CADMIUM AND CADMIUM COMPOUNDS

First Listed in the *First Annual Report on Carcinogens* as *Reasonably Anticipated to be Human Carcinogens* updated to *Known to be Human Carcinogens* in the *Ninth Report on Carcinogens*

CARCINOGENICITY

Cadmium (CAS No. 7440-43-9) and Cadmium Compounds are *known to be human carcinogens* based on sufficient evidence of carcinogenicity from studies in humans, including epidemiological and mechanistic information which indicate a causal relationship between exposure to cadmium and cadmium compounds and human cancer. In several cohort studies of workers exposed to various cadmium compounds, the risk for death from lung cancer is elevated (reviewed in IARC V.58, 1993). Although confounding factors, such as co-exposure to arsenic, were present in several of these studies, it is unlikely that the increase in lung cancer risk is completely explained by exposure to arsenic). Follow-up analysis of some of these cohorts has not definitively eliminated arsenic as a possible confounding factor, but has confirmed that cadmium exposure is associated with elevated lung cancer risk under some industrial circumstances (Sorahan and Lancashire, 1997; Sorahan et al., 1995). In some early cohort studies, an increased risk of mortality from prostate cancers was found in cadmium-exposed workers but later cohort studies have not confirmed this observation. Additional epidemiological evidence (case-control studies, geographic distribution studies, etc.) suggests an association between cadmium exposure in human populations and prostate (van der Gulden et al., 1995; Garcia Sanchez et al., 1992; Shigematsu et al., 1982; Bako et al., 1982); renal (Mandel et al., 1995; Kolonel, 1976); and bladder (Siemiatycki et al., 1994) cancers.

The evidence that cadmium and cadmium compounds are human carcinogens is supported by experimental animal studies that have shown cadmium and cadmium compounds induce malignant tumor formation by multiple routes of exposure at various sites in multiple species of experimental animals. Inhalation of a variety of cadmium compounds has repeatedly been reported to produce dose-dependent increases in pulmonary adenocarcinomas in rats, and occasionally to produce pulmonary tumors in mice but not in hamsters (reviewed in IARC V.58, 1993). Intratracheal instillation of cadmium compounds produces malignant lung tumors in rats (IARC V.58, 1993). Oral exposure to cadmium chloride produces dose-related increases in leukemia and benign testicular tumors in rats. In several studies, single or multiple injections (s.c., i.m., or i.p.) of a variety of soluble and insoluble cadmium compounds have caused local sarcomas in rats and mice (IARC V.58, 1993; Waalkes and Rehm, 1994a). Subcutaneously injected cadmium compounds produce a variety of tumors including prostate tumors in rats, testicular tumors in rats and mice, lymphomas in mice, adrenal tumors in hamsters and mice, and lung and liver tumors in mice (IARC V.58, 1993; Waalkes and Rehm, 1994a, b, c; Waalkes et al., 1994). Based on the carcinogenicity of a wide variety of cadmium compounds, it appears that it is ionic cadmium that is the active, carcinogenic species (reviewed in IARC V.58, 1993). Studies in animals and in isolated cells or tissues suggest that ionic cadmium, or compounds that release ionic cadmium cause genetic damage and are carcinogenic. Thus, the carcinogenic potential of a given cadmium compound would depend on the degree to which the compound releases ionic cadmium under the conditions of exposure.

ADDITIONAL INFORMATION RELEVANT TO CARCINOGENESIS OR POSSIBLE MECHANISMS OF CARCINOGENESIS

Increases in chromosomal aberrations have been observed in lymphocytes of workers exposed to cadmium in industrial settings. Many studies of cultured animal cells have shown that cadmium compounds damage genetic material. DNA strand breaks, mutations, chromosomal damage, cell transformations, and disrupted DNA repair have been observed in *in vitro* studies. The accumulated information supports the conclusion, also reached by IARC, that ionic cadmium is the active, genotoxic form of the metal or metal compounds (IARC V.58, 1993). No data are available that indicate the mechanisms thought to account for cadmium carcinogenesis in experimental animals would not also operate in humans.

