Draft
Lead Report

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Acronyms, Abbreviations, Symbols and Glossary

µ	symbol for micro
µg/dL	micrograms per deciliter
µg/g	microgram per gram
µg/L	micrograms per liter
µg/m^3	microgram per cubic meter
ADI	acceptable daily intake
Anglesite	Lead sulfate ore mineral, $PbSO_4$.
Annual Workplan	Category of contaminated sites DTSC is actively working on to remediate the contamination, either in a lead role or support capacity
ARB	California Air Resources Board
ATSDR	Agency for Toxic Substances and Disease Registry
BLL	Blood Lead Level, most common method of measuring lead exposure
BLM	U.S. Bureau of Land Management
BMPs	Best Management Practices
Cal Sites	DTSC database containing information on properties in California where hazardous substances have been released or where the potential for a release exists
CCR/T22	California Code of Regulations, Title 22
CDC	Centers for Disease Control and Prevention
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act (also referred to as Superfund)
Cerrusite	Lead carbonate ore mineral, $PbCO_3$
CFR	Code of Federal Regulations
cm	centimeter
CPSC	U.S. Consumer Product Safety Commission
CRTs	Cathode Ray Tubes
CWA	Federal Clean Water Act
CWC	California Waste Codes
CZARA	Coastal Zone Act Reauthorization Amendment
DPR	California Department of Pesticide Regulation
DOC	California Department of Conservation
DTSC	California Department of Toxic Substances Control
DWR	California Department of Water Resources
EP	Erythrocyte protoporphyrin
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FR	Federal Register
Galena	Lead sulfide ore mineral, PbS
g/gallon	grams per gallon
H&SC	California Health and Safety Code
HUD	U.S. Department of Housing and Urban Development
IARC	International Agency for Research on Cancer
IEUBK	Integrated Exposure Uptake Biokinetic exposure model used extensively by U.S. EPA to establish requirements for the lead paint abatement program
kg/L	kilograms per liter
LeadSpread	DTSC exposure model used for activities such as developing remediation goals
Litharge	yellow lead oxide, PbO, used in storage batteries, glass and as a pigment
Massicot	yellow crystalline mineral form of lead oxide, PbO
m	meter
MCL	Maximum Contaminant Level
mg/day	milligrams per day
mg/(m^2year)	Milligram per square meter, year

Acronyms, Abbreviations, Symbols and Glossary

mg/kg	milligram per kilogram
MMAD	Mass Median Aerodynamic Diameter; the point in a particle size distribution wherein half of the mass occurs on either side of the MMAD value
nano	Prefix used in SI units of measurement, equivalent to one-billionth part
NCHS	National Center for Health Statistics
ng/m^3	nanogram per cubic meter
NHANES	National Health and Nutrition Examination Survey
NPDES	National Pollutant Discharge Elimination System
NPS	non-point source
OAL	Office of Administrative Law
OEHHA	California Office of Environmental Health Hazard Assessment
OSHA	U.S. Occupation Safety and Health Administration
OSWER	Office of Solid Waste and Emergency Response, provides policy, guidance and direction for U.S. EPA's solid waste and emergency response programs.
Pb	symbol for lead
PbB	Blood lead
pg/g	Picogram per gram
pH	A numerical designation of relative acidity and alkalinity
POTW	Publicly Owned Treatment Works
ppm	parts per million
PVC	Polyvinyl chloride
RCRA	Resource Conservation and Recovery Act
Regional Water Boards	California Regional Water Quality Control Boards
RfD	Reference Dose
SIC Codes	Standard Industrial Classification Codes
SLI Batteries	Starting, lighting, and ignition batteries, used in most on-road vehicles, tractors, marine craft, aircraft and military vehicles
SDWA	Federal Safe Drinking Water Act
STLC	Soluble Threshold Limit Concentration. Threshold used in California to determine if a waste is hazardous according to the toxicity criteria
SWRCB	California State Water Resources Control Board
TRI	Toxic Chemical Release Inventory
TSCA	Federal Toxic Substances Control Act
TTLC	Total Threshold Limit Concentration. Threshold used in California to determine if a waste is hazardous according to the toxicity criteria
U.S.	United States
U.S. DHHS	U.S. Department of Health and Human Services
U.S. DOE	U.S. Department of Energy
U.S. EPA	U.S. Environmental Protection Agency
U.S.C.	United States Code, contains public laws passed by the U.S. Congress
U.S.C.A.	United States Code Annotated
USDA	United States Department of Agriculture
USGS	United States Geologic Survey

EXECUTIVE SUMMARY

This report examines lead in the environment, discussing the ways that lead appears in the environment, the means whereby exposure occurs, the health effects associated with that exposure, and the ways exposure can be minimized. Considerable research has been conducted recently regarding the health effects of lead, particularly as it affects children. New information about the low concentrations of lead that can adversely affect sensitive populations has prompted several changes in laws and regulations that provide additional protection. Accordingly, the Department of Toxic Substances Control (DTSC) proposes changes to the hazardous waste threshold that protects these sensitive populations from lead exposure.

Problem

The current hazardous waste thresholds for lead were developed over twenty years ago, before many of the initiatives to limit exposure to lead had been completed, and before the current findings regarding health effects were completely known. At the time that DTSC's hazardous waste standards were developed, the acceptable blood lead level for children was 30 µg/dL, a level now associated with a variety of physical and cognitive deficits. Currently, the federal Centers for Disease Control and Prevention consider child blood lead levels at or above 10 µg/dL to be elevated.

One of California's hazardous waste standards is the total threshold limit concentration (TTLC). The TTLC for lead is intended to protect receptors from direct exposure, primarily through ingestion, which is the type of exposure of greatest concern with lead and certain sensitive receptors. The TTLC in use today assumes that 1,000 mg/kg is protective of children likely to ingest soil containing lead. However, as shown by the recently developed health-based screening values for soils at school sites and near residential lead-paint sites, soil-lead concentrations that protect children who are exposed to lead in soil range from 255 mg/kg to 400 ppm, depending upon the model and assumptions used. The models and assumptions used to develop these values consider the most recent information regarding the health effects of lead and the exposure likely to occur.

Background

Lead is a ductile, dense metal, resistant to corrosion and a poor electrical conductor. Lead has a low melting point and readily forms alloys with other metals. It can absorb a broad range of radiation and is easily separated from its highly concentrated ores. This range of physical properties gives rise to the many uses of lead throughout history and today.

Lead has a long history of use, dating to the earliest civilizations. In fact, some of the ancient uses of lead, such as for plumbing, cosmetic and medicinal purposes, are still used today, albeit under extreme warnings and restrictions. Early uses in the U.S. were primarily for ammunition, brass and pewter, paints and protective coatings, glass and crystal, ceramic glazes, and water lines and pipes. Ultimately, lead use expanded to include machine bearings, cable covering, caulking, solder, fuel additives and lead acid storage batteries.

Over time, as concern over the health effects associated with lead began to grow, health and environmental regulations were enacted to restrict the use of lead in certain products and activities in the U.S. In the last twenty-five years, lead-based paint, leaded gasoline, leaded can solder and lead-containing plumbing materials were among the products that were gradually restricted or phased out of use. The use of lead, however, is far from obsolete. An important commodity, lead is now primarily used to manufacture lead acid batteries. Lead also continues to be used in paint, glass, ceramics, pigments, casting metals, metal products, solder, and other minor uses.

As a result of its extensive use throughout history, lead is ubiquitous in the environment. Manmade sources of lead in the environment far exceed natural sources. Some records estimate a 2,000-fold increase in lead deposition since pre-Roman times. In the past, emissions from leaded gasoline represented the primary source of lead air emissions. Now, because leaded fuel is still used by some off-road sectors, mobile sources continue to contribute to air emissions of lead, as well as industrial emissions, such as mining and smelting. In California, for the year 2000, emissions from industrial stationary sources were estimated at 31,000 pounds per year. In the same year estimates of another 1.6 million pounds per year of lead were attributed to area source emissions, primarily in the form of dusts.

Emissions of lead to soil and water can occur directly, as in the application of sewage sludge to land and the discharge of treated effluent. However, lead contamination in soil and water also occurs via deposition of lead from other sources, such as from the dispersal and deposition of air emissions, or the weathering and flaking of lead-based paint. Because elemental lead is insoluble and most lead compounds have low solubility, once lead is deposited it tends to accumulate unless mobilized by solutions with low pH or by physical transfer.

Exposure and Health Effects

Once lead is released into the environment, it becomes available for exposure to receptors. The many pathways of exposure are complex, but the most common pathways for human exposure are through ingestion of lead-contaminated food, water, soil, paint chips and dust, and through inhalation of lead-containing fumes and fine particles. The most common sources of exposure for children are dust and paint chips from lead-based paint. For adults, over 90% of cases of elevated blood lead levels can be attributed to occupational exposure, through inhalation of lead fumes and particles and ingestion of lead-contaminated dust and other lead-contaminated materials in the workplace. Other potential sources of exposure for the general population include lead-contaminated soils and dust at shooting ranges, molten lead and lead solder used in hobbies (such as stained glass making), lead glazes for pottery, art paints, glassblowing coloring agents, plastic food wrap pigments, certain cosmetics and folk remedies, and inadequately glazed lead in earthenware used for food storage or cooking.

The most common method of measuring lead exposure is the blood lead level (BLL), the amount of lead measured in whole blood. Because the half-life of lead in blood is about 36 days, BLLs usually reflect recent exposure. BLLs are also useful because the frequency and severity of symptoms increase as the BLLs increase. As an added benefit, BLL levels have been tracked for a number of years through the National Health and Nutrition Examination Survey (NHANES), allowing an evaluation of changes in BLLs through time. For example, such longitudinal studies revealed a dramatic decrease in BLLs after the phaseout of leaded gasoline. The most recent data show the U.S. average for blood lead is 2.9 micrograms per deciliter. For children 1-2 years old, the most recent data show the mean level is 3.1 micrograms per deciliter. The Centers for Disease Control has defined elevated child blood lead levels as 10 micrograms per deciliter, recommending various forms of education, follow-up testing, and intervention when child BBLs reach or exceed 10 micrograms of lead per deciliter of blood.

An extensive body of medical observation and scientific research developed over many years, much of it focused during the last thirty years, has revealed many toxic effects associated with lead exposure, although a threshold at which biochemical effects are known to occur has not yet been established. In addition to neurotoxic and neurodevelopmental effects, lead also can cause adverse effects on the hematopoietic, renal, cardiovascular, gastrointestinal, and reproductive systems. Lead has also been tentatively identified as a carcinogen. And, while more advanced stages of lead poisoning may result in severe outcomes such as damage to the brain and kidney, severe anemia, and spontaneous abortions, low level or early exposure to lead may produce nondescript symptoms such as fatigue, loss of appetite,

reduced attention span, insomnia, and constipation. It is not known whether all of the effects of lead exposure are reversible.

Lead is distributed primarily in the blood, soft tissue, and bones. The latter contain 95% of the total body burden of lead in adults and 73% in children. The half-life of lead in the bones can be more than 20 years, as compared to approximately 25 to 28 days in the blood. Hence, lead can be released into the bloodstream from the bones long after an initial exposure. Lead interferes with bodily processes by combining with sulfhydryl groups on proteins. It inhibits the synthesis of hemoglobin and disrupts mitochondrial function in the nervous system.

Children are particularly sensitive to lead exposure because their frequent hand-to-mouth behaviors and tendency to get dirty and ingest foreign substances increases their rate of intake of lead contaminated media. Furthermore, infants and young children have a higher rate of absorption of ingested lead into the gastrointestinal system, about 50% compared to 5%-15% for adults. Certain nutritional deficiencies, such as calcium and zinc deficiencies, which tend to be more prevalent in children, also enhance the absorption of lead. Finally, since children are growing more rapidly than adults, both physically and developmentally, they are more susceptible to the adverse biochemical effects of lead. Child exposure to lead is associated with decreased intelligence; reduced short-term memory; reading disabilities; and deficits in vocabulary, fine motor skills, reaction time, and hand-eye coordination; as well as effects on children's hematopoietic, renal, and gastrointestinal systems.

Protective Laws and Regulations

As the evidence regarding the serious and cumulative impacts associated with lead exposure has mounted over the last twenty years, state and federal requirements have been implemented to reduce exposure to lead. In some instances these requirements have been revised over the years to provide additional protection for human health and the environment, particularly to protect children from exposure to lead. During this time, complex models to characterize exposure and predict the risks associated with that exposure were also developed and refined. Two of these models are the Integrated Exposure Uptake Biokinetic model (IEUBK), used extensively by U.S. Environmental Protection Agency (U.S. EPA) to establish requirements for lead paint abatement, and LeadSpread, developed and used by DTSC for activities such as developing remediation goals.

One regulatory requirement that has not changed in twenty years, however, is the set of thresholds used to determine if a waste is hazardous according to the toxicity criteria for lead. The Total Threshold Limit Concentration (TTLC) and the Soluble Threshold Limit Concentration (STLC) are the thresholds used in California to determine if a waste is considered hazardous due to its lead content.

The TTLC for lead is intended to protect receptors from direct exposure, primarily through ingestion of lead, the type of exposure of greatest concern with sensitive receptors. The TTLC in use today assumes 1,000 mg/kg is protective of children likely to ingest soil containing lead. However, as shown by recently developed health-based screening values for soils at school sites and near residential lead-paint sites, soil-lead concentrations that protect children who are exposed to lead in soil range from 255 mg/kg to 400 ppm, depending upon the model and assumptions used.

Proposed Solutions

To reflect the considerable information that has become available regarding the health effects associated with even low exposures to lead, DTSC is proposing to update the TTLC threshold. The TTLC is the level that protects receptors from direct exposure to hazardous waste through ingestion, inhalation and dermal contact. In the case of hazardous waste containing lead, ingestion and inhalation are the routes of exposure of greatest concern, and ingestion by children is a pathway of particular concern since children

are particularly sensitive to the effects of lead exposure and more likely to ingest nonfood substances. Updating the TTLC to consider recent knowledge about child lead exposure will be more protective of such sensitive receptors, and ensure consistency with other updated requirements that consider the effects of child lead exposure.

DTSC has identified four different approaches for updating the TTLC to reflect more current findings about the impacts of lead. One option updates the original TTLC calculation, two are health-based approaches using the LeadSpread and IEUBK exposure models, and the fourth replaces the current test procedure with an extraction test that estimates the bioavailability factor for ingested lead. A fifth alternative considers no changes in the TTLC.

Recommendation and Future Activities

DTSC has found that the STLC for lead remains protective in light of the new information regarding the health impacts associated with exposure to lead, but the TTLC is obsolete and does not provide the protection intended for TTLCs. DTSC recommends updating the TTLC to reflect current information about the health effects of lead and to provide a more protective threshold that is consistent with soil-lead values developed using models that consider child exposure to lead.

Workshops to present this report and discuss the alternatives for adjusting the TTLC are planned. DTSC anticipates interest and participation in these workshops from its sister Departments, Boards and other state organizations, in addition to environmental and community organizations, industry, and the regulated community.

CHAPTER 1 – LEAD IN OUR SOCIETY

Lead is a gray, malleable, ductile metal whose usefulness has been valued since ancient times. A very dense metal, lead has a low melting point and is easily separated from its highly concentrated ores. These features, coupled with its ease of fabrication likely resulted in its earliest uses[1]. In addition lead is resistant to corrosion and a poor electrical conductor. It readily forms alloys with other metals[2] and can absorb a broad range of radiation[3]. This range of physical properties gives rise to the many uses of lead throughout history and today.

Historical Uses

Ubiquitous in our world today, lead traces its history to ancient times. The Latin word for lead, "plumbum," is reflected in the chemical symbol for lead, Pb. Similarly, the plumbing trade owes its name to plumbum because of the use of lead water pipes dating from the Roman Empire. Evidence of lead pollution from smelting operations goes back at least 5,000 years, attributable to the early use of lead for various industrial products, such as building materials, pigments for ceramic glazes, pipes, windows and decorative fixtures. The use of lead compounds for medicinal and cosmetic purposes may date back as far as 6,000 years to ancient Egyptian culture[4].

The early history of the U.S. saw lead used primarily for ammunition, brass and pewter, paints and protective coatings, glass and crystal, ceramic glazes, and water lines and pipes. In the twentieth century, the use of lead paralleled certain technological advances. Developments in applications of electricity and telecommunications resulted in the use of lead for machine bearings, cable covering, caulking and solder. Similarly, as motor vehicles became commonplace, the demand for lead increased due to its use in lead acid storage batteries and fuel additives.[5]

In the 1970s, amid growing concern over the health effects associated with lead exposure, environmental regulations restricted the use of lead in certain consumer products and activities in the U.S. Two of the most notable of these are lead-based paint and leaded gasoline.

Lead-Based Paint

Lead has long been used as a component of paint, primarily as a pigment and for its ability to inhibit and resist corrosion. In 1904 lead paint on porch railings and walls was first implicated as a source of lead poisoning among children in Australia, ultimately resulting in legislation to restrict the use of lead paint in Queensland in 1922[6]. As concern regarding the manufacture and application of lead paint continued to grow, other countries restricted its use. In 1922 the Third International Labor Conference of the League of Nations recommended banning interior uses of white lead (as lead paint was referred to at that time)[7]. By 1934 a number of countries, including France, Belgium, Austria, Tunisia, Greece, Czechoslovakia, Great Britain, Sweden, Poland, Spain, Yugoslavia and Cuba, had restricted the interior use of lead paint[8].

In the U.S., paint manufacturers set a voluntary standard in 1960, limiting the amount of lead in paint to five percent. In 1971 the Lead-Based Paint Poisoning Prevention Act initiated a national effort to reduce the hazards associated with exposure to lead-based paint. This program, administered by the federal Department of Housing and Urban Development (HUD), began by prohibiting the use of paint containing more than one percent lead in any residential building receiving public assistance. This program has evolved over time, through subsequent amendments, to define and regulate lead paint abatement activities, to require abatement in federally-owned housing, and to establish standards for taking abatement action. The Residential Lead-Based Paint Hazard Reduction Act of 1992 (Title X of the

Housing and Community Development Act of 1992) shifted the focus of the federal lead paint abatement activities to identification and control of lead paint hazards. Many states have related lead paint programs and are authorized to implement the federal requirements. In California, the Department of Health Services (DHS) Lead Poisoning Prevention Branch oversees many lead paint abatement requirements.

The 1973 amendments to the Lead-Based Paint Poisoning Prevention Act directed the federal Consumer Product Safety Commission (CPSC) to determine acceptable levels of lead in paint. The CPSC initially established a maximum allowable lead content in paint of 0.06 percent of the weight of the total nonvolatile content of the paint or the weight of the dried paint film (600 ppm). Eventually, paint in excess of this lead content was determined to be a hazardous product and was banned in 1978 for specified consumer products, including toys and other articles intended for children, and furniture. Similarly, such paints were banned from use in areas where consumers have or will have direct access to the painted surface, including residences, schools, hospitals, parks, playgrounds and public buildings.[9]

Leaded Gasoline

First added to gasoline in the 1920s, tetraethyl lead was used to increase the octane rating of gasoline, thereby reducing engine "knock." Other lead alkyl compounds, namely tetramethyl lead and dimethyldiethyl lead, are also effective antiknock additives. Engine knock occurs when gasoline vapor fails to combust evenly in the combustion chamber and detonates, resulting in a fluctuation in pressure in the cylinder. Lead alkyls added to the fuel decompose in the combustion chamber to form lead oxides, which prevent the chemical reactions that result in knocking. To avoid fouling the combustion chamber with lead deposits, other chemicals, such as ethylene dichloride and ethylene dibromide, are also added to the fuel to react with the lead oxide forming lead halides, which are emitted with the exhaust.[10] In the early development of leaded gasoline, approximately 0.8 to 1.0 g of lead was added to each liter of gasoline (3.0 – 3.8 g/gallon), a formulation still used in some developing countries[11]. Before the phase-out of leaded gasoline in the U.S., approximately 250,000 tons of organic lead was added each year to gasoline produced in this country[12].

In the 1970s a general concern over air emissions from mobile sources resulted in the development and use of emissions control devices, such as catalytic converters. However, the noble metal catalysts used, typically platinum, were disabled by the presence of lead in the exhaust, so unleaded fuels were developed to be used with these devices[13]. In 1973 U.S. EPA determined that leaded gasoline damaged emissions control equipment and required that unleaded gasoline be made available to consumers. In this regulation, U.S. EPA defined unleaded fuels as containing not more than 0.05 gram of lead per gallon of gasoline[14].

U.S. EPA also determined in the 1970s that the lead emissions resulting from leaded gasoline posed a significant health risk to urban populations, especially to children[15]. It has been estimated over the life of a vehicle, 75% of the lead consumed with leaded gasoline was emitted as particulate matter in the exhaust[16]. (The remaining 25% of the lead was deposited in the engine and exhaust system, with most of the engine deposits eventually transferred to the lubricating oil and removed from the system[17].) Small particles (<0.1 um) can remain airborne for up to 7 to 30 days and travel thousands of miles from the original source, whereas larger particles, which are formed by the agglomeration of smaller particles, spend less time airborne and don't travel as far[18]. The average size of the particulate emissions from leaded fuel (with 1.8 g lead per gallon) was estimated to be <0.25 um, creating a potential for widespread contamination resulting from the airborne deposition of particulate matter containing lead[19].

Concern regarding exposure to lead from exhaust prompted U.S. EPA to implement a gradual phaseout, beginning in 1973, to reduce the amount of lead added to gasoline. This program placed a limit of 0.5 g/gallon for the average amount of lead in all gasoline (including leaded and unleaded gas). In 1982, the limit was changed to 1.10 g Pb/gallon in leaded gasoline alone. In 1985 the lead phaseout called for a

limit of 0.5 g Pb/gallon by July of 1985 and 0.1 g Pb/gallon by January 1986[20]. The program also included a system for banking and trading lead credits among refineries, as well as extensive reporting requirements.

According to U.S. EPA, the phaseout was a success. In the 1970s the average amount of lead in leaded gasoline ranged from 1.8 g/gallon to over 2.0 g/gallon[21]. By 1988 the amount of lead used in gasoline was less than 1% of the amount of lead used in 1970, the peak year for lead usage[22]. In 1990 the Amendments to the Clean Air Act prohibited the sale of leaded gasoline for use in motor vehicles, effective December 31, 1995. In regulations promulgated in 1996 U.S. EPA clarified that unleaded gasoline may contain trace amounts of lead, but no gasoline may be sold as motor vehicle fuel that either contains any lead additives, or more than 0.05 g of lead per gallon of gasoline[23].

Other Restricted Uses

In the U.S. lead is no longer used to solder cans containing food; however, lead solder may still be used overseas and appear in the U.S. in imported canned products. Similarly, in 1997 imported plastic miniblinds were found to contain lead. Concern regarding the formation of lead-containing dust from photo-oxidation of the blinds, and subsequent exposure of children to the dust, resulted in a ban on lead-containing miniblinds issued by the CPSC. Lead has also been restricted in the components of water systems. In 1986 U.S. EPA restricted the use of lead solder or flux exceeding 0.2% lead, and the use of lead pipes, faucets and other plumbing fittings with more than 8% lead.

Current Lead Uses

Today lead continues to be considered an important commodity, used for an admittedly narrower variety of products than in the past. Although production of lead in the U.S. has declined slightly in recent history, for example, U.S. mine production declined by 9% from 1999 to 2000[24], consumption has remained relatively constant, even increasing over certain reporting periods[25]. In 1998 U.S. consumption of lead represented about 29% of worldwide consumption[26]. This demand for lead continues despite the decline in many lead-containing products because the uses of lead have shifted dramatically to a single predominant end use: the manufacture of lead acid batteries.

Batteries

In the U.S. a trend toward increased demand for lead acid batteries has helped to offset a reduced demand for lead in many other product sectors. As shown in Table 1, approximately 87% of the lead consumed in the U.S. in 2000 was attributed to storage batteries[27].

Lead acid batteries typically consist of a number of cells, each of which contains positive and negative electrodes, separators and a sulfuric acid electrolyte, all enclosed in a container. The electrodes are composed of plates formed from a lead alloy grid (often an alloy of lead and antimony) coated with a lead oxide-lead sulfate paste, which is the active material of the electrode[28]. Lead acid batteries are considered secondary cells because they are rechargeable. Both of the half-cell reactions that occur within the lead acid battery are reversible, so the battery is capable of both discharging and recharging from several hundred to thousands of times[29].

Lead acid batteries fall into two primary categories: starting, lighting and ignition (SLI) batteries and industrial batteries. SLI batteries, which account for 83% of the battery sector, are used in most on-road vehicles, such as passenger vehicles, light trucks, buses, commercial vehicles and motorcycles[30]. SLI batteries are also used in tractors, marine craft, aircraft and military vehicles[31]. Industrial batteries, which account for 17% of batteries, are divided into two types: motive power and stationary power. Motive power, or traction batteries, comprise 39% of the industrial battery total and are used for vehicles such as

industrial trucks and forklifts, airline ground equipment mining vehicles and railroad cars[32]. Stationary power batteries, 61% of the industrial battery total, are used for telecommunications, uninterruptable power supplies, and control and switchgear equipment[33].

The consumption of lead for batteries increased from about 650,000 metric tons in 1980 to over 1,400,000 metric tons in 1998, 88% of apparent U.S. lead consumption. The demand for lead-acid batteries is projected to continue to grow over the next few years with most of the growth projected for industrial batteries (5%-8%), attributable primarily to development of infrastructure for wireless telecommunications[34].

Table 1 – Lead Consumption in the U.S. in 2000

Uses	Lead consumed (metric tons)[a]	% of total
Storage batteries (grids, posts and oxides)	1,490,000	86.6%
Ammunition, shot and bullets	63,500	3.6%
Other oxides (paint, glass and ceramics, other pigments and chemicals)	52,400	3%
Casting metals (machinery, vehicles and equipment, and nuclear radiation shielding)	35,100	2%
Sheet lead (building construction, storage tanks, medical radiation shielding)	23,800	1.4%
Type metal, and other metal products (including foil, collapsible tubes, annealing, plating, galvanizing and fishing weights)	21,700	1.3%
Miscellaneous uses	14,000	0.8%
Solder	11,500	0.7%
Brass and bronze, billets and ingots	3,670	0.2%
Pipes, traps and other extruded products (building construction, storage tanks)	2,010	0.1%
Bearing metals (machinery, electrical and electronic equipment, motor vehicles), includes terne metal (sheet iron or steel plated with a lead-tin alloy)	1,480	0.09%
Caulking lead, building construction	1,140	0.07%
Cable covering, power and communication	---[b]	---
Total	**1,720,000**[c]	---

[a]Source: Smith 2000; Smith 2001
[b]Amount withheld to avoid disclosing proprietary data
[c]Does not add to total due to rounding

Other Current Uses

As shown in Table 1, other uses for lead consume a small fraction of the lead produced when compared to batteries. The second largest consumption of lead is attributed to ammunition, including lead shot and bullets. Although some regulations have been issued requiring the use of non-toxic shot in certain wildlife refuge areas for certain animals[35], most uses of lead for hunting and fishing are still allowed.

