	1.60 (1.50–1.60)	8,372
	2005–2006	0.310 (0.294–0.327)	0.270 (0.250–0.280)	0.501 (0.460–0.560)	1.02 (0.910–1.13)	1.53 (1.34–1.75)	8,407
	2007–2008	0.315 (0.300–0.331)	0.270 (0.260–0.280)	0.500 (0.460–0.560)	1.00 (0.900–1.13)	1.51 (1.30–1.77)	8,266
Age group 1–5 Years	1999–2000	Not calculated	<LOD	0.300 (<LOD–0.300)	0.400 (0.300–0.400)	0.400 (0.300–0.400)	723
	2001–2002	Not calculated	<LOD	<LOD	<LOD	0.300 (<LOD–0.300)	898
	2003–2004	Not calculated	<LOD	<LOD	0.200 (0.200–0.300)	0.200 (0.200–0.400)	910
	2005–2006	Not calculated	<LOD	<LOD	<LOD	0.230 (0.210–0.250)	968
	2007–2008	Not calculated	<LOD	<LOD	0.210 (<LOD–0.230)	0.240 (0.220–0.260)	817
6–11 Years	1999–2000	Not calculated	<LOD	0.300 (<LOD–0.300)	0.400 (0.300–0.400)	0.400 (0.400–0.500)	905
	2001–2002	Not calculated	<LOD	<LOD	<LOD	0.400 (0.300–0.400)	1,044
	2003–2004	Not calculated	<LOD	0.200 (<LOD–0.200)	0.300 (0.200–0.300)	0.300 (0.300–0.300)	856
	2005–2006	Not calculated	<LOD	<LOD	0.220 (0.200–0.300)	0.260 (0.230–0.280)	934
	2007–2008	Not calculated	<LOD	<LOD	0.230 (0.210–0.240)	0.260 (0.240–0.280)	1,011
12–19 Years	1999–2000	0.333 (0.304–0.366)	0.300 (<LOD–0.300)	0.300 (0.300–0.400)	0.800 (0.600–0.900)	1.10 (0.900–1.10)	2,135
	2001–2002	Not calculated	<LOD	0.300 (<LOD–0.300)	0.400 (0.400–0.500)	0.800 (0.600–1.10)	2,231
	2003–2004	Not calculated	0.200 (<LOD–0.200)	0.300 (0.300–0.300)	0.600 (0.500–0.700)	0.900 (0.800–1.10)	2,081
	2005–2006	Not calculated	<LOD	0.250 (0.240–0.270)	0.520 (0.500–0.700)	0.960 (0.820–1.08)	1,996
	2007–2008	Not calculated	<LOD	0.260 (0.240–0.270)	0.520 (0.400–0.670)	0.960 (0.730–1.19)	1,074

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-4. Geometric Mean and Selected Percentile Blood Concentrations ($\mu\text{g/L}$) of Cadmium in the U.S. Population from 1999 to 2008

Group	Survey years	Geometric mean ^a (95% CI)	Selected percentiles (95% CI)				Sample size
			50 th	75 th	90 th	95 th	
≥ 20 Years	1999–2000	0.468 (0.426–0.513)	0.400 (0.300–0.400)	0.600 (0.600–0.700)	1.00 (1.00–1.10)	1.50 (1.40–1.60)	4,207
	2001–2002	Not calculated	0.300 (0.300–0.400)	0.600 (0.500–0.600)	1.10 (0.900–1.20)	1.60 (1.30–1.80)	4,772
	2003–2004	0.378 (0.359–0.398)	0.400 (0.300–0.400)	0.600 (0.600–0.700)	1.20 (1.20–1.30)	1.80 (1.60–1.90)	4,525
	2005–2006	0.373 (0.352–0.395)	0.330 (0.310–0.350)	0.610 (0.570–0.660)	1.17 (1.06–1.26)	1.72 (1.53–1.95)	4,509
	2007–2008	0.376 (0.354–0.399)	0.330 (0.310–0.350)	0.600 (0.550–0.670)	1.16 (1.02–1.30)	1.70 (1.50–1.96)	5,364
Gender							
Males	1999–2000	0.403 (0.368–0.441)	0.400 (0.300–0.400)	0.600 (0.500–0.600)	1.00 (0.900–1.10)	1.30 (1.20–1.50)	3,913
	2001–2002	Not calculated	0.300 (<LOD–0.300)	0.400 (0.400–0.500)	0.900 (0.900–1.10)	1.40 (1.20–1.80)	4,339
	2003–2004	0.283 (0.266–0.300)	0.300 (0.200–0.300)	0.500 (0.500–0.500)	1.10 (1.00–1.20)	1.60 (1.50–1.60)	4,131
	2005–2006	Not calculated	0.240 (0.220–0.260)	0.470 (0.420–0.530)	1.02 (0.910–1.12)	1.53 (1.27–1.86)	4,092
	2007–2008	0.299 (0.283–0.317)	0.240 (0.230–0.260)	0.470 (0.420–0.540)	1.05 (0.930–1.19)	1.60 (1.30–1.90)	4,147
Females	1999–2000	0.421 (0.386–0.460)	0.300 (0.300–0.400)	0.600 (0.500–0.600)	1.00 (0.800–1.00)	1.30 (1.10–1.40)	4,057
	2001–2002	Not calculated	0.300 (0.300–0.400)	0.500 (0.500–0.600)	1.00 (0.900–1.10)	1.40 (1.20–1.60)	4,606
	2003–2004	0.326 (0.300–0.300)	0.300 (0.300–0.300)	0.600 (0.500–0.600)	1.10 (1.00–1.20)	1.60 (1.50–1.70)	4,241
	2005–2006	0.329 (0.311–0.349)	0.290 (0.280–0.310)	0.530 (0.480–0.580)	1.02 (0.870–1.18)	1.54 (1.33–1.79)	4,315
	2007–2008	0.331 (0.316–0.348)	0.290 (0.280–0.310)	0.530 (0.480–0.570)	0.980 (0.860–1.10)	1.43 (1.29–1.63)	4,119
Race/ethnicity							
Mexican Americans	1999–2000	0.395 (0.367–0.424)	0.400 (0.300–0.400)	0.400 (0.400–0.500)	0.700 (0.700–0.900)	1.10 (0.900–1.30)	2,742
	2001–2002	Not calculated	<LOD	0.300 (0.300–0.400)	0.600 (0.500–0.700)	1.00 (0.700–1.30)	2,268
	2003–2004	0.235 (0.26–0.255)	0.200 (0.200–0.300)	0.400 (0.300–0.400)	0.600 (0.500–0.800)	1.00 (0.800–1.50)	2,085
	2005–2006	Not calculated	0.220 (0.200–0.240)	0.350 (0.300–0.400)	0.580 (0.510–0.680)	0.820 (0.710–1.00)	2,236
	2007–2008	Not calculated	0.220 (0.210–0.230)	0.350 (0.320–0.370)	0.570 (0.510–0.660)	0.870 (0.690–1.02)	1,712

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-4. Geometric Mean and Selected Percentile Blood Concentrations ($\mu\text{g/L}$) of Cadmium in the U.S. Population from 1999 to 2008