The sensitivity of cells or tissues to cadmium appears to be related, at least in part, to expression of the metallothionein (*MT*) gene. The *MT* protein produced following activation of the *MT* gene functions to sequester cadmium. Activation of the *MT* gene can limit the genotoxic effects of cadmium. Differential expression of the pulmonary *MT* gene appears to be the basis for the sensitivity of rats and the insensitivity of mice to lung tumors induced by inhaled cadmium. Additionally, other target tissues of cadmium carcinogenesis in rodents show minimal basal expression or poor activation upon stimulation of the *MT* gene (Oberdrster et al., 1994).

PROPERTIES

Cadmium is a silver-white, blue-tinged, malleable metal or grayish-white powder. It has an atomic weight of 112.41 and belongs to group IIB of the periodic table. Cadmium is a rare element and not found in nature in its pure state. The cadmium minerals do not occur in quantities that support mining. Cadmium is generally the by-product of processing sulfide ores for zinc, lead, and copper (IARC V.58, 1993).

It is soluble in acid, ammonium nitrate, and hot sulfuric acid and insoluble in cold and hot water. Cadmium carbonate occurs as a white amorphous powder that is soluble in acids, potassium cyanate, and ammonium salts and insoluble in ammonia and water, both cold and hot. Cadmium chloride occurs as small, white-to-colorless, hexagonal crystals. It is soluble in water and acetone and insoluble in ethanol. Cadmium fluoroborate is extremely hygroscopic and very soluble in water. When heated to decomposition, it emits toxic fumes of cadmium, hydrofluoric acid, and other fluorinated compounds. Cadmium nitrate occurs as white, amorphous pieces or hygroscopic needles. It is very soluble in acids and soluble in ethanol, acetone, water, and ammonia. Cadmium oxide occurs as a colorless amorphous powder or brown-red cubic crystals. It is soluble in acids, ammonium salts, and alkalies, but is insoluble in cold and hot water. When heated to decomposition, it emits toxic fumes of cadmium. Cadmium sulfate occurs as white rhombic crystals. It is soluble in water but insoluble in alcohol, acetone, and ammonia. Cadmium sulfide is a yellow-orange or brown powder. It forms a colloid in hot water; it is soluble in acids and ammonia and insoluble in cold water. When heated to decomposition, cadmium sulfate and cadmium sulfide emit toxic fumes of cadmium and sulfur oxides (SO_x).

Cadmium metal is available in purities ranging from 99.5%-99.999% in the following grades: technical, powder, pure sticks, ingots, slabs, and high-purity crystals with less than 10 ppm impurities. Cadmium carbonate is available in a commercial grade that has a purity of about 98%, with lead, zinc, and iron as impurities. Reagent-grade cadmium carbonate, with purities ranging from 99.9%-99.995%, is also available. Commercial cadmium chloride is a mixture of hydrates that is similar to the dihydrate form of cadmium chloride. The commercial grade available in the United States typically contains about 51% cadmium and 0.005% each of

iron and copper; higher purity grades (99.9%) are also available. Cadmium fluoroborate is available commercially in a 50% aqueous solution. Cadmium nitrate is available in technical and reagent grades (purity > 99%) with typical impurities of chloride, sulfate, copper, iron, lead, zinc, and arsenic. Commercial-grade cadmium oxide is available in the United States with a purity of 99.7%; common contaminants are lead and thallium. Cadmium sulfate is available in technical and C.P. grades. Cadmium sulfide is available in technical, N.D., high-purity (single crystals), and commercial grades. Typically, cadmium sulfide is available in mixtures depending upon its use as either a pigment or a phosphor. Cadmium is available in bars, sheets, wire, or gray, granular powder (HSDB, 1997).

USE

The principal use of cadmium metal is the coating and electroplating of metals to prevent corrosion. Relative cadmium usage in 1991 was estimated as 45% for batteries, 20% for coating and plating, 16% for pigments, 12% for plastic and synthetic products, and 7% for alloys and other metal products. From 1988 to 1991, the relative consumption of cadmium increased about 5% per year for batteries, decreased about 10% for coating and plating, remained at approximately 15% for pigments, remained at an average of 12% for plastic and synthetic products, and remained at an average of 9% for alloys (USDOI, 1991). Cadmium sulfide (CdS) and cadmium selenium sulfide (CdSeS) are used in pigments, primarily for plastics, and as stabilizers for poly(vinyl chloride) (PVC). Cadmium is also used in nickel-cadmium (Ni-Cd) batteries (electrode material), fungicides, alloys, and other uses (IARC V.58, 1993; Carson et al., 1986; ATSDR, 1997-H010).