The third most significant use of lead in the U.S. is the demand for lead oxides for paint, pigment, and glass and ceramics. Lead is incorporated into the glass for video display equipment and other cathode ray tubes (CRTs) to provide shielding from radiation emitted from these devices. The lead content of

discarded CRTs exceeds the state's hazardous waste criteria and DTSC is actively promoting recycling of CRTs to avert these wastes from hazardous waste disposal. Lead for paint and glass pigments fell from 90,000 in 1978 to 50,000 metric tons in 1998[36].

In addition, although the use of lead in paint and pigments is restricted, the ban is not complete and lead continues to be used in these products, although the amount currently used is unclear. From 1972 to 1993, use of lead in paints and allied products decreased 80%. The amount of lead consumed over this period decreased from 55,000 tons to 9,000 tons[37]. Because the CPSC regulation restricting lead in paint contains a number of exclusions from the ban and its definitions, lead paint can still be used for industrial and commercial buildings and equipment, motor vehicles and boats, graphic arts coatings, artist paints, appliances and metal furniture (except children's furniture)[38].

For example, in motor vehicle paint, lead may be found in the pigments for white, orange, yellow and metallic coatings[39]. In other paints white lead (lead carbonate) was used to produce white pigments until it was largely replaced by titanium dioxide. However, some pigments still use lead compounds. A popular orange pigment is molybdate orange, containing lead chromate, lead sulfate and lead molybdate, and chrome yellow pigments, containing lead chromate and lead sulfate, are still in use[40]. Red lead (lead oxide) and litharge (yellow lead oxide) are also commonly used to inhibit corrosion in steel structures[41]. An estimated 89% of steel bridges carrying public roads (185,928 bridges of a total of 208,505) have a lead-based coating on them[42]. A study conducted in 1991 estimated 80% of steel bridges that were repainted between 1985 and 1989 were coated with lead-based paint, and up to 40% of steel in industrial facilities are coated with lead-based paint[43].

Other uses for lead include casting, bearings and sheet lead for machinery and equipment, motor vehicles, building construction, and a variety of forms of radiation shielding, including nuclear radiation shields and shields for medical testing and equipment and electronic equipment. Lead is also still used in solders, particularly for printed circuit boards in the electronics industry, and containers, including tanks and collapsible tubes. Many of these lead uses, however, are being replaced by plastic substitutes, for example in building construction, cable coverings and containers[44]. Lead for solder use decreased from 70,000 in 1978 to 15,000 metric tons in 1998[45]. Other metals and plastics are also used for packaging and protective coatings and tin has replaced lead solder in potable water systems in the U.S.[46]. New uses for lead are being developed that take advantage of lead's unique physical properties, including piezoelectric ceramics for transducers and sensors in ultrasound technologies, precision glass for medical and military uses, and high temperature superconductors.

Finally, some old uses of lead continue in overseas markets. For example, leaded gasoline continues to be used for motor vehicles in some, primarily developing countries. In addition although alkyl lead has not been manufactured in the U.S. since 1994, it continues to be imported for the production of leaded gasoline in this country. Because the ban on the sale of leaded gasoline applies only to fuel for motor vehicles, which are defined as self-propelled vehicles designed for transporting persons or property on a street or highway[47], leaded fuel may continue to be produced and sold for a variety of other non-road vehicles, including aircraft, car racing, marine engines and farm equipment[48]. Most leaded gasoline in the U.S. is used for general aviation aircraft (piston engines) and racing cars[49] although the amount used is difficult to determine because the U.S. Department of Energy (U.S. DOE) discontinued the tracking of leaded gasoline in 1993. Alkyl lead has been identified as a persistent bioaccumulative and toxic chemical by U.S. EPA, who has developed a draft National Action Plan for alkyl lead to do the following:

- characterize the use and exposure attributed to alkyl lead,
- encourage voluntary reduction of leaded gasoline in the aviation and racing industries, and
- assist with international efforts to reduce the use of leaded gas worldwide.

Lead Production

Lead in the U.S. is produced from both primary and secondary sources. Primary lead production in the U.S. in 2000 occurred principally from mines in Missouri and Alaska with additional mine production contributed by Idaho and Montana and other states[50]. In 2000 U.S. mine production of 457,000 metric tons of recoverable lead, ranked third, worldwide, behind Australia and China[51].

Most of the lead produced in the U.S., however, originates from secondary sources and lead is notable for having the highest recycling rate of any metal[52]. A survey conducted by the U.S. Geological Survey (USGS) revealed that secondary lead accounted for 77% of lead refinery production in 2000[53], amounting to a lead content of 1,130,000 metric tons[54]. Scrap lead acid batteries represent the major source of lead scrap for secondary lead production accounting for 90% of the lead produced from secondary sources[55]. The remaining 10% of lead scrap comes from old scrap, including castings, sheet lead, solder, and miscellaneous fabricated parts, and new scrap, including industrial scrap, drosses and residues[56].

From 1990 to 1995 an average of nearly 95% of lead-acid batteries were recycled annually[57]. Approximately 60% to 70% of spent lead acid batteries are collected for recycling by battery manufacturers, typically retrieving spent batteries from retailers when delivering new supplies. The remaining 30% to 40% of spent batteries are collected by scrap dealers who offer them for sale on the open market. Some batteries or their components are exported for recycling. SLI batteries have an average lifespan of about 4 years and represent about 88% of spent batteries; industrial motive batteries represent about 8% of spent batteries and last an average of 6 years, while stationary batteries have an average lifespan of about 10 years and represent about 4% of spent batteries[58].

Most of the components of a lead acid battery are recovered for reuse. The sulfuric acid is drained and treated to produce various products, including regenerated battery electolytes, fertilizers, laundry detergents, paper processing chemicals and pH control solutions for wastewater treatment. The shredded plastic casings are used to produce new battery casings. The recoverable lead components of the battery include lead alloy from the grids and posts, lead oxide from the electrode paste, which is desulfurized, and other lead compounds from the cell reactions. The lead components are separated from the other battery parts and processed in a variety of furnaces to recover a lead product that meets customer specifications[59].

The recycling of lead acid batteries is encouraged by a number of state and federal programs that prohibit the disposal of lead acid batteries to municipal waste facilities and require retailers and others to accept spent batteries[60]. In California spent lead acid batteries are considered hazardous waste, due to the lead and acid content of the batteries, and are subject to hazardous waste management requirements when they are disposed. When the spent batteries are recycled, however, the handling requirements for the waste are relaxed[61]. California law requires retailers to accept spent batteries when a new battery is purchased[62] and in some cases battery wholesalers and household hazardous waste collection facilities will accept spent lead acid batteries for recycling.

CHAPTER 2 – LEAD IN THE ENVIRONMENT

Although lead occurs naturally on earth, it is a relatively rare trace metal when compared with other more abundant metals, such as aluminum, iron, copper and zinc. However, because lead occurs in highly concentrated ores that are readily accessible, because it is easily separated from these ores and has been highly useful for many centuries, and because lead is quite persistent, its prevalence exceeds its natural abundance.

Natural Occurrence

Found throughout the world, lead ore deposits in North America are estimated to represent about one-third of the world's reserves of lead. The most important ore mineral for mining is galena (lead sulfide or PbS) with 87% lead. Anglesite ($PbSO_4$, lead sulfate) with 68% lead and cerrusite ($PbCO_3$, lead carbonate) with 77.5% lead are products of the weathering of galena and are also important lead ore minerals[63]. In the U.S. most lead mining occurs in Missouri and Alaska with additional mining activity in Colorado, Montana, Idaho, Washington and Nevada[64]. Overall, the average concentration of lead in the earth's crust has been estimated to be 12.5 ppm, being slightly more concentrated in granite and shale at 20 ppm and depleted in basalt at 5 ppm. Average concentrations in sandstone and carbonates are reported to be 7 and 9 ppm, respectively[65].

Median concentrations of lead in air reported by Kabata-Pendias (1984) ranged between 0.19 and 1.2 ng/m^3 at the South Pole. In air over West Germany, lead concentrations have been measured from 120 to 5,000 ng/m^3 and 45 to 13,000 ng/m^3 in air over North America[66]. Median concentrations of lead in soil in the U.S. at a depth of 20 cm were measured at 15-16 ug/g[67].

Because lead has been in use since ancient times, the concentrations of lead in the surficial media of the earth do not represent natural sources of lead alone. Chronological records of lead deposition in polar ice strata, sediments, and tree rings show an up to 20-fold increase in lead deposition occurring in the last 150 years, reflecting the increase in lead use during the industrial revolution, and up to a 2,000 fold increase since pre-Roman times[68]. For example, examination of polar ice strata indicate the prehistoric concentrations of lead in Greenland and Antarctica were 1.4 and 1.2 pg/g, respectively, while present concentrations are 200 and 5-6 pg/g[69]. Similarly, the natural atmospheric concentration of lead has been estimated to be 0.000076 ug/ m^3 [70].

Natural sources of lead in the environment include weathering and erosion of lead-containing minerals, resulting in lead being incorporated into soils, leached into water, taken up by plants and windblown into the atmosphere. Other natural sources of atmospheric lead include volcanic eruptions, seaspray, emissions by plants, and forest fires[71]. Estimates of the contribution of natural sources to lead in environmental media vary, but indicate a small amount of lead in the environment originates from natural sources, particularly when compared to anthropogenic sources[72].

Anthropogenic Sources

Man-made lead emissions arise from the various uses for lead through time. In these emissions, lead is often released to air in the form of particles of varying sizes, which, depending upon the particle size and atmospheric conditions, are eventually deposited to land and bodies of water. In other instances, soils and

water may become contaminated with lead as the result of mining, pesticide use and past industrial or disposal activities.

Air Emissions

A variety of sources and activities contribute to air emissions of lead, including mobile, area and stationary sources. In the recent past the major source of lead-containing air emissions was the consumption of leaded gasoline in motor vehicles. In 1984 over 89% of an estimated total of 39,000 tons of lead emitted in that year was attributed to gasoline consumption[73]. In the years following the reduction of the use of alkyl lead in gasoline, ambient air concentrations of lead declined, as shown in Figure 2-1. During that time the contribution of mobile sources to lead air emissions also declined, as shown in Table 2-1.

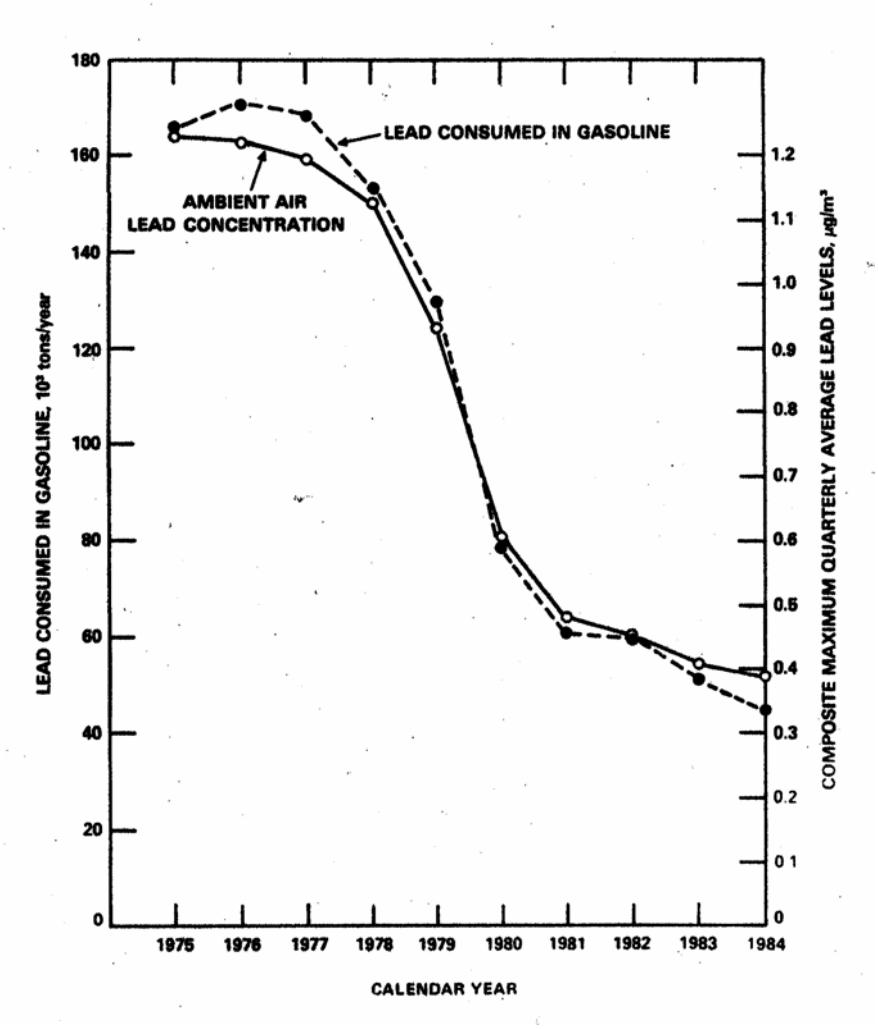

Figure 2-1 – Lead consumed in gasoline and ambient lead concentrations, 1975-1984
Source: U.S. EPA, 1986, p. 5-18

Table 2-1 – National Lead Emission Estimates (tons), 1979-1989

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

*The sums of subcategories may not equal total because of rounding.
Source: U.S. DHHS, 1999, p. 383.

As described in Chapter 1, leaded gasoline is still consumed by a variety of mobile sources, including general aviation aircraft, cars used for racing and other off-road vehicles and equipment. It is difficult to determine, however, the amount of leaded fuel still used. After September 1993, sales of leaded motor fuel in the U.S. were no longer reported in the Petroleum Marketing Annual reports produced by the U.S. Department of Energy[74]. Aviation fuel sales are reported, but not all aviation fuel contains alkyl lead.

Some information describing lead emissions attributed to non-road mobile sources are available to characterize these sources. Table 2-1 shows that during the phaseout of leaded gasoline, in 1989 around 31% of total lead emissions were attributed to transportation sources. More recent emissions attributable to these sources can be estimated from the draft National-Scale Air Toxics Assessment Data, collected for 1996, the year the phaseout was completed and leaded gasoline was prohibited for on-road vehicles. This assessment compiles emissions estimates from state and local regulatory agencies for specified air toxic priority pollutants, including lead compounds, from outdoor sources. The data includes emissions attributed to non-road mobile sources, which includes mobile sources not found on roads or highways, namely, aircraft, trains, and construction and agricultural machinery and equipment. Although this preliminary data is still in a draft form undergoing scientific review and is thought to underestimate the actual volume of lead emissions, it can be used to provide an indication of the relative percentages of lead compound emissions attributed to various sources on an aggregated basis[75].

National totals for the states that have provided estimates for the National-Scale Air Toxics Assessment are summarized in Table 2-2. These estimates show that for the U.S. around 22% of total lead emissions are attributed to non-road mobile sources, according to the models used in this assessment. It is likely that the lead emissions from these non-road mobile sources arise from the continued consumption of leaded gasoline.

Table 2-2 – 1996 Emissions of Lead Compounds
(National-Scale Air Toxics Assessment Draft Data for Scientific Peer Review)

	Major Emissions		Area and Other Emissions		Onroad Mobile Emissions		Nonroad Mobile Emissions		Total Emissions
	tons/yr	% of total	tons/yr	% of total	tons/yr	% of total	tons/yr	% of total	tons/yr
Sum of all Statewide Totals	1,050	46%	695	31%	18.6	<1%	505	22%	2,270

Source: Internet web site, accessed 12/31/01: http://www.epa.gov/ttn/atw/nata/tablemis.html

The 1996 California Toxics Inventory results are depicted in Table 2-3. This evaluation reported categories slightly different from the National-Scale Air Toxics Assessment, but still indicate a sizable percentage, 33%, of lead emissions are attributable to other mobile sources. Other mobile sources in the 1996 California Toxics Inventory refer to off-road equipment, such as commercial and industrial equipment, and lawn and garden equipment, farm equipment, off-road recreational vehicles, recreational boats, ships, trains and aircraft.

Table 2-3 – 1996 California Toxics Inventory (revised 8/28/00) - Lead

	Stationary Sources		Area Sources		Mobile Sources		Other Mobile Sources		Natural Sources		Total
	tons/yr	% of total	tons/yr	% of total	tons/yr	% of total	tons/yr	% of total	tons/yr	% of total	tons/yr
Sum of California Counties	23.5	51%	2.95	6%	-	-	15.3	33%	4.5	10%	46.2

Source: Internet web site, accessed 12/27/01: http://www.arb.ca.gov/toxics/cti/cti.htm

Some sources have reported that leaded aviation gasoline accounts for a large majority of the lead emissions currently attributable to fuel consumption[76]. This assertion is confirmed by an analysis that projects emissions and concentrations of mobile source air toxics from 1996 to 2007[77]. Building upon the work initiated by the National-Scale Air Toxics Assessment, this study expands the analysis to project future emissions of hazardous air pollutants from mobile sources. The mobile sources considered include both highway vehicles and non-road vehicles, such as lawn mowers and other small engines, non-road equipment, airports, marine vessels and railroads[78]. As Table 2-4 shows, the aggregate emission projection results for non-road mobile sources greatly exceed those for on-road sources, by over 95%. Table 2-5 shows that 99.8% of the lead emissions generated by non-road sources are contributed by airports due to the use of leaded fuel for general aviation aircraft[79].

Table 2-4 – Summary of Mobile Source Emission Projection Results for theContiguous 48 States and the District of Columbia

Hazardous Air Pollutant	Estimated 1996 emissions (tons/year)			Projected 2007 emissions (tons/year)		
	On-road	Non-road	All Mobile	On-road	Non-road	All Mobile
Lead	18.9	527.2	**546.1**	22.0	585.2	**607.2**

Source: U.S. EPA, 2001, p. 22.

Table 2-5 – Lead Emission Projections for Different Non-road Engine Categories (tons/year)*

	2-stroke gasoline	4-stroke gasoline	Non-road diesel	Marine diesel	Railroad	Airports	Total non-road
Estimated 1996 lead emissions	0.0	0.0	0.0	1.0	0.0	526.1	**527.2**
Projected 2007 lead emissions	0.0	0.0	0.0	1.2	0.0	584.0	**585.2**

*(Contiguous 48 States and the District of Columbia)
Source: U.S. EPA, 2001, p. 27.

In addition to mobile sources, a variety of industries and processes also produce lead air emissions. Some of these emissions, particularly those generated by heat-producing process steps, are controlled by air pollution control technology. Fugitive emissions, especially those generated by material handling, are more difficult to control. These stationary sources of lead emissions include lead and other metal production and smelting, including mining and metal refining; coal, oil and waste combustion and incineration; and various manufacturing processes, including battery production and recycling, and lead-containing glass manufacture. To estimate lead air emissions, U.S. EPA compiled lead emission factors for all industries and processes expected to contribute to lead air emissions[80]. The industries and processes included in this evaluation are listed below:

- Primary and secondary lead smelting
- Pressed and blown glass
- Primary and secondary copper production
- Lead-acid battery production
- Primary zinc smelting
- Lead oxides in pigments
- Secondary aluminum operations
- Lead cable coating
- Miscellaneous lead products (ammunition, type metal, other metallic lead products, abrasive grain)
- Stationary internal combustion
- Stabilizers in resins
- Incineration (municipal waste; industrial and commercial waste; sewage sludge; medical waste; hazardous waste)

- Iron and steel foundries
- Frit manufacturing
- Ore mining, crushing and grinding
- Ceramics and glazes
- Brass and bronze processing
- Solder manufacturing
- Stationary external combustion (residential heating; utility, industrial and commercial fuel combustion)
- Electroplating (including printed circuit boards)
- Drum and barrel reclamation
- Asphalt concrete
- Scrap tire incineration and open burning
- Application of paints
- Crematories
- Shooting ranges and explosive ordnance sites
- Pulp and paper industry
- Rubber products
- Portland cement manufacturing

The emission factors developed for these industries and processes are used to estimate the amount of lead air emissions generated, often for reporting purposes. For example, the Toxic Chemical Release Inventory (TRI), established under Section 313 of the Emergency Planning and Community Right-to-Know Act of 1986, is a national database that identifies facilities, chemicals manufactured and used at these facilities, the amounts of these chemicals released in various waste streams, and other waste management activities from manufacturing and federal facilities[81]. The TRI was expanded under the Pollution Prevention Act of 1990 to further require the reporting of chemicals recycled, combusted for energy recovery, and treated on- and off-site. Additional industries were added in 1997 by U.S. EPA rulemaking, including mining. Facilities that have ten or more employees and that operate in certain industry sectors are required to report to the TRI database environmental releases and waste management activities for specified chemicals that exceed threshold quantities. Table 2-6 lists the Standard Industrial Classification (SIC) Codes for common industries expected to submit TRI reports for lead and lead compounds[82].

Table 2-6 – Industry and Process Sources of Lead and Lead Compounds

SIC Code	Industry Description
10	Metal mining (except 1011 – Iron ores; 1081 – Metal mining services; and 1094 – Uranium, radium, vanadium ores)
12	Coal mining (except 1241 – Coal mining services)
20	Food and kindred products
21	Tobacco products
22	Textile mill products
23	Apparel and other finished products made from fabrics and similar materials
24	Lumber and wood products, except furniture
25	Furniture and fixtures
26	Paper and allied products
27	Printing, publishing and allied industries
28	Chemicals and allied products
29	Petroleum refining and related industries
30	Rubber and miscellaneous plastics products
31	Leather and leather products
32	Stone, clay, glass, and concrete products

33	Primary metal industries
34	Fabricated metal products, except machinery and transportation equipment
35	Industrial and commercial machinery and computer equipment
36	Electronic and other electrical equipment and components, except computer equipment
37	Transportation equipment
38	Measuring, analyzing, and controlling instruments; photographic, medical and optical goods; watches and clocks
39	Miscellaneous manufacturing industries
4911	Electric services (limited to facilities that combust coal and/or oil for the purpose of generating power for distribution)
4931	Electric and other services combined (limited to facilities that combust coal and/or oil for the purpose of generating power for distribution)
4939	Combination utilities, not elsewhere classified (limited to facilities that combust coal and/or oil for the purpose of generating power for distribution)
4953	Refuse systems (limited to facilities regulated under RCRA)
5169	Chemicals and allied products, not elsewhere classified
5171	Petroleum bulk stations and terminals
7389	Business services, not elsewhere classified (limited to facilities primarily engaged in solvent recovery services on a contract or fee basis)
--	Federal facilities

Source: 66 FR 4500

Facilities must report the amounts of the listed chemicals released on-site to air, water, and land, and injected underground, and the amounts of chemicals that are recycled, combusted for energy recovery, treated, or disposed, both on- and off-site. The reported data represents the number of pounds of a listed chemical released or managed on-site and off-site in a calendar year. However, because only facilities with ten or more full-time employees in certain industries sectors using specified chemicals above reporting thresholds are required to report, the data is likely to underestimate true emissions.

TRI reports for industry releases are available for lead and lead compounds and can be sorted by a number of variables, including industry, geographical area and year. Lists of facilities reporting specified releases are also available and included in Appendix 2-A for lead and lead compounds in California for 1999. Table 2-7 contains the TRI industry release air emissions data for California for lead and lead compounds in the years 1995 through 1999. As seen in this table, the trend over time has been one of steady or declining emissions with the exception of 1999.

Table 2-7 – TRI Total Air Emissions (in pounds) for California – Lead and Lead Compounds reported in 1999-1995

SIC code	Industry	1999		1998		1997		1996		1995	
		Lead	Lead cmpd	Lead	Lead cmpd	Lead	Lead cmpd	Lead	Lead cmpd	Lead	Lead cmpd
28	Chemicals	-	309	-	310	-	315	-	86	-	122
29	Petroleum	1	21	1	51	-	467	3	413	4	280
30	Plastics	10	476	10	1,439	10	507	10	772	10	28
32	Stone/Clay/Glass	-		-	-	-	-	5	-	5	-
33	Primary metals	1,023	1,492	970	4,898	921	4,942	583	4,851	918	5,660
34	Fabricated metals	14	-	12	-	14	-	15	-	20	-
35	Machinery	56	-	56	-	29	-	499	-	499	-
36	Electrical equip	16	3,523	0	2,778	-	3,026	54	3,421	274	3,675
37	Transportation equipment	39		64	-	75	-	83	-	101	-
38	Measure/Photo	-	10	-	10	-	286	-	500	-	500
20-39	Multiple codes	14	5	14	84	16	1,224	260	1,724	542	126
-	No reported codes	29,205	-	-	-	1	-	1	-	-	-
10	Metal mining*	-	323	0	1,599	-	-	-	-	-	-
4953/ 7389	RCRA solvent recovery*	832	255	10	510	-	-	-	-	-	-
	Total	**31,210**	**6,414**	**1,137**	**11,679**	**1,066**	**10,767**	**1,513**	**11,767**	**2,373**	**10,391**

*New industries added in 1998 reporting year.
Source: Internet web site, accessed 12/14/01: http://www.epa.gov/triexplorer/explorer.htm

Recently, U.S. EPA determined lead and lead compounds are persistent and bioaccumulative chemicals resulting in the need for enhanced information about releases and waste management activities associated with these chemicals. In January 2001 U.S. EPA lowered the TRI reporting threshold for lead and lead compounds from 25,000 pounds of lead or lead compounds manufactured or processed, or 10,000 pounds of lead or lead compounds otherwise used, to 100 pounds[83]. As a result, specified industries that manufacture, process or otherwise use 100 pounds or more of lead or lead compounds must report releases and waste management activities beginning in calendar year 2001. A number of new facilities were expected to begin reporting this information by July 1, 2002. New facilities reporting due to the reduced threshold for lead and lead compounds are not reflected in the TRI data contained in this report.

An additional, and major, source of information regarding stationary source air emissions of lead in California comes from the state's Air Resources Board (ARB). In California, ARB compiles the state's emissions inventory and performs air quality and emissions inventory special studies. The state's lead emissions inventory includes data for stationary sources, which include both point and area-wide sources. Stationary point source emissions are based on estimates made by facility operators and local air pollution control districts. Point sources refer to a single facility. Area-wide sources include source categories associated with human activity and emissions that take place over a wide geographic area. ARB staff is responsible for estimating emissions from area source categories.

The ARB's emission inventory for 2000 estimates that there were approximately 31,145 pounds per year of lead emitted from stationary point sources in California. These lead emissions range statewide from 9,408 pounds per year in Los Angeles County to negligible lead emissions in Merced, Napa, Solano, and

Trinity Counties. Sources include industrial inorganic chemicals, secondary nonferrous metals, national security, storage batteries, electronic components, signs and advertising displays, steam supply, electric services, gold ores, metal ores, blast furnaces and steel mills, glass containers, fabricated structural metal, sawmills and planing mills, minerals, and wood products. Lead emissions of 500 pounds per year, or more, from stationary point sources only occur in nine counties: Los Angeles, Sonoma, San Bernardino, Alameda, Orange, San Joaquin, Tuolumne, Inyo and Mendocino.