Group	Survey years	Geometric mean ^a (95% CI)	Selected percentiles (95% CI)				Sample size
			50 th	75 th	90 th	95 th	
Non-Hispanic blacks	1999–2000	0.393 (0.361–0.427)	0.300 (0.300–0.400)	0.600 (0.500–0.600)	1.00 (0.800–1.10)	1.40 (1.10–1.50)	1,842
	2001–2002	Not calculated	<LOD	0.400 (0.400–0.500)	1.00 (0.900–1.00)	1.40 (1.20–1.50)	2,219
	2003–2004	0.304 (0.275–0.337)	0.300 (0.300–0.300)	0.500 (0.400–0.600)	1.00 (0.900–1.20)	1.50 (1.30–1.70)	2,292
	2005–2006	0.307 (0.290–0.326)	0.260 (0.250–0.280)	0.490 (0.440–0.570)	1.03 (0.880–1.21)	1.50 (1.23–1.79)	2,193
	2007–2008	0.333 (0.316–0.352)	0.280 (0.270–0.300)	0.550 (0.480–0.620)	1.20 (1.02–1.36)	1.81 (1.45–2.13)	1,746
Non-Hispanic whites	1999–2000	0.376 (0.470–0.209)	0.400 (0.300–0.400)	0.500 (0.500–0.600)	1.00 (0.900–1.10)	1.30 (1.20–1.40)	2,716
	2001–2002	Not calculated	<LOD	0.500 (0.500–0.600)	0.900 (0.900–1.10)	1.40 (1.20–1.80)	3,806
	2003–2004	0.313 (0.296–0.331)	0.300 (0.300–0.300)	0.600 (0.500–0.600)	1.10 (1.00–1.20)	1.60 (1.50–1.70)	3,478
	2005–2006	0.321 (0.300–0.343)	0.270 (0.250–0.300)	0.540 (0.470–0.610)	1.08 (0.930–1.23)	1.64 (1.40–1.94)	3,310
	2007–2008	0.210 (0.303–0.341)	0.270 (0.260–0.290)	0.520 (0.470–0.580)	1.05 (0.920–1.20)	1.55 (1.30–1.80)	3,461

^aThe proportion of results below the LOD was too high to provide a valid result.

CI = confidence interval; LOD = limit of detection

Source: CDC 2011

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-5. Geometric Mean and Selected Percentile Urine Concentrations (Creatinine Corrected) ($\mu\text{g/g}$ Creatinine) of Cadmium in the U.S. Population from 1999 to 2008

Group	Survey years	Geometric mean ^a (95% CI)	Selected percentiles (95% CI)				Sample size
			50 th	75 th	90 th	95 th	
Total, age 6 and older	1999–2000	0.181 (0.157–0.209)	0.219 (0.199–0.238)	0.423 (0.391–0.446)	0.712 (0.645–0.757)	0.933 (0.826–1.07)	2,257
	2001–2002	0.199 (0.181–0.218)	0.212 (0.194–0.232)	0.404 (0.377–0.440)	0.690 (0.630–0.754)	0.917 (0.813–0.998)	2,689
	2003–2004	0.210 (0.201–0.219)	0.208 (0.189–0.226)	0.412 (0.381–0.438)	0.678 (0.650–0.716)	0.940 (0.833–1.04)	2,543
	2005–2006	0.189 (0.169–0.210)	0.180 (0.160–0.200)	0.370 (0.310–0.430)	0.650 (0.590–0.720)	0.910 (0.770–1.08)	2,576
	2007–2008	0.193 (0.177–0.210)	0.190 (0.180–0.210)	0.370 (0.330–0.410)	0.660 (0.580–0.740)	0.960 (0.850–1.06)	2,627
Age group							
6–11 Years	1999–2000	Not calculated	0.085 (0.063–0.107)	0.147 (0.123–0.182)	0.210 (0.171–0.316)	0.300 (0.184–0.607)	310
	2001–2002	0.075 (0.059–0.094)	0.100 (0.083–0.112)	0.166 (0.136–0.192)	0.233 (0.206–0.281)	0.291 (0.221–0.440)	368
	2003–2004	0.090 (0.078–0.104)	0.091 (0.075–0.104)	0.126 (0.111–0.156)	0.200 (0.147–0.350)	0.308 (0.178–0.415)	287
	2005–2006	0.081 (0.072–0.092)	0.080 (0.070–0.090)	0.130 (0.110–0.140)	0.170 (0.150–0.190)	0.200 (0.180–0.240)	355
	2007–2008	0.084 (0.076–0.092)	0.080 (0.080–0.090)	0.120 (0.110–0.140)	0.180 (0.150–0.240)	0.260 (0.180–0.430)	394
12–19 Years	1999–2000	0.071 (0.051–0.098)	0.093 (0.084–0.106)	0.147 (0.130–0.163)	0.215 (0.204–0.240)	0.283 (0.222–0.404)	648
	2001–2002	0.078 (0.067–0.091)	0.091 (0.085–0.101)	0.136 (0.123–0.143)	0.191 (0.175–0.234)	0.280 (0.234–0.321)	762
	2003–2004	0.086 (0.077–0.096)	0.084 (0.074–0.097)	0.122 (0.113–0.135)	0.176 (0.154–0.198)	0.234 (0.187–0.274)	724
	2005–2006	0.076 (0.071–0.081)	0.080 (0.070–0.0990)	0.120 (0.110–0.130)	0.50 (0.140–0.180)	0.210 (0.160–0.240)	701
	2007–2008	0.070 (0.062–0.079)	0.070 (0.070–0.080)	0.110 (0.100–0.110)	0.150 (0.130–0.160)	0.180 (0.160–0.200)	376
≥ 20 Years	1999–2000	0.267 (0.247–0.289)	0.288 (0.261–0.304)	0.484 (0.433–0.545)	0.769 (0.727–0.818)	1.07 (0.927–1.17)	1,299
	2001–2002	0.261 (0.236–0.289)	0.273 (0.247–0.303)	0.481 (0.426–0.518)	0.776 (0.691–0.850)	0.979 (0.874–1.12)	1,559
	2003–2004	0.268 (0.255–0.281)	0.270 (0.247–0.292)	0.490 (0.444–0.538)	0.767 (0.688–0.830)	1.02 (0.909–1.14)	1,532
	2005–2006	0.240 (0.216–0.267)	0.250 (0.200–0.260)	0.440 (0.400–0.500)	0.730 (0.660–0.820)	1.02 (0.850–1.18)	1,520
	2007–2008	0.247 (0.227–0.270)	0.250 (0.220–0.270)	0.430 (0.390–0.70)	0.740 (0.670–0.840)	1.05 (0.930–1.16)	1,857

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-5. Geometric Mean and Selected Percentile Urine Concentrations (Creatinine Corrected) ($\mu\text{g/g}$ Creatinine) of Cadmium in the U.S. Population from 1999 to 2008

