Nomination Dossier on Lead

Submission by The United States to the Working Group of the

Sound Management of Chemicals (SMOC)

to consider lead as a candidate substance for development of a NARAP

This nomination dossier is a working document and

is not an official governmental or CEC document

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Substance Nomination Dossier

Lead

Identity, CAS number(s), and Description

Lead (pronounced "led", CAS number 7439-92-1) is a naturally occurring, bluish-gray metal originating in the earth's crust. It is an element with atomic number 82, and it has an atomic (molecular) weight of 207.20. It has a vapor pressure of 1.77 mm Hg at 1000°C, 10 mm Hg at 1162°C, 100 mm Hg at 1421°C, and 400 mm Hg at 1630°C (ATSDR 1993). Its group number is 14. It has a melting point of 327.4°C and a boiling point of 1740°C (ATSDR 1993). Its specific gravity is 11.3 (the specific gravity for water is 1.0). Its chemical abbreviation is Pb. Other names for lead include C.I. 77575, C.I. pigment metal 4, KS-4, Glover, Lead S2, Olow (Polish), plumbum, and Omaha.

Lead is odorless, tasteless, and has no known physiologic value. It does not dissolve in water and does not burn (ATSDR 1993). It is most commonly used in the production of batteries, ammunition, roofing materials, some metal products (e.g., sheet lead, solder, and pipes), some medical equipment (e.g., radiation shields, electronic ceramic parts), and military equipment (engine blades, tracking systems) (ATSDR 1993).

As lead is released to the environment, it often encounters anions to form compounds such as lead nitrate ($Pb(NO_3)_2$) and lead acetate (PbAc). These compounds may not resemble the metallic form of lead and may have properties that differ from lead, such as the ability to burn (ATSDR 1993).

Sources

Currently, lead-containing paint is one of the greatest sources of public health concern regarding lead exposure in the United States, especially in young children (CDC 1991). Prior to 1950, many house paints included up to 50 percent lead by dry weight (US HUD 1995). While these types of paints remained available through the 1970s, use of lead-based paint in housing began to decrease in the 1950s, and the Consumer Product Safety Commission (CPSC) ruled in 1978 that paint used for residences, toys, furniture, and public areas must not contain more than 0.06 percent lead by weight. However, housing components continue to remain covered with lead-based paint throughout the nation's housing stock, especially in older homes. The risk that lead-based paint poses to human health is increased when the paint is in a deteriorated state or is found on accessible, chewable, impact, or friction surfaces (US EPA 1986; CDC 1991). In this state, the lead in paint can contaminate dust or soil in the vicinity or can be directly ingested by children with pica tendencies for paint chips. In a national survey, approximately 83 percent of privately-owned, occupied US housing units built prior to 1980 (approximately 64 million units) were estimated to contain lead-based paint, with 18 percent containing more than five square feet of deteriorated lead-based paint (US EPA 1995).

Industrial processes are considered the major source of lead emissions to the atmosphere, accounting for 73 percent of total emissions in 1996 (US EPA 1998). Of these, the largest contributor is metals processing, which accounted for 52 percent of total emissions in 1996 (US EPA 1997c). Lead concentrations are highest in the vicinity of stationary sources such as ferrous and non-ferrous smelters and battery manufacturing plants (US EPA 1998). Such concentrations can result in considerable exposures to humans residing close enough to them. Approximately 230,000 children have been estimated

to reside near enough to a smelter to result in significant lead exposure (ATSDR 1988).

At one time, automobiles were the largest source of lead emissions (US EPA 1998). EPA issued its first reduction standards for lead in gasoline in 1973, calling for a gradual phasedown to 0.1 g/gal by 1986. Unleaded gasoline was introduced to the United States in 1975, accounting for 50 percent of gasoline sales by 1982 (US EPA 1997c). The US Clean Air Act banned the sale of any remaining leaded fuel effective January 1, 1996. As a result of these actions, the percentage of total US lead emissions attributed to on-road vehicles declined from 78 percent in 1970 to 0.5 percent in 1996 (US EPA 1997c). While leaded gasoline in relation to on-road sources is no longer considered a contributing source of lead in the US environment, non-road transportation sources, such as aircraft, contributed 14 percent to total lead emissions to the atmosphere in 1996 (US EPA 1998). The years of lead emissions from vehicular gasoline combustion resulted in large amounts of lead deposited in soil and dust. This lead is still present as lead's elemental property prevents it from dissipating, biodegrading, or decaying (CDC 1991).