The ARB's 2000 emission inventory estimates that there were approximately 1,682,872 pounds per year (lbs/yr) of lead emitted from area sources in California. Lead emissions statewide range from 175,504 lbs/yr in Los Angeles County to 1,861 lbs/yr in Alpine County. The major sources of lead emissions from area sources are listed below:

- Unpaved road travel dust 690,448 lbs/yr
- Paved road travel dust 564,865 lbs/yr
- Fugitive dust from unpaved roads 119,482 lbs/yr
- Construction and demolition building construction dust 71,094 lbs/yr
- Construction and demolition road construction dust 65,231 lbs/yr
- Military aircraft jet fuel 30,974 lbs/yr
- Fugitive agricultural dust 13,230 lbs/yr

Table 2-8 summarizes the total stationary point source data and area source data by county for 2000.

Table 2-8 - ARB's 2000 Emission Inventory for Lead (pounds/yr)

County	Stationary Point Source Emissions	Area Source Emissions	Total	County	Stationary Point Source Emissions	Area Source Emissions	Total
Los Angeles	9,408	175,504	**184,912**	Placer	92	17,290	**17,382**
Imperial	365	148,018	**148,383**	Trinity	-	17,295	**17,295**
San Bernardino	3,109	114,347	**117,456**	El Dorado	75	16,184	**16,259**
San Diego	243	110,492	**110,735**	Humboldt	178	15,946	**16,124**
Riverside	331	95,427	**95,758**	Sonoma	4,098	11,597	**15,695**
Fresno	723	79,688	**80,411**	Kings	41	15,216	**15,257**
Kern	634	68,881	**69,515**	San Mateo	1	15,118	**15,119**
Orange	1,969	58,714	**60,683**	Plumas	69	14,645	**14,714**
Santa Clara	19	42,772	**42,791**	Madera	419	14,248	**14,667**
Sacramento	78	40,013	**40,091**	Santa Barbara	80	13,732	**13,812**
Tulare	24	37,217	**37,241**	Nevada	-	13,357	**13,357**
San Joaquin	1,932	30,766	**32,698**	Tehama	4	11,801	**11,805**
Inyo	952	29,567	**30,519**	Sierra	38	11,443	**11,481**
Alameda	3,000	26,466	**29,466**	Colusa	45	11,144	**11,189**
Mono	67	27,042	**27,109**	Santa Cruz	8	11,144	**11,152**
Merced	0	26,321	**26,321**	Lake	523	9,792	**10,315**
Stanislaus	34	23,893	**23,927**	San Benito	67	8,930	**8,997**
Solano	0	22,757	**22,757**	Sutter	1	8,804	**8,805**
Lassen	117	22,455	**22,572**	Calaveras	-	8,351	**8,351**
San Luis Obispo	3	22,328	**22,331**	Glenn	8	8,256	**8,264**
Shasta	274	21,635	**21,909**	Tuolumne	1,078	6,939	**8,017**
Ventura	206	21,218	**21,424**	San Francisco	1	7,992	**7,993**
Monterey	4	20,814	**20,818**	Mariposa	-	7,895	**7,895**
Yolo	5	19,636	**19,641**	Marin	1	6,489	**6,490**
Mendocino	546	18,336	**18,882**	Yuba	41	6,052	**6,093**
Siskiyou	-	18,658	**18,658**	Del Norte	6	5,438	**5,444**
Contra Costa	209	18,448	**18,657**	Amador	-	5,152	**5,152**
Modoc	-	17,788	**17,788**	Napa	0	3,935	**3,935**
Butte	19	17,625	**17,644**	Alpine	-	1,861	**1,861**
				Totals	**31,145**	**1,682,872**	**1,714,017**

Source: ARB Emission Inventory Data, 2000.

Appendix 2-B contains additional details of ARB's emission inventory data for 2000, including definitions of the source categories. Table 1 in Appendix 2-B lists the stationary source data by county, including the industry source categories. Table 2 in Appendix 2-B shows Area Source Data by county, providing the total lead emissions in pounds per year by county, the distribution of those emissions by air basin(s) within the county, and the source categories.

Other Emissions – Soil and Water

Other emissions of lead to environmental media include emissions directly to land, including soil and water. The sources of lead emissions to soil vary, ranging from industrial activity and processes to deposition from air emissions. A recent literature review found the three most commonly identified sources of elevated soil lead concentrations are lead-based paint on exterior surfaces, such as buildings; point source emitters, such as smelters, batteries and mine tailings; and leaded gasoline emissions from automobiles[84].

Lead from paint can contaminate soil as exterior coats of paint weather and "chalk," if the paint is allowed to deteriorate and peel or flake, or if painted surfaces are sanded, scraped or otherwise abraded. Soil lead concentrations of 1,000 ppm within 3 meters of houses coated with lead paint are typical[85]. The pattern of soil contamination near a residence shows in general a higher concentration of lead near the foundation of the structure than at more remote locations. The concentration levels of lead in soil near

residences vary widely, however. The National Survey of Lead-Based Paint in Housing, conducted by HUD, was followed by a U.S. EPA study of soil lead concentrations, whose data are shown in Table 2-9. This study found wide variation in soil lead concentrations and, because soil lead data near public housing units was limited, draws few conclusions about the soil conditions near public housing. The conclusions of the study did establish the age of the unit as the strongest predictor of soil lead concentration. The other major predictor of soil lead concentration is the location of the housing unit, with units in the northwestern regions of the country showing the highest concentrations and units in the western and southern regions having the lowest concentrations[86].

Table 2-9 – Lead Measurements in Housing Soil Samples

Housing Type	Entrance Samples		Dripline Samples		Remote Samples	
	Range of concentrations (ppm)	Mean*	Range of concentrations (ppm)	Mean*	Range of concentrations (ppm)	Mean*
Private Housing Units	3 – 6,800	85	1 – 22,900	74	1-6,900	46
Public Housing Units	8 – 520	55	10 – 870	55	5 – 600	44

*Weighted sample geometric mean.
Source: U.S. EPA, May 1996, pp. 10-11.

As described in Chapter 1, lead-based paint is still used on some steel structures, such as bridges, often for corrosion resistance. Maintenance for bridges requires removal of old paint, the method most commonly used was open abrasive blasting, which resulted in emissions and/or occupational exposure. Open blasting, however, is no longer used for bridges; the residue from blasting is currently contained and properly disposed[87]. In addition to bridges, an estimated 12,000 water storage tanks and aboveground petroleum and natural gas tanks coated with lead-based paint require repainting each year[88].

Common point sources of lead emissions include smelters, incinerators, mining operations, and lead-acid battery facilities and waste disposal sites[89]. For example, the mining and smelting of lead has been a source of soil contamination for thousands of years. Levels up to 30,000 ppm have been found in soils within 100 meters of a smelter in England[90]. In the U.S., up to 2,200 ppm lead was found in soils near a smelter in Missouri[91]. Much of this contamination is attributed to aerial deposition although high concentrations of lead were also found in ore handling areas.

Deposition of lead particles from air emissions to soil and water is a common source of lead contamination. Depending upon the size of the particles and atmospheric conditions, airborne lead can travel a considerable distance from the point of emission and be transferred from air to land, either as dry particles or incorporated into precipitation. Evidence for long range deposition of lead has been reported in many studies although the sources of the lead are unknown. At the most remote global sites, reported deposition rates were 0.04 mg/(m^2 year) at the South Pole, 0.72 mg/(m^2 year) in northwestern Canada, and 0.63 mg/(m^2 year) in northern Michigan[92]. Deposition rates from 3.1 to 31 mg/(m^2 year) have been reported in remote rural locations, while deposition rates in suburban and industrial locations ranged between 27 and 140 mg/(m^2 year)[93]. In Yosemite National Park, Elias et al. (1980) reported between 0.34 and 1.01 mg/(m^2 year)[94].

As an example of deposition resulting from a known source of lead emissions, elevated lead concentrations near highways are largely attributable to fallout of particulate matter containing lead from the exhaust of vehicles using leaded gasoline. An estimated 40% of the lead emitted as part of exhaust is large enough to be deposited near the roadway[95]. In the 1960s, and later, evidence began appearing on soils and vegetation near highways containing from 50 to 2,000 ppm lead in surface soil within 25 meters of the curb, decreasing with distance from the roadway. Isotope studies were used to confirm that this lead originated from gasoline-burning vehicles[96].

Recent research has implicated lead wheel weights as a source of lead on and near roadways. Lead wheel weights are commonly used to balance motor vehicle wheels. These weights can be dislodged from the vehicle wheels and deposited in streets and along curbs, often in locations where rapid changes in vehicle speed or direction occur. Once the wheel weights are deposited in streets, they can be abraded and may contribute significant amounts of lead to streets and adjoining soil. Lead deposition on streets in Albuquerque attributed to wheel weights was estimated to be 3,730 kg/year[97].

Agricultural activities also can contribute to lead in soil and water. Lead arsenate was used as an insecticide in orchards in past years, leaving elevated lead concentrations in orchard soils. Similarly, applications of sewage sludge to land may result in lead contamination of soils because sewage sludge often contains metal contaminants, including lead, which typically occurs at concentrations of less than 1,000 mg/kg[98].

As with soil, the sources of emissions of lead to water include direct industrial or process wastewater emissions and the wet and dry deposition of airborne emissions.

Wastewater, derived from municipal and industrial sources, is typically treated although toxic elements may remain in effluent waters at elevated concentrations. Most trace metals, including lead, however, are adsorbed by suspended solids, which are subsequently filtered or removed from the wastewater by flocculation. The resulting sewage sludge may have elevated concentrations of metals, as described above. Concentrations of lead in the primary effluent is reported to range from <0.2 to 6.0 ppm with a median < 0.2 ppm; and in secondary effluent to range from 0.003 to 0.35 ppm with a median of 0.008 ppm[99].

Urban runoff and runoff from contaminated soils or lead containing wastes represents another source of emissions to water[100]. In addition water supplies may contain lead that has leached from pipes, solder or plumbing fixtures.

As described for air emissions, the TRI database also includes on-site releases to water, land and injected underground. Again, because only certain industries above specified thresholds are required to report, this data is not comprehensive. Table 2-10 shows the amount of lead and lead compounds released onsite and offsite to water, land and Publicly-Owned Treatment Works (POTWs) in California in 1999.

Table 2-10 – TRI On-site and Off-site Reported Releases for Lead and Lead Compounds (in pounds), California, 1999

SIC code	Industry	Surface Water Discharges		Releases to Land		Total Off-Site Releases*	
		Lead	Lead cmpds	Lead	Lead cmpds	Lead	Lead cmpds
28	Chemicals	-	6	-	-	-	712
29	Petroleum	-	76	-	-	7,539	1,250
30	Plastics	0	2	-	-	2,164	1,994
33	Primary metals	3	2	512	0	48,222	914,147
34	Fabricated metals	0	-	-	-	10	-
36	Electrical equipment	-	611	-	0	2,083	27,070
37	Transportation equip.	4	-	-	-	171	-
20-39	Multiple codes	5	36	5	-	500	17,931
-	No reported codes	-	-	-	-	8,666	3
10	Metal mining**	-	-	-	222,983	-	-
4953/ 7389	RCRA/Solvent Recovery**	0	-	691,449	1,714,657	310,158	1,005
	Total	**12**	**733**	**691,966**	**1,937,640**	**379,513**	**964,112**

*Total off-site releases include transfers off-site to disposal, metals and metal compounds transferred off-site for solidification/stabilization and for wastewater treatment, including discharge to POTWs.
**New industries added in 1998 reporting year.
Source: Internet web site, accessed 12/14/01: http://www.epa.gov/triexplorer/explorer.htm

Some of the releases characterized by the TRI data represent emissions of lead directly into environmental media with no further treatment or containment, such as the releases to surface water in which the data indicate the amount of lead in pounds released to streams or other surface water bodies. The other releases listed, however, do not necessarily represent uncontrolled releases to environmental media. On-site and off-site releases to land include releases to landfills, as well as landfarming and other disposal techniques. Similarly, discharges to POTWs typically must meet specified limits established by the POTW and may undergo further treatment.

Another method of characterizing releases into environmental media is to evaluate sites contaminated with lead that are undergoing cleanup in California. DTSC maintains an automated database, called "CalSites," containing information on properties in California where hazardous substances have been released, or where the potential for a release exists. One category within the database, the Annual Workplan category, includes confirmed release sites at which DTSC is actively working to remediate the contamination, either in a lead role or support capacity. These confirmed sites are generally high priority, have high potential risk, and include military facilities, state "funded" or Responsible Party lead, and National Priority List sites.

As of September 13, 2001, the CalSites database contained 463 sites for all categories with a potential or confirmed detection of lead contamination. Table 2-11 shows the distribution of these 463 sites by county. Sixty-one of these sites are in the Annual Workplan category as shown in Table 2-12.

Table 2-11 - Sites Where Lead Is Potentially Present or Confirmed Present, by County

County	# of sites	County	# of sites
Los Angeles	84	El Dorado	5
Alameda	70	San Diego	5
Contra Costa	38	Stanislaus	5
Santa Clara	31	Butte	3
San Francisco	30	Riverside	3
Sacramento	20	Santa Barbara	3
San Mateo	20	Amador	2
Solano	11	Humboldt	2
Kern	15	Imperial	2
Ventura	12	Kings	2
Nevada	11	Merced	2
Orange	9	Shasta	2
San Joaquin	9	Siskiyou	2
Placer	8	Calaveras	1
San Bernardino	8	Lassen	1
Santa Cruz	8	Mendocino	1
Fresno	7	San Benito	1
Monterey	7	Tehama	1
Napa	7	Tulare	1
Marin	6	Tuolumne	1
Sonoma	6	Yolo	1
		Total	**463**

Source: CalSites Database, for All Categories as of 9/13/01

Table 2-12 – Active Sites Where Lead Is Confirmed Present, by County

County	# of sites	County	# of sites
Los Angeles	12	Marin	1
Alameda	10	Monterey	1
Contra Costa	8	Nevada	1
Sacramento	8	Orange	1
Kern	4	San Diego	1
San Bernardino	3	Santa Barbara	1
San Francisco	2	Santa Clara	1
Solano	2	Siskiyou	1
Sonoma	2	Stanislaus	1
Lassen	1		
		Total	**61**

Source: CalSites Database, for Annual Workplan Category as of 9/13/01

The 61 active sites in Table 2-12 are represented by the following categories:

• Miscellaneous	23 sites
• Military Facilities	15 sites
• Plating Shops	7 sites
• Railroad Yards	6 sites
• Marine/Port Facilities	3 sites
• Drum Reconditioners	2 sites
• Landfills/Dumps	2 sites
• Residential/Schools	2 sites
• Utilities	1 site

A random sample of 13 sites (or 21%) selected from these categories give an indication of the types of facilities where lead is a contaminant. The 13 facilities and sampling results are summarized in Table 2-13. More detailed information gathered from the sites' project managers are listed in Appendix 2-C.

Table 2-13 – Lead Sampling Results from Selected Sites

County	Facility	Sample source*	Sample results
Los Angeles	Former tire manufacturing facility	Soil	85 - 97 mg/kg (one at 3,720 mg/kg)
Los Angeles	Former lead smelter & dye casting	Soil	8 - 76,000 ppm
Los Angeles	Former oil refinery	Soil	1,000 - 4,400 ppm at surface 5-10 ppm at 5 ft. depth
Los Angeles	Military facility	Soil GW	0.35 - 2, 220 mg/kg 0.07 - 105.4 ug/L
Los Angeles	Elementary & intermediate school site	Soil	5.6 - 189 mg/kg
Alameda	Former electroplating facility	Soil SW GW	1.2 - 130 mg/kg 0 - 0.055 mg/kg 0 - 0.4 mg/kg
Alameda	Former military facility	Soil	680 - 81,800 mg/kg
Contra Costa	Currently vacant, former railcar refurbishing site	Soil	3 - 9,800 mg/kg at surface 23,800 ppm at 2 ft. depth 1,430 mg/kg at 10 ft. depth
Contra Costa	Apartments, former railcar maintenance site	Soil	68 - 4,020 mg/kg at surface 55-16,780 mg/kg at 1.5 ft depth
Sacramento	Former manufactured gas plant	Soil	3,000 ppm
Sacramento	Former rocket assembly & testing site	Soil	2.3 - 14 ppm
Kern	Military facility	Soil GW SW	0.075 - 1,510 mg/kg 0.001 - 20 mg/L 0.0051 - 0.12 mg/L
San Bernardino	Former steel production plant	Soil	2.4 - 76 mg/kg

*GW means groundwater; SW means surface water
Source: Communication with site project managers.

Lead in Waste Streams

Another potential source of lead in the environment is from waste streams. While the majority of hazardous wastes are managed in compliance with requirements designed to prevent releases to the environment, on occasion accidents and mistakes in handling, either intentional or inadvertent, occur. Such incidents may ultimately become a contaminated site in need of remediation.

As described in Chapter 1, lead is ubiquitous in many products and thus in waste streams. Lead is or has been used to produce a variety of products, including lead-acid batteries, paint, solder, automobiles, munitions, cathode ray tubes, ceramics and crystals, and vinyl mini-blinds, which all eventually become wastes. In addition, lead can be found in many industrial wastes, such as ashes from co-generation plants and municipal waste incinerators, spent abrasive blasting material, metal drosses, sludges, and slags, spent foundry sands, refinery tank bottoms, machining scrap, spent etching solutions, used oil, galvanizing wastes, sewage sludges, auto shredder waste and contaminated soil.

In addition to information about chemical releases, U.S. EPA's TRI database also provides information about waste quantities generated and managed by the reporting industries. Facilities must report the amounts of chemicals that are recycled, combusted for energy recovery, treated, or disposed, both on- and off-site. The TRI data represents the amount of a specific chemical in pounds reported in the waste, however, not the amount of the entire waste stream. In addition, only certain industries report TRI data, so this database is not comprehensive.

Because TRI data can be sorted by industry, those industries that generate the most lead and lead compounds in their waste on an annual basis in California can be identified. As shown in Tables 2-14 and 2-15, which depict total lead and lead compounds in waste managed from 1995 through 1999, certain industry groups clearly predominate in the amount of lead released as waste. Appendix 2-A contains more detailed TRI reports showing the breakdown among waste management activities.

The largest industry reporting lead in wastes in that period of time for the lead category, on an annual basis in California, was the industry group classified as primary metals. This industry group includes companies engaged in smelting and refining ferrous and nonferrous metals from ore, pig, or scrap; in rolling, drawing, and alloying metals; in manufacturing castings and other basic metal products; in manufacturing nails, spikes, and insulated wire and cable; and in producing coke. Most of the lead managed by this industry group was recycled on- and off-site. The next largest industry group reporting lead in waste was fabricated metals, which includes companies engaged in fabricating ferrous and nonferrous metal products, fabricated structural metal products, metal forgings, metal stampings, ordnance, and miscellaneous metal and wire products. Almost all of the lead-containing waste managed by this industry was recycled offsite. The two new industry groups that started reporting in 1998, metal mining and RCRA solvent recovery operations, also reported lead-containing waste in large quantities.

Table 2-14 – Lead in Waste Managed (in pounds), California, 1995 – 1999

SIC Code	Industry	Total Waste Managed 1999	Total Waste Managed 1998	Total Waste Managed 1997	Total Waste Managed 1996	Total Waste Managed 1995
29	Petroleum	7,539	1,441	-	1,209	3,017
30	Plastics	15,439	18,250	16,865	2,450	21,725
32	Stone/Clay/Glass	-	-	-	2,700	2,700
33	Primary metals	1,549,220	907,336	1,286,600	850,032	1,813,307
34	Fabricated metals	284,817	245,194	225,885	404,968	345,225
35	Machinery	1,862	2,993	3,113	2,692	3,960
36	Electrical equip.	128,063	96,209	135,972	340,232	174,416
37	Transp. equip.	127,452	152,389	150,149	110,342	83,513
20-39	Multiple codes	161,009	286,531	487,809	136,554	120,906
-	No reported codes	36,830	-	17,532	12,582	16,081
	Original Industries Subtotal	**2,312,231**	**1,710,343**	**2,323,925**	**1,863,761**	**2,584,850**
10	Metal mining*	-	439,575	-	-	-
4953/ 7389	RCRA/solvent recovery*	1,187,255	233,858	-	-	-
	Total	**3,499,486**	**2,383,776**	**2,323,925**	**1,863,761**	**2,584,850**

*New industries added in 1998 reporting year.
Source: Internet web site, accessed 01/07/02: http://www.epa.gov/triexplorer/explorer.htm

Table 2-15 – Lead in Waste Managed (in pounds), for Lead Compounds California, 1995 – 1999

SIC Code	Industry	Total Waste Managed 1999	Total Waste Managed 1998	Total Waste Managed 1997	Total Waste Managed 1996	Total Waste Managed 1995
28	Chemicals	200,806	231,000	360,900	442,526	220,434
29	Petroleum	1,200	3,779	11,155	5,665	12,522
30	Plastics	2,019	2,532	2,043	1,939	2,079
33	Primary metals	2,988,957	4,056,183	5,357,605	4,485,268	2,359,587
36	Electrical equip.	23,207,669	21,456,561	23,195,338	22,691,169	20,375,766
38	Measure/photo.	29,010	29,100	14,100	14,100	15,100
20-39	Multiple codes	951,850	803,921	757,299	47,491	21,839
-	No reported codes	37	23	-	-	13
	Original Industries Subtotal	**27,381,548**	**26,583,099**	**29,698,440**	**27,688,158**	**23,007,340**
10	Metal mining*	223,306	1,423,434	-	-	-
4953/ 7389	RCRA/solvent recovery*	1,714,910	2,409,127	-	-	-
	Total	**29,319,764**	**30,415,660**	**29,698,440**	**27,688,158**	**23,007,340**

*New industries added in 1998 reporting year.
Source: Internet web site, accessed 01/07/02: http://www.epa.gov/triexplorer/explorer.htm

The data for lead compounds reveals some similarities, although the largest amount of waste is reported by the industry defined as electronic and other electrical equipment and components, except computer equipment, which includes establishments engaged in the manufacturing of machinery, apparatus, and supplies for the generation, storage, transmission, transformation, and utilization of electrical energy. The primary metals industry represents the second largest industry reporting this waste and the chemicals and allied products industry is the third largest industry reporting this waste. This latter industry group includes companies producing basic chemicals, such as acids, alkalies, salts, and organic chemicals; chemical products to be used in further manufacture, such as synthetic fibers, plastics materials, dry colors and pigments; and finished chemical products to be used for ultimate consumption, such as drugs, cosmetics, and soaps, or as supplies for other industries, such as paints, fertilizers, and explosives. Again, the two new industry groups, metal mining and RCRA solvent recovery operations, also produced this waste in large quantities.

California's hazardous waste manifests provide another source of information about generated wastes. Data gathered from manifests may be used to indicate the quantities of lead-bearing wastes produced annually in this state, the disposition of these waste streams, and, to some extent, the nature of these wastes. Because there is no specific California waste code for lead-containing wastes in general, the evaluation focused on the wastes manifested from 1993 to 2000 bearing the federal waste code for lead, D008. These wastes are characterized as hazardous due to the federal toxicity characteristic for lead.

As shown in Table 2-16, from 1993 to 2000, California generators manifested between 78,516 and 110,284 tons of D008 waste annually (with a mean of 90,187 tons and a standard deviation of 10,558 tons). In total, about 721,500 tons of D008 wastes were manifested in that time period. Some of the waste reported on manifests as D008 wastes were also identified by California Waste Codes (CWC). The major waste code categories are included in Table 2-16. Of the total wastes manifested from 1993 to 2000, where the manifest included a disposition code, 261,213 tons were recycled, 159,082 tons were treated (including incineration), and 100,736 tons were disposed to land.

Table 2-16 – California Manifest Data for Lead-Containing Waste Codes (tons) – 1993 – 2000

Waste	1993	1994	1995	1996	1997	1998	1999	2000	Total
Total D008 waste	78,516.29	88,926.83	79,567.83	91,109.18	92,302.46	110,284.52	98,135.19	82,610.18	721,495.48
Other inorganic solid waste (CWC 181) % of total	35,445.10	51,682.23	52,927.17	62,817.16	59,870.43	68,505.59	50,624.54	50,321.86	432,194.08 60%
Contaminated soil (CWC 611) % of total	21,461.09	23,280.45	10,687.92	9,687.18	13,617.18	16,999.47	17,302.63	17,437.18	130,473.10 18%
Other organic solids (CWC 352) % of total	8,550.62	1,938.64	1,615.71	3,021.03	5,273.74	6,742.92	4,622.31	4,227.47	35,992.44 5%
Aqueous soln with metals (CWC 132) % of total	1,010.20	526.02	724.03	3,358.62	1,869.73	1,999.65	14,821.26	836.98	25,146.49 3%
Ash (CWC 571) % of total	27.98	747.92	5,701.46	3,117.18	3,007.15	2,996.99	2,673.43	466.59	18,738.70 3%
Off-spec. surplus inorganics (CWC 141) % of total	1,486.31	1,687.44	1,618.28	1,651.29	2,077.16	2,528.49	2,288.72	2,174.14	15,511.83 2%

Source: Haznet, 2001.

Physical and Chemical Properties

Lead is a dense, corrosion-resistant and malleable blue-gray metal with a low melting point and high boiling point. Comprised of a mixture of isotopes with mass numbers 204 (15%), 206 (23.6%), 207 (22.6%) and 208 (52.3%), the average atomic weight of lead varies depending upon its origin. Lead has four electrons in its outer shell, but most commonly forms ionic compounds corresponding to the +2 oxidation state. Lead also forms covalent compounds corresponding to the +4 oxidation state, but less commonly than the divalent forms[101].

Lead occurs primarily as lead sulfide (galena) in magmatic rocks, commonly in association with zinc sulfide (sphalerite), but may also occur as the metal. Often lead and zinc are mined together and separated later. Although thermodynamically unstable in most aqueous environments, lead sulfide will only slowly dissolve while metallic lead is very resistant to dissolution. In a finely divided state or under acidic conditions, the rate of dissolution is expected to be higher as more mineral surface area is available for reaction. Within the normal limits of redox potential and pH found at the earth's surface, however, virtually the only thermodynamically stable oxidation state for lead is the divalent state, +2. Hence, the environmental chemistry of lead is largely controlled by acid/base reactions and not redox reactions. In contrast, among organolead compounds the tetravalent state (+4) is predominant. Such organolead compounds also often undergo photolysis when exposed to light[102]. Physical and chemical properties of lead and some selected lead compounds are summarized in Table 2-17. A more complete listing is available in Appendix 2-D.