Group	Survey years	Geometric mean ^a (95% CI)	Selected percentiles (95% CI)				Sample size
			50 th	75 th	90 th	95 th	
Gender							
Males	1999–2000	0.154 (0.131–0.182)	0.174 (0.158–0.191)	0.329 (0.293–0.382)	0.617 (0.537–0.700)	0.788 (0.696–0.929)	1,121
	2001–2002	0.159 (0.143–0.177)	0.168 (0.157–0.182)	0.334 (0.304–0.364)	0.532 (0.491–0.653)	0.757 (0.690–0.856)	1,334
	2003–2004	0.173 (0.161–0.187)	0.162 (0.143–0.185)	0.325 (0.300–0.352)	0.591 (0.560–0.631)	0.740 (0.678–0.795)	1,277
	2005–2006	0.160 (0.145–0.177)	0.150 (0.130–0.160)	0.300 (0.260–0.340)	0.362 (0.560–0.340)	0.820 (0.710–0.990)	1,271
	2007–2008	0.160 (0.146–0.175)	0.160 (0.140–0.180)	0.290 (0.260–0.330)	0.520 (0.430–0.650)	0.740 (0.600–0.960)	1,327
Females	1999–2000	0.211 (0.170–0.261)	0.267 (0.239–0.308)	0.473 (0.423–0.551)	0.783 (0.690–0.917)	1.09 (0.813–1.38)	1,136
	2001–2002	0.245 (0.216–0.278)	0.263 (0.228–0.297)	0.479 (0.414–0.541)	0.792 (0.687–0.884)	0.985 (0.876–1.16)	1,355
	2003–2004	0.252 (0.238–0.266)	0.253 (0.227–0.288)	0.487 (0.438–0.533)	0.802 (0.716–0.906)	1.06 (0.940–1.21)	1,266
	2005–2006	0.220 (0.193–0.252)	0.220 (0.180–0.250)	0.420 (0.370–0.470)	0.690 (0.590–0.850)	0.990 (0.780–1.31)	1,305
	2007–2008	0.231 (0.211–0.254)	0.230 (0.200–0.270)	0.440 (0.390–0.480)	0.770 (0.700–0.860)	1.09 (0.970–1.17)	1,300
Race/ethnicity							
Mexican Americans	1999–2000	0.175 (0.137–0.223)	0.181 (0.144–0.225)	0.331 (0.266–0.418)	0.612 (0.441–0.828)	0.843 (0.674–1.13)	780
	2001–2002	0.156 (0.136–0.177)	0.170 (0.150–0.184)	0.282 (0.263–0.340)	0.501 (0.388–0.614)	0.693 (0.507–0.839)	682
	2003–2004	0.160 (0.147–0.181)	0.159 (0.140–0.183)	0.296 (0.256–0.311)	0.531 (0.418–0.667)	0.718 (0.562–0.950)	614
	2005–2006	0.162 (0.146–0.181)	0.150 (0.140–0.170)	0.280 (0.220–0.340)	0.480 (0.430–0.510)	0.570 (0.520–0.640)	652
	2007–2008	0.162 (0.144–0.183)	0.160 (0.130–0.180)	0.280 (0.220–0.340)	0.530 (0.390–0.640)	0.720 (0.520–1.07)	515
Non-Hispanic blacks	1999–2000	0.183 (0.140–0.240)	0.201 (0.168–0.241)	0.414 (0.343–0.472)	0.658 (0.516–0.827)	0.873 (0.722–0.962)	546
	2001–2002	0.190 (0.156–0.232)	0.195 (0.174–0.225)	0.385 (0.336–0.449)	0.676 (0.559–0.850)	0.917 (0.725–1.08)	667
	2003–2004	0.190 (0.173–0.210)	0.185 (0.168–0.207)	0.338 (0.288–0.431)	0.700 (0.500–0.818)	0.865 (0.708–1.10)	717
	2005–2006	0.171 (0.159–0.183)	0.160 (0.140–0.180)	0.320 (0.290–0.370)	0.550 (0.500–0.580)	0.700 (0.610–0.730)	692
	2007–2008	0.180 (0.164–0.197)	0.170 (0.160–0.190)	0.330 (0.290–0.390)	0.600 (0.470–0.700)	0.770 (0.690–0.900)	589

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-5. Geometric Mean and Selected Percentile Urine Concentrations (Creatinine Corrected) ($\mu\text{g/g}$ Creatinine) of Cadmium in the U.S. Population from 1999 to 2008

Group	Survey years	Geometric mean ^a (95% CI)	Selected percentiles (95% CI)				Sample size
			50 th	75 th	90 th	95 th	
Non-Hispanic whites	1999–2000	0.175 (0.146–0.209)	0.219 (0.191–0.250)	0.432 (0.387–0.470)	0.729 (0.666–0.783)	1.00 (0.826–1.16)	760
	2001–2002	0.205 (0.184–0.229)	0.224 (0.208–0.242)	0.421 (0.382–0.470)	0.719 (0.668–0.784)	0.931 (0.806–1.05)	1,132
	2003–2004	0.220 (0.209–0.235)	0.221 (0.197–0.253)	0.434 (0.398–0.476)	0.687 (0.647–0.767)	1.00 (0.830–1.08)	1,070
	2005–2006	0.193 (0.169–0.221)	0.180 (0.160–0.220)	0.390 (0.310–0.480)	0.680 (0.630–0.750)	0.930 (0.800–1.07)	1,041
	2007–2008	0.199 (0.178–0.221)	0.200 (0.180–0.220)	0.380 (0.340–0.440)	0.700 (0.630–0.810)	1.03 (0.880–1.13)	1,095

^aThe proportion of results below the LOD was too high to provide a valid result.

CI = confidence interval; LOD = limit of detection

Source: CDC 2011

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-6. Geometric Mean and Selected Percentile Urine Concentrations (µg/L) of Cadmium in the U.S. Population from 1999 to 2008

Group	Survey years	Geometric mean ^a (95% CI)	Selected percentiles (95% CI)				Sample size
			50 th	75 th	90 th	95 th	
Total, age 6 and older	1999–2000	0.193 (0.169–0.220)	0.232 (0.214–0.249)	0.475 (0.436–0.519)	0.858 (0.763–0.980)	1.20 (1.06–1.33)	2,257
	2001–2002	0.210 (0.189–0.235)	0.229 (0.207–0.255)	0.458 (0.423–0.482)	0.839 (0.753–0.919)	1.20 (1.07–1.28)	2,690
	2003–2004	0.211 (0.196–0.226)	0.210 (0.200–0.230)	0.450 (0.400–0.500)	0.800 (0.730–0.880)	1.15 (0.980–1.26)	2,543
	2005–2006	0.191 (0.170–0.216)	0.200 (0.170–0.220)	0.400 (0.360–0.460)	0.780 (0.700–0.860)	1.05 (0.960–1.17)	2,576
	2007–2008	0.185 (0.173–0.198)	0.180 (0.170–0.200)	0.380 (0.360–0.400)	0.700 (0.50–0.770)	1.00 (0.920–1.12)	2,627
Age group							
6–11 Years	1999–2000	Not calculated	0.078 (0.061–0.101)	0.141 (0.115–0.173)	0.219 (0.178–0.233)	0.279 (0.211–0.507)	310
	2001–2002	0.061 (<LOD–0.081)	0.077 (0.067–0.092)	0.140 (0.112–0.160)	0.219 (0.184–0.262)	0.282 (0.260–0.326)	368
	2003–2004	0.077 (0.065–0.090)	0.080 (0.060–0.090)	0.120 (0.100–0.160)	0.190 (0.160–0.310)	0.310 (0.170–0.610)	287
	2005–2006	0.066 (0.056–0.078)	0.060 (0.050–0.080)	0.110 (0.090–0.130)	0.180 (0.130–0.240)	0.240 (0.160–0.290)	355
	2007–2008	0.064 (0.058–0.071)	0.060 (0.050–0.070)	0.110 (0.090–0.130)	0.180 (0.140–0.210)	0.230 (0.180–0.310)	394
12–19 Years	1999–2000	0.092 (0.067–0.126)	0.128 (0.107–0.148)	0.202 (0.183–0.232)	0.329 (0.272–0.372)	0.424 (0.366–0.596)	648
	2001–2002	0.109 (0.087–0.136)	0.135 (0.114–0.157)	0.210 (0.189–0.247)	0.327 (0.289–0.366)	0.442 (0.366–0.480)	762
	2003–2004	0.121 (0.119–0.134)	0.130 (0.110–0.150)	0.200 (0.160–0.190)	0.300 (0.260–0.360)	0.390 (0.330–0.490)	724
	2005–2006	0.099 (0.090–0.109)	0.110 (0.100–0.120)	0.170 (0.150–0.190)	0.240 (0.210–0.280)	0.310 (0.250–0.430)	701
	2007–2008	0.089 (0.079–0.100)	0.080 (0.070–0.110)	0.150 (0.140–0.170)	0.260 (0.200–0.300)	0.330 (0.280–0.410)	376
≥20 Years	1999–2000	0.281 (0.253–0.313)	0.306 (0.261–0.339)	0.551 (0.510–0.621)	0.979 (0.836–1.13)	1.31 (1.13–1.57)	1,299
	2001–2002	0.273 (0.249–0.299)	0.280 (0.261–0.308)	0.545 (0.493–0.607)	0.955 (0.855–1.06)	1.28 (1.20–1.43)	1,560
	2003–2004	0.260 (0.238–0.284)	0.210 (0.210–0.300)	0.260 (0.470–0.580)	0.890 (0.800–0.990)	1.25 (1.09–1.46)	1,532
	2005–2006	0.241 (0.213–0.272)	0.250 (0.220–0.290)	0.490 (0.440–0.560)	0.860 (0.790–0.930)	1.12 (1.06–1.32)	1,520
	2007–2008	0.232 (0.215–0.251)	0.240 (0.210–0.260)	0.450 (0.410–0.490)	0.790 (0.730–0.870)	1.13 (0.990–1.44)	1,857