Cadmium carbonate and cadmium chloride have been used as fungicides for golf courses and home lawn turf (ATSDR, 1997-H010). By 1997, all cadmium pesticides had undergone voluntary cancellation (OPP, 1997). Cadmium carbonate is used as a catalyst in organic reactions and as a source of cadmium in other reactions. The intermetallic compounds, including cadmium sulfide (CdS), cadmium selenide (CdSe), and cadmium telluride (CdTe), have been used as semiconductors for photoconductors, photovoltaic cells, and infrared windows (ACGIHa, 1986; Kirk-Othmer V.4, 1978). Cadmium salts, especially the oxide and anthranilate, are used as anthelmintics in treating swine and poultry (Budavari, 1996). Cadmium chloride is used in photocopying, printing, dyeing, electroplating baths, and the manufacture of special mirrors and of cadmium yellow. A cadmium fluoroborate bath is used for electrodeposition of cadmium on high-strength steels to avoid the problem of hydrogen embrittlement inherent in cyanide plating (Kirk-Othmer V.4, 1978). Cadmium nitrate and cadmium oxide are used in the production of cadmium salts, photographic emulsions, coloring glass and porcelain, and in the laboratory as reagents. Cadmium oxide also finds use in plating baths, in electrodes for storage batteries, in phosphors, as a nematocide, as a starting material for PVC heat stabilizers, and as an additive in nitrite rubbers and plastics such as Teflon®. Cadmium sulfate and cadmium sulfide are used in pigments, fluorescent screens, in photoelectric cells, and in electroplating. Cadmium sulfide is the most widely used cadmium compound and is used primarily as a pigment (Sax, 1987; cited by IARC V.58, 1993; Kirk-Othmer V.4, 1978).

PRODUCTION

Cadmium is chiefly recovered as a by-product of smelting domestic and imported zinc concentrates; as such, its production is dependent upon the demand for zinc. TRI95 (1997) listed ASARCO Incorporated, Denver, CO (cadmium refinery); Big River Zinc Corp., Sauget, IL; Jersey Miniere Zinc Co., Clarksville, TN; and Zinc Corp. of America, Bartlesville, OK, as

producers in 1995. These producers recovered and produced cadmium as a by-product of smelting domestic and imported zinc concentrates, and one company (ASARCO, Inc.) in Colorado refined cadmium from other sources such as lead smelter baghouse dust (Llewellyn, 1993). In 1997, only two companies produced primary cadmium in the United States—the electrolytic plants in Sauget, IL, and Clarksville, TN (USDOI, 1997).

Production for 1972 through 1977 ranged from 4.1 million to 7.5 million lb (Kirk-Othmer V.4, 1978). The 1979 TSCA Inventory reported that in 1977, there were 14 producers of cadmium producing 6.3 million lb and 18 importers importing 2.6 million lb (TSCA, 1979). From 1980 through 1989, U.S. annual cadmium production averaged 3.3 million lb (range 2.2-4.2 million lb), imports averaged 5.6 million lb (4.2-7.0 million lb), and exports averaged 0.45 million lb (0.02-1.4 million lb). In the period 1990-1997, production remained about the same (average 3.3 million lb, range 2.2-4.5 million lb), but imports were lower (average 3.0, range 1.7-4.5 million lb) and exports were higher (average 1.1 million lb, range 0.02-3.2 million lb) (USDOI, 1985, 1987, 1988, 1990, 1991, 1997; Plachy, 1997).

EXPOSURE

Consumption of food, drinking water, incidental ingestion of soil or dust contaminated with cadmium, inhalation of cadmium-containing particles from ambient air, inhalation of cigarette smoke, or working in an occupation involving exposure to cadmium fumes and dusts are all means of human exposure to cadmium (ATSDR, 1997-H010). Food is the major source of cadmium exposure for nonsmokers. Drinking water normally has very low levels of cadmium.

Occupational exposure to cadmium and cadmium compounds varies with the process in which cadmium is being used. IARC (V.58, 1993) summarized various occupational processes and exposure levels determined by ambient air monitoring and biological monitoring (IARC V.58, 1993).