Table 2-17 – Physical and Chemical Properties of Lead and Lead Compounds

Property	Lead Pb	Lead oxide PbO	Lead phosphate $Pb_3(PO_4)_2$	Lead sulfate $PbSO_4$	Lead sulfide PbS
Molecular weight	207.20	223.21	811.54	303.26	239.26
Color	Bluish-gray	Reddish-yellow; yellow above 489 ^oC	White	White	Black, blue or silvery
Density (g/cm^3)	11.34 @ 20 ^oC	9.3 (litharge) 8.0 (massicot)	6.9 – 7.3	6.2	7.5
Melting point (^oC)	327.4	886 (litharge)	1,014	1,170	1,114
Boiling point (^oC)	1,740	1,472 (decomposes)	No data	No data	1,281 (with sublimation)
Solubility (water)	Insoluble	10 mg/L at 20 ^oC	0.14 mg/L at 20 ^oC	42.5 mg/L at 25 ^oC	0.86 mg/L at 13 ^oC

Source: U.S. HHS, 1999, pp. 362 – 365; U.S. EPA, Vol. II, 1986, pp. 3A-1 – 3A-2.

Solubility products (K_{sp}) of selected lead compounds are given in Table 2-18. Most of these minerals are too soluble to be important in natural environments. Of these minerals, $PbSO_4$ (anglesite) is the most stable below pH 6.0 and $PbCO_3$ (cerussite) is generally the most stable at higher pHs.

Table 2-18 – Solubility Product Constants of Selected Lead Minerals

Compound	log K_{sp}
PbO(yellow)	-15.1
PbO(red)	-15.3
$Pb(OH)_2$	-19.84
$PbSO_4$ (anglesite)	-7.8
$PbCO_3$	-13.1
$Pb_3(CO_3)_2(OH)_2$	-24.5
PbS (galena)	-27.5
$Pb_3(HPO_4)_2$	-43.5
$PbHPO_4$	-9.9
PbMoO4 (wulfenite)	-16.04

Source: Adapted from Lindsay, W. L., 1979, Chemical Equilibria in soils. John Wiley & Sons, New York, N.Y.

It has been suggested that lead solubility may be primarily controlled by lead phosphate minerals in the natural environment. Lead phosphate minerals are known to be highly insoluble. If phosphate (PO_4^{3-}) activity is controlled by the mineral strengite ($FePO_4 \cdot H_2O$) and ferric iron (Fe^{3+}) activity in acid soils, then the activity of phosphate will be such that the activity of Pb will be controlled by $Pb_5(PO_4)_3Cl$ (chloropyromorphite). At higher soil pHs, phosphate activity, and hence, lead activity, will most likely be controlled by calcium phosphate minerals present in the soil.

Below pH 7.7, the lead ion Pb^{2+} predominates over hydrolysis species of Pb. Above pH 7.7, $PbOH^+$ is the predominant hydrolysis specie. In solution, lead readily forms soluble complexes with chloride (Cl^-). Chloride complexes with lead will be important in waters with high chloride concentrations, such as seawater. Formation constants (K) for important species of lead are given in Table 2-19. The lead ion also forms soluble complexes with various chelates, such as EDTA and citric acid.

Table 2-19 – Formation Constants for Soluble Lead Complexes

Equilibrium Reaction	Log K*
$Pb^{2+} + OH^- = PbOH^+$	6.30
$Pb^{2+} + 2OH^- = Pb(OH)_2(aq)$	10.25
$Pb^{2+} + Cl^- = PbCl^+$	1.60
$Pb^{2+} + 2Cl^- = PbCl_2(aq)$	1.78

* Equilibrium constants assume 0 ionic strength and 25°C

Source: Adapted from Lindsay, W. L., 1979, *Chemical Equilibria in Soils*. John Wiley & Sons, New York, N.Y.

Lead may be removed from the soil solution by either precipitation of above described minerals or by adsorption to soil organic matter and solid phases, primarily clays and iron and manganese oxide minerals. Very low measured soil solution concentrations of lead suggest that lead solubility in soils is primarily controlled by adsorption processes.

Environmental Fate

As discussed earlier in this chapter, a variety of sources, some of them obsolete, have discharged lead to the environment and continue to contribute to lead emissions. The behavior of lead in the environment helps to determine the extent of the potential exposure that may result from the various lead emissions. Important factors include the movement of lead through and among environmental media, transformation of lead compounds under ambient conditions and the persistence of lead in environmental media.

Air

Air emissions of inorganic lead consist primarily of particles that are dispersed through the air and ultimately deposited to soil, dust, water or biota. Lead-contaminated dust can become re-entrained and undergo a similar dispersion and deposition process, as demonstrated by the air emissions data, which attribute the major sources of lead emissions in California to dust.

The movement of lead in the atmosphere is associated primarily with the particle size distribution of the emissions and atmospheric conditions, such as air turbulence. In general atmospheric particles can be classified into three groups, nuclei mode (<0.1 um), accumulation mode (0.1 – 2 um) and large particle mode (>2 um). Lead particles near a source tend to occur in the nuclei and large particle modes. The large particles are typically deposited near the source, while the nuclei mode particles either attach to surfaces or agglomerate while airborne to form accumulation mode particles, which then can travel great distances[103].

For example, it has been estimated that about 35% of the lead consumed in gasoline is emitted as small particles (<0.25 um mass median aerodynamic diameter (MMAD)) and 40% as large particles (>10 um MMAD). The small particles may agglomerate and remain airborne for 7 to 30 days, traveling thousands of miles from the source, while the larger particles do not remain in the atmosphere as long[104]. Hence, although the use of leaded gasoline has been restricted in the U.S. and other developed countries, its continued use for off-road vehicles and in developing countries can affect regions far from the original sources of emissions.

Deposition of lead particles occurs either through dry or wet mechanisms. Dry deposition involves movement from the air stream first to the boundary layer and then to an impact with a surface. Large particles tend to accelerate downward, independent of windspeed or surface characteristics, eventually achieving a constant velocity. Smaller particles move through other mechanisms, often depending on windspeed or turbulent eddies, which are typically affected by weather and surface irregularities. Wet deposition involves particles that either form the nucleus of raindrops within clouds, or particles that are collected by raindrops as they fall through the atmosphere.

The relative importance of wet and dry deposition for airborne lead particles varies, most likely with weather conditions, topography and airborne particle size distributions and concentrations. Some sources indicate that wet deposition removes more lead from the atmosphere than dry deposition with ratios of wet to dry deposition ranging from 1.63 to 2.5[105]. Other sources estimate global amounts of wet and dry

deposition to be roughly equal. For example, of an estimated global atmospheric lead deposition total of 410 x 10^6 kg/year, 208 x 10^6 kg/year has been attributed to wet deposition and 202 x 10^6 kg/year to dry deposition[106].

Water

As shown in Table 2-17, elemental lead is insoluble and most other lead compounds have low water solubilities. The occurrence of dissolved lead in surface water depends primarily on the pH of the water, and the dissolved salt content of the water. In theory more dissolved lead can be expected in soft waters with low pH. Calculations show that at pH<5.4 the total lead solubility is about 30 ug/L in hard water and about 500 ug/L in soft water[107]. In water with pH near 6.5 and an alkalinity of about 25 mg bicarbonate ion/L of water, common in areas of the northeastern U.S., lead concentrations of 330 ug/L could be stable[108].

In most natural water, however, lead tends to form compounds of low water solubility with anions in the water, such as hydroxides, carbonates, sulfates and phosphates. These compounds ultimately precipitate out of the water and either adsorb and accumulate in sediments or are incorporated into organic matter and other solid particles carried in the water. In river water most lead is expected to be in an undissolved form. The ratio of lead in suspended solids to dissolved lead ranges from 4:1 in rural streams to 27:1 in urban streams[109].

Soil

Much of the accumulation of lead in soil is directly related to atmospheric deposition and in most soils, lead is not expected to move appreciably. Research has shown that lead deposited on soils from automobile emissions remains in the top few centimeters of soil. In addition, land subject to treatment with sewage sludge containing a high concentration of metals shows little movement of lead below the application zone.

The behavior of lead in soil depends on solubility, adsorption or cation exchange with soil minerals, and the formation of organic-metal complexes or chelates. These processes are affected by a variety of factors such as the pH of the soil, particle size and type of the soil, organic and inorganic soil components, the cation exchange capacity of the soil, and the amount of lead in the soil[110]. Lead tends to form stable complexes with organic soil matter, and can be immobilized by ion exchange with hydrous oxides or clays in soil.

In acid soils with little clay and greater organic matter content, greater leaching of lead may be expected as organic lead complexes become more soluble. The mobilization potential of lead in soils will be also be enhanced in soil high in chloride due to the formation of lead chloride complexes. Addition of certain chelates to soil can further enhance lead solubility and mobilization potential. Lead may be expected to leach in soils when the concentration of lead approaches or exceeds the cation exchange capacity of the soil, the soil contains constituents that can form soluble complexes with lead, and the leaching solution is acidic.

Overall, however, the movement of lead downward into soil is largely facilitated by mechanical action, such as soil tilling. In addition erosion of lead-containing soil by wind and water can result in lead movement, including transfer of lead to surface waters.

Biota

Lead has been found to bioconcentrate in plants and animals, but biomagnification has not been reported. The highest lead concentrations species have been observed near the sites of lead emissions, such as

mining, smelting and refining facilities, areas with heavy auto and truck traffic, battery recycling facilities, sewage sludge disposal areas, hunting grounds, and urban and industrialized areas[111].

Plant uptake of lead from soil can occur under conditions similar to those that increase soil leachability. Plant surfaces can become contaminated from atmospheric deposition and lead deposited on foliage surfaces can also be taken up by the plant and incorporated into its internal tissue. Terrestrial animals accumulate lead through inhalation of lead particles, and ingestion of contaminated species. The highest concentration of lead occurs in the oldest organisms.

Among aquatic species, the highest concentration of lead occurs in the benthic organisms and algae; the lowest lead concentrations appear in the highest level predators, such as carnivorous fish[112]. In studies of aquatic life the highest bioconcentration factors were found among algae, mussels and oysters, and were lower for fish[113]. Because lead is toxic to aquatic species, it has been suggested that organisms higher on the food chain may experience lead poisoning[114]. That is, the species occupying positions higher on the food chain are do not exhibit correspondingly high concentrations of lead because these fish are killed by the toxic effects of lead; only those fish with lower lead concentrations are able to survive and serve as subjects for lead measurements.

CHAPTER 3 – LEAD IN OUR LIVES: EXPOSURE AND HEALTH EFFECTS

As shown in the previous chapter, anthropogenic sources of lead account for the most common sources of environmental lead, largely because lead has been widely used throughout history and remains persistent in the environment. These sources also are the most prevalent sources of human lead exposure, including exposure of the general population and occupational exposure. The health effects associated with exposure to lead vary widely depending upon the level of exposure, the physiological system affected and the receptor.

Exposure

Lead is so widespread that most people, if not all, are exposed to it in one medium or another. The many pathways of exposure to lead are complex and often overlapping. Exposure commonly occurs through ingestion of lead-contaminated food, water, soil, paint chips, and dust; inhalation of lead-containing fumes and fine lead particles; and, to a lesser extent, through dermal absorption of certain lead compounds. Figure 3-1 depicts the primary pathways that result in exposure of humans to lead.

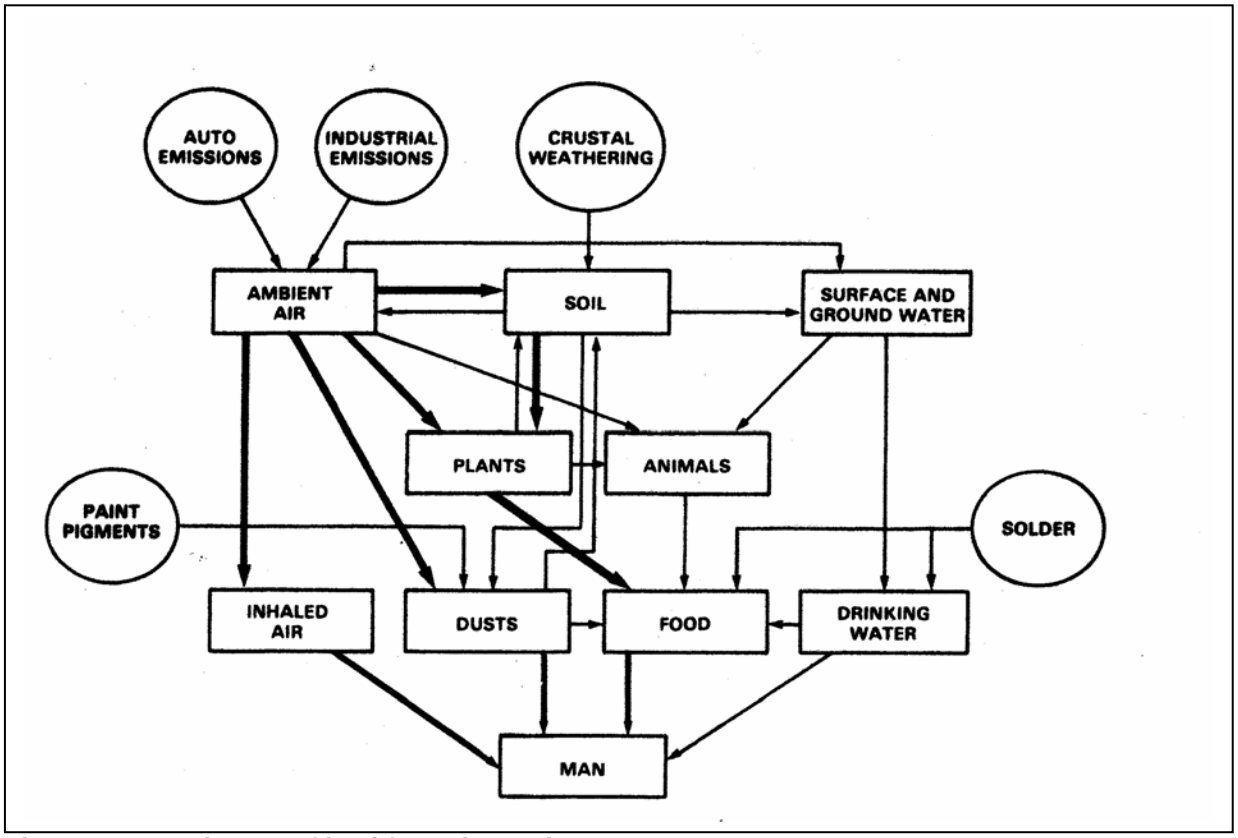

Figure 3-1 – Pathways of lead from the environment to man
Source: U.S. EPA, 1986, p. 7-42

The most common sources of exposure for children are dust and paint chips from lead-based paint[115]. For adults, over 90% of cases of elevated blood lead levels can be attributed to occupational exposure, through inhalation of lead fumes and particles and ingestion of lead-contaminated dust and other lead-contaminated materials in the workplace[116]. Other potential sources of exposure for the general population include lead-contaminated soils and dust at shooting ranges, molten lead and lead solder used in hobbies (such as stained glass making), lead glazes for pottery, art paints, glassblowing coloring agents, plastic food wrap pigments, certain cosmetics and folk remedies, and inadequately glazed lead in earthenware used for food storage or cooking.

Inhalation

In the past most inhaled lead was attributed to motor vehicle emissions. During the phase-down of leaded gasoline, from 1976 to 1995, ambient concentrations of lead in air in the U.S. declined by 97%, although from 1994 to 1995 national average lead concentrations did not change although emissions of lead declined by 1%[117]. Current emissions of airborne lead are dominated by industrial sources, such as lead mining, smelting, or lead acid battery recycling. This exposure pathway also includes airborne particles from lead-contaminated soil and dust. As discussed in Chapter 2, lead in soil remains largely immobile, resisting movement through leaching under typical conditions. Hence, surface contamination is likely to remain at the soil surface and soils contaminated by past emissions, such as the deposition of motor vehicle emissions from previous consumption of leaded fuel, are likely to remain contaminated. Such soils can become airborne, representing an inhalation pathway for lead-laden dust.

Air lead concentrations vary due to a variety of factors, including proximity to emission sources, the presence of contaminated soils and dusts, and weather conditions. Historically, air lead concentrations tended to be higher in urban areas and near smelting and mining operations. Estimates of the atmospheric concentration of lead has been known to range between 0.3-1.1 μg/m3 in urban areas and 0.15-0.3 μg/m3 in rural areas[118]. Estimates of exposure to air lead concentrations depend upon conditions including whether exposure occurs indoors or outdoors, level of activity, season, and the type of monitoring used.

Those exposed to the inhalation pathway of lead exposure at levels beyond background exposure include populations located near emission sources and areas where lead-containing soils or dusts are likely to become airborne. When inhaled air contains lead particulates, a portion of the lead is deposited and absorbed in the pulmonary and upper respiratory tract and a portion is deposited and absorbed in the gastrointestinal tract, since particles deposited in the airways are cleared by the mucociliary apparatus and swallowed. Once particulate lead is deposited in the lower respiratory tract, it is almost completely absorbed.

In adult humans the pulmonary deposition rate of lead in ambient air ranges from 30% to 50% with most of the amount deposited thought to be absorbed over a short time period[119]. In addition the absorption rate varies with breathing rate and particle size. Children inhale a higher daily volume of air per unit body weight and area proportionate to adults and the pulmonary deposition rate for children has been estimated to be as much as 2.7-fold greater per unit body mass than for adults[120].

Ingestion

As shown in Figure 3-1, ingestion of lead occurs through many pathways. Some exposure occurs through ingestion of food and water containing lead, although in recent years, limits have been imposed to reduce the incidence of lead from these sources. Ingestion of lead also occurs when lead-containing soils and dusts are ingested. Soil and dust become contaminated with lead in a number of ways, both

natural and anthropogenic. Lead also can be ingested when non-food items containing lead are ingested, as in the case of lead-based paint chips.

The absorption and bioavailability of ingested lead in humans depends upon a number of factors, including age and health status; ingestion patterns; and particle size, solubility and species of lead. Estimates of the rate of absorption of dietary lead vary, but all agree that absorption is higher in children than adults, a trait observed in humans and other mammals[121]. One source reports the dietary absorption rate of lead in the intestinal tract for children is about 50%, whereas adults absorb up to 10-15% of lead ingested with meals[122]. Another source estimates young children absorb 30-40% of ingested lead while adults absorb 5-15%, retaining less than 5% of this amount[123]. Yet another source estimates adults absorb about 11% of lead that reaches the digestive tract and children absorb from 30 to 75 percent[124]. For adults ingesting lead under fasting conditions, however, the absorption rate can be 60% or higher[125]. In addition lead absorption tends to be higher in individuals with nutritional deficiencies, especially iron, calcium, zinc and vitamin D deficiencies, and compromised health. Lead absorption also appears to increase during the latter half of pregnancy. Acid in the gastrointestinal system solubilizes ingested lead, resulting in absorption. Lead transport across digestive tissue appears to mimic that of calcium[126]. Lead that is not absorbed in the gastrointestinal system is excreted.

Food

Depending upon the food item, a variety of sources can contribute lead to foods. Deposition of airborne lead particles onto food crops can result in elevated lead concentrations on the plant's surfaces, which persists and accumulates during the life of the crop. The lead can then be ingested directly if the plant surfaces are not thoroughly cleansed before food processing or consumption. Such surface contamination is not easily removed by rainfall or harvest washing[127].

Lead has also been found in internal plant tissues, indicating that plants can take up lead from the surrounding environment. For example, foliar uptake of lead from surface contamination may occur in some plant species. Edible plants grown in soil containing lead may also take up available lead from the soil though the plant's root system[128]. Because lead tends to adsorb to soil organic matter, the bioavailability of lead in soil to plants depends upon the soil conditions, such as the soil pH and level of organic matter.

Table 3-1 shows lead concentrations in samples of raw edible plants grown in agricultural soils undisturbed by other human activities. This data is used to represent background concentrations of lead in these food crops. Actual lead levels in food crops may be higher if the crops are grown near lead sources.

Table 3-1 – Lead Concentrations in Edible Plants

Plant Species	Mean µg/g wet weight
Wheat	0.037
Potato	0.009
Field corn	0.022
Sweet corn	0.0033
Soybean	0.042
Peanut	0.010
Onion	0.005
Rice	0.007
Carrot	0.009
Tomato	0.002
Spinach	0.045
Lettuce	0.013

Source: U.S. EPA, 1986, p. 7-33.

Studies of lead in food crops generally show that the lead concentration is lowest in crops, such as fruit or corn, where the edible portion of the plant grows above ground, but is protected from lead deposition. Lead levels are typically highest in crops whose edible portion is exposed to airborne deposition, such as leafy greens and grains. Root crops, such as carrots, potatoes or onion, are protected from deposition but accumulate lead in the root portion of the plants and usually have lead levels somewhat between the other crop types[129].

Studies of lead concentrations in soil and uptake of lead by plants indicate that the amount of lead in internal plant tissue correlates directly with the amount of lead in the soil[130]. These studies did not find bioaccumulation of lead by the plants studied[131].

Among aquatic species, the highest lead concentrations are found in algae and benthic organisms with lower concentrations among species higher on the food chain[132]. As depicted in Table 3-2, reported bioconcentration factors vary widely among aquatic species. Table 3-3 shows the concentration of lead in various species of shellfish.

Table 3-2 – Bioconcentration Factors Among Aquatic Species

Species	Bioconcentration factor
Crassostrea virginica (oyster)	6,600
Senenastrum capricornutum (freshwater algae)	92,000
Salmo gairdneri (rainbow trout)	726
Fish	42
Oysters	536
Insects	500
Algae	725
Mussels	2,570

Source: U.S. DHHS, 1999, p. 391.

Table 3-3 – Lead Concentrations in Shellfish

Species	Mean lead level* (µg/g)	Range of mean lead levels** (µg/g)
Clam, hardshell	0.24	0.7-0.8
Clam, softshell	0.29	0.4-0.5
Oyster, Eastern	0.11	0.6-0.7
Oyster, Pacific	0.06	0.6-0.7
Clams, overall	0.26	---
Oysters, overall	0.09	---
Crab, blue, body/claw	---	0.6-0.7
Crab, dungeness, body/claw	---	0.7-0.8
Lobster, American, claw/tail	---	0.5-0.6
Lobster, spiny, Atlantic, tail	---	0.6-0.7
Lobster, spiny, Pacific, tail	---	0.5-0.6
Shrimp, several species, tail	---	0.6-0.7
Range of mean lead levels for molluscs and crustaceans	---	0.4-0.8

*Results of 1985-86 FDA shellfish survey for lead – wet weight basis
**Results of 1978 National Marine Fisheries Service survey of trace elements in selected shellfish
Source: U.S. FDA, 1998.

Plants containing elevated lead concentrations can also affect livestock and other food sources. For example, an increased lead concentration was observed in the blood of cattle grazing near a lead smelter and was attributed to consumption of contaminated forage[133]. Animals can be directly affected, too, as in the instance of lead transferred to the milk and meat of cattle that had licked parts of burned and discarded lead storage batteries[134]. Some of the lead in meat can also be attributed to animals consuming soil that contains lead[135]. Normal lead concentrations in beef in Sweden are <0.005 mg/kg and lead concentrations in milk seem to vary with the time since exposure and the cow's state of gestation[136]. Table 3-4 shows typical concentrations of lead in foods in the U.S., including meat and dairy products, based on studies conducted by the federal Food and Drug Administration (FDA) in the 1980s[137].

Table 3-4 – Typical Lead Concentrations in Foods

Food Group	Concentration (µg/g)
Dairy Products	0.003 - 0.083
Meat, fish and poultry	0.002 - 0.159
Grain and cereal products	0.002 - 0.136
Vegetables	0.005 - 0.649
Fruit and fruit juices	0.005 - 0.223
Oils, fats and shortenings	0.002 - 0.028
Sugar and adjuncts	0.006 - 0.073
Beverages	0.002 - 0.041 (µg/L)

Source: U.S. DHHS, 1999, p. 403.

The FDA conducts a total diet study (TDS) of various residues, contaminants and nutrients in a market basket collection of typical foods to estimate intake of these substances in representative diets. In the early 1990s the FDA updated its total diet study and since that time over 260 food items have been evaluated quarterly for lead and other substances. Table 3-5 presents selected results from the most recent TDS, an evaluation of market baskets from 1991 through 1999[138]. The complete results of this most recent TDS are presented in Appendix 3-A.

In a food safety compliance program administered by the FDA Center for Food Safety Applied Nutrition, the FDA is expanding its database on background levels of lead in foods. This research focuses on foods and ceramics, particularly imported items, to identify any correlation between environmental and manufacturing practices in developing countries and elevated levels of lead in foods[139]. Although regulatory action is not anticipated to be part of this effort, the program's protocol specifies that any atypically high sample results for lead in foods should be reported to the FDA Center for Food Safety Applied Nutrition. Lead levels greater than 0.25 ppm are considered to be atypically high for this program[140].

Table 3-5 – U.S. FDA Total Diet Study: Results for Lead

Food Description	Mean (mg/kg)	Std deviation (mg/kg)	Minimum (mg/kg)	Maximum (mg/kg)
Cabbage, fresh, boiled	0	0	0	0
Iceberg lettuce, raw	0	0.001	0	0.006
Pear, raw	0	0.002	0	0.008
Tomato, red, raw	0	0	0	0
Banana, raw	0.001	0.003	0	0.008
Broccoli, fresh/frozen, boiled	0.001	0.003	0	0.014
Cantaloupe, raw	0.001	0.004	0	0.014
Corn, fresh/frozen, boiled	0.001	0.004	0	0.014
Eggs, boiled	0.001	0.003	0	0.013
Oatmeal, quick, cooked	0.001	0.002	0	0.009
Plums, raw	0.001	0.003	0	0.011
Strawberries, raw	0.001	0.003	0	0.009
Whole milk, fluid	0.001	0.003	0	0.011
Apple, red, raw	0.002	0.004	0	0.013
Green peas, fresh/frozen, boiled	0.002	0.005	0	0.020
Orange, raw	0.002	0.004	0	0.012
Beef steak, loin, pan-cooked	0.003	0.006	0	0.020
Grapes, red/green, seedless, raw	0.003	0.005	0	0.019
Peach, raw	0.003	0.007	0	0.030
Wheat cereal, farina, quick, cooked	0.003	0.006	0	0.022
White rice, cooked	0.003	0.007	0	0.030
Whole wheat bread	0.005	0.006	0	0.018
Sandwich cookies w/crème filling, commercial	0.010	0.010	0	0.040
Sauerkraut, canned	0.010	0.017	0	0.069
Collards, fresh/frozen, boiled	0.011	0.008	0	0.040
Raisins, dried	0.011	0.011	0	0.030
Pineapple, canned in juice	0.012	0.008	0	0.030
Spinach, fresh/frozen, boiled	0.012	0.012	0	0.040
Chocolate chip cookies, commercial	0.013	0.005	0	0.023
Chocolate snack cake w/chocolate icing	0.015	0.006	0	0.025
Chocolate syrup dessert topping	0.015	0.007	0	0.030
Sweet potato, fresh, baked	0.014	0.015	0	0.064
Teething biscuits	0.014	0.008	0	0.030
Pear, canned in light syrup	0.019	0.016	0	0.067
Dill cucumber pickles	0.022	0.018	0	0.064
Honey	0.023	0.011	0	0.040
Sweet potatoes, strained/junior	0.023	0.007	0.010	0.039
Fruit cocktail, canned in heavy syrup	0.025	0.017	0.008	0.064
Peach, canned in light/medium syrup	0.026	0.016	0	0.057
Milk chocolate candy bar, plain	0.027	0.021	0	0.110
Liver, beef, fried	0.030	0.019	0.014	0.080
Sweet cucumber pickles	0.031	0.023	0.007	0.115
Shrimp, boiled	0.032	0.048	0	0.210

Source: FDA, *Total Diet Study Statistics on Element Results*, 2001.