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-6. Geometric Mean and Selected Percentile Urine Concentrations (µg/L) of Cadmium in the U.S. Population from 1999 to 2008

Group	Survey years	Geometric mean ^a (95% CI)	Selected percentiles (95% CI)				Sample size
			50 th	75 th	90 th	95 th	
Gender							
Males	1999–2000	0.199 (0.165–0.241)	0.227 (0.193–0.263)	0.462 (0.381–0.539)	0.892 (0.748–1.15)	1.41 (0.980–1.83)	1,121
	2001–2002	0.201 (0.177–0.229)	0.223 (0.191–0.257)	0.445 (0.393–0.481)	0.870 (0.741–1.03)	1.22 (1.12–1.38)	1,335
	2003–2004	0.206 (0.190–0.222)	0.210 (0.190–0.230)	0.440 (0.390–0.490)	0.790 (0.700–0.870)	1.01 (0.890–1.25)	1,277
	2005–2006	0.195 (0.176–0.217)	0.210 (0.190–0.230)	0.400 (0.360–0.440)	0.800 (0.730–0.890)	1.17 (1.01–1.30)	1,271
	2007–2008	0.179 (0.162–0.197)	0.180 (0.160–0.200)	0.360 (0.320–0.400)	0.670 (0.540–0.780)	0.950 (0.800–1.14)	1,327
Females	1999–2000	0.187 (0.153–0.229)	0.239 (0.220–0.255)	0.492 (0.456–0.540)	0.806 (0.705–0.980)	1.10 (1.01–1.19)	1,136
	2001–2002	0.219 (0.192–0.251)	0.234 (0.202–0.265)	0.466 (0.433–0.519)	0.817 (0.733–0.886)	1.17 (0.918–1.36)	1,355
	2003–2004	0.216 (0.195–0.238)	0.210 (0.200–0.240)	0.450 (0.400–0.530)	0.820 (0.700–0.960)	1.20 (1.02–1.37)	1,266
	2005–2006	0.188 (0.160–0.221)	0.190 (0.170–0.200)	0.400 (0.350–0.480)	0.750 (0.640–0.860)	0.980 (0.830–1.20)	1,305
	2007–2008	0.191 (0.177–0.207)	0.190 (0.170–0.200)	0.400 (0.370–0.430)	0.740 (0.670–0.840)	1.09 (0.940–1.38)	1,300
Race/ethnicity							
Mexican Americans	1999–2000	0.191 (0.157–0.233)	0.202 (0.167–0.221)	0.438 (0.351–0.551)	0.813 (0.686–0.977)	1.12 (0.886–1.38)	780
	2001–2002	0.160 (0.135–0.189)	0.181 (0.171–0.198)	0.321 (0.285–0.362)	0.559 (0.430–0.733)	0.766 (0.633–1.15)	683
	2003–2004	0.175 (0.151–0.203)	0.170 (0.150–0.210)	0.350 (0.290–0.430)	0.680 (0.520–0.820)	1.04 (0.820–1.20)	614
	2005–2006	0.173 (0.152–0.193)	0.180 (0.160–0.200)	0.340 (0.300–0.380)	0.560 (0.500–0.630)	0.780 (0.660–0.900)	652
	2007–2008	0.160 (0.141–0.182)	0.170 (0.140–0.210)	0.320 (0.290–0.380)	0.570 (0.520–0.640)	0.730 (0.640–0.840)	515
Non-Hispanic blacks	1999–2000	0.283 (0.208–0.387)	0.312 (0.243–0.412)	0.633 (0.498–0.806)	1.22 (0.892–1.38)	1.48 (1.30–1.72)	546
	2001–2002	0.277 (0.229–0.336)	0.302 (0.257–0.354)	0.580 (0.476–0.713)	1.04 (0.843–1.38)	1.51 (1.28–1.74)	667
	2003–2004	0.265 (0.237–0.295)	0.270 (0.220–0.320)	0.550 (0.440–0.640)	0.960 (0.810–1.17)	1.52 (1.06–1.82)	717
	2005–2006	0.236 (0.240–0.210)	0.240 (0.210–0.260)	0.480 (0.420–0.530)	0.830 (0.670–0.930)	1.04 (0.870–1.26)	692
	2007–2008	0.246 (0.218–0.277)	0.260 (0.220–0.300)	0.460 (0.420–0.530)	0.840 (0.690–0.980)	1.40 (0.900–1.85)	589

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-6. Geometric Mean and Selected Percentile Urine Concentrations ($\mu\text{g/L}$) of Cadmium in the U.S. Population from 1999 to 2008

Group	Survey years	Geometric mean ^a (95% CI)	Selected percentiles (95% CI)				Sample size
			50 th	75 th	90 th	95 th	
Non-Hispanic whites	1999–2000	0.175 (0.148–0.206)	0.220 (0.194–0.246)	0.455 (0.388–0.510)	0.797 (0.714–1.01)	1.17 (0.963–1.47)	760
	2001–2002	0.204 (0.179–0.231)	0.221 (0.191–0.255)	0.445 (0.394–0.479)	0.813 (0.717–0.875)	1.17 (0.989–1.24)	1,132
	2003–2004	0.209 (0.192–0.226)	0.200 (0.190–0.220)	0.440 (0.390–0.500)	0.790 (0.700–0.860)	1.13 (0.940–1.26)	1,070
	2005–2006	0.185 (0.159–0.216)	0.200 (0.160–0.230)	0.400 (0.330–0.480)	0.780 (0.670–0.920)	1.05 (0.940–1.25)	1,041
	2007–2008	0.177 (0.161–0.195)	0.170 (0.150–0.190)	0.370 (0.330–0.400)	0.690 (0.620–0.780)	1.00 (0.880–1.12)	1,095

^aThe proportion of results below the LOD was too high to provide a valid result.

CI = confidence interval; LOD = limit of detection

Source: CDC 2011

6. POTENTIAL FOR HUMAN EXPOSURE

As a part of the New York City Health and Nutrition Examination Survey (NYC HANES), 2004 blood cadmium levels were evaluated in 1,811 New York City adults (age 20 years and older). The variables used in this study were sex, age, race/ethnicity, place of birth, family income, education, and smoking status (see [Table 6-7](#) for detailed results of this study). The geometric mean blood cadmium concentration in New York City adults was 0.77 µg/L, slightly higher than the 1999–2000 estimated national mean of 0.47 µg/L with heavy smokers having the highest geometric mean blood cadmium level of 1.58 µg/L, higher than any other subgroup. The reason for the elevated blood cadmium levels in nonsmoking, New York City adults is not known, although it was speculated that higher shellfish consumption may be the cause of elevated blood cadmium levels in Asian subgroup (McKelvey et al. 2007).