Like the United States, Canada has instituted a ban on the sale of leaded gasoline. Mexico introduced unleaded gasoline in 1990.

While drinking water generally has non-detectable levels of lead upon release from treatment plants, lead can be introduced into drinking water from sources of lead in the distribution system, such as water pipes, connectors, and lead-containing solder. In addition, water can become lead-contaminated upon contact with lead or brass components of water fountains, coolers, faucets, and other fixtures. Leaching of lead from water systems into drinking water is increased when the water has high acidic levels (ATSDR 1993). This exposure is more likely to occur from water sources that collect acid rain but do not control for the water's acidity, such as some small community systems and private wells. Through authority of the 1986 Safe Drinking Water Act, EPA banned the use of lead-containing materials and solders in new plumbing and plumbing repairs.

Dietary intake of lead has declined over recent years, partially as a result of the phase-out of lead-soldered food cans (Adams 1991). Dietary lead intake of children decreased from 34–44 µg/day in 1980 to 5 µg/day by 1988. In the 1991 Total Diet Study, sponsored by the US Food and Drug Association (FDA), daily dietary lead intake for two year old children is estimated to be 2 µg/day (Bolger et al. 1996). The FDA has worked to reduce lead exposure to children by establishing specifications for lead in foods and food additives; controlling amounts of lead in canned foods, bottled water, and dietary supplements; and controlling exposure through use of ceramic pitchers, dinnerware, and decorated glass (Adams 1991). The FDA issued a final rule prohibiting use of lead solder in the manufacturer of food storage cans effective 27 December 1995, with existing stocks (at safe lead levels) sold no later than 27 June 1996 (US DOI 1995a). The gradual phase-out and ban of lead additives in gasoline in the 1970s through the 1990s has also contributed to reduced dietary intake of lead, as leaded gasoline emissions are no longer deposited from the air to vegetation, soils, and drinking water, or to house dust that can eventually reenter the air and come into contact with food.

In industrial applications involving lead, lead may be obtained from its primary form (mined ore) or from a secondary form (recycled materials containing lead). In 1998, US production of refined lead from primary sources is forecast to be 325,000 metric tons, while lead production by recovery from secondary sources is forecast to be 1,080,000 metric tons (US Department of Commerce and the McGraw-Hill Companies 1998). In 1995, lead production (mine output) was estimated to be 394,000 metric tons in the United States (US DOI 1995a), 164,348 metric tons in Canada, and 210,415 metric tons in Mexico (US DOI 1995b).

The forecasted United States lead usage in 1998 is 1,680,000 metric tons, with approximately 87 percent of this total going toward lead-acid battery manufacturing (US Department of Commerce and the McGraw-Hill Companies 1998). Non-battery uses of lead declined at an average rate of 4 percent between 1992 and 1996, with the primary use being the manufacture of ammunition (shot and bullets) (US Department of Commerce and the McGraw-Hill Companies 1998).

Presence in the Environment

Lead generally is present only at very low, background levels within the earth's crust (ATSDR 1988). While background surface soil-lead concentrations vary in different geological regions, natural soil-lead concentrations derived from crustal rock range from <10–30 µg/g (ATSDR 1993). It is the release of lead into the atmosphere by anthropogenic emissions that is the primary source of lead in the environment (ATSDR 1993).

In 1995, facilities reporting to the EPA's Toxics Release Inventory (TRI) released approximately 3 million pounds of lead and 14 million pounds of lead compounds to the environment (US EPA 1997b). Approximately 87 percent of these releases were to land, 12 percent to air, and less than 1 percent to water or underground. In this same year, these facilities transferred approximately 67 million pounds of lead and 312 million pounds of lead compounds to off-site locations, with approximately 93 percent transferred to recycling, 5 percent to disposal, and 2 percent to treatment (US EPA 1997b). When rating the 647 chemicals monitored by the TRI in terms of total amount of production-related waste in 1995, lead compounds were rated among the top ten chemicals, with 861 million pounds (US EPA 1997b).