The amount of lead ingested from dietary sources is declining, largely due to the phaseout of lead-soldered cans, as described further below. In the past, estimates of dietary lead ingestion in adults ranged from 82-95 µg/day[141]. Table 3-6 contains data from the FDA's total diet study that shows from 1982-1984 to 1994-1996 daily intakes of lead from food declined markedly for both children, by 96%, and adults, by 93%[142].

Table 3-6 – FDA's Total Diet Study Daily Lead Intakes (µg/day)

	1982-1984	1994-1996	% change
Children - 2-5 years	30	1.3	96%
Adults	38	2.5	93%

Source: U.S. FDA, 1998.

Estimates of the relationship between the amount of lead ingested per day and the resulting blood lead levels vary between adults and children and apply primarily to lower ingestion concentrations. Some evidence suggests that the process for transfer of lead from the gastrointestinal tract to blood is saturable, that is, uptake occurs until a given saturation point is reached[143]. Hence, blood lead levels are directly correlated to lead ingestion, but are not necessarily proportional to the amount of lead ingested, particularly at higher concentrations. U.S. EPA has estimated for children an increase of 0.16 µg Pb/dL blood for each µg Pb/day ingested, and for adults, an increase of 0.04 µg Pb/dL blood for each µg Pb/day ingested[144]. Using these estimates, the FDA developed provisional tolerable total intake levels (PTTIL) for lead for different population groups, depicted in Table 3-7, which were used to develop levels of concern for the consumption of shellfish[145].

Table 3-7 – Provisional Tolerable Total Intake Levels (PTTIL)

Population	PTTIL (µg Pb/day)
Children, ages 0-6 years	6
Children, over 7 years	15
Pregnant women	35
Adults	75

Source: FDA, 1993.

Lead in foods occurs primarily from environmental sources, or from processing and packaging activities. As noted, crops, livestock and other food sources can become contaminated with lead due to contact with atmospheric deposition, soil, sediments or water during growth, harvesting or collection. In addition, contact with lead-contaminated media during preparation and processing activities can further introduce lead to food items. If machinery is used during processing, to grind or crush foods, for example, lead from metallic machine parts or industrial greases may also contribute lead to foods. In addition lead-containing water used during food preparation may introduce lead. Finally, in some instances lead in packaging materials may leach into foods, particularly under acidic conditions. The best known of these is lead-soldered cans.

Food packaging

In the past, solder containing lead was commonly used to seal cans and was the greatest source of lead in foods at the time, estimated to contribute more than 66% of the lead in canned foods and beverages[146]. Migration of lead from the solder into foods is increased by acidic foods, the presence of oxygen, and length of time the food is in contact with the solder seam[147]. The lead concentration ranges in foods

shown in Table 3-4 includes both fresh foods and canned foods with lead-soldered seams. As depicted in Table 3-8, lead concentrations in albacore tuna from cans with lead solder exceed the levels in fresh tuna by a factor of 4,000, whereas lead levels in tuna from cans without lead solder exceed the levels in fresh fish by a factor of 20.

Table 3-8 – Lead Concentrations in Human Food from a Marine Food Chain (ng/g fresh weight)

Source	Concentration (ng/g)
Albacore muscle, fresh	0.3
Albacore muscle from die-punched unsolder can	7.0
Albacore muscle, lead-soldered can	1400
Anchovy from albacore stomach	21
Anchovy from lead-soldered can	4200

Source: U.S. EPA, 1986, p. 7-47.

During the 1970s efforts by the FDA to reduce lead intake from foods by children resulted in industry-wide efforts to remove lead from infant foods, primarily by discontinuing lead-soldered cans for infant formula. Eventually, the FDA established action levels for lead in infant foods and other foods, and the use of three-piece lead-soldered cans was phased out in the U.S. by 1991. The overall mean level of lead in canned food declined from 0.31 ppm in 1980 to 0.04 ppm in 1988[148].

Although lead-soldered cans are no longer used in the U.S., older canned foods may still be stored in pantries and foods canned in such cans may still be imported from other countries. In 1997 an FDA investigation found over 100 lead-soldered cans in ethnic grocery stores in California[149]. In addition other food packages may pose a hazard of lead exposure. In one report children with elevated blood lead levels had consumed sticky tamarind candy that had been packaged in candy wrappers with lead levels from 16,000 to 21,000 ppm[150]. Some plastic food wrappers are printed with inks containing lead chromate pigments. In one study of bread bags, the wrappers contained a mean concentration of 26 mg of lead/2,000 cm^2 of bag and 39% of respondents surveyed reused the bags with 16% turning the bags inside out when reusing them[151].

Water

Lead in drinking water represents another source of lead ingestion. Lead enters drinking water in two ways; either from lead in the source water, or from corrosion of lead-containing plumbing components in the water distribution system. As described in Chapter 2, although lead can occur in source water from naturally occurring sources, most lead in surface water and groundwater is attributed to anthropogenic sources. U.S. EPA estimates that less than 1% of the public water systems have source water entering the distribution system at levels exceeding 5 µg/L[152]. Exposure to lead in drinking water occurs when the water is consumed directly, or when beverages or foods are prepared or cooked using the water, either in the home or in commercial preparations.

Lead concentrations in water consumed domestically, however, can be higher due to the presence of lead in the water distribution system and household plumbing. Lead in water distribution systems occurs primarily from corrosion of lead-containing components of the water distribution and delivery systems,

including water service mains, connections such as lead goosenecks or pigtails, lead service lines, lead solder, lead pipes and lead-containing alloys such as leaded brass faucets. The amount of lead that leaches from plumbing into drinking water depends upon a variety of factors, including the corrosivity of the water; the number, age and quality of the lead-soldered joints in the plumbing system; the length and diameter of the lead service line; the temperature of the water; and, in general, the contact time between the water and lead in the distribution system. The concentration of lead in water increases with the time that water remains in contact with the lead-containing components in the water distribution system. When lead-containing plumbing systems are flushed of first draw water, the levels of lead decline markedly[153].

Although the Safe Drinking Water Act Amendments of 1986 restricted the use of lead solder or flux exceeding 0.2% lead, and lead pipes, faucets, and other plumbing fittings with more than 8% lead, the incidence of existing lead plumbing components prior to this restriction is likely to be sizeable. For example, before this restriction, solder containing up to 50% lead was commonly used to connect copper piping. In addition, U.S. EPA estimated in 1991 about 10 million lead service lines and/or connections are in use in the U.S. and about 20% of all public water systems have some lead service lines and/or connections in their systems[154]. In a well-publicized occurrence in Washington, DC, elevated lead levels were found in 4,075 of 6,118 residences tested. This instance became noteworthy because the water agency did not adequately notify residents or U.S. EPA of its findings[155].

The average lead concentrations in drinking water in the U.S. in 1988 was reported to be 17 µg/L and the national mean concentration of lead in drinking water was 29 µg/L[156]. Even with the restriction on lead content in solder and fittings, some lead may leach into water, particularly when water is corrosive and/or in contact with the lead-containing components for extended time periods. As shown in Table 3-9, even plumbing systems using plastic pipes and containing no lead solder showed elevated lead concentrations.

Table 3-9 – Lead Concentrations in One Liter First-Draw Samples (µg/L)

Source	Concentration (µg/L)
Copper pipe	9
Galvanized pipe	4.2
Plastic pipe	4.5

Source: U.S. DHHS, 1999, p. 398.

To control lead in drinking water U.S. EPA in 1991 promulgated a maximum contaminant level (MCL) goal of zero for lead[157]. Because the occurrence of lead in drinking water is highly variable, depending largely on the presence of lead in the water distribution and plumbing systems rather than the source water, U.S. EPA also changed the approach for the regulatory requirement, or MCL, for lead. In 1991 the MCL for lead was changed from 50 µg/L in source water to a treatment technique approach that requires public water systems to control the corrosiveness of the system's source water. In addition U.S. EPA established an action level for lead of 15 µg/L, above which treatment steps must be taken. For example, when more than 10% of samples collected at consumers' taps exceed the action level for lead, the water system must implement additional measures, which may include source water treatment, lead service line replacement and public education requirements.

Soils and dusts

As noted in Chapter 2, most of the lead in soils, particularly in urban areas and adjacent to residences, arises from anthropogenic sources. Deposition of airborne lead from past combustion of leaded fuel, from lead recycling or smelting, and from flaking and weathering of lead-based paint all contribute to elevated concentrations of lead in soil. Household dusts can be affected if interior surfaces are coated with lead paint, resulting in lead-containing dust from wear and scraping of painted surfaces. Similarly, other lead-contaminated dusts, such as soil dust, street dust or occupational dust, that are tracked into residences on shoes, clothing or hair, or blown into residences by drafts and breezes, can contribute to lead-containing interior dusts. One researcher estimates 50-70% of household dust is thought to derive from outdoor soils[158].

Studies have shown a correlation between exterior soil lead concentration and interior contamination, as well as a strong association between soil and dust lead concentrations and children's blood lead levels[159]. In one study lead-containing dust collected in window wells and a child's age were the strongest predictors of BLLs among children[160]. In another instance, children of a worker who restored chemically stripped furniture showed elevated blood lead levels associated both with take-home contamination by the worker, as well as lead paint in the home[161].

Dusts accumulate on exposed surfaces and become lodged in fabrics and carpets and must be removed frequently to reduce exposure. The concentration of lead in household dust varies widely depending upon the location and condition of the housing; U.S. EPA estimates a mean concentration of lead in household dust of 300 μg/g[162]. Residents are exposed to lead from such dusts through the pathways of inhalation and, more frequently, ingestion.

With the phaseout of lead-soldered cans and the control of lead in drinking water systems, ingestion of lead from contaminated soil and dust, and flaking and weathered lead paint is rising in importance. Ingestion of soils and dusts among the general population commonly occurs when foods are contaminated during food storage, meal preparation and food consumption.

Children in particular often fail to wash dirt and dust from their hands prior to eating or snacking. In addition young children ingest dirt and dust from a variety of typical childhood behaviors, including:

- finger- and thumb-sucking;
- frequent hand-to-mouth exploratory behavior involving toys and other non-food objects;
- mouthing non-food items and surfaces that collect dust, such as windowsills;
- eating dropped or soiled food; and
- playing, often vigorously, in locations that have contact with soils and dirt, such as playgrounds, parks and sidewalks.[163] "Vacant" lots also seem to be irresistible informal play areas to children.

Because the amount of soil ingested from normal child behavior is difficult to determine and depends largely on the assumptions used, estimates usually encompass a wide range of values. One researcher estimates a range of from 14 mg/day to 1,800 mg/day[164]. Another source reports children under 3 years of age ingest approximately 30-100 mg of soil and dust each day[165]. Soil ingestion is common and normal among children, especially young children, as many of the behaviors that result in soil ingestion decline after 5 or 6 years of age[166]. Some people, especially children, however, also exhibit a tendency toward the deliberate consumption of non-food items such as soil or paint chips, a condition known as pica.

Although the prevalence of pica is difficult to determine, soil-pica has been defined as the recurrent ingestion of large amounts of soil (from 1,000 – 5,000 mg/day). One researcher notes that rates of soil ingestion of 5,000 mg/day have been reported, although the frequency of this level of soil ingestion was not studied[167]. Those at greatest risk for soil-pica are young children, 6 and under, and developmentally disabled persons[168]. Pica can also include the ingestion of other nonfood items, such as paint chips or plaster. An instance of fatal lead poisoning in 2000 was attributed in part to such pica behavior. In this case a 2-year-old was hospitalized with a BLL of 391 µg/dL after she was observed removing and ingesting painted wall plaster. Later samples contained 5% and 12% lead. High levels of lead were also found in dust in a window well and peeling paint on an outdoor porch where the child had played.[169]

As described in Chapter 2, soil lead concentrations of 1,000 mg/kg near houses coated with lead-based paint are typical. Although the pattern of soil contamination near a residence shows in general a higher concentration of lead near the foundation of the structure than at more remote locations, the concentration levels of lead in soil near residences vary widely. Data from a U.S. EPA study of soil lead concentrations near residences are shown in Table 3-10.

Table 3-10 – Lead Measurements in Housing Soil Samples

Housing Type	Entrance Samples		Dripline Samples		Remote Samples	
	Range of concentrations (ppm)	Mean*	Range of concentrations (ppm)	Mean*	Range of concentrations (ppm)	Mean*
Private Housing Units	3 – 6,800	85	1 – 22,900	74	1-6,900	46
Public Housing Units	8 – 520	55	10 – 870	55	5 – 600	44

*Weighted sample geometric mean.
Source: U.S. EPA, May 1996, pp. 10-11.

Miscellaneous Sources

Some lead exposure also occurs from a variety of products that contain lead, including foods and some products used as medicinal remedies. Although the use of lead for medicinal and cosmetic purposes dates back to ancient times, in some cultures and immigrant populations traditional and folk remedies containing lead are still in use today. For example, remedies known as alarcon, greta and pay-loo-ah are often used as laxatives or for stomach or intestinal ailments and typically contain high lead concentrations[170]. Typically, greta is 99% lead oxide[171], in one case, greta powder was found to contain 770,000 ppm of lead[172]. Some calcium supplements were also found to be contaminated with lead[173].

Some eye cosmetics, namely kohls and surmas, still contain from 16% to 80% lead sulfide[174]. In one case kohl applied to the lower eyelid of a 7-month-old child resulted in a blood lead level of 39 µg/dL[175]. In addition some progressive hair dyes are made with lead acetate and may contain lead concentrations from 2,300 to 6,000 µg/g[176]. Because lead acetate is soluble and easily transferred to hands and other surfaces during and after application, such hair dyes may expose anyone who comes in direct contact with the dye, or its residues, to lead. Dermal absorption of lead by intact skin is low, however, typically less than 1%[177], with one study reporting an average non-occupational dermal absorption rate of 0.06%[178].

Foods and beverages can also accumulate lead from crockery, dishes and leaded crystal. Some glazes used on pottery or dishes, both antique and new, contain lead and can leach into foods, particularly acidic foods and when the vessel and food are heated. Lead glazes can also release lead through the abrasive activities of normal usage, such as scrubbing, scraping or washing and rinsing[179]. When lead-containing glazes are improperly applied or fired at low temperatures, large quantities of lead may leach from the vessel into food. Even properly applied glazes, however, can release lead. Some handcrafted earthenware vessels and ceramics are of particular concern since they are commonly made with lead-containing glazes and are often fired at lower temperatures. For example, fruit candy and jams packaged in jars with a lead-based glaze were found to contain elevated lead concentrations, which were attributed to the jars[180]. Tableware that contains lead in glazes, paints, decals and other decorations are best used for display or with a liner that protects foods.

In California tableware containing lead is regulated by federal and state requirements. As shown in Table 3-11, tableware that leaches lead at levels that range from 0.5 ppm to 3 ppm cannot be sold in California. Tableware with leaching levels from 0.1 to 0.226 ppm can only be sold in California with a Proposition 65 warning that the product may expose the consumer to lead. The leaching standards are based on the amount of lead that leaches from the tableware after exposure for 24 hours to a 4% acetic acid solution[181].

Table 3-11 – Standards for Lead in Tableware (ppm)

Type of Tableware	FDA/California Tableware exceeding these levels cannot be sold	Proposition 65 Tableware exceeding these standards must carry a warning
Flatware (plates)	3.0	0.226
Small holloware (bowls)	2.0	0.1
Large holloware (serving dishes)	1.0	0.1
Cups or mugs	0.5	0.1
Large pitchers, jugs	0.5	0.1

Source: DHS, 2003.

Similarly, leaded crystal glassware can leach lead into foods and beverages, especially decanters and pieces that contain liquids and foods for long periods of time and crystal with a high lead content. One study showed that after four months, the concentration of lead in port wine stored in crystal decanters with 32%, 32% and 24% lead oxide content increased from 89 µg/L to 5,331 µg/L, 3.061 µg/L and 2,162 µg/L, respectively[182]. Old wines bottled with leaded foil capsules may also present a risk of lead exposure, particularly if the foil cap has been corroded. The lead content of wine poured over residue left by a corroded lead foil cap increased from 200 µg/L to 1,200 µg/L[183]. After one study estimated 3-4 percent of wines tested could be contaminated during pouring by the lead capsule, the FDA banned the use of these lead capsules in 1996[184].

Other sources of lead exposure include smoking, hobbies and certain products made with lead. Tobacco contains about 2.5 to 12.2 µg of lead per cigarette, as estimated by the World Health Organization. Approximately 2% to 6% of the lead in tobacco is inhaled by the smoker[185]. Hobbies that employ lead

or lead solder, such as stained glass, electronics, the use of indoor shooting ranges, and the manufacture of leaded materials, such as fishing weights may also result in lead exposure[186].

Finally, many products are manufactured with lead, among other components, and the use of these products can result in inadvertent lead exposure. For example, although the candle-making industry voluntarily agreed in 1974 to stop making candles with wicks that have been stiffened with lead, recent tests revealed a small percentage of candles still use lead-containing wicks. Total lead content of the wicks ranged from approximately 24,000 μg to 118,000 μg[187].

Lead is also commonly used as a pigment in colored plastics, or a stabilizer in the manufacture of polyvinyl chloride (PVC). These materials are used in a variety of products that have resulted in exposure to lead, including wire coatings and vinyl miniblinds[188]. Brass often contains some portion of lead, added to increase the malleability of the metal. Products made of brass may also contain, and release, lead, resulting in warnings regarding items such as brass keys[189].

Measuring Lead Exposure

Quantifying human exposure to lead involves both environmental and biological measurement and monitoring methods. As summarized above, environmental measurements assess the levels of lead contained in various media that pose as sources of lead exposure. Examples include concentrations of lead in air, soils, water and foods. When combined with assumptions regarding the uptake of these sources, human exposure due to these media can be estimated.

Biological monitoring evaluates the levels of lead in the systems of biological receptors as an indicator of lead exposure. A variety of methods can be used, including measurements of lead in blood, urine, serum, cerebrospinal fluid, bones, teeth and hair. Biological effects of exposure can also be evaluated, as with the measurement of erythrocyte protoporphyrin (EP) concentration[190]. The reliability and usefulness of these methods as measurements of lead exposure vary. For example, although increased EP concentrations often indicate the inhibition of heme synthesis by lead, other reasons for elevated EP concentrations exist, including anemia[191].

The most common method of measuring lead exposure is the BLL, measured in whole blood collected through venipuncture. Because the half-life of lead in blood is about a month, BLLs can be used for screening since they typically reflect recent exposure, particularly in children, and allow for intervention and control of current lead exposure. BLLs are also useful as a diagnostic technique since the frequency and severity of symptoms increase as the BLLs increase, a phenomenon not as well-documented with other biological measurements[192]. As an added benefit, BLLs have been tracked for a number of years through the National Health and Nutrition Examination Survey (NHANES), allowing an evaluation of changes in BLLs through time. X-ray fluorescence is useful as a non-invasive, accurate method of measuring lead concentrations in bones, which represents more long-term exposure to lead[193].

NHANES

Originally begun in 1960, NHANES is administered by the National Center for Health Statistics (NCHS) of the U.S. Department of Health and Human Services (U.S. DHHS) to characterize chronic disease, health and nutrition among Americans. Originally conducted as discrete surveys and followups, NHANES now conducts interviews annually, surveying approximately 5,000 persons per year through interviews and medical examinations[194].

As described above, for many years leaded gasoline and lead solder in food tins were a major source of lead exposure, through inhalation of ambient lead, ingestion of lead contaminated soils and dust, and

food. With the elimination of lead in gasoline and food tins, however, NHANES measured a precipitous drop (78%) in the mean BLLs of the U.S. population between 1976 and 1991, from 12.8 to 2.9 µg/dL. The prevalence of BLLs over 10 µg/dL, furthermore, decreased from 77.8% to 4.3%[195]. Subsequent NHANES studies indicate BLLs continue to decline. Recent data from 1991-1994 show the geometric mean BLL for the general U.S. population is 2.3 µg/dL. NHANES data is also reported for various age groups as depicted in the following tables. Table 3-12 shows the decline in BLLs and prevalence of elevated BLLs from 1976-1994 for children aged 1-5 years[196]. Table 3-13 shows the mean BLLs and incidence of elevated BLLs (greater than 10 µg/dL) among children by age for NHANES data from 1991-1994, the most recent data evaluated.[197].

Table 3-12 – Elevated Blood Lead Levels Among Children Aged 1-5 Years

Reporting Period	Mean blood lead level (µg/dL)	Percentage with BLLs > 10 µg/dL
NHANES II 1976-1980	15	88%
NHANES III - Phase 1 1988-1991	3.6	8.9%
NHANES III – Phase 2 1991-1994	2.7	4.4%

Source: Juberg, 2000, p. 30.

Table 3-13 – Incidence of Elevated Blood Lead Levels Among Children –1991-1994

Age of Child	Geometric mean blood lead level (µg/dL)	Percentage with BLLs > 10 µg/dL
1-2	3.1	5.9%
3-5	2.5	3.5%
6-11	1.9	2.0%

Source: CDC, 1997, Table 1.

Although the overall percentage of children with elevated BLLs has declined dramatically, about a million children under 6 still have BLLs greater than 10 µg/dL. Despite the national decline of elevated BLLs, localized exposure continues to be a problem, particularly for certain children in high-risk situations. The NHANES results indicate that lead levels are more likely to be elevated among younger rather than older children, and those that are poor, non-Hispanic blacks, living in large metropolitan areas or older housing[198]. In addition an estimated 2-3% of children with elevated BLLs are exposed to lead carried into the home from the workplace of adult caregivers[199]. The American Academy of Pediatrics supports the CDC guidelines for universal screening in certain areas and targeted screening for children at high risk[200].

California Data

From 1992 to 2001 in California 11,417 cases of childhood elevated BLLs were identified. These cases include children that are younger than 21 with at least one blood lead measurement equal to or greater than 20 µg/dL or 2 test measurements equal to or greater than 15 µg/dL. However, this case definition does not include all children with elevated BLLs greater than or equal to 10 µg/dL, which is CDC's level of concern. The number of children with BLLs greater than or equal to 10 µg/dL is estimated at 2 to 3 times the number of cases[201].

The actual number of children with BLLs greater than 10 µg/dL is difficult to determine because California's reporting regulations only require laboratories to report BLLs above 25 µg/dL or higher. Reporting of values between 10 µg/dL and 25 µg/dL is voluntary. The numbers of children with elevated BLLs are also likely to be underreported because of low screening rates. The best available numbers suggest that approximately 20% of California children at risk for lead poisoning are being screened[202].

There are numerous sources for lead exposure in California children. However, lead-contaminated paint was named as a source of lead exposure for over 60% of children with elevated BLLs during the period 1998-2000, where one or more sources of lead exposure were identified during the case investigation. Soil and dust exposures are also usually due to contamination of these sources by lead paint[203].

Health Effects

Lead provides no benefit to the human body; its effects are strictly detrimental. Due to extensive medical observation and scientific research over many years, the toxic effects of lead are well known. Some of these effects may occur at BLLs so low, there is no established clear threshold below which biochemical effects clearly do not occur. For this reason, U.S. EPA has not developed a reference dose (RfD) for inorganic lead[204] and CDC has not developed a minimal risk level (MRL) to humans. Instead, to reflect the considerable information regarding sometimes subtle effects at low BLLs, CDC developed a strategy for monitoring such effects. In 1991 CDC defined a blood lead concentration of 10 µg/dL in children as a level that triggers community prevention activities, education and follow-up. At 20 µg/dL medical and environmental evaluation are recommended and children with BLLs greater than or equal to 15 µg/dL should receive individual case management[205].

The U.S. Department of Labor, Occupational Health and Safety Administration (OSHA) also requires monitoring of BLLs among workers and specifies action when such levels exceed 40 µg/dL, whereas a limit of 30 µg/dL is recommended by the American Conference of Governmental Industrial Hygienists. International limits include a level of concern of 20 µg/dL, recommended by the World Health Organization, and a biological tolerance level for workers of 40 µg/dL, established in Germany[206].

While the most publicized effects of lead on humans are the neurotoxic and neurodevelopmental effects, lead also can cause adverse effects on most of the systems in the body, including the hematopoietic, renal, cardiovascular, gastrointestinal, and reproductive systems, as well as being tentatively identified as a carcinogen. In addition, while more advanced stages of lead poisoning may result in severe situations such as damage to the brain and kidney, severe anemia, spontaneous abortions and death, low level or early exposure to lead may produce nondescript symptoms such as fatigue, loss of appetite, reduced attention span, insomnia, and constipation[207]. The general nature of these symptoms often makes it difficult to identify lead as the culprit. At a blood lead concentration of 10 µg/dL, adverse effects include[208]:

- impairment of developing central nervous system and organs in fetuses,

- cognitive impairment and behavioral disorders in young children,
- blood pressure increases in adults, and
- impairment of calcium function and homeostasis in sensitive populations.

Figure 3-2 depicts some of the health effects associated with various BLLs. A summary of the results of research and studies involving health effects arising from lead exposure and associated internal lead doses is contained in Appendix 3-B. Most of the information regarding health effects in this section is derived from U.S. EPA's *Air Quality Criteria for Lead*[209] and the U.S. DHHS's *Toxicological Profile for Lead*[210] unless otherwise stated.