Vahter et al. (1996) studied the dietary intake and uptake of cadmium in nonsmoking women consuming a mixed diet low in shellfish (n=34) or with shellfish once a week or more (n=17). The shellfish diets, with a median of 22 µg Cd/day, contained twice as much cadmium as the mixed diets, which had a median of 10.5 µg Cd/day. In spite of the differences in the daily intake of cadmium, there were no statistically significant differences in the blood cadmium concentrations of the shellfish group (0.25 µg/L) and the mixed diet group (0.23 µg/L) or in the urinary cadmium concentrations of the shellfish and mixed diet groups (0.10 µg/L in both groups). These results indicate a lower absorption of cadmium in the shellfish group than in the mixed diet group or a difference in kinetics. The authors suggested that a higher gastrointestinal absorption of cadmium in the mixed diet group could be explained in part by their lower body iron stores as measured by the concentrations of serum ferritin (S-fer). A median S-fer concentration of 18 µg/L was measured for the mixed diet group compared to a median of 31 µg/L for the shellfish group.

Except in the vicinity of cadmium-emitting industries or incinerators, the intake of cadmium from drinking water or ambient air is of minor significance (Elinder 1985a). Cadmium is removed from waste water and sewage through precipitation to hydroxide or carbonate compounds and ultimate separation (Schulte-Schrepping and Piscator 2002). EPA requires water suppliers to limit the cadmium concentration in water to <5 µg/L (EPA 2006a).

IARC (1993) reports that the total body burden of non-occupationally exposed adult subjects has been estimated to range from 9.5 to 50 mg in the United States and Europe. People living near sources of cadmium pollution may be exposed to higher levels of cadmium. Ambient air cadmium concentrations in industrialized areas was estimated between 15 and 150 ng/m³ (Morrow 2001). During a study conducted

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-7. Blood Cadmium Concentrations, Geometric Means, Adjusted Proportional Change in Means, and 95th Percentiles in New York City Adults in Population Subgroups

Variable	Number ^a	Results		
		Crude weighted geometric mean blood cadmium (µg/L)	Adjusted proportional change in mean blood cadmium (µg/L) ^b	Crude weighted 95 th percentile blood cadmium (µg/L)
Total:	1,811	0.77	—	1.88
Male	762	0.76	1.00	1.95
Female	1,049	0.79	1.07	1.83
20–39 years old	903	0.76	1.00	1.82
40–59 years old	673	0.84	1.16	2.19
≥60 years old	235	0.77	1.15	1.52
White, non-Hispanic ^c	529	0.73	1.04	1.71
Black, non-Hispanic ^c	390	0.80	1.11	1.97
Asian, non-Hispanic ^c	231	0.99	1.41	2.36
Hispanic ^c	630	0.73	1.00	1.73
Place of birth:				
United States	882	0.76	1.00	1.95
Outside the United States	923	0.79	1.02	1.73
Family income (\$ U.S.):				
<20,000	610	0.86	1.00	2.33
20,000–49,999	566	0.77	0.94	1.76
50,000–74,999	256	0.74	0.92	1.76
≥75,000	304	0.69	0.91	1.43
Education:				
<Bachelors	1,252	0.82	1.09	2.02
Bachelors or greater	551	0.69	1.00	1.43
Smoking status:				
Never smoked	1,036	0.66	1.00	1.28
Former smoker	310	0.71	1.07	1.32
Current smoker	449	1.22	1.88	3.00

^aTotals do not all equal 1,811 because of missing data.

^bThe exponential β coefficient from a log-linear multiple regression that includes all covariates in the table. Sample size for adjust analysis is 1,707, after excluding study participants for whom covariate data are missing.

^cExcludes 27 participants who self-classified as "other".

Source: McKelvey et al. 2007

6. POTENTIAL FOR HUMAN EXPOSURE

in Germany between March and May 2000, cadmium levels in child-mother pairs, as a function of ambient air quality, were compared between populations in the urban, industrialized area of Duisburg and the rural area of North Rhine Westphalia. Cadmium levels in the ambient air of Duisburg-South ranged from 1.5 to 31 ng/m³, compared to 0.5 ng/m³ in the rural area of Westphalia. Cadmium levels in the blood and urine of mothers in the industrialized area were higher than in the rural areas. Cadmium levels in the blood and urine of the children did not differ between the two areas. In the industrialized area, regression analysis indicated a significant influence of cadmium in ambient air on cadmium in blood (Wilhelm et al. 2005).

It has been estimated that tobacco contains 1.7 µg cadmium per cigarette, and about 10% is inhaled when smoked (Morrow 2001; NTP 2005). Tobacco leaves naturally accumulate large amounts of cadmium (Morrow 2001). During a study monitoring cadmium levels in 331 cigarette packs from over 20 areas around the world, it was found that the mean cadmium level per cigarette was 1.15 µg/cigarette ±0.43 (AM±ASD) or 1.06 µg/cigarette ±1.539 GM±GSD. Cigarettes from Mexico had the highest mean level of cadmium with an AM±ASD of 2.03 µg/cigarette ±0.33 or a GM±GSD of 2.00 µg/cigarette ±1.190. Cigarettes from India had the lowest mean levels of cadmium with an AM±ASD of 0.35 µg/cigarette ±0.09 or a GM±GSD of 0.34 µg/cigarette ±1.284 (Watanabe et al. 1987). The amount of cadmium absorbed from smoking one pack of cigarettes per day is about 1–3 µg/day (Lewis et al. 1972a; Nordberg et al. 1985), roughly the same as from the diet. This large contribution is due to the greater absorption of cadmium from the lungs than from the gastrointestinal tract (Elinder 1985a). Direct measurement of cadmium levels in body tissues confirms that smoking roughly doubles cadmium body burden in comparison to not smoking, with kidney concentrations averaging 15–20 µg/g wet weight for nonsmokers and 30–40 µg/g wet weight for heavy smokers at the age of 50–60 (Ellis et al. 1979; Hammer et al. 1973; Lewis et al. 1972a, 1972b). Ellis et al. (1979) found an increase in kidney cadmium of 0.11±0.05 mg per pack-year (AM±ASD) of smoking and an increase in liver cadmium concentration of 0.077±0.065 µg/g per pack-year (AM±ASD). Because excretion of cadmium is very slow, half-lives of cadmium in the body are correspondingly long (17–38 years) (Wester et al. 1992).

Workers in a variety of occupations may be exposed to cadmium and cadmium compounds. Occupations with potential exposure to cadmium are listed in [Table 6-8](#) (IARC 1993).

Highest levels of occupational exposure would be expected to occur in operations involving heating cadmium-containing products by smelting, welding, soldering, or electroplating, and also in operations associated with producing cadmium powders (OSHA 1990). The primary route of occupational exposure

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-8. Occupations with Potential Exposure to Cadmium and Cadmium Compounds

Occupation	
Alloy production ^a	Phosphorous production
Battery production ^a	Pigment production and use ^a
Brazing	Plastics production ^a
Coating	Plating
Diamond cutting	Printing
Dry color formulation	Semiconductor and superconductor production
Electroplating	Sensors production
Electrical contacts production	Smelting and refining ^a
Enameling	Solar cells production
Engraving	Soldering
Glasswork	Stabilizer production
Laser cutting	Textile printing
Metallizing	Thin film production
Paint production and use	Transistors production
Pesticide production and use	Welding

^aActivity with high risk because atmospheric concentrations of cadmium are high and the number of workers employed is significant.

Source: IARC 1993

6. POTENTIAL FOR HUMAN EXPOSURE

is through inhalation of dust and fumes, and also incidental ingestion of dust from contaminated hands, cigarettes, or food (Adamsson et al. 1979).

Concentrations of airborne cadmium found in the workplace vary considerably with the type of industry and the specific working conditions. Processes that involve high temperatures can generate cadmium oxide fumes that are absorbed very efficiently through the lungs (IARC 1993). Deposition and absorption of dust containing different compounds depend upon particle size (IARC 1993). These exposures can be controlled through use of personal protective equipment and good industrial hygiene practices, and through operating procedures designed to reduce workplace emissions of cadmium (OSHA 1990).