In 1995, Canadian facilities reporting to Canada's National Pollutant Release Inventory (NPRI) released approximately 1,572 metric tons (3.4 million pounds) of lead and lead compounds to the environment, with 52 percent released to land, 42 percent to air, and 5 percent to water (Environment Canada 1997). In this same year, these facilities transferred approximately 2,063 metric tons (4.5 million pounds) of lead and lead compounds to off-site locations, with approximately 75 percent transferred to landfills (Environment Canada 1997).

Lead accumulates over time in both humans and the environment. From 3–4 million tons of lead were estimated to exist in paint, while a similar amount is expected to exist in dust, soil, and water as a result of atmospheric fallout due to anthropogenic sources (US EPA 1986).

Outdoor airborne lead levels have continued to decline over the years as leaded gasoline emissions have declined. Ambient lead concentrations have declined 97 percent from 1977 to 1996 (US EPA 1998). From 1987 to 1996, the maximum quarterly average ambient lead concentrations decreased 75 percent in populated areas, from 0.16 $\mu g/m^3$ to 0.04 $\mu g/m^3$ (US EPA 1998). In remote areas, such as Antarctica, ambient air lead levels were measured at 7.6×10^{-5} $\mu g/m^3$ (Maenhaut et al. 1979). EPA has set a National Ambient Air Quality Standard of 1.5 $\mu g/m^3$ for lead (representing a quarterly average concentration). As early as 1988, the average ambient air lead concentration across all US cities within the National Air Surveillance Network was lower than this standard. Airborne lead levels are considered to be 0.3-0.8 times lower indoors than outdoors (ATSDR 1993).

In water, lead levels are influenced by acidity and salinity. Concentrations of dissolved lead in groundwater tend to be low, as lead forms compounds such as carbonates, sulfates, and phosphates when encountering anions in the water. These compounds then tend to precipitate out of the water column. The total solubility of lead in hard water is approximately 30 µg/L, compared to approximately 500 µg/L in soft water (ATSDR 1993). While lead levels as high as 890 µg/L have been measured in surface water

in the United States, the mean measured levels across 50,000 surface water stations was 3.9 µg/L (Eckel and Jacob 1988).

Presence in Biota

Lead levels in plants and animals are highest when a lead-emitting point source is located near to their living area. Plant surfaces can contain lead due to atmospheric deposition, while internal plant tissues can contain lead as a result of biological uptake from soil and leaf surfaces. Uptake from soil is heightened when the soil is more acidic and when organic matter content is reduced (ATSDR 1993).

Animals and wildlife encounter lead by inhaling contaminated air and by ingesting contaminated soil and plants. Animals high on the food chain have more opportunities to consume lead-contaminated food than other species may. As lead tends to accumulate over time, many species tend to increase their body burdens of lead as they age (ATSDR 1993).

Within aquatic organisms, lead concentrations have been found to be highest in benthic organisms and algae, while upper trophic-level predators, such as carnivorous fish, had lower concentrations. While lead is considered toxic to all aquatic biota, organolead compounds are considered to be more toxic than inorganic lead forms and tend to bioconcentrate in aquatic biota (ATSDR 1993).

Presence in Human Populations

Lead enters the human body primarily via ingestion and inhalation, with dermal absorption playing a very minor role (Moore et al. 1980). Approximately 99 percent of lead ingested into an adult body leaves the body through human waste (i.e., feces, urine, and sweat) (Reilly 1991), compared to only 30–40 percent of lead ingested into the body of a child up to two years of age (Ziegler et al. 1978). Lead absorption by the body is dependent on nutrient intake; low calcium, zinc, and iron levels have been shown to enhance lead absorption in the small intestines (Mushak and Crochetti 1996). Lead that is absorbed by the body is distributed via the blood to "soft tissues" (e.g., liver, kidneys, muscles, brain) and, over the course of several weeks, also moves into "hard tissues" (e.g., bones, teeth). Approximately 94 percent of lead in an adult's body is estimated to be stored in bones and teeth, compared to only 73 percent in a child's body (ATSDR 1993). While lead can remain in bones and teeth for decades, it can eventually move back into blood and soft tissue. Between 40–70 percent of lead in an adult's blood may be attributed to mobilized bone-lead stores (Gulson et al. 1995; Smith et al. 1996). Certain states of physiological stress that cause calcium mobilization from teeth and bones, such as pregnancy, also can result in mobilization of lead into blood. This result is of particular concern for pregnant women, as lead mobilized from maternal bone stores can be transferred via the placenta to the unborn fetus.