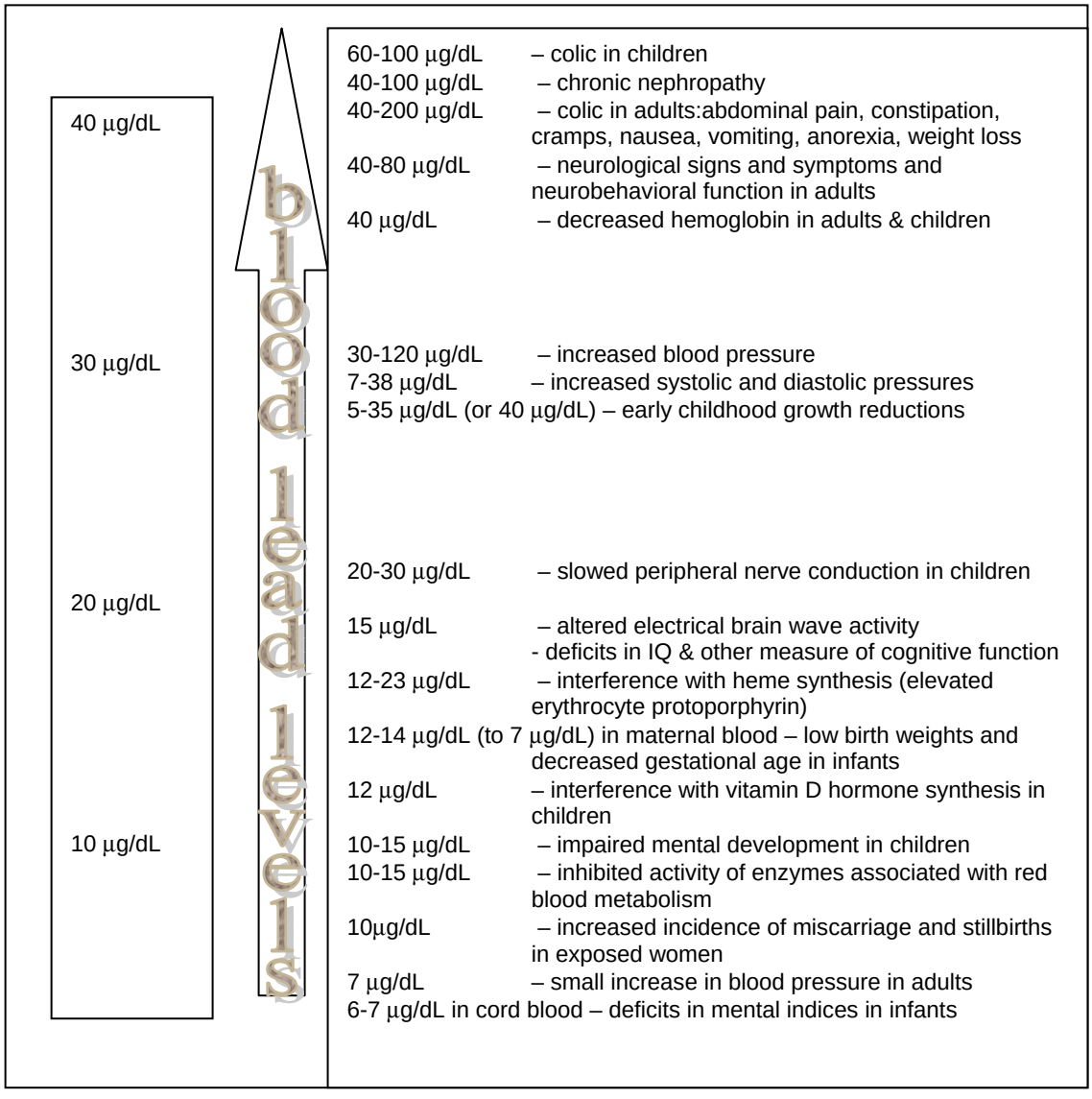

Figure 3-2 – Some of the relationships between blood lead levels and health effects
Source: 56 FR 26468

Inorganic lead is not metabolized by humans; rather it forms complexes, typically with proteins, and then is absorbed, distributed and excreted. Lead enters the bloodstream and is deposited and redistributed to other tissues in the body, eventually reaching a steady state when consistent exposure occurs over a period of time. Fluctuations in exposure can result in fluctuations in blood lead content of the blood, tissues and bone in the body. Distribution of inorganic lead in the body does not seem to be affected by the route of exposure or method of lead uptake. Once lead is absorbed, it is distributed primarily in one of three compartments: the blood and soft tissue (kidney, bone marrow, liver, and brain), which represent the active pool of lead in the body, and mineralizing tissue (teeth and bones), which serves primarily as the storage pool for lead in the body. Bones contain about 94% of the total body burden of lead in adults and 73% in children. For children under 10 years of age, the total body burden is estimated to be less than 0.1 mg/kg, increasing with age to levels ranging from 1.4-5.7 mg/kg in an older adult[211].

Estimates of the half-life of lead in blood, soft tissue and bones vary, depending on factors such as the size, type and location of the bones. For example, the distribution of lead in bone varies, particularly with age, since lead will tend to accumulate in those areas of bone undergoing active calcification at the time of exposure. In addition two compartments in bones have been indicated, inert and labile, accounting for the variability in the half-life of lead in bone. Because lead can form stable complexes with phosphate, lead can compete with calcium in the formation of the calcium-phosphate salt, hydroxyapatite, that forms the basic matrix structure for bones.

One study estimates the half-life of lead in the non-labile portion of the bones can be more than 20 years[212]. Another source cites the half-life of lead in bone as a range of 5 years to decades[213]. Lead in the labile bone compartment establishes a state of equilibrium for lead among the bones, blood and soft tissue compartments. Hence bone provides a source of endogenous lead exposure within the body, releasing lead into the bloodstream from the storage sites in the bones, sometimes long after an initial exposure. Release of lead from the bones to the other compartments can occur in response to triggers such as infection, pregnancy or a wasting disease, or with some bones, aging. Lead that has been stored in the dentin in teeth does not appear to move back into the blood stream; hence, lead in teeth can indicate cumulative exposure to the point the teeth are shed or tested.

As described in an earlier section, concentrations of lead in the blood (BLLs, PbB), measured as micrograms per deciliter (µg/dL), are often used as a measure of exposure. Because the half-life of lead in blood is about a month (reported in various sources as 28 to 36 days[214], approximately 25 to 28 days[215], and 35 days[216]), BLLs generally indicate recent exposure. Because blood can reabsorb lead from tissues and bones, however, when intermittent exposure occurs, blood lead concentrations represent both recent and past exposures. The half-life of lead in soft tissue falls between the half-life ranges for blood and bone, and is estimated at about 40 days by one source[217]. The liver and kidney take up lead, as well as the brain and muscle tissue, to a lesser extent.

Blood lead that is not retained is excreted primarily through the kidneys and gastrointestinal tract. Lead is also excreted in breast milk and can be taken up by nursing infants[218]. In adults most of the lead taken up by the body will be excreted. Up to 99% of lead in adults is excreted within weeks of exposure, unless the exposure persists, whereupon more lead will be accumulated. In young children, however, only about 32% of the lead taken up by the body will be excreted; the remainder is distributed in the blood, tissue and bones of the body[219].

Effects on Children

One of the segments of the U.S. population at greatest risk of lead exposure, and resulting lead poisoning, are children, typically those in older urban neighborhoods, where lead from lead-based paint may be present in paint chips, dust, and soils. Children with residential exposure are at much higher risk than adults in the same setting for several reasons. Since children are much more likely to play in dirt, place dusty or dirty hands or other objects in their mouths, and occasionally eat dirt, paint chips, or the plaster from walls, they have higher rates of intake of lead-contaminated media. Furthermore, as reported earlier, while adults absorb an estimated 5-15% of ingested inorganic lead, infants and young children typically absorb about 50%[220].

Absorption increases during fasting conditions and children are more likely than adults to ingest lead during activities not associated with food or eating. Also, adults excrete a much larger proportion of lead taken in compared to children. In addition, certain nutritional deficiencies such as calcium, iron or zinc deficiency, which tends to be more prevalent in children, also enhance the absorption of lead or its effects. Some of the health effects associated with lead occur at lower blood lead thresholds than adults. Finally, since children are growing more rapidly than adults, both physically and developmentally, they are more susceptible to the adverse biochemical effects of lead. Because lead exposure is implicated in effects on neurological function, the developing brains of children are thought to be particularly susceptible to the effects of lead.

Studies of children have associated lead exposure with decreased intelligence, reduced short-term memory, reading disabilities, and deficits in vocabulary, fine motor skills, reaction time, and hand-eye coordination[221]. Given the complexities of evaluating intelligence, and all the variables that impact intelligence and behavior, there is some controversy if slight to moderate levels of blood lead can affect, reversibly or not, children's neurobehavior and development. Generally, however, epidemiological studies indicate a statistical association between blood lead concentrations and IQ: children have shown IQ score deficits of about 5 points at BLLs of 50-70 μg/dL, 4 points at levels of 30-50 μg/dL, and 1-2 points at levels of 15-30 μg/dL[222]. One source estimates a 1-3 point decline in IQ for every 10 μg/dL increase in BLLs[223]. Although the mechanism of neurotoxicity is not well understood, the large number of studies in the U.S. and across Europe have resulted in highly consistent outcomes and dose-response relationships despite differences in populations.

In addition to neurobehavioral effects, lead can affect children's hematopoietic, renal, and gastrointestinal systems. Elevated BLLs can cause anemia in children: reduced heme synthesis is seen at BLLs of 40 μg/dL, while severe anemia can appear at concentrations of 70 μg/dL[224]. With regard to the renal system, acute nephropathy can occur during the early stages of excess lead exposure in children. Gastrointestinal effects, such as abdominal pain, constipation, cramps, nausea, vomiting, anorexia, and weight loss, may be seen at BLLs over 60 μg/dL[225].

With children, as with adults, no clear threshold has been established at which even the first symptoms of lead exposure can be detected and some adverse cognitive effects are noted at BLLs as low as 10 μg/dL. Until the early 1970s, the BLL limit for children was 60 μg/dL. As research into the effects of lead exposure, especially among children, expanded, and study methods improved, the recognized level for toxicity continued to decline to 30 μg/dL in 1978, 25 μg/dL in 1985, ultimately to the current goal of 10 μg/dL[226].

Fetuses are also susceptible to the effects of lead exposure since the placenta does not form a barrier to lead. Although cord blood lead concentrations are typically somewhat lower than maternal blood lead concentrations, such exposure can result in potentially adverse neurological effects *in utero*, as well as

post-natally. In 1984 an estimated 400,000 fetuses were exposed to blood lead concentrations likely to result in developmental effects[227].

Effects on Adults

Lead exposure to adults most commonly occurs in the workplace. Occupations that may result in lead exposure include the following[228]:

- mining, smelting and refining operations;
- battery manufacture and recycling;
- steel welding and spray coating;
- lead grinding or cutting;
- radiator repair;
- production of paint, ceramics and glazes, enamel, rubber and plastics;
- car refinishing and paint removal and abatement;
- plumbing and general construction;
- firearms training instructors, students, and firing range custodians.

From 0.5 to 1.5 million workers nationwide are exposed to occupational lead, more than 200,000 in California alone[229].

Workers' exposure most often results in damage to the central nervous system, cardiovascular system, reproductive system, hematological system, and the kidneys[230]. Nonspecific symptoms such as stomach pain, headaches, anxiety, irritability, and poor appetite may not be recognized as symptoms of lead poisoning. In addition some symptoms appear at relatively low lead levels. For example, adverse health effects such as hypertension, subclinical nervous system deficits and adverse reproductive outcomes have been reported at BLLs less than 40 μg/dL, prompting the U.S. DHHS to recommend that BLLs be reduced to 25 μg/dL for all adults[231].

Family members also can be exposed by lead dust carried home on the exposed worker's clothes, shoes, skin, hair or vehicle. This secondary type of exposure can be reduced or avoided if the workers wear uniforms or personal protective clothing that are left at the workplace and stored in special containers, separate from the workers' street clothes and shoes. Some employers provide showers for workers to use at the end of their shifts.

Women can be especially susceptible to lead exposure because some studies suggest pregnancy, lactation and osteoporosis may increase demineralization of bone, resulting in redistribution of lead from bone to blood and tissues. During pregnancy, it has been theorized that demineralization occurs to form the fetal skeleton. Lead absorption may also increase during pregnancy. Increased bone demineralization also occurs during aging. Because an increase in loss of bone lead is more pronounced in aging women than men, osteoporosis may account for this finding.

Effects in the Body

The effects of lead in the body occur primarily after lead is absorbed and generally do not vary with the route of exposure. Cellular disturbances caused by lead exposure affect most bodily systems, although the precise mechanisms of action for lead's effects in the body are often unknown. In general lead interferes with bodily processes by inhibiting or mimicking the activity of calcium and by combining with sulfhydryl groups on proteins, inhibiting enzymes involved in a number of pathways in the body, including enzymes required for the synthesis of hemoglobin, myoglobin, and cytochrome. For example, low concentrations of lead in blood inhibit the synthesis of hemoglobin, thereby shortening erythrocytes'

life spans. Lead also disrupts mitochondrial function in the nervous system and produces lesions in the kidney[232].

Most of the findings about the health effects associated with lead in humans arise from epidemiological studies, primarily from occupational exposures. In most of these studies lead exposure, and dose, is determined by BLL. More recently a number of studies focusing on children's exposure to lead, mostly in residential settings, also have been undertaken. Because it is difficult to control for confounding or interfering factors in such studies, conclusions from them must be developed and interpreted carefully. For example, in the past less was known about the health effects associated with low BLLs, so in older studies the BLLs in the control group may be considered elevated according to current standards. Supporting evidence, largely regarding the types of effects and mechanisms of action, has been gathered from numerous animal studies in which both blood lead concentrations and applied doses are typically known. Many of these human and animal studies have been replicated; results are compiled in Appendix 3-C.

As mentioned earlier, many symptoms of lead exposure are generalized and include loss of appetite, metallic taste, malaise, weakness, insomnia, vertigo, irritability, muscle and joint pain, tremors and colic. When such symptoms appear alone, the reasons for such ailments are difficult to determine and additional confirmation of lead exposure, such as a measurement of blood lead, is needed. Organ- and system-specific effects are discussed below.

Neurotoxicity

Lead neurotoxicity may be the best-documented effect of lead exposure, especially as a result of occupational exposure. A large number of studies of occupationally exposed adults report a variety of neurological ailments. Some studies suggest lead exposure may be responsible, even at low blood lead levels, for non-overt neurologic impairment, such as increased irritability and fatigue, decreased ability to process information, and store information in short term memory[233].

For example, at BLLs ranging from 40-120 µg/dL, resulting from acute, intermediate and chronic lead exposure, symptoms such as malaise, forgetfulness, irritability, lethargy, headache, fatigue, impotence, dizziness, weakness and paresthesia (burning or tingling in limbs) have been observed. Effects related to slower motor nerve conduction and memory problems have been noted among workers with BLLs >40 µg/dL. For instance, increases in work-related accidents, poor performance in work speed and dexterity tests and interpersonal conflicts have been observed, as well as verbal and visual impairments and increased rates of depression, confusion, anger and tension. These and other behavioral effects such as nervousness, moodiness and decline in coping mechanisms were observed at blood lead concentrations ranging from >40 µg/dL to 50-80 µg/dL. Postural balance also appears to be affected, particularly associated with cumulative lead exposure in the 2-9 years prior to testing.

At BLLs over 100 µg/dL, lead encephalopathy can appear, manifested initially as dullness, irritability, poor attention span, headache, muscular tremor, memory loss and hallucinations, eventually progressing to ataxia, convulsions, delirium, paralysis, coma, and death[234]. Although the severest symptoms of lead encephalopathy appear at high blood lead concentrations (460 µg/dL), signs of encephalopathy have been observed at lower blood lead levels, ranging from 50->300 µg/dL.

The symptoms of lead encephalopathy in children are similar to adults, and include hyperirritability, ataxia, convulsions, stupor, coma, and sometimes death. These symptoms are associated with BLLs of approximately 90-800 µg/dL, although some acute encephalopathy has been observed at lower BLLs of <70 µg/dL. These lower BLLs suggest children are more sensitive than adults to the neurological effects

of lead. The damage resulting from lead encepahlopathy is associated with permanent impairment of neurological and cognitive function.

Even asymptomatic children with elevated BLLs (ranging from 40-60 µg/dL to >70-200 µg/dL) showed a pattern of lower IQ, by about 5 IQ points, and cognitive deficits. Some studies have also documented IQ deficits at lower BLLs, associating a deficit of approximately 4 IQ points with tooth dentin levels that exceed 20-30 ppm (a blood lead concentration of roughly 30-50 µg/dL), and an estimate of a reduction of 9 IQ points with each 10 µg/dL increase in blood lead.

Lead exposure also seems to affect behavior in children resulting in inattention and hyperactivity. Some studies also suggest lead exposure can affect hearing in children. Finally, peripheral nerve function in children is also affected by lead exposure, with apparent peripheral neuropathy observed at BLLs of 60-136 µg/dL.

Hematological Effects

The effects of lead on heme in blood are widely documented. As stated earlier, lead interferes with the synthesis of heme by inhibiting enzymes involved in this biosynthesis, namely, -aminolevulinic acid dehydratase (ALAD) and ferrochelatase, resulting in increased erythrocyte protoprophyrin (EP), among other effects. This enzyme interference impairs production of hemoglobin and leads to the reduced life span of red blood cells. Most of the lead in blood is associated with erythrocytes and about half of the erythrocyte lead is bound to hemoglobin; lead appears to compete with calcium for a bonding site in the red blood cell.

Numerous studies have shown inhibition of the activity of ALAD at low blood lead concentrations in adults, children, and pregnant mothers and their newborns with no observed threshold. For example, this effect was observed over the blood lead range of 3-34 µg/dL. Decreased hemoglobin levels resulting from impaired heme synthesis is estimated to be associated with BLLs of about 50 µg/dL in occupationally exposed adults, and 40 µg/dL in children. Severe anemia appears to be correlated with BLLs of at least 80 µg/dL[235].

Reproductive Effects

There seems to be little argument regarding the effect of high levels of blood lead on reproductive health, in both males and females. High lead exposure can lead to sterility, miscarriages, and stillbirths, in women, along with neonatal morbidity and mortality from exposure of the fetus *in utero*. Some evidence also suggests that women exposed to high levels of lead as children may experience reproductive impairment later in life. Pregnant women are considered a sensitive population when discussing lead exposure because they serve as surrogates for the fetus, which is as sensitive to the impacts of lead exposure as children, if not more so. Levels as low as 10 µg/dL in umbilical cord blood are reported to adversely affect neurological development[236].

High lead exposure in men adversely affects fertility, semen quality, and appears to also lead to miscarriages and stillbirths in their spouses[237]. A number of studies reported decreases in sperm production, counts and motility, as well as dead and abnormal sperm, in workers with blood lead ranges of 40-98 µg/dL.

Renal and Hepatic Effects

Well-documented adverse effects of lead on the kidney result in generalized kidney disease as a result of disruption of the function of the tubular structures[238]. Nephropathy resulting from early or acute lead exposure includes outcomes such as nuclear inclusion bodies, alteration of mitochondria, enlarged

proximal tubular epithelial cells, Fanconi's syndrome (proximal tubule defects causing urinary changes such as aminoaciduria and glucosuria) and other urinary changes, namely, increased sodium and decreased uric acid excretion. These defects seem to be reversible as lead exposure declines.

Chronic nephropathy results in typically irreversible symptoms including progressive interstitial fibrosis, tubule dilation and defects in the tubular epithelial cells, reduction in glomerular filtration and azotemia (accumulation of waste products in blood). Lead exposure also appears to cause kidney disease associated with gout and essential hypertension. Although low-level lead exposure seems to affect renal function (tubular damage was observed at BLLs of <40 μg/dL), more definitive symptoms such as aminoacuria occur at higher blood lead concentrations (35-80 μg/dL in children) with full Fanconi syndrome occurring in some children with lead encephalopathy (150 μg/dL).

There is also some evidence that lead exposure affects liver function. For example, lead inhibits formation of the heme-containing protein cytochrome P-450 resulting in reduced activity of hepatic oxygenases. Lead also affects vitamin D metabolism, a process that takes place within the liver and kidney.

Growth and Development

Growth in children of all ages, can also be inhibited by lead exposure, even among infants exposed *in utero*. Analyses of NHANES data and other studies have shown that BLLs are a statistically significant predictor of a child's height, weight and chest circumference at concentrations ranging from 4-35 μg/dL. The relationship between BLLs and height is especially strong, even at the lowest concentrations, again, suggesting no threshold for this effect.

Even low levels of prenatal lead exposure are associated with developmental effects such as reduced birth weight, reduced gestational age and mental development and other neurobehavioral deficits. These effects were observed at maternal BLLs of about 14-21 μg/dL and cord blood levels of approximately 12-17 μg/dL. Although increased BLLs were also associated with minor congenital anomalies, such as hemangiomas, lymphangiomas and undescended testicles, no connection was observed between lead levels and major congenital problems.

Gastrointestinal Effects

While many of the gastrointestinal symptoms resulting from lead exposure can be vague, particularly at low lead levels, early or acute exposure consistently results in colic, characterized by:

- abdominal pain
- constipation
- cramps
- nausea
- vomiting
- anorexia
- weight loss

Colic is commonly associated with high BLLs of from 100-200 μg/dL in adults, but has also been observed at BLLs of 40-60 μg/dL. In children colic is associated with BLLs of 60-100 μg/dL.

Cardiovascular Effects

Although a number of studies have observed an association between lead exposure and hypertension, the data is insufficient to establish a conclusive causal relationship. These studies include both occupational exposure and exposure of the general population, and some analyses indicate a weak association between

blood lead and blood pressure, particularly for men and children. Other observed cardiac effects include degenerative changes in heart muscle, ECG abnormalities and an association between lead levels in the aorta heart-related deaths.

Carcinogenicity

The International Agency for Research on Cancer (IARC) has classified lead and inorganic lead compounds as possible carcinogens, based on sufficient animal evidence, although the human evidence is inadequate[239]. U.S. EPA classified lead as a probable human carcinogen (B2), based on the same sufficient animal evidence of renal tumors associated with dietary and subcutaneous exposure of soluble lead salts, primarily acetates and phosphates[240]. The human carcinogenicity data is considered inadequate; two of four epidemiological studies did not find any correlation between lead exposure and cancer mortality. Furthermore, none of the studies contained quantitative exposure information, data regarding the potential contribution of smoking, or controls for simultaneous exposure to other metals, such as arsenic, cadmium and zinc[241]. Some recent studies suggest that occupational lead exposure increases the risk of cancer[242].

Effects on Wildlife and Plants

Lead and Plant Toxicity

As discussed above, plants are exposed to lead through surface deposition, from rainfall, dust, and soil, and through their roots. Lead absorption and transport through the plant varies widely among species, and between different breeds of the same species. Generally, plants more readily absorb lead from soils with low pH or low organic content. The interaction of lead with other metals in the soil is fairly complex and will affect the uptake of lead by different plants.

As with humans and animals, lead appears to have no value to plant life. Lead inhibits plant growth, reduces photosynthesis, and reduces mitosis and water absorption. Lead apparently inhibits photosynthesis by blocking protein sulfhydryl groups and affecting phosphate levels in living cells[243].

In 1994, the Oak Ridge National Laboratory recommended that "to be considered unlikely to represent an ecological risk to terrestrial plants …", lead in soil should not exceed 50 mg/kg. This study also concluded that minimum soil concentrations causing phytotoxicity were between 100 and 400 mg/kg[244].

Lead and Animal Toxicity

In addition to human receptors, a variety of domestic and wild animals may be at risk from exposure to excessive concentrations of lead. These include fowl that ingest, either directly or indirectly, lead shot left by hunters; domestic livestock near smelters, refineries, and battery reclamation facilities; zoo animals and domestic livestock held in enclosures coated with lead-based paints; wildlife that forage near heavily traveled roads; aquatic life near mine drainage areas and waters polluted by atmospheric lead fall-out; and invertebrates living in lead-contaminated soils, among others.

There appears to be a widely varied response among aquatic species to lead exposure. Among sensitive species, the following observations were made: dissolved waterborne lead was more toxic than total lead and organic lead compounds were more toxic than inorganic compounds. The effects were most pronounced in warmer water, with low pH, in relatively soft waters, and in younger developmental stages and after prolonged exposure[245].

Similar studies of amphibians and reptiles also revealed the drastic effects of lead poisoning. In adult leopard frogs, lead poisoning produced sloughing of the outer skin; sluggishness; decreased muscle tone,

decreases in red blood cells, white blood cells, neutrophils, and monocytes; erosion of the stomach lining; and, just before death, excitement, salivation, and muscle twitching. Lethal and sub-lethal effects were noted in bullfrogs, salamanders, and tadpoles at concentrations of 0.5 to 1.5 mg/L[246].

In birds, the most severe instances of lead poisoning have been observed in wild fowl ingesting shot scattered from hunters' rifles. Over one million ducks and geese die annually from lead poisoning produced by the ingestion of shotgun pellets. The lead solubilizes in the highly acidic gizzards of the fowl and results in damage to the nervous system; muscular paralysis, inhibition of heme synthesis, and damage to the kidneys and liver[247].

With mammals, as with other wildlife, there is a significantly different response to lead exposure between different species. Generally, however, the effects of lead poisoning are more pronounced with organolead compounds than with inorganic forms, the younger developmental stages are the most sensitive, and the effects of the lead are exacerbated by elevated temperatures and by diets deficient in minerals, fats, and proteins. It should be noted that data on the effects of lead on mammalian wildlife are scarce, and that most of the available information comes from observed effects on laboratory animals or domestic livestock. These observations reveal that, in severe lead poisoning, death is preceded by impairment of the central nervous system, gastrointestinal tract, and muscular and hematopoetic systems. Signs of poisoning include vomiting, lassitude, loss of appetite, uncoordinated body movement, convulsions, and stupor. Non-fatal symptoms include depression, anorexia, colic, disturbed sleep patterns, diarrhea, anemia, visual impairment, blindness, renal malfunction, impaired peripheral nervous control, reduced growth, and learning impairment[248].

With regard to the correlation of soil concentrations of lead and risk to wildlife, the U.S. Bureau of Land Management (BLM) developed the following risk management criteria for western U.S. BLM lands, for different species:

deer/mouse: 302 mg/kg,
rabbit: 44 mg/kg,
bighorn sheep: 425 mg/kg,
white-tailed deer: 354 mg/kg,
mule deer: 438 mg/kg,
elk: 361 mg/kg,
mallard: 152 mg/kg,
Canada goose: 55 mg/kg, and
trumpeter swan: 59 mg/kg.

By contrast, the criterion for people camping in the same lands was set at 1,000 mg/kg[249].

CHAPTER 4 – LEAD IN LAWS AND REGULATIONS

For as long as lead has been produced, humans have experienced the health effects associated with lead exposure. Even some of the earliest uses of lead inspired observations of lead's poisonous nature. Vitruvius in ancient Rome noted: ". . . it seems that water should not be brought in lead pipes if we desire to have it wholesome."[250] Despite these concerns, Romans continued lead production and it continues to this day.

As lead production expanded in modern times, awareness of the extent of lead emissions and the associated health effects resulted in restrictions on some of the uses of lead and limitations on lead emissions. Reflecting the ubiquity of lead's uses and presence in the environment, the agencies and resulting requirements applicable to lead are numerous and varied. Many laws and regulations have been developed in the U.S., particularly since the 1970s, to address lead emissions and prevent adverse effects from exposure to those emissions. More recently, research has been focused on the mechanisms of lead's health effects and the growing realization of children's susceptibility to lead. As a result, some laws, regulations and standards have been updated to reflect concern over childhood lead exposure.