Data from the National Occupational Exposure Survey (NOES), conducted by NIOSH from 1981 to 1983, estimated the number of workers potentially exposed to various chemicals in the workplace during the same period (NIOSH 1990); these data are summarized in [Table 6-9](#). The NOES database does not contain information on the frequency, level, or duration of exposure of workers to any of the chemicals listed. It provides only estimates of workers potentially exposed to the chemicals.

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).

Children are most likely to be exposed to cadmium in from ingestion of food (NTP 2005). There are no data on gastrointestinal absorption of cadmium in children, although very limited evidence exists that cadmium absorption from the gut may be greater in young animals. Oral absorption is discussed in more detail in Section 3.4.1.2. A study performed in Cincinnati, Ohio, investigated cadmium in human milk

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-9. Estimated Number of Workers Potentially Exposed to Various Chemicals in the Workplace in 1981–1983

Chemical	Number of workers potentially exposed
Cadmium sulfide	45,562
Cadmium oxide	15,727
Cadmium (pure)	335
Cadmium dust (form unknown)	3,893
Cadmium powder (form unknown)	486
Cadmium sulfate	1,313
1:1 Cadmium salt of carbonic acid	164
Cadmium (form unknown)	88,968
Total	153,486

Source: NIOSH 1990

6. POTENTIAL FOR HUMAN EXPOSURE

and found a mean concentration of 19 ppb (0.019 ppm) (Jensen 1983). The NHANES 1999–2008 reported cadmium levels in blood (see Table 6-4) and urine (see Tables 6-5 and 6-6) for children in different age groups (CDC 2011). The NYC HANES did not test for blood cadmium levels in children, although the blood cadmium levels in adults were slightly higher than the national average (McKelvey et al. 2007). Results of the U.S. FDA Total Diet Study (Capar and Cunningham 2000) reported cadmium levels in infant and junior foods ranged from no detection to 0.090 mg/kg. According to the National Human Exposure Assessment Survey (NHEXAS), children in EPA Region V (Great Lakes Region) have a mean dietary cadmium exposure of 17 (± 1.8) $\mu\text{g}/\text{kg}$ for minority children and 21 (± 2.2) $\mu\text{g}/\text{kg}$ for non-minority children (Pellizzari et al. 1999).

Except in the vicinity of cadmium-emitting industries or incinerators, the intake of cadmium from drinking water or ambient air is of minor significance (Elinder 1985a). Ambient air cadmium concentrations in industrialized areas has been estimated between 15 and 150 ng/m^3 (Morrow 2001). Cadmium levels in the ambient air of Duisburg-South, Germany ranged from 1.5 to 31 ng/m^3 , compared to 0.5 ng/m^3 in the rural are of Westphalia. Cadmium levels in the blood and urine of mothers in the industrialized area were higher than in the rural areas. Cadmium levels in the blood and urine of the children did not differ between the two areas. In the industrialized area, regression analysis indicated a significant influence of cadmium in ambient air on cadmium in blood (Wilhelm et al. 2005). Children in the homes of parents who smoke also can be exposed to cadmium through the inhalation of environmental tobacco smoke. There is potential for cadmium originating from second-hand smoke to settle onto surfaces; thus, there is a possibility that children may ingest cadmium from contaminated surfaces by the hand-to-mouth pathway. Although no data were found, children playing near hazardous waste sites could be exposed to cadmium in soil by hand-to-mouth activity and/or soil pica. No case studies were found on accidental poisoning of children by swallowing cadmium-containing batteries or by ingesting cadmium-containing household pesticides, which also are potential routes of exposure. No information was found concerning differences in the weight-adjusted intakes of cadmium by children.

In the Workers' Home Contamination Study conducted under the Workers' Family Protection Act (DHHS 1995), several studies were identified that reported home contamination with cadmium originating from parental occupation in a lead smelter. In a study of 396 children of ages 1–9 years living <900 m from a primary lead smelter, 380 children (96%) had blood cadmium (CdB) levels $>0.0089 \mu\text{g}/\text{L}$ (Carvalho et al. 1986). The geometric mean and standard deviation were 0.087 $\mu\text{mol}/\text{L}$ and 2.5, respectively. No significant relationship was found between parental occupation in the smelter and CdB in children, but a significant relationship was found between presence of smelter dross in the house and

6. POTENTIAL FOR HUMAN EXPOSURE

elevated CdB in children. Higher CdB was significantly associated with shorter distance from the home to the smelter. In a similar study of 263 children (ages 1–9 years), living <900 m from a primary lead smelter, the mean cadmium in hair was significantly higher at 6.0 ppm for children whose fathers worked in lead smelters than the concentration of 3.7 ppm for children whose fathers had other jobs (Carvalho et al. 1989). In a study of 9 children from families of lead workers and 195 children (ages 4–17 years) from other families, the children from the families of lead workers had significantly higher geometric mean urinary cadmium (CdU) ($0.34 \mu\text{g/L} \pm 2.6$) than children from other families ($0.13 \mu\text{g/L} \pm 2.2$). The CdB levels of children from families of lead workers were higher than those of the children from other families, but the difference was not statistically significant (Brockhaus et al. 1988). Maravelias et al. (1989) measured the CdBs of 514 children (ages 5–12) from four schools located within various distances (500–1500 m) from a lead smelter. The geometric mean and geometric standard deviation CdB was $0.36 \mu\text{g/L} \pm 1.4$, respectively, with a range of 0.1–3.1 $\mu\text{g/L}$. Children from the school closest to the smelter had higher CdB levels than children from other schools, but no relationship was found between childrens' CdB and parental employment in the smelter.

The placenta may act as a partial barrier to fetal exposure to cadmium. Cadmium concentration has been found to be approximately half as high in cord blood as in maternal blood in several studies including both smoking and nonsmoking women (Kuhnert et al. 1982; Lauwerys et al. 1978; Truska et al. 1989). Accumulation of cadmium in the placenta at levels about 10 times higher than maternal blood cadmium concentration has been found in studies of women in Belgium (Roels et al. 1978) and the United States (Kuhnert et al. 1982); however, in a study in Czechoslovakia, the concentration of cadmium in the placenta was found to be less than in either maternal or cord blood (Truska et al. 1989). Baranowska (1995) also measured the concentrations of cadmium and lead in human placenta and in maternal and neonatal (cord) blood to assess the influence of a strongly polluted environment on the content of metals in tissues and on the permeability of the placenta to cadmium and lead. Samples for the study were collected from women living in the industrial district of Upper Silesia, one of the most polluted regions in Poland. The mean (range) concentration of cadmium in the air was $11.3 (2.1\text{--}25.4) \text{ ng/m}^3$ ($0.0113 [0.0021\text{--}0.0254] \mu\text{g/m}^3$). The mean concentrations of cadmium were 4.90 ng/mL ($0.00490 \mu\text{g/mL}$) in venous blood, $0.11 \mu\text{g/g}$ in placenta, and 1.13 ng/mL ($0.00113 \mu\text{g/mL}$) in cord blood. The researcher concluded that the placenta is a better barrier for cadmium than for lead, based upon the relative decrease in metal concentrations from placenta to cord blood. The mechanism by which the placenta transports the essential metals, copper and zinc, while limiting the transport of cadmium is unknown, but may involve the approximately 1,000-fold higher concentration of zinc in the placenta and the higher affinity of cadmium than zinc for metallothionein (Goyer and Cherian 1992). Timing and level

6. POTENTIAL FOR HUMAN EXPOSURE

of cadmium exposure may influence the uptake of cadmium by the placenta, perhaps explaining the conflicting human studies. Galicia-García et al. (1995) performed analyses of cadmium in maternal, cord, and newborn blood for 50 births in a Mexico City hospital. Multiple regression analyses applied to the data indicated a significant association between cord and newborn blood and between cord and maternal blood, but not among maternal and newborn blood. Birth weight of the newborns was found to be inversely associated with cord blood cadmium levels and smoking habits.