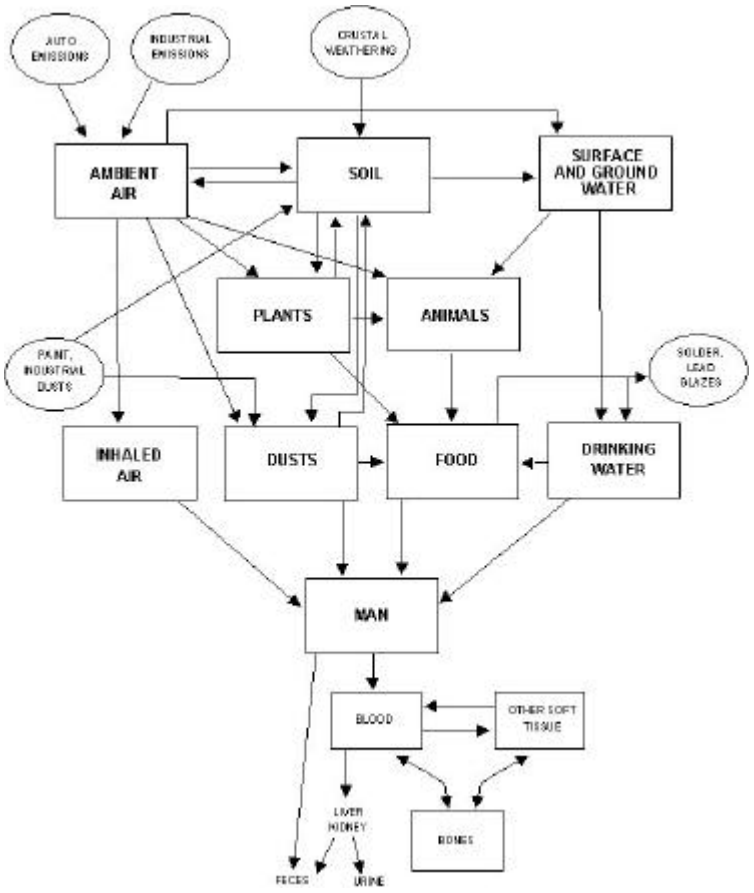
The most common measure of body lead burden is blood-lead concentration, typically expressed as micrograms of lead per deciliter of blood (µg/dL) (ATSDR 1988). In Phase 2 of the Third National Health and Nutrition Examination Survey (NHANES III), conducted from 1991-1994, the geometric mean blood-lead concentration was estimated at 2.3 µg/dL (95 percent confidence interval: 2.1–2.4 µg/dL) for persons in the United States aged 1–74 years. This is a decline from 2.8 µg/dL measured in Phase 1 of NHANES III (1988–1991) and 12.8 µg/dL measured in NHANES II (1976–1980). For US children aged 1–5 years, NHANES III Phase 2 estimated the geometric mean blood-lead concentration at 2.7 µg/dL (95 percent confidence interval: 2.5–3.0 µg/dL), which is a decline from 3.6 µg/dL measured in Phase 1 of NHANES III and 15.0 µg/dL measured in NHANES II (CDC 1997; Pirkle et al. 1994).

Phase 2 of NHANES III has also estimated that 5.9 percent of US children aged 1–2 years have blood-

lead concentrations at or above 10 µg/dL, the blood-lead concentration considered elevated by CDC. For US children aged 1–5 years, the estimate is 4.4 percent (CDC 1997). The percentage of children with elevated blood-lead concentration is generally higher for children in lower-income households and for African-American children.

Figure 1 illustrates the major sources and reservoirs of lead, how lead is introduced into the human environment, and various pathways of human exposure.