Standards Applicable to Lead

A number of statutory and regulatory standards applicable to lead have been promulgated. These standards apply to different media and have been developed to provide protection from exposure to emissions of lead and other forms of lead in the environment, often by limiting emissions of lead to the environment. These standards include regulatory threshold concentrations used to determine if a waste is hazardous and concentrations used for cleanup levels and site screening. Table 4-1 summarizes the standards applicable in California and Table 4-2 summarizes federal standards. Some of these standards are described in more detail below.

Regulatory Thresholds

Hazardous Waste

A waste that has been determined to be hazardous is subject to a variety of requirements relevant to the waste's management, including, but not limited to treatment, storage, transportation, disposal, and associated permitting and recordkeeping provisions. In California a waste is considered hazardous if it meets any of the following conditions:

- it meets the statutory definition of a hazardous waste,
- it is classified as a listed or characteristic hazardous waste according to the federal criteria,
- it exhibits any of the criteria of a hazardous waste in California, including toxicity, reactivity, ignitability or corrosivity.

Because the Department of Toxic Substances Control (DTSC) is authorized to implement the federal Resource Conservation and Recovery Act (RCRA) requirements in California, the state's hazardous waste provisions include federal regulations in addition to state requirements. The hazardous waste criteria are a good example of a provision that incorporates both state and federal requirements. Among the tests for determining if a waste is hazardous in California are the two thresholds established by the state's regulations: the Soluble Threshold Limit Concentration (STLC) and the Total Threshold Limit Concentration (TTLC). In addition, the state's regulations specify consideration of the federal criteria, including the hazardous waste listings and the federal threshold for wastes considered hazardous due to hazardous characteristics, as determined by the Toxicity Characteristic Leaching Procedure.

Table 4-1 – Overview of Standards, Regulatory levels, and Guidance Levels for Lead – California

Regulatory Level	Reference and Authority	Toxicological Basis, Routes of Exposure and Pathways	Models, Assumptions and Parameters	Comments
Total Threshold Limit Concentration, TTLC				
1,000 mg of lead/Kg of waste	Ref: 22 CCR 66261.23(a)(2) Auth: H&SC, 25141 & 25159	Basis: STLC of 5 mg/L Route: Ingestion Pathway: Direct contact	Assumptions: 100- fold uncertainty factor and children should not be exposed to soil-lead concentrations above 1,000 mg/kg	TTLC = 100 x STLC, adjusted to coincide with 1,000 mg/kg threshold for child exposure
Soluble Threshold Limit Concentration, STLC				
5 mg of lead/L of waste extract solution (using Waste Extraction Test)	Ref: 22 CCR 66261.23(a)(2) Auth: H&SC, Sections 25141 & 25159	Basis: MCL of 50 µg /L Route: Ingestion of drinking water Pathway: Drinking water derived from groundwater or surface water	Models: Battelle (1976) and U.S. EPA (1980) Assumptions: 100-fold attenuation factor as reported by Battelle (1976) and U.S. EPA (1980)	STLC = 100 x MCL
Statutory Limit:				
350 mg of lead/Kg of waste	H&SC, Section 25157.8	Unknown	Unknown	A waste that meets the criterion specified in this statute but is not otherwise a California hazardous waste is required to be disposed of in a Class I (hazardous waste) landfill. However, some landfill operators have balked at accepting such waste, as it is technically not a hazardous waste. In addition, manifesting of such waste does not appear necessary for the same reason.
School Sites Initial Screening Value, Proposed:				
255 mg/Kg, corresponding to the 99th percentile		Basis: Threshold blood lead concentration of 10-µg/dL Routes: Ingestion, inhalation and dermal contact Pathways: Dietary intake, drinking water, soil and dust ingestion, inhalation, and dermal contact	Models: LeadSpread, Version 7 Assumptions/Parameters: Regional and statewide air concentrations of lead; 15 µg lead/L in drinking water	Exposure scenario assumes exposure to children at school sites developed on "formerly residential or uncontaminated commercial properties."
Accreditation, Certification, and Work Practices for Lead-Based Paint and Lead Hazards				
400 ppm in children's play areas;1000 ppm in other areas	Ref: 17 CCR 35036 Auth: H&SC sections 105250(a)&(b), 124160(b), 124165	unknown	unknown	Definition of Lead-Contaminated Soil

Table 4-2 – Overview of Standards, Regulatory levels, and Guidance Levels for Lead – Federal

Regulatory Level	Reference and Authority	Toxicological Basis, Routes of Exposure and Pathways	Models, Assumptions and Parameters	Comments
Toxicity Characteristic for D008:				
5 mg/l (using the Toxicity Characteristic Leaching Procedure)	40 CFR Section 261.24	Basis: MCL of 50 µg /L in drinking water. Route: Ingestion of drinking water Pathway: Drinking water derived from groundwater or surface water	Models: Battelle (1976) and U.S. EPA (1980) Assumptions: 100-fold dilution attenuation factor	Threshold = DAF X MCL
Toxic Substances Control Act (TSCA) Values:				
400 ppm of lead in bare soil in children's play areas or 1200 ppm average for bare soil in the rest of the yard	40 CFR Section 745.65	Basis: The level at which 5% of potentially exposed children will have blood lead concentrations in excess of 10 µg/dL Routes: Inhalation and ingestion Pathways: Air, drinking water, food (including homegrown food), soil, and dust	Models: IEUBK model with default parameters and guidance manual for soil concentrations	The purpose of this standard is to provide residential dust and soil threshold values for lead above which children may be at risk of having elevated blood lead levels.
CERCLA Sites and RCRA Corrective Action Screening Levels:				
400 ppm for lead in residential soil	Guidance only	Basis: Blood lead concentration of 10-µg/dL Routes: Ingestion and inhalation	Model: IEUBK model Assumptions: Use site-specific information, if available. Takes into account multiple environmental sources of lead.	The exposure scenario assumes exposure of children to lead from various environmental media.

A waste exhibits the characteristic of toxicity for lead if, among other criteria, it contains concentrations of lead that exceed the regulatory threshold concentrations, listed below:

Threshold	Concentration
Total Threshold Limit Concentration (TTLC)	1,000 mg/kg
Soluble Threshold Limit Concentration (STLC)	5.0 mg/L
Toxicity Characteristic for D008 (as determined by the Toxicity Characteristic Leaching Procedure)	5.0 mg/L

The STLCs and TTLCs, originally proposed in 1978, were intended to identify those wastes that "pose a substantial threat to human health and the environment if not disposed in a controlled and systematic manner."[251] As depicted in Table 4-1, the STLC for lead focused on ingestion of drinking water as the route of exposure, considering the exposure pathway of drinking water derived from groundwater or surface water. The STLC was developed by applying a 100-fold attenuation factor to the maximum contaminant level (MCL) for lead, which was 50 μg of lead/L of drinking water at the time the threshold was developed.

The TTLC for lead was based upon the STLC value for lead of 5 mg/L multiplied by an uncertainty factor of 200. For most of the TTLCs promulgated, an uncertainty factor of 100 was imposed "to take into account the likelihood that not all of a particulate persistent and bioaccumulative toxic substances [sic] placed in the environment would be available for ingestion or uptake by organisms." According to this procedure, the resulting TTLC for lead would have been 500 mg/kg. This value, however, was increased to 1,000 mg/kg based upon a report produced by the Department of Health Services in 1979 suggesting that concentrations above 1,000 mg/kg should be unacceptable due to potential for ingestion by children exhibiting pica behavior[252].

Another source asserts that the initial TTLC of 500 mg/kg for lead was considered too low when compared with mean western soil lead levels ranging from 7 to 700 mg/kg, and with British data for soil criteria, which defined soil with greater than or equal to 1,000 mg/kg of lead as unacceptable in areas where children may consume soil[253]. Both reasons for adjusting the TTLC, however, cite as a rationale for doubling the calculated TTLC the 1,000 mg/kg figure as a level that would be protective for children ingesting lead-contaminated soil by children.

A summary of more recent research recommending soil lead standards is depicted in Table 4-3, accompanied by the basis for the recommendations[254]. The recommended values that are based on protection of children with pica, namely, <100 ppm and <150 ppm, are far below the value of 1,000 mg/kg intended to protect children with pica, which was used to adjust the original calculation of the TTLC.

Table 4-3 – Soil Lead Standard Recommendations

Standard (ppm)	Comments
<100	Protect pica children
<150	Prevent lead toxicity (10 µg /dL)
<150	Protect pica children
300	Keep acceptable daily intake (ADI) <50 µg Pb/day (street dust standard)
300	Keep blood lead level below 25 µg /dL
500	Keep blood lead level below 25 µg /dL
600	Permit an increase in blood lead of 5 µg /dL above existing levels
250	Protect children where there is no grass cover
900	Based upon an ADI of 200 µg Pb/day
1,000	Allows dust to contribute 2.5 – 3.0 µg /dL (housedust) above existing levels

Source: DHHS, 1992

As shown in Table 4-2, the federal hazardous waste criterion for determining if a waste is considered to exhibit the characteristic of toxicity for lead is measured using an extraction test known as the Toxicity Characteristic Leaching Procedure (TCLP). This test is analogous to the test used to determine if a waste exceeds the STLC. California's regulations require that both the state and federal thresholds must be considered, in addition to the other criteria and listings, when characterizing a waste. The ensuing regulatory requirements can be different for wastes that are hazardous according to the federal criteria, known as RCRA wastes, and those wastes that are hazardous according to the state's criteria, known as non-RCRA wastes.

In some instances wastes that are considered hazardous according to the federal criteria are exempted or excluded from federal regulations. These otherwise RCRA wastes usually are regulated as non-RCRA wastes if California has not adopted the same exclusions or exemptions. For example, residential lead-based paint waste typically exhibits the toxicity characteristic for lead according to state and federal criteria. U.S. EPA, however, determined that the federal household hazardous waste exclusion applies to these wastes[255], thereby allowing such wastes to be disposed as non-hazardous wastes.

California's statutory and regulatory requirements for household hazardous wastes are different from the federal regulations, so RCRA wastes subject to the federal exclusion are considered non-RCRA wastes according to the state's requirements[256]. In California, authority for implementing regulations that establish alternative management standards for lead-painted wood debris that is a hazardous waste was recently restored[257]. Alternative management standards for this waste stream could provide disposal options similar to those provided by the federal exclusion, and, in addition, allow DTSC to maintain recordkeeping and tracking abilities that ensure appropriate disposal of the wastes. The federal exclusion does not allow U.S. EPA to retain hazardous waste authority for the household hazardous waste, leaving U.S. EPA to "encourage" handlers of the waste to follow safe handling measures.

Because the regulatory requirements differ, often dramatically, depending upon the classification of a waste, it is important to accurately assess the waste. In the case of lead-containing waste, this typically involves evaluating the total and soluble concentrations of lead in the waste. Because California's regulations allow a generator to use knowledge regarding the waste to determine its classification, various rules of thumb have evolved to allow a generator to enhance that knowledge using assumptions.

One of those assumptions is that the relationship between the total and soluble concentrations, using the waste extraction test (WET), differs by a factor of ten; that is, if the total lead concentration does not exceed 50 mg/kg, the soluble concentration is not likely to exceed 5 mg/L (using the WET). Comparisons by DTSC's Hazardous Materials Laboratory reveals the factor to be more in the neighborhood of thirty, but the actual relationship between total and soluble concentrations cannot be assumed since it depends upon many factors, including the form of the lead or lead compounds undergoing testing and other constituents that may be present during the test. Such assumptions are not always reliable and should be confirmed for individual situations before being used.

That rules of thumb are not reliable can be amply illustrated by the example of old DTSC guidance for lead painted building debris[258]. This information, contained in a guidance document that DTSC rescinded in 2001, stated that DTSC did not generally expect intact painted building material to exhibit a characteristic of a hazardous waste. This expectation was based on assumptions derived from the demolition of large, mostly concrete structures, in which the concentration of lead in a representative sample of the large amount of demolition debris was found to be small. While this outcome has been confirmed for some situations similar to the original assumptions, it is typically not true for wood debris containing lead-based paint and cannot even be generalized for concrete structures since the results vary widely depending upon maintenance activities. Although DTSC's old information specified that the waste classification should consider the ratio of the mass of all materials in the waste to the lead content of the paint (or more clearly, one should take a representative sample of the waste under consideration), some users erroneously interpreted the guidance to mean that building debris with lead-based paint was not hazardous.

Another threshold that is significant to the management of waste containing lead is a statutory requirement. This provision requires that any waste with a total lead concentration that exceeds 350 ppm must be disposed in a hazardous waste disposal facility[259]. This requirement does not affect the classification of hazardous waste containing lead. Wastes that are not otherwise classified as hazardous, but that exceed this threshold, must still be disposed to hazardous waste disposal facilities.

Cleanup and Screening Levels

For most site remediation activities, remediation goals or cleanup levels are typically determined on a site-specific basis using a health-based analysis that takes many factors into account, including, but not limited to the characteristics of the site, the contaminant(s) of concern and the expected receptors. In some instances, however, general cleanup goals or site-screening levels are developed using health-based models that assume conservative scenarios to determine the model parameter values. Some of the cleanup and screening levels summarized in Tables 4-1 and 4-2 are described below.

School Site Initial Cleanup Value, Proposed

Under specified conditions, state law requires a school district to "enter into agreement with DTSC to oversee response action if a preliminary endangerment assessment discloses the presence of a hazardous material release, or threatened release, or the presence of naturally occurring hazardous materials at a proposed school site at concentrations that could pose a significant risk to children or adults". Although the law does not require screening values for hazardous substances, at school sites, DTSC has proposed a screening value. This value of 255 mg/kg represents soil lead levels that would result in 1% of children exposed to the soil having BLLs exceeding 10 μg/dL, given specific assumptions about the exposure scenario and the relationship of direct exposure to blood lead concentrations.

The soil lead screening value was developed using DTSC's LeadSpread model (Version 7) and assumes exposure of children at school sites developed on formerly residential or uncontaminated commercial properties. The routes of exposure considered are ingestion, via dietary intake, drinking water and soil and dust ingestion, inhalation and dermal contact. Regional and statewide air concentrations of lead were used, as well as a value of 15 µg lead/L in drinking water. The homegrown produce pathway was not considered.

Lead Paint in Soil

As part of the many federal initiatives to control exposure to lead-based paint, U.S. EPA established residential dust and soil threshold values for lead above which children may be at risk of having elevated BLLs. These values specify interior loadings of 40 µg lead in dust per square foot on floors and 250 µg lead in dust per square foot on interior window sills. The exterior concentration standards are 400 ppm of lead in bare soil in children's play areas or 1200 ppm average for bare soil in the rest of the yard[260]. These concentrations use the Integrated Exposure Uptake Biokinetic Model (IEUBK) and assume a threshold at which 5% of exposed children will have BLLs in excess of 10 µg/dL, considering exposure via inhalation of air and ingestion of drinking water, food (including homegrown food), soil and dust.

California's Department of Health Services, Childhood Lead Poisoning Prevention Branch has developed regulations that implement accreditation, certification and work practices for lead-based paint and lead hazards[261]. These regulations define lead hazards, including lead contaminated-soil and lead-contaminated dust. Lead-contaminated soil is bare soil with lead in excess of 400 ppm in children's play areas, and 1,000 ppm in all other areas. Lead-contaminated dust is defined as dust in excess of the amounts listed below:

Location	concentration
interior floor surfaces	50 µg/ft^2
interior horizontal window surfaces	250 µg/ft^2
exterior floor	800 µg/ft^2
exterior horizontal window surfaces	800 µg/ft^2

CERCLA Sites and RCRA Corrective Action Screening Levels

U.S. EPA developed a screening level for lead in residential soil of 400 ppm. This screening level was defined as a level of contamination above which there may be enough concern to warrant site-specific study of risks. U.S. EPA stated that "…generally, OSWER will attempt to limit exposure to soil lead levels that a typical (or hypothetical) child or group of similarly exposed children would have an estimated risk of no more than 5% of exceeding a 10 µg/dL BLL."[262]

Recent research indicates that soil lead standards in other countries are consistent with the schools value and residential lead paint soil values. Examples of soil lead standards from a variety of locations are depicted in Table 4-4.

Table 4-4 – Soil Lead Standards for Residential Land Use

Location	Standard (ppm)	Comments
U.S.	500	600 µg /g repealed, 500 µg /g interim standard
Minnesota	500	Proposed emergency rule, interim 1,000 µg /g standard
OME, Canada	375	Sandy soil
	500	Non-sandy soil
Netherlands	50	Reference value
	150	Further investigation
	600	Clean-up value
England	500	Redevelopment of industrial lands
London	500	Dust standard

Source: U.S. DHHS, 1992

Some additional international cleanup goals include the following values[263]:

- proposed European Economic Commission standards for Maximum Allowable Concentrations in soils treated with sewage sludge: 50 (100) ppm dry weight (the value in parentheses is mandatory)
- clean-up levels in Holland, Japan, Quebec: 600 ppm
- Ontario (Canada) clean-up levels: 500 ppm for residential and park lands; 1,000 ppm for commercial and industrial lands.

Requirements Applicable to Lead

Environmental Laws

A variety of requirements address releases and exposures associated with water, air, wastes and other environmental media.

Drinking water

The California Safe Drinking Water Act of 1995 (and amended in 1996 and 1997) established the Drinking Water Regulatory Program within DHS. The legislative intent was for the program to provide the orderly and efficient delivery of safe drinking water within the state and to give the establishment of drinking water standards and public health goals greater emphasis and visibility within DHS[264].

Under federal environmental laws, the Safe Drinking Water Act is found in 42 U.S.C.A. sections 300(f) et seq. In passing the Safe Drinking Act in California, the Legislature's intent was to establish primary drinking water standards at least as stringent as those under the federal act. These standards are found in the CCR/T22, chapter 17.5, "Lead and Copper", beginning with section 64670.

U.S. EPA considers drinking water lead concentrations at or above 15 parts lead per billion parts water (ppb) to be unsafe and recommends that such outlets be removed from service immediately until the level of lead contamination is reduced to below 15 ppb. In 1986, the Safe Drinking Water Act of 1974 was amended to ban all future use of lead pipe and lead solder.

In 1987, U.S. EPA identified refrigerated water fountains as another potential source of lead contamination. The Lead Contamination and Control Act was approved in 1988 to address this concern. The Act required that water coolers with lead-lined tanks be repaired or removed; banned the manufacture and sale of water coolers that were not lead free, and required that lead problems in schools' drinking water be identified and resolved. To comply with the Act, the California Department of Education issued three advisories in December 1987, March 1988, and June 1989 to alert school superintendents to the potential of lead in refrigerated water fountains. The advisories described methods for testing drinking water and for reducing the threat of lead contamination.

DTSC's primary activities related to drinking water source assessment and protection are included in two programs mandated by federal law.

- Under TSCA, U.S. EPA regulates the treatment, disposal (including incineration, landfill, alternative technology), and storage of hazardous chemical substances.
- The RCRA Program has been delegated to DTSC. This program regulates the treatment, transportation, storage and disposal of hazardous waste.

The Office of Environmental Health Hazard Assessment's (OEHHA) functions and responsibilities related to drinking water source assessment and protection include developing health-protective exposure standards for different media (air, water, land) to recommend to regulatory agencies, including drinking water chemical contaminant standards for DHS. OEHHA's Water Toxicology Unit performs major risk assessment and hazard evaluations relating to chemical contaminants in drinking water. These activities include developing health advisories, action levels, proposed MCLs and public health goals for chemical substances, additives, and pollutants in drinking water and on chemical monitoring activities for the drinking water supply. The program also provides education to the public and other governmental agencies regarding drinking water contamination and regulatory standards development.

OEHHA is also responsible for implementing the Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65). This initiative statute prohibits businesses from discharging into drinking water sources chemicals identified by the state to cause cancer or reproductive toxicity, which includes lead. It also requires warnings to be provided whenever exposures to those chemicals are anticipated to occur[265].

Federal water programs are administered primarily by U.S. EPA. The U.S. Army Corps of Engineers, U.S. Bureau of Reclamation, U.S. Department of Agriculture (USDA) and other federal agencies play complementary roles. USGS principally compiles information that assists others in their water protection efforts.

Several federal programs related to drinking water source assessment and protection are administered by U.S. EPA. The primary purpose of the Safe Drinking Water Act (SDWA) is to ensure the safety of drinking water served to the public. The SDWA includes the Wellhead Protection Program, the Sole Source Aquifer Program, and the Underground Injection Control Program. (Congress passed the SDWA in 1974 and it was amended in 1986 and 1996.)

Other federal environmental laws to protect water supplies include, but are not limited to: the Clean Water Act (CWA), which ensures protection of surface waters designated, in part, for use as drinking water; RCRA; CERCLA, otherwise known as "Superfund"; and the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). These laws provide authorities, financial support, and technical assistance to protect sources of drinking water, especially ground water.

Ground and Surface Water

A number of state agencies, boards, departments, and offices share responsibility with federal and local agencies for ground and surface water protection in California.

The State Water Resources Control Board (SWRCB) formulates and adopts the State's policy for water quality control, assisting and overseeing the nine Regional Water Quality Control Boards (Regional Water Boards), and, in conjunction with the courts, administers California's system of water rights (California Code of Regulations, Title 23). With SWRCB approval the Regional Water Boards formulate, adopt, and implement water quality control policies and plans within their jurisdictions. Collectively, the nine regions cover all of California. Each Regional Water Board designates beneficial uses of surface and ground water resources and establishes water quality objectives to reasonably protect existing and potential beneficial uses of water resources in its region, as well as implements programs to achieve compliance with the water quality objectives. Beneficial uses, water quality objectives, and the implementation program are specified in each region's Water Quality Control Plan, as called for in the California Water Code, section 13240.

Regional Water Board activities related to drinking water source assessment and protection include:

- Basin Planning: Each Regional Water Board has adopted one or more Water Quality Control Plans (Basin Plans) for its jurisdiction, which is based upon surface water hydrologic basin boundaries. The Basin Plans identify existing and potential beneficial uses of marine, ground, and surface waters; establish water quality objectives to protect the beneficial uses; specify implementation programs to achieve these objectives; and describe surveillance and monitoring activities to evaluate the effectiveness of the water quality program. Basin Plans contain standards for surface water and ground water quality that are independently established by each Regional Water Board as water quality objectives necessary to protect the identified beneficial uses. Thus, there are differences both among and within Regions, depending upon the particular ground water basin and the assigned beneficial uses.

- National Pollutant Discharge Elimination System (NPDES) and Waste Discharge Requirements: Under the authority of the federal Clean Water Act (CWA), the NPDES program regulates point source discharges to surface waters such as wetlands, lakes, rivers, estuaries, bays and oceans. In California, the Porter-Cologne Water Quality Control Act regulates any discharge of waste that may affect water quality in California[266].
- Waste Discharges to Land: SWRCB has adopted regulations that implement provisions of the Porter-Cologne Act[267]. These regulations apply to all hazardous and non-hazardous wastes discharged to land, including surface impoundments. The chapter 15 regulations prescribe siting standards, construction standards, ground water and vadose zone monitoring requirements, and closure and post-closure procedures and requirements. Protecting ground and surface water from the migration of contaminants from solid waste disposal facilities is the responsibility of SWRCB and Regional Water Boards, which, thereby, require all solid waste disposal facilities to conform to waste discharge requirements adopted by a Regional Water Board.
- Hazardous Waste Facility Monitoring: Under a Memorandum of Agreement with DTSC, SWRCB and the Regional Water Boards carry out a ground water monitoring and surveillance program, perform water quality-related review work, and develop regulations, standards, and guidelines pursuant to RCRA.
- Non-Point Source Pollution: The CWA was amended in 1987 to include Section 319, which required the states to develop and implement non-point source management programs. SWRCB subsequently adopted a "Non-point Source Management Plan" in 1988, and by early 1990, had organized a multi-faceted, surface and ground water, non-point source program which focused on agriculture, mining, urban runoff, construction, and pesticides. The non-point source program seeks to reduce or eliminate surface and ground water pollution through the implementation of management measures to control non-point source pollution at its source.
- Coastal Zone Act Reauthorization Amendment (CZARA) of 1990: The two primary federal statutes that establish a framework for addressing non-point source (NPS) pollution are the CWA section 319 (1987) and CZARA section 6217. Together, they encourage states to assess water quality problems associated with NPS pollution and to develop programs to control NPS sources of pollution. CWA section 319 requires that states develop an assessment report and a management program specifying NPS controls. CZARA section 6217(a) requires states to establish coastal NPS programs to develop and implement management measures for NPS pollution to restore and protect coastal waters. California received $5.4 million of federal funding under the CWA in 1997 to carry out its NPS program.

  In 1988, SWRCB adopted the California NPS Management Plan that outlined a three-tiered approach for address polluted runoff: (1) voluntary implementation of Best Management Practices (BMPs), (2) regulatory-based encouragement of BMPs, and (3) effluent limitations. In response to CZARA section 6217, SWRCB, the Regional Water Boards, and the California Coastal Commission initiated a joint effort to improve the statewide NPS program and comply with CZARA requirements. As a result, California is working to enhance its statewide NPS program by better utilizing existing state authorities and programs, pursuing watershed approaches, and encouraging voluntary cooperation.

The Department of Pesticide Regulation (DPR) regulates the use and management of pesticides to prevent pollution of surface water bodies and ground water aquifers that may be used for drinking water supplies, as mandated in the State Pesticide Contamination Prevention Act (1986). DPR is responsible for regulating the sale and use of pesticides, evaluating and mitigating environmental and human health impacts of pesticide use, and promoting alternative pest control strategies. The DPR program relies on authorities in the California Food and Agriculture Code sections 13141 et seq.

Additional authorities in the California Pesticide Contamination Prevention Act require DPR to carry out specific activities to prevent ground water contamination. Prevention is the preferred goal because once ground water has become contaminated, cleanup activities are very difficult, expensive, and time-consuming. This Act requires:

- pesticide registrants to submit specific information to DPR regarding the impacts of their products on ground water;
- DPR to identify pesticides that have the potential to pollute ground water to be put on a ground water protection list; and
- DPR to conduct a monitoring program for pesticides in soil and ground water.

The Air Resources Board (ARB) regulates emissions of air pollutants than can affect the quality of surface and ground water.

The Department of Water Resources (DWR) is responsible for preparing and updating the California Water Plan to guide development and management of the state's water resources. SWRCB and the Regional Water Boards must consider this plan in their decisions. In addition, the Porter-Dolwig Ground Water Basin Protection Law gives DWR authority to initiate or participate in investigations, studies, and plans and to design criteria for projects to prevent degradation of ground water throughout the state[268].

The Department of Conservation (DOC) is responsible for preventing contamination of ground water due to the drilling, operation, maintenance, and abandonment of oil, gas, and geothermal wells. This includes both extraction and injection wells[269].