The analysis of Weidenberg et al. (2011) on the bioavailability of cadmium present in inexpensive jewelry suggests that substantial quantities of cadmium may be released from jewelry that is mouthed or swallowed and that the amount released was greatly varied from item to item.

6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

The greatest potential for above-average exposure of the general population to cadmium is from smoking, which may double the exposure of a typical individual. Smokers who are exposed to cadmium in the workplace are at highest risk (CDC 2005). Individuals living near zinc or lead smelting operations, municipal incinerators, or other industrial processes emitting cadmium to the air will also have above-average exposure (Elinder 1985a). Exposures through inhalation are diminishing due to pollution controls at such facilities, but exposure resulting from soil contamination may continue to be significant. Persons who have corrosive drinking water and cadmium-containing plumbing, who habitually consume cadmium-concentrating foods (kidney, liver, and shellfish), or who ingest grains or vegetables grown in soils treated with municipal sludge or phosphate fertilizer all may have increased exposure (Elinder 1985a). The 2004 NYC HANES indicated that the New York City Asian population, especially those born in China, had higher concentrations of cadmium in blood. The authors speculate that this might be due to higher consumption of fish and shellfish (McKelvey et al. 2007).

Multiple pathways of exposure may exist for populations at hazardous waste sites contaminated with cadmium (ingestion of contaminated drinking water or garden vegetables, inhalation of airborne dust, incidental ingestion of contaminated soil).

Persons who consume large quantities of sunflower kernels can be exposed to higher levels of cadmium. Reeves and Vanderpool (1997) identified specific groups of men who were likely to consume sunflower kernels. The groups included baseball and softball players, delivery and long-distance drivers, and line workers in sunflower kernel processing plants.

6. POTENTIAL FOR HUMAN EXPOSURE

Recreational and subsistence fishers that consume appreciably higher amounts of locally caught fish from contaminated waterbodies may be exposed to higher levels of cadmium associated with dietary intake (EPA 1993a). Cadmium contamination has triggered the issuance of several human health advisories. As of December 1997, cadmium was identified as the causative pollutant in five fish and shellfish consumption advisories in New York and another in New Jersey. EPA is considering including cadmium as a target analyte and has recommended that this metal be monitored in fish and shellfish tissue samples collected as part of state toxics monitoring programs. EPA recommends that residue data obtained from these monitoring programs be used by states to conduct risk assessments to determine the need for issuing fish and shellfish consumption advisories for the protection of the general public as well as recreational and subsistence fishers. Under the same program, EPA has issued a statewide advisory in Maine for cadmium in moose (EPA 1998).

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 cadmium 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 cadmium.

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 chemical and physical properties of cadmium and its salts are known well enough to permit estimation of the environmental fate of the compounds (Elinder 1985a, 1992). Additional information on properties does not appear to be crucial for evaluating potential fate.

6. POTENTIAL FOR HUMAN EXPOSURE

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 2009, became available in February of 2011. This database is updated yearly and should provide a list of industrial production facilities and emissions.

The production volume, producers, import/export quantities, and uses of cadmium in the United States are well documented (SRI 2007; USGS 2007, 2008). Recycling of cadmium from spent batteries is increasing, and there are some data to suggest that there is still a large portion of cadmium being disposed of as municipal waste (USGS 2007). More data concerning the amount of municipal disposal would be helpful. Disposal of cadmium-containing wastes is regulated by the federal government, and data are available for industrial disposal practices (EPA 1982a; HSDB 2008; U.S. Bureau of Mines 1990). Most releases of cadmium are not from production of the metal or its compounds, but from combustion or smelter emissions, land application of sewage sludge and fertilizers, and other sources; estimates of these releases have been made (TRI09 2011).

Environmental Fate. Cadmium partitioning among media occurs, and this partitioning depends on local environmental conditions (Elinder 1985a, 1992). Cadmium may be subject to long-range transport in air and water (EPA 1980d). Cadmium is persistent in all media, although it may form organic complexes in soil and water under certain environmental conditions (EPA 1979). These processes, which are important for determining the environmental fate of cadmium, seem to be relatively well understood. Therefore, additional information on environmental fate does not appear to be essential to evaluate potential human exposure to cadmium.

Bioavailability from Environmental Media. Factors that control the bioavailability of cadmium from air, water, soil, and food have been investigated. Intestinal absorption of cadmium from food is low, about 5–10% (McLellan et al. 1978; Newton et al. 1984; Rahola et al. 1973), but the absorption of cadmium from soil is not known. Absorption from the lungs is somewhat greater, averaging about 25% (Nordberg et al. 1985). Estimates of dermal absorption of cadmium from soil and water on human skin have been made (Wester et al. 1992). There is some evidence that bioavailability of cadmium to plants and worms from contaminated soil is greater following remediation (Van Gestel et al. 1988). Additional information on the factors influencing bioavailability, particularly from remediated soil, are needed to assess residual risk to populations in the vicinity of reclaimed hazardous waste sites.

6. POTENTIAL FOR HUMAN EXPOSURE

Food Chain Bioaccumulation. Sufficient data are available to indicate that cadmium is concentrated in plants, aquatic organisms, and animals (Alloway et al. 1990; Beyer 1986; Handy 1992a, 1992b; Kuroshima 1992; Naqvi and Howell 1993; Roseman et al. 1994; Suresh et al. 1993; Vos et al. 1990). In vertebrates, cadmium accumulates in the liver and kidneys (Harrison and Klaverkamp 1990; Sileo and Beyer 1985; Vos et al. 1990). There is strong evidence for food chain bioaccumulation, but the potential for biomagnification is uncertain. Additional studies on biomagnification are needed to provide data for more accurate evaluation of the environmental impact of cadmium contamination.

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

Current ambient air quality surveys testing for cadmium concentrations in rural and urban locations in the United States is lacking. Since the major source of exposure to cadmium is through dietary intake and since cadmium emissions to air are not expected to increase, there may be less interest in these data. There are several long-range atmospheric transport studies, but since these were conducted Europe and Russia, they only illustrate the potential for cadmium contamination via atmospheric deposition in the United States (Reimann et al. 1997; Shevchenko et al. 2003; Vidovic et al. 2005). There is also minimal data on current levels of cadmium in agricultural soils of the United States and the identification of the sources of cadmium levels, whether they are native geochemistry, phosphate fertilizers, atmospheric deposition, etc. (Xue et al. 2000). Continuing monitoring efforts in all media would allow more precise estimation of current sources and levels of human exposure and would assist in identifying major sources contributing to current exposure.

Exposure Levels in Humans. Cadmium has been detected in human blood, urine, breast milk, liver, kidney, and other tissues, both in occupationally exposed individuals and in the general population (CDC 2011; McKelvey et al. 2007; OSHA 1990). The NHANES and NYC HANES provide current data on the levels of cadmium in humans (CDC 2011; McKelvey et al. 2007). Other large-scale surveys concentrating on urban, agricultural, and suburban communities would be beneficial in understanding cadmium exposure to the U.S. population. Also, more information is needed on the specific exposure levels for different cadmium salts to determine if cadmium sulfides, for example, are associated with less harmful effects than cadmium oxides (Chettle and Ellis 1992).

6. POTENTIAL FOR HUMAN EXPOSURE

This information is necessary for assessing the need to conduct health studies on these populations.

Exposures of Children. Cadmium has been measured in maternal and neonatal (cord) blood and in placenta (Baranowska 1995; Galicia-García et al. 1995; Kuhnert et al. 1982; Lauwerys et al. 1978; Roels et al. 1978; Truska et al. 1989), but the resulting data are sometimes conflicting with respect to the uptake of cadmium by the placenta. Research on the effects of timing and level of exposure on cadmium uptake by the placenta might help to explain these conflicting human studies. More recent data would be useful, both from women and children living in unpolluted areas (for background levels) and in polluted areas such as those near existing or former lead smelters.