Figure 1



Pathways of Lead from the Environment to Humans, Main Organs of Absorption and Retention, and Main Routes of Excretion

(Sources: US EPA 1986; US EPA 1996)

Transport and Environmental Fate

The atmosphere is commonly the initial recipient of lead released to the environment (ATSDR 1993). Lead in the atmosphere can differ according to particle size, with large particles (>2 µm) settling rapidly near the emission source and small particles having the potential to remain suspended for longer periods and to travel considerable distances before depositing.

Natural chemical and physical processes such as weathering, runoff, and precipitation permit lead to be transferred continuously between air, water, and soil (ATSDR 1993); this is illustrated in Figure 1.

Soil and sediments are important sinks of lead in the environment. Historically, atmospheric deposition of lead has been the primary source of lead in soil (ATSDR 1993). Such lead generally deposits within the top 2–5 centimeters of soil (US EPA 1986). While some of the deposited lead could have originated from point sources many miles away, most lead comes from point and mobile sources in the vicinity of the soil. For example, urban and residential soils can become lead-contaminated as a result of vehicular emissions,

or the demolishing, renovating, or weathering of nearby buildings containing lead-based paint.

As a result of runoff, contaminated soil can contribute to contamination of any nearby sediments (Case et al. 1989). Soil-lead tends to remain in the soil, sorbed to organic matter, until the soil is disturbed or eroded. Organic lead complexes in soil are more soluble and can leach out or be absorbed by plants more easily at a soil pH of 4–6 compared to a higher pH. The lead which plants absorb from soil is returned to the soil when the plants decay where they have grown.

Toxicity

Lead accumulates in humans over time, being stored in tissues such as bone and teeth. This lead can mobilize back into the blood stream over time as well. As a result, if a person has been exposed to lead in the past, it is difficult to quantify a dose-response relationship, and therefore, how a person's exposure to lead is associated with adverse health effects. Even exposure to low levels of lead over an extended period of time can lead to significant accumulations of lead in the human body (ATSDR 1993).

Tables 1 and 2 present estimates of LOAELs (lowest-observed-adverse-effect levels) for lead in the blood of children and adults, respectively, as reported in the scientific literature. These tables originally appeared in NAS (1993). Additional summary information on human health effects associated with lead exposure can be found in Appendix B of US EPA (1997a).

The effects of lead in the human body are modulated by its distribution in the body, its affinity for various binding sites, and differences in cellular composition and structure within tissues and organs. As a result, there is no single, well-defined mechanism that explains the toxicological activity of lead in all tissues (US EPA 1986). Lead-induced alterations of ion transport in the human body result in effects that disturb the development and function of many organ systems, particularly the central nervous system, thereby resulting in many of the health effects associated with lead exposure.

While the particular lead compounds, routes of exposure, and lead levels that are associated with cancer risks in humans are currently unknown, EPA has classified lead as a probable human carcinogen (Group B2), while the International Agency for Research on Cancer has classified lead as a possible human carcinogen (Group 2B). These classifications are primarily the result of evidence found in animal studies.

As understanding of childhood lead poisoning has improved over the years, the blood-lead concentration considered toxic has consistently declined. The minimal level at which children's blood-lead concentration is considered elevated has declined from 60 µg/dL prior to the 1960s, to 30 µg/dL by 1975, to 25 µg/dL by 1985, to its current level of 10 µg/dL. Recently, blood-lead concentrations as low as 10 µg/dL have been associated with adverse effects in children, such as decreased intelligence and impaired neurobehavioral development (Davis and Svendsgaard 1987; Mushak et al. 1989). In adults, blood-lead concentrations of 25 µg/dL or higher are considered elevated by the Adult Blood Lead Epidemiology and Surveillance Program, operated by the National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention.