Schools Programs

The Lead-Safe Schools Protection Act of 1992[270] requires DHS to do the following:

- conduct a survey of public elementary schools and public day care facilities for the purpose of predicting lead contamination in paint, soil and drinking water;
- provide each participating school with the results of environmental testing for lead;
- recommend statewide standards for control of lead hazards in California public schools and day care facilities;
- consider the feasibility of statewide lead testing or other lead-related activities in schools and evaluate lead abatement technologies; and
- in cooperation with the California Department of Education, develop voluntary guidelines to minimize lead hazards in the course of repair and maintenance and abatement procedures.

The Act also requires public elementary schools and day care facilities to do the following:

- notify teachers, staff, and parents of the results of environmental testing for lead if they participated in the DHS survey (described above);
- use DHS-certified lead-related construction personnel when abating lead hazards; and
- prohibit the use of lead paint, lead plumbing and solders, or other potential sources of lead contamination in new construction and modernization or renovation projects[271].

School Property Evaluation and Cleanup

DTSC's Schools Property Evaluation and Cleanup Division is responsible for assessing, investigating and cleaning up proposed school property sites. This division ensures that selected properties are free of contamination where properties were previously contaminated and that they have been cleaned up to a level that protects the students and faculty who will occupy the new school. All proposed school sites that will receive state funding for acquisition or construction are required to go through a rigorous environmental review and cleanup process under DTSC's oversight[272].

This division has also developed new regulations, pursuant to Education Code section 17210(g), that streamline the environmental review process for proposed school sites. These regulations allow school districts to submit limited soil sampling data for specific contaminants during a Phase I Environmental Assessment and clarify the environmental assessment requirements in the Education Code. These regulations became effective February 10, 2003, and are codified in CCR/T22, division 4.5, chapter 51.5[273].

Toxic Air Contaminants

The Toxic Air Contaminant Identification and Control Act created California's program to reduce exposure to air toxics[274]. Established in 1983, this program established a two-step process of risk identification and risk management to address the potential health effects from air toxic substances and to protect public health. During the identification step, ARB and OEHHA determine if a substance should be formally identified as a toxic air contaminant (TAC) in California. ARB staff assesses for the potential for human exposure to a substance and OEHHA staff evaluates the health effects. In the risk management step, ARB reviews the emission sources of an identified TAC to determine if any regulatory action is necessary to reduce the risk.

ARB and OEHHA evaluated lead for identification as a TAC under this program. In 1997, ARB approved the listing of inorganic lead as a TAC for which a threshold exposure level could not be identified. The threshold exposure level is the level below which adverse health effects are not expected to occur. Lead is the first identified TAC for which non-cancer effects with no threshold have been identified[275].

In 1999, the Children's Environmental Health Protection Act added requirements to protect the health of infants and children. As part of this law, ARB, working with OEHHA, must evaluate all ambient air quality standards to determine that they are protective of the public and children's health in particular. In addition during the identification of TACs, the law requires OEHHA to consider whether the contaminant may cause infants and children to be susceptible to illness. Also, ARB must determine if the control measures for TACs are adequate to protect public health, especially that of infants and children, or if additional control measures are needed[276].

Leaded Gas Restrictions

The first federal Clean Air Act of 1963 empowered the federal Department of Health, Education and Welfare to define air quality criteria based on scientific studies and provided grants to state and local air pollution control agencies. This act was amended by the Motor Vehicle Air Pollution Control Act of 1965 which authorized the federal government to regulate air pollution and directed the Department of Health, Education and Welfare to establish auto emission standards.

The federal Air Quality Act, enacted in 1967, established a framework for defining air quality control regions based on meteorological and topographical factors of air pollution. The act also allows the State of California a waiver to set and enforce its own emissions standards for new vehicles based on California's unique need for more stringent controls. In 1970, the federal Clean Air Act was amended to establish the basic U.S. program for controlling air pollution.

In 1988, the California Clean Air Act was enacted, establishing the framework for air quality management in California for the next 20 years. In 1990, federal Clean Air Act amendments were signed, relying largely on several elements of the California Clean Air Act such as curbing toxic air pollutants and vehicle emissions[277]. The federal Clean Air Act and corresponding U.S. EPA regulations prohibited

gasoline containing lead or lead additives (leaded gasoline) as a motor vehicle fuel after December 31, 1995[278]. This ban ended a 25-year effort to phaseout lead from gasoline.

Health Laws

California has adopted several laws to address the problems associated with lead exposures to children:

- The Childhood Lead Poisoning Prevention Act of 1991 established the Childhood Lead Poisoning Prevention Branch within DHS and requires the branch to compile information, identify target areas, and analyze information to design and implement a program of medical follow-up and environmental abatement to reduce childhood lead exposure[279].
- The law that requires reporting of elevated BLLs declares childhood lead exposure the most significant childhood environmental health problem in the state. It mandates medical laboratories to report cases of children with elevated BLLs. It also mandates DHS to establish childhood lead poisoning prevention activities, including identification and selection of target areas for childhood lead screening programs, field trials of alternative lead abatement technologies, implementation of a program to identify and follow-up on high risk children, provision of environmental abatement and continued programs to reduce the incidence of excessive childhood lead exposure[280].
- The law that establishes lead exposure screening requires blood lead screening for children covered by health insurance[281].
- The law regarding lead in children's toys prohibits the manufacture, sale or exchange of toys with lead content in excess of the amount permitted by federal regulations[282].
- The Lead-Safe Schools Protection Act implements a lead poisoning prevention and protection program for California schools to survey and ascertain risk factors that predict lead contamination in public schools. It establishes guidelines for notification and advisement regarding survey findings, utilization of state-certified workers for activities to remediate lead-hazards and prohibits the use of potential sources of lead contamination in public schools[283].
- The law regarding real estate disclosure requires the disclosure of known lead-based paint hazards upon sale of a property[284].

Lead Paint Requirements

A number of federal laws applicable to the control of lead-based paint have been promulgated through time.

The Lead-Based Paint Poisoning Prevention Act, 42 U.S.C. 4822 (1971), initiated programs to screen children and begin the control of lead-based paint hazards in residential housing. The act directed the U.S. DHHS to:

- prohibit the use of lead-based paint in residential structures constructed or rehabilitated by the federal government or federal assistance in any form,
- establish a national program to encourage and assist states and cities to conduct mass screenings to identify children with elevated BLLs and make sure they receive medical treatment,
- investigate the children's residences for sources of lead, and
- order abatement of the residences if necessary.

The Lead Contamination Control Act of 1988 authorized CDC to provide grants to states to administer a program for preventing childhood lead poisoning for fiscal years 1990 and 1991[285]. Using this grant money, states were to:

- screen infants and children for lead,
- refer cases of elevated BLLs to the state for treatment and provide environmental case management, and
- provide education to communities with the highest risk for elevated BLLs

This act also addresses lead in drinking water, requiring states to establish programs to test and eliminate lead in water from schools and day care centers by July 1989 and to provide public notification of drinking water analyses. U.S. EPA distributed grants to states to assist local education agencies in meeting the requirements of the act.

In 1992 Congress passed the Residential Lead-Based Paint Hazard Reduction Act, the most comprehensive federal lead poisoning prevention legislation, as Title X of the Housing and Community Development Act. Title X redefines the federal response to lead poisoning by directing several federal agencies to establish a coordinated effort to reduce lead hazards. The main agencies responsible for Title X are the Department of Housing and Urban Development, U.S. EPA, and the Occupational Safety and Health Administration of the U.S. Department of Labor. This law amended TSCA to include a provision for lead exposure reduction, Title IV[286] providing authority for regulations, which went into effect March 6, 1996[287].

The National Lead Laboratory Accreditation Program, found in TSCA section 405(b), establishes protocols, criteria, and minimum performance standards for laboratory analysis of lead in paint, dust, and soil. The Hazard Standards for Lead in Paint, Dust, and Soil, found in TSCA section 403, establishes standards for lead-based paint hazards and lead dust cleanup levels in most pre-1978 housing and child-occupied facilities. The Training & Certification Program for Lead-Based Paint Activities, specified in TSCA sections 402 and 404, ensure that individuals conducting lead-based paint abatement, risk assessment, or inspection are properly trained and certified, that training programs are accredited, and that these activities are conducted according to reliable, effective and safe work practice standards. TSCA section 404 directed U.S. EPA to develop a program that would allow states and tribes to develop their own lead-based paint training and certification programs to operate in lieu of the federal program specified in TSCA section 402. The Pre-Renovation Education Rule, found in TSCA section 406(b), ensures that owners and occupants of most pre-1978 housing are provided information concerning potential hazards of lead-based paint exposure before certain renovations are begun on that housing. The Disclosure Rule requires disclosure of known lead-based paint and/or lead-based paint hazards by persons selling or leasing housing constructed before the phaseout of residential lead-based paint use in 1978[288]. Although California already has laws about lead disclosure[289], this new federal law is more extensive.

California's lead accreditation and certification program began in June 1994. At that time, new childhood lead poisoning prevention legislation required DHS to create a program to certify lead-related construction trades-people and accredit lead-related construction training providers. A subsequent revision to the regulations established work practice standards for lead-related construction and amended the previously established accreditation and certification requirements[290].

California’s occupational lead poisoning prevention laws establish a program to register and monitor laboratory reports of adult lead toxicity cases, monitor reported cases of occupational lead poisoning to ascertain lead poisoning sources, conduct investigations of take-home exposure cases, train employees and health professionals regarding occupational lead poisoning prevention, and recommend means for lead poisoning prevention[291]. There are three major elements of this program.

1. Surveillance and Case Follow-up:

- operates the California Occupational Blood Lead Registry, which is a compilation of laboratory reports of individuals with elevated BLLs; and
- investigates reported cases of occupational lead poisoning to ensure that the workers receive appropriate medical follow-up, that conditions in the workplace that resulted in lead poisoning are

corrected, and that household members at risk from "take-home" lead exposure are identified to local health agencies.

2. Outreach, Education and Training:
- develops and provides education, training, materials and other resources about the hazards of lead in the workplace and occupational lead poisoning prevention to workers, employers, health professionals and others; and
- conducts statewide outreach activities, including broad and targeted public health campaigns on the hazards of lead in the workplace and home. As part of these activities, the program designs, conducts and evaluates public health intervention projects directed toward targeted industries, occupations or other high-risk groups.

3. Technical Assistance and Exposure Control:
- conducts investigations and research to identify sources of and risk factors for lead exposure in the workplace, and to evaluate the effectiveness of control measures;
- provides technical assistance and consultation to employers, workers, industry groups, labor unions, health agencies and the general public regarding occupational lead poisoning prevention; and
- makes recommendations for the prevention and control of occupational lead poisoning.

California also has a law for lead-related activities in construction work to establish standards that protect the health and safety of employees who engage in lead-related construction work, including construction, demolition, renovation and repair[292].

Consumer Laws

State law directs DHS to establish a program, separate from the federal program, to protect the public from unsafe tableware, and to develop standards for lead and cadmium release from tableware[293]. This legislation was written because the U.S. FDA's program was thought to be inadequate to protect Californians from the importation and sale of unsafe tableware.

Senate Bill 460 (Ortiz) Chapter 931, Statutes of 2002, requires reporting of all blood lead analysis results, creates authority for local building code officials to enforce regulations against lead hazards, allows DHS and local agencies to take action against illegal activities of training providers, certified workers, and non-certified individuals who should be certified; and allows DHS and local agencies to issue cease and desist orders to people creating lead hazards, such as uncontained removal of lead-based paint.

Toys

State law establishes the following requirements[294]:
- prohibits the manufacture, sale or exchange of toys containing lead in excess of the amount permitted by federal regulations contained in Section 1500.17 of Title 16 of the Code of Federal Regulations adopted pursuant to the Federal Hazardous Substances Act, Chapter 30, beginning with section 1261, of title 15 of the United States Code, and
- empowers DHS to embargo any toy found to be in violation of this article.

Lead Solder

Health and Safety Code (H&SC) section 116880 authorizes DHS to adopt building standards to implement H&SC section 116875 that prohibits the use of solder in any plumbing fitting or fixture that is not lead free. The corresponding federal environmental laws are found in 42 U.S.C.A. section 300g-6.

Lead Acid Batteries

According to H&SC sections 25215-25215.5, dealers are required to accept a spent lead acid battery from a consumer when that consumer purchases a new lead acid battery from the dealer. Manufacturers of these batteries are required to notify distributors, wholesales, and dealers of the batteries it manufactures of the requirements of H&SC sections 25215.2 and 25215.3.

Additionally, a DTSC fact sheet, *Management of Spent Lead Acid Batteries*, June 2001, indicates that the public can also take their spent lead acid batteries to a household hazardous waste collection point or to some recycling centers.

Miniblinds

The Consumer Product Safety Commission (CPSC), an independent federal regulatory agency created by Congress in 1972 under the Consumer Product Safety Act, began operation in 1973. CPSC was established to protect the public against unreasonable risks of injuries associated with consumer products. In a June 25, 1996 news release, the CPSC stated that after testing and analyzing imported vinyl miniblinds, it determined that some of the blinds can present a lead poisoning hazard for young children. Twenty-five million non-glossy, vinyl miniblinds that have lead added to stabilize the plastic in the blinds are imported each year from various countries.

CPSC found that over time, the plastic deteriorates from exposure to sunlight and heat to form lead dust on the surface of the blind. In some blinds, the levels of lead in the dust was so high that a child ingesting dust from less than one square inch of blind a day for about 15 to 30 days could result in blood levels at or above the 10 µg/dL amount CPSC considers dangerous to children. In homes where children 6 years old and younger live, CPSC recommended that consumers remove the vinyl blinds.

CPSC asked the Window Covering Safety Council, which represents the industry, to immediately change the way it produces vinyl miniblinds by removing the lead. Manufacturers made the change and new miniblinds without lead were expected to be available to consumers within approximately 90 days following a June 1996 press release[295].

CHAPTER 5 – LEAD IN CONCLUSION

Extensive use of lead in a multitude of applications, both historically and in modern times, has left a dubious legacy to subsequent generations. Although significant steps have been taken to reduce exposure to lead, particularly from air emissions and food, sensitive populations remain at risk from exposure to lead in the environment, and, potentially, hazardous wastes that contain lead. The Department of Toxic Substances Control has the authority to classify wastes containing lead as hazardous, and to specify safe and appropriate management of those wastes.

Past and current emissions of lead are persistent and bioaccumulative. Although some transformations can occur, inorganic lead largely tends to remain where it has been deposited, unless exposed to acidic conditions or physically transferred. This is a particular concern in the instance of lead deposited to soil from industrial and automotive emissions, and from the weathering and flaking of lead-based paint. Exposure to lead-containing soil occurs when soil or its resulting dust is ingested or inhaled. Elevated soil-lead concentrations have been observed near roads, industrial sources and buildings, such as residences, coated with lead-based paint. Lead-contaminated soil is of concern to DTSC because it can be considered hazardous waste in certain situations.

The health risks associated with exposure to lead have been well-documented over a long history. Recent findings, however, show that there is no known threshold for lead since some effects, including cognitive impacts in children, occur at very low blood lead concentrations, even below the currently accepted level of concern of 10 µg/dL. Children are at particular risk for exposure to lead, primarily because of the following factors:

- children are more sensitive to the types of neurological damage incurred by lead, in part because their neural pathways are still developing;
- children absorb more lead than adults, in part because much of the lead that can be ingested by children is ingested in the absence of food; and
- children are more readily exposed to soil lead due to typical hand-mouth behaviors.

Recent studies also indicate that the health effects associated with lead are not always reversible.

As the evidence regarding the serious and cumulative impacts associated with lead exposure has mounted over the last 20 years, state and federal requirements have been implemented to reduce exposure to lead. In some instances these requirements have been revised over the years to provide additional protection for human health and the environment, particularly to protect children from exposure to lead. Examples of restrictions placed on lead include the phaseout of leaded gasoline, elimination of lead solder in cans, limitations and abatement of lead-based paint, and limitations on the use of lead in plumbing systems.

Over this time, BLLs in children have been monitored and correlated to health effects, resulting in actions to gradually reduce the level of concern from 60 µg/dL to 10 µg/dL. During this time, complex models to characterize exposure and predict the risks associated with that exposure were also developed and refined. Two of these models are the Integrated Exposure Uptake Biokinetic model (IEUBK), used extensively by U.S. EPA to establish requirements for the lead paint abatement program, and LeadSpread, developed and used by DTSC for activities such as developing remediation goals.

The LeadSpread model calculates concentrations of lead in blood in adults and children using the following exposure pathways:

- dermal contact with soil and/or dust at a site
- ingestion of soil and/or dust at a site

- inhalation of background air inhalation
- inhalation of dust from a site
- ingestion of drinking water
- ingestion of a defined market basket of food
- ingestion of home-grown produce.

The model uses site-specific and background for variables such as the background lead concentration in air, lead concentration in site soil/dust, lead concentration in drinking water, percentage of ingested produce homegrown, and respirable soil/dust from site (μg /m^3). Other critical variables, such as the proportionality constants, bioavailability factors, the quantity of media that the child is exposed to per day, and duration of exposure, are typically constant. By fixing all inputs but soil lead concentration, the soil lead level estimated to be associated with a limit of 10 μg/dL of lead in blood at a specified percentile of the above distribution can be calculated.

The IEUBK Model calculates a lognormal probability distribution of blood lead concentration in children who have been exposed to various environmental media (air, soil, dust, and diet). The model has four components – exposure, uptake, biokinetic, and probability distribution. The exposure component calculates media-specific contact rates using data including the concentrations of lead in various environmental media to which the child is exposed, the quantity of media that the child is exposed to per day, and duration of exposure. The uptake component calculates the amount of lead inhaled or ingested that is transferred to the blood plasma. The biokinetic component models the transfer of blood between plasma/extracellular fluid and various organs and excretion pathways. The probability distribution component calculates the lognormal probability distribution for the calculated blood lead concentration. The IEUBK model can provide various outputs, including geometric blood lead concentrations by age, percent of children with blood lead levels exceeding a specified level of concern, average media-specific daily lead uptake rates, and media-specific remediation goals. Appendix 5-A contains additional detailed information about the structure and differences between the IEUBK and LeadSpread models.

Although a variety of regulatory changes have occurred in response to the emerging information about the impacts of lead exposure, one requirement that has not changed in 20 years is the set of thresholds used to determine if a waste is hazardous according to the toxicity criteria. The thresholds used in California for non-RCRA wastes are the Total Threshold Limit Concentration (TTLC) and the Soluble Threshold Limit Concentration (STLC). To reflect the considerable additional information that has become available regarding the health effects associated with even low exposures to lead, DTSC is proposing to update the TTLC threshold.

TTLC

The current TTLC for lead was developed over 20 years ago, before many of the initiatives to limit exposure to lead had been completed, and before the current findings regarding health effects were known. At the time of the development of the TTLC, the acceptable BLL for children was 30 μg/dL, a level now associated with a variety of physical and cognitive deficits.

DTSC is focusing on the TTLC for lead because it was developed to protect receptors from direct exposure to hazardous wastes; exposure of the type the most sensitive receptors most commonly encounter. In general the TTLCs are intended to protect those expected to encounter hazardous waste, such as landfill workers, from direct exposure through dermal contact, inhalation and ingestion. In the case of lead, the original basis for the TTLC considered the potential for ingestion of lead-contaminated soil by children and sought to provide protection for these sensitive receptors. Exposure through

ingestion and inhalation by others is also a concern, but children (and pregnant women) remain the most sensitive receptors and, hence, become the focus of the discussion. Similarly, the TTLCs are applicable to any potentially hazardous wastes and waste streams, including sludges, liquids and other solids, not only to media such as soil that contains wastes or constituents. Because sensitive receptors are more likely to encounter soil contaminated with lead than other potentially hazardous lead-containing waste streams, such soils become the focus of consideration when discussing classification of lead-containing wastes.

The TTLC originally proposed in 1978, and in use today, assumes 1,000 mg/kg is protective of children likely to ingest soil containing lead. This value reflects the information available at that time. However, as shown by the recently developed health-based screening values for soils at school sites and near residential lead-paint sites, soil-lead concentrations that protect children who are exposed to lead in soil range from 255 mg/kg to 400 ppm, depending upon the model and assumptions used. The models and assumptions used to develop these values consider the most recent information regarding the health effects of lead and the exposure likely to occur. LeadSpread was used to develop the value of 255 mg/kg for school sites, and the IEUBK was used to develop the value of 400 ppm for soil near residences.

Because direct contact with lead, particularly ingestion, is the route of exposure of greatest concern among sensitive receptors, primarily children, and because the TTLC is the threshold developed to address such direct contact, DTSC has determined that the TTLC should be updated to reflect the more current findings about the health impacts associated with exposure to lead. DTSC has identified four different approaches for updating the TTLC. One option updates the original TTLC calculation; two are health-based approaches using the LeadSpread and IEUBK exposure models, and the fourth replaces the current test procedure with an extraction test that estimates the bioavailability factor for ingested lead. A fifth approach considers no change in the TTLC.

The practical effect of changing the TTLC is likely to be mixed since both total and soluble concentrations of lead must be considered when classifying a waste. If one uses the factors that estimate the relationship between a waste's total lead concentration and soluble lead concentrations, a waste that does not exceed the STLC for lead would likely have a very low total concentration of lead, levels near those protective of children with pica. For example, applying the factor of ten or, even thirty, to a waste with a soluble concentration of lead of less than 5 mg/L, using the WET, would yield an estimate of total lead concentration in the range of 50 to 150 mg/Kg. (As described earlier, because the relationship between soluble and total concentrations is not always reliable, the actual total concentration of lead would depend upon a variety of factors and such a relationship should be verified if it is used to characterize a wastestream.) Hence, if the TTLC were adjusted to be consistent with the school sites soil-lead value, and lowered from 1,000 mg/Kg to 255 mg/Kg, any additional wastestreams that would be captured by lowering the TTLC are likely to have already been considered hazardous by comparison of the soluble lead concentration with the STLC of 5 mg/L. New wastestreams identified by adjusting the TTLC are likely to only occur if the adjusted value of the TTLC approaches 150 mg/Kg. Generators who compare only the total lead concentration to the TTLC value, neglecting to compare soluble concentrations to the STLC, may erroneously assume that merely changing the TTLC value will change the amounts of waste classified as hazardous. While this may occur as a result of an adjusted TTLC, depending upon the value of the new TTLC, it is more likely to occur as generators correct errors in their waste classification procedures by comparing soluble concentrations of lead to the existing STLC.

Other positive and negative aspects specific to each of the alternatives are outlined below.

Alternative 1 – Update the original TTLC calculation by applying the uncertainty factor of 100 to the STLC value to arrive at a new TTLC of 500 mg/kg. This approach would not double the resulting value

since the original premise for doing so is no longer valid. Current models show soil-lead levels that protect children range from 255 mg/kg to 400 ppm, rather than 1,000 mg/kg, as was thought 20 years ago.
Pro:

- Methodology is consistent with the historical TTLC calculations and with other TTLC values.
- Approach acknowledges that doubling the value to 1,000 mg/kg is not protective of children with pica, as previously assumed in 1978

Con:

- Methodology is not health-based
- Resulting value of 500 mg/kg may not be protective of pica children

Alternative 2 – Update the original TTLC using DTSC's LeadSpread model and conservative assumptions for the model's parameters and a target BLL for children of 10 µg/dL.
Pro:

- Methodology incorporates a health-based approach.
- Methodology and resultant standard would be consistent with DTSC cleanup levels.
- The LeadSpread model is a simplified approach developed and used extensively by DTSC for other values, such as remediation goals for site mitigation and corrective action.
- The approach incorporates current data regarding the exposure to and health effects of lead for sensitive receptors.

Con:

- LeadSpread has not yet been used to establish regulatory levels.

Alternative 3 – Update the original TTLC using the Integrated Exposure Uptake Biokinetic model (IEUBK) and conservative assumptions for the model's parameters and a target BLL for children of 10 µg/dL.
Pro:

- Methodology incorporates a health-based approach.
- Methodology and resultant standard would be consistent with federal lead paint standards.
- IEUBK has been used extensively by the federal lead poisoning prevention programs to assess impacts of lead exposure to children and establish exposure thresholds.
- The approach incorporates current data regarding the exposure to and health effects of lead for sensitive receptors.
- The approach is more flexible than LeadSpread and can predict geometric mean blood levels in test populations of children.

Con:

- Considerable data requirements.
- May require additional evaluation to determine effects of sampling, methods of estimating amount of soil and dust ingested, and variations in lead bioavailability in soil and dust.

Alternative 4 - Replace the current test procedure for determining total lead concentrations in soil and wastes by an *in vitro* physiologically based extraction test to estimate the bioavailability factor for ingested lead.

Pro:

- Determines the amount of lead absorbed by the body from the waste or soil rather than using the total concentrations in waste or soil as a surrogate for exposure.

Con:
- Emerging science currently undergoing development, which may delay implementation.
- Approach has not been peer-reviewed or used to establish regulatory levels.

Alternative 5 – No change in the TTLC.

Pro:
- Requires no rulemaking.

Con:
- TTLC remains dated and inconsistent with current knowledge about the health effects associated with lead exposure.

STLC

The STLC for lead is intended to protect receptors from exposure to lead that could leach into water sources and be ingested with drinking water. DTSC considered a number of factors when the STLCs were developed, including the leaching action of rain, surface and ground water, and landfill leachate; the movement of resulting extractant from the disposal area; and attenuation or dilution of toxic substances in the extractant through soil absorption or through mixing with ground or surface waters.

As described in Chapter 4, the STLC was originally based on the MCL, which had a value of 50 µg/L. Since that time, the structure of the MCL for lead was changed, so that the current drinking water requirement for lead is to require water systems to control the corrosiveness of the water. If the concentration of lead in more than 10% of tap water samples exceeds the action level of 0.015 mg/L, the water system must take additional steps.

Unlike the new findings regarding the health impacts associated with exposure to lead, no additional data has become available that indicates lead is likely to leach under conditions other than the acidic conditions that formed the basis for the original STLC. Because the new findings regarding lead focus on the health effects associated with lead and do not affect the assumptions regarding the STLC for lead, DTSC determined that the STLC remains protective as originally proposed. DTSC is not suggesting changes for the STLC for lead.

For this evaluation of the TTLC and STLC for lead, DTSC has determined that the reasonable worst case exposure scenario would be exposure of children to soil or waste materials disposed in a residential setting. The DTSC rationale is that one cannot assume that waste, not otherwise classified as a hazardous waste, necessarily would be disposed in a Class III landfill. Such waste, including lead-contaminated soil, could ultimately expose both human and ecological receptors to the contaminants present in the waste, if it is improperly managed.

Future Activities and Outreach

This report summarizes available information about lead in various waste streams and the environment, and the potential impacts associated with exposure to lead, highlighting the current findings that were not available when the hazardous waste thresholds were originally developed. DTSC has found that the STLC for lead remains protective in light of the new information regarding the health impacts associated with exposure to lead, but the TTLC is obsolete and does not provide the protection intended for TTLCs.

DTSC plans to hold workshops to present this report and discuss the alternatives for adjusting the TTLC. DTSC anticipates interest and participation in these workshops from its sister Departments, Boards and other state organizations, in addition to environmental and community organizations, industry, and the regulated community.

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