The National Occupational Exposure Survey (NOES) conducted in 1981-1983 estimated that 93,681 workers were potentially exposed to cadmium or cadmium-containing substances; of these, 16,450 were female (18% of the surveyed workforce) (NIOSH, 1990). The industrial segments with the largest numbers of potentially exposed workers were “fabricated metal products” (15,077 workers, 4637 of which were female).

In its proposed rule for occupational cadmium exposure, OSHA in 1990 estimated that approximately 512,000 U.S. workers were exposed to cadmium, 81% of whom were exposed below a time-weighted average (TWA) of 20 $\mu g/m^3$ (70% below a TWA of 5 $\mu g/m^3$) (ATSDR, 1997-H010).

The four major sources of occupational exposure are smelting of zinc and lead ores; producing, processing, and handling of cadmium powders; welding or remelting of cadmium-coated steel; and working with solders that contain cadmium (ATSDR, 1997-H010). The major route of occupational exposure to cadmium is inhalation of dust and fumes and incidental ingestion of dust from contaminated hands, cigarettes, or food.

The metallothionein (MT) gene expression has been developed for use as a biomarker of heavy metal exposure (Ganguly et al., 1996). Reverse transcription-PCR (RT-PCR) was used to determine the level of MT-specific mRNA in blood samples from exposed workers and control subjects. There was a strong correlation between the airborne cadmium that registered on personal monitors and the blood levels of cadmium.

An association has been observed between the increase of the cadmium concentration in the urine, and abnormal values of several renal biomarkers (i.e., functional markers such as β_2-microglobulin in serum, urinary excretion of low and high molecular weight proteins; cytotoxicity markers such as tubular antigens and enzymes in urine; biochemical markers such as eicosanoids, glycosaminoglycans, and sialic acid in urine). Results of three studies support the conclusion that low-molecular-weight proteinuria indicates an exposure to a concentration of cadmium higher than the recommended biological threshold limit value. The authors noted that additional studies on biomarkers other than microproteinuria are needed to assess the validity of the use of biomarkers for identification of occupational exposure to cadmium (Lauwerys et al., 1995).

Exposure to cadmium may be monitored in blood or urine by atomic absorption spectrometry and polarography. Cadmium concentrations of 0.1 μg/L can be determined using graphite furnace atomic absorption spectroscopy. These methods are explained in detail by IARC (V.58, 1993, pp. 124-125). Neutron activation analysis (*in vivo* and *in vitro*) and x-ray fluorescence (*in vivo*) are used as techniques to measure cadmium in tissue (ATSDR, 1997-H010). The *in vivo* techniques are used to measure occupational exposure.

The U.S. Toxic Chemical Release Inventory collects cadmium data in two categories—"cadmium" and "cadmium compounds," and individual facilities may report releases in each category. In 1995, atmospheric releases of "cadmium" as reported in the Toxic Chemical Release Inventory totaled 11,939 lb (5.427 Mg). Of 45 facilities surveyed in 1995, 25 reported air emissions of cadmium. Sixteen reported emissions of less than 50 lb (0.023 Mg); 5 reported approximately 250 lb (0.113 Mg); 2 reported approximately 1000 lb (0.454 Mg); 1 reported 1881 lb (0.853 Mg); and one reported 6640 lb (3.012 Mg) (TRI95, 1997). Industries reporting releases of cadmium included the following categories: inorganic (SIC 2819) and agricultural chemicals (SIC 2879); plastic materials and resins (SIC 2821); national security (SIC 9711); and hydraulic cement (SIC 3241). Primary metal industries reporting releases were copper (SIC 3331); metal products, nec (SIC 3399); and nonferrous metals, nec (SIC 3339). Industries involved in recycling nonferrous metals (SIC 3341); metalworking (SIC 3351, 3356, 3362, 3369, and 3444); and electroplating (SIC 3353 and 3471) reported releases. The manufacturers of the following products also reported releases of cadmium or cadmium-containing compounds: batteries (SIC 3691 and 3692); rubber and plastic footwear (SIC 3021); and steel pipes and tubes (SIC 3317). Blast furnaces and steel mills (SIC 3312) are other sources in the iron and steel industry (TRI95, 1997).