There are some current data concerning cadmium exposure in children (Capar and Cunningham 2000; CDC 2011; Pellizzari et al. 1999). The NHANES 1999–2008 reported cadmium levels in blood (see [Table 6-4](#)) and urine (see [Table 6-5](#)) for children in different age groups (CDC 2011). The NYC HANES did not test for blood cadmium levels in children, although the blood cadmium levels in adults were slightly higher than the national average (McKelvey et al. 2007). Results of the U.S. FDA Total Diet Study (Capar and Cunningham 2000) reported cadmium levels in infant and junior foods ranged from no detection to 0.090 mg/kg. According to the NHEXAS, children in EPA Region V (Great Lakes Region) have a mean dietary cadmium exposure of 17 (± 1.8) $\mu\text{g}/\text{kg}$ for minority children and 21 (± 2.2) $\mu\text{g}/\text{kg}$ for non-minority children (Pellizzari et al. 1999).

Some body burden data are available for children living near lead smelters (Lagerkvist and Lundstrom 2004; Leroyer et al. 2001; Jin et al. 2002). However, none of the studies took place in the United States. Body burden data from children living in polluted and unpolluted regions (for background levels) of the United States are needed.

Current information on whether children are different in their weight-adjusted intake of cadmium via oral, inhalation, and dermal exposures was not located. A study to determine this information would be useful. Also, no information was found on childhood specific means to reduce cadmium exposure.

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

6. POTENTIAL FOR HUMAN EXPOSURE

Exposure Registries. The State of New York has established the Heavy Metals Registry for surveillance of occupational heavy metals absorption. Cadmium levels >10 $\mu\text{g/L}$ in blood and 5 $\mu\text{g/L}$ in urine are reported to the registry. The number of adults with reportable levels has varies per year, but there have always been <50 adults reported per year. Between 1995 and 2003, the number of reportable adults was <5 , and these exposures are due mostly to exposure for people working as jewelers and casting machine operators (NYS Dept of Health 2006).

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

6.8.2 Ongoing Studies

The Federal Research in Progress (FEDRIP 2008) 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 6-10](#).

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-10. Ongoing Studies on Cadmium

Investigator	Affiliation	Research description	Sponsor
Birnbaum ER	Caldera Pharmaceuticals, Inc., Los Alamos, New Mexico	Biomarkers of response to environmental stressors	National Institute of Environmental Health Sciences
Chen Z	X-Ray Optical Systems, Inc. East Greenbush, New York	Direct measurement of trace elements in body fluids	National Center for Research Resources
Dweik BM	Giner, Inc., Newton, Massachusetts	Field-deployable monitor to assess personal exposure to multiple heavy metals	National Institute of Environmental Health Sciences
Fallin MD	Johns Hopkins University, Baltimore, Maryland	AGE-related epigenetic changes - environmental causes and disease consequences	National Institute of Environmental Health Sciences
Fox MA	Johns Hopkins University, Baltimore, Maryland	Environmental exposure to metal mixtures and kidney disease	National Institute of Environmental Health Sciences
Larkin PM	Ecoarray, Inc. Alachua, Florida	Developing and using sheepshead minnow microarrays for ecotoxicology	National Institute of Environmental Health Sciences
Mo J	Kumetrix, Inc, Union City, California	Automatic multi-analyte in-situ bioassay for monitoring exposure to toxic metals	National Institute of Environmental Health Sciences
Polette-Niewold LA	Mayan Pigments, Inc.	SBIR phase II: One-step environmentally-friendly synthesis of novel organic/inorganic hybrid pigments	National Science Foundation
Santra S	University of Central Florida	Selective detection of toxic heavy metal ions using highly sensitive quantum dot probes	National Science Foundation
Basta N; Raun WR	Oklahoma State University	Chemistry and bioavailability of waste constituents in soils	U.S. Department of Agriculture
Basta NT	Oklahoma State University	Heavy metal and trace element chemistry in soils: Chemical speciation and bioavailability	U.S. Department of Agriculture
Basta NT; Lower SK; Lanno R	Ohio State University	Heavy metal and trace element biogeochemistry in soils: Chemical speciation, bioavailability, and toxicity	U.S. Department of Agriculture
Bleam WF; Helmke PA	University of Wisconsin	Verifying and quantifying the specific complexation of metals to humic substances	U.S. Department of Agriculture
Chaney RL	Beltsville Agricultural Research Center	Characterization and remediation of potential trace element and phosphate risks from contaminated soils	U.S. Department of Agriculture
Chaney RL	Beltsville Agricultural Research Center	Risk assessment and remediation of soil and amendment trace elements	U.S. Department of Agriculture

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-10. Ongoing Studies on Cadmium

Investigator	Affiliation	Research description	Sponsor
Chaney RL; Daniels WL	Virginia Polytechnic Institute	Effects of long-term biosolids applications on phytoavailability of soil cadmium and zinc	U.S. Department of Agriculture
Chang AC; Page AL	University of California, Riverside	Chemistry and bioavailability of waste constituents in soils	U.S. Department of Agriculture
Eick MJ	Virginia Polytechnic Institute	Trace element and ligand adsorption/ desorption from soil constituent surfaces	U.S. Department of Agriculture
Hopkins DG	North Dakota State University	Influence of geologic materials and pedogenic processes on trace elements in soil landscapes	U.S. Department of Agriculture
Hunt JR; Lykken GI	University of North Dakota	Whole body counting and radiotracer methods in research on mineral requirements in human nutrition	U.S. Department of Agriculture
Kpomblekou- Ademawou K; Ankumah RO	Tuskegee University	Trace elements in broiler littered soils: Fate and effects on nitrogen transformation	U.S. Department of Agriculture
Martinez CE	Pennsylvania State University	Chemical and biogeochemical processes involved in trace and toxic element cycling in soils	U.S. Department of Agriculture
Morrissey MT	Oregon State University	Characterization of the cadmium health risk, concentrations and ways to minimize cadmium residues in shellfish	U.S. Department of Agriculture
Schwab AP; Joern B; Johnston C	Purdue University	Chemistry and bioavailability of waste constituents in soils	U.S. Department of Agriculture
Sparks DL	University of Delaware	Rates and mechanisms of metal and metalloid sorption/surfaces	U.S. Department of Agriculture
Thomas, DG; Kennedy TS	Oklahoma State University	Maternal dietary nutrients and neurotoxins in infant cognitive development	U.S. Department of Agriculture
Williams PL	University of Georgia	Environmental health impacts of soil contamination	U.S. Department of Agriculture
	National Risk Management Research Laboratory	Biomonitoring of source water quality	U.S. Environmental Protection Agency
Petterson L	National Exposure Research Lab Environmental Sciences Division Characterization and Monitoring Branch	Efficient monitoring of heterogeneous media and electronic wastes	U.S. Environmental Protection Agency

6. POTENTIAL FOR HUMAN EXPOSURE

Table 6-10. Ongoing Studies on Cadmium

Investigator	Affiliation	Research description	Sponsor
Petterson L	National Exposure Research Lab Ecosystems Research Division Ecosystems Assessment Branch	Geochemical and interfacial applications for assessing ecological toxicant exposures	U.S. Environmental Protection Agency
Nolan P	Office of Regional Administrator Office of Environmental Measurement and Evaluation	Lower Merrimack River fish tissue study	U.S. Environmental Protection Agency
Janes D	Office of Research and Development National Health and Environmental Effects Research Lab Mid-Continent Ecology Division	Risks of heavy metals to aquatic organisms from multiple exposure routes	U.S. Environmental Protection Agency

Sources: FEDRIP 2008; SI/EPA 2007