Table 1

Lowest-Observed-Adverse-Effect Levels (LOAELs) of Lead in the Blood of Children

LOAEL (µg/dL)	Neurologic Effects	Heme-Synthesis Effects	Other Effects
<10–15 (pre- and post-natal)	Deficits in neurobehavioral development (Bayley and McCarthy Scales), electro-physiologic changes,[a,b] and lower intelligence quotient[c,d]	ALA-D inhibition[e]	Reduced gestational age and birthweight; reduced size up to age 7–8 years[a,b,e]
15–20		Erythrocyte protoporphyrin increase[a,e]	Impaired vitamin D metabolism, Py-5'-N inhibition[a,e]
<25	Longer reaction time (studied cross-sectionally)[b,e]	Reduced hematocrit (reduced Hb)[f]	
30	Slower nerve conduction[e]		
40		Increasing CP-U and ALA-U[c]	
70	Peripheral neuropathies[a,e]	Frank anemia[a,e]	
80–100	Encephalopathy[a,e]		Colic, other gastrointestinal effects, kidney effects[e]

Source: Table 2-4 of NAS (1993).
[a] From CDC 1991
[b] From US EPA 1990a,b
[c] From Bellinger et al. 1992
[d] From Dietrich et al. 1993
[e] From ATSDR 1988
[f] From Schwartz et al. 1990
ALA-D = delta-aminolevulinic acid dehydratase

Table 2

Lowest-Observed-Adverse-Effect Levels (LOAELs) of Lead in the Blood of Adults

LOAEL (µg/dL)	Heme Synthesis and Hematologic Effects	Neurologic Effects	Other Effects (Renal, Reproductive, Cardiovascular)
<10	ALA-D inhibition		
10–15			Increased blood pressure
15–20	Erythrocyte protoporphyrin increase in females		
25–30	Erythrocyte protoporphyrin increase in males		
40	Increased ALA and CP in urine	Peripheral nerve dysfunction (slower nerve conduction)	
50	Reduced hemoglobin production	Overt subencephalopathic neurologic symptoms	Altered testicular function
60			Female reproductive effects
80	Frank anemia		
100–120		Encephalopathic signs and symptoms	Chronic nephropathy

Source: Table 2-5 of NAS (1993), adapted from Volume IV of US EPA 1986.
ALA-D = delta-aminolevulinic acid dehydratase
ALA-D = delta-aminolevulinic acid
CP = coproporphyrin

Risk Management Experience

On 20 February 1996, the 29 member countries of the Organisation for Economic Cooperation and Development (OECD) issued a Declaration on Risk Reduction of Lead. This declaration noted the findings of risk assessments in the member countries, the willingness of industry to cooperate in risk reduction, and the reductions in lead exposure that resulted from proper application and management of risk reduction programs. Through this declaration, the OECD pledges its support to continue cooperation among member countries on risk reduction efforts, to monitor the environment for lead levels, to work with industry in implementing voluntary risk reduction activities, to share information on lead exposure among all countries, and to continue to raise the issue of lead exposure at an international level. The OECD recognizes that gasoline combustion, food packaging, food consumption practices, paint, building materials, drinking water, use of lead shot and sinkers in hunting and fishing activities, and certain occupational practices, are among the most likely contributors to lead exposure in many countries. In 1997, the G7 Environment Leaders of the Eight committed to fulfill and promote the OECD declaration on an international level.

Starting in the mid-1970s, atmospheric lead concentrations in US cities began to decline as a result of US EPA regulation under the US Clean Air Act. This included: (1) the listing of lead as a "criteria air pollutant" and the setting of a National Ambient Air Quality Standard for Lead of 1.5 $\mu g/m^3$ (90-day average) (40 CFR 50.12), and (2) the phaseout of the use of lead additives in gasoline, which allowed the introduction of lead-sensitive, emission control-equipped vehicles in the United States. These actions contributed to a tremendous decline in average blood-lead concentrations in the US population. According to NHANES II, nearly a 40 percent decline in average blood-lead concentration was observed between 1976 and 1980 alone; this decline corresponded to an approximate 50 percent decrease in the use of leaded gasoline in the United States (CDC 1991; Annest 1983). The decline in average blood-lead concentration is likely due to the considerable reductions in airborne lead levels and reduced contamination of soil and food that resulted from the decline in use of leaded gasoline. EPA regulation of the amount of lead in gasoline continued through the 1980s, until Title II of the 1990 amendments to the Clean Air Act (42 USC 7545) instituted a controlled phase-out of leaded gasoline by 31 December 1995.

Previous discussion mentioned the ruling by the CPSC to reduce lead content in house paint to no more than 0.06 percent by weight. This followed voluntary manufacturer reductions to 1 percent lead by weight in the late 1950s and reductions to 0.5 percent mandated by the US Department of Housing and Urban Development (HUD) in the 1970s under the Lead-Based Paint Poisoning Prevention Act. While these efforts reduced the amount of lead in paint within the nation's housing stock, it did not address the presence of lead in existing paint films. In response to Title X of the Housing and Community Development Act of 1992, HUD has prepared guidelines on identifying and controlling existing lead-based paint hazards, as improper control procedures can actually increase the threat of lead-based paint exposure (US HUD 1995). Currently, under Section 403 of the Toxic Substances Control Act, as amended under Title X, EPA is preparing regulations to set standards for lead levels in paint, dust, and soil (US EPA 1997a).

In 1991, through the National Primary Drinking Water Regulations for Lead and Copper (56 FR 26460), the US EPA set an action level of 15 parts per billion (ppb) for lead content in drinking water and a maximum contaminant level goal of 0 ppb at the tap. At the local level, efforts to reduce the corrosivity of drinking water can reduce lead exposures when lead sources are present in the distribution system; such efforts have been estimated to cost only 25 percent of the value of the health benefits associated with the resulting lead exposure reduction. Efforts by the city of Boston in the 1970s to reduce corrosivity of

drinking water considerably reduced the amount of lead in tap water. Earlier investigations showed a relationship between high amounts of lead in tap water and increased blood-lead concentration in Boston children under age six years (Worth et al. 1981).

While government regulatory action has contributed significantly toward reducing lead exposures to the US population over the last 20–30 years, voluntary actions taken in the private sector have also contributed to reduced lead exposures. Some actions by the private sector have already been discussed, such as the voluntary decline of lead content in paint that occurred in the 1950s and the manufacture of vehicles that did not require lead additives in gasoline. Voluntary actions also reduced the percentage of food cans containing lead solder, from over 90 percent in 1979 to less than 5 percent in 1990 (Adams 1991), leading to the FDA's 1995 ban on lead solder in food containers. Over the same period, foods imported into the United States in lead-soldered containers have also been dramatically reduced. The private sector has also voluntarily ended the practice of using lead solder to solder copper pipes to transport water in new homes.

Conclusions

Over the years, as the world has become highly industrialized and as products have been manufactured for human use and consumption, considerable amounts of lead have been introduced into the environment. Given the propensity for lead to accumulate over time in various environmental media (e.g., soil, dust, and sediments), the potential for undue human exposure tends to increase in the absence of appropriate and effective remediation. In recent years, the scientific community has been made aware of the toxic effects of lead and lead compounds, especially in sensitive populations such as young children and women of childbearing age (due to potential risks to the fetus in the event of pregnancy). Lead contributes to a variety of adverse effects in the human body, especially to the central nervous system, and can even cause death at sufficiently high levels. Considerable research has been done to investigate the extent to which lead is present in the environment and how this exposure and the resulting increased body burden of lead in the population contributes to increased occurrence of certain health effects.

US regulatory efforts of the past 20 years, as well as efforts in the private sector, have contributed to substantial declines in lead levels that are present in human blood, especially in children. Although elevated blood-lead concentrations remain unacceptably high in some subpopulations (determined by such factors as socioeconomic status and educational attainment), the estimated geometric mean blood-lead concentration in the US population declined more than 80 percent in the 20 years where lead emissions from gasoline combustion and sources of dietary lead continually declined. Currently, the presence of lead-containing paint in the national housing stock remains one of the greatest sources of public health concern regarding lead exposure in the United States, especially to young children. In addition, as levels of exposure related to health effect concerns continue to decline, ways to reduce exposure to even lower levels will depend on whether currently-unidentified secondary sources of exposure for children, such as those related to the child's hygiene and food-related behaviors, can be identified (Freeman et al. 1997).

The overwhelming evidence of lead exposures present in the human environment and the effects that these exposures have on human health warrant continued action at the international level. The experience that many countries have accumulated on risk reduction should be shared with the entire international community.

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