The total air release of "cadmium compounds" reported separately in 1995 was 41,113 lb (18.65 Mg). Of 109 facilities reporting releases to air in 1995, 24 reported individual releases greater than 200 lb (0.091 Mg), 6 of which reported releases greater than 1000 lb (0.454 Mg) (TRI95, 1997). The industries reporting releases of cadmium compounds in air were classified in the following categories based on the type of product manufactured: metals (SIC 3331 and 3339), chemicals (including inorganic chemicals, nec) (SIC 2819), and custom compound purchased resins (SIC 3087). Reduction of air emissions is evident by atmospheric releases of cadmium (4775 lb) and cadmium compounds (39,889 lb) reported to the U.S. EPA in 1996 (TRI96, 1998).

Industry has increased cadmium recovery at primary smelters so release to the environment from industrial operations is expected to decrease (USDOI, 1991). There is significant inhalation exposure in industrial areas where zinc, lead, or copper smelters are located. The mean annual airborne concentration of cadmium in an area about 1 km from a zinc smelter was 0.023 $\mu g/m^3$. In the United States, ATSDR estimated that about 2 million lb of cadmium are emitted during cadmium production and up to another 2 million lb are emitted from the use of cadmium. Burning of fossil fuels, such as coal or oil, and the incineration of municipal waste materials contributes to the cadmium emitted in the air (ATSDR, 1997-H010).

Atmospheric cadmium is generally in the form of particulate matter. Combustion processes produce very fine cadmium-containing particles. The principal species in the air are cadmium oxide and some cadmium salts, which occur as stable forms in the environment. Cadmium compounds in atmospheric emissions are dispersed by winds and deposited by wet or dry processes (ATSDR, 1997-H010).

Cadmium concentrations in ambient air are generally less than 0.005 $\mu g/m^3$, but concentrations up to 0.5 $\mu g/m^3$ have been detected in air near cadmium-emitting facilities (Elinder, 1985a; cited by ATSDR, 1997-H010). Ambient air concentrations in rural areas are usually less than 1 ng/m^3, and in urban areas range from 3-40 ng/m^3 (ATSDR, 1997-H010; IARC V.58, 1993).

Cadmium intake from ambient air is less than 0.8 µg/day in non-industrial areas (WHO, 1989; cited by WHO, 1993).

In 1995, the total release of "cadmium" to surface water as reported in the Toxic Chemical Release Inventory was 458 lb (0.208 Mg). Of 9 facilities reporting releases to water, 8 reported a release of less than 100 lb (0.045 Mg) and 1 reported a release of 250 lb (0.113 Mg). Industries reporting releases of cadmium to water were classified into the following categories based on type of product produced: organic fibers (SIC 2824), petroleum refining (SIC 2911), electronic connectors (SIC 3678), chemicals (SIC 2869), primary metals (SIC 3339), secondary metals (SIC 3341), metalworking (SIC 3351, 3356, and 3362), and storage batteries (SIC 3691). The total release of "cadmium compounds" to waters in the United States was 650 lb (0.295 Mg): 21 facilities reported minor releases, and only one reported a release greater than 200 lb (0.091 Mg). The industries reporting releases of cadmium compounds to water were classified as manufacturers of primary metals (SIC 3331 and 3339) (TRI95, 1997). In 1996, a total of 1010 lb of cadmium and a total of 3614 lb of cadmium compounds were released to surface water (TRI96, 1998). Cadmium solubility in water depends on the acidity of the water. At lower pHs, suspended particles or sediment-bound cadmium dissolve (Ros and Slooff, 1987; cited by WHO, 1993).

The total reported U.S. land disposal releases of "cadmium" in 1995 were 19,938 pounds (9.044 Mg) based on reports from nine facilities, with releases from individual facilities ranging from <100 to 9946 lb (<0.045 to 4.51 Mg) (TRI95, 1997). In 1995, only five facilities were responsible for the total land releases of 49,119 lb of "cadmium compounds" (TRI95, 1997). In 1996, the total land release of "cadmium compounds" was 10-fold higher (502,027 lb). Five facilities were responsible for 97.3% of total land releases in 1996. An inorganic pigments manufacturer, not among the top five in 1995, was responsible for 67.4% of total releases to land in 1996. Total land release of "cadmium" was also higher in 1996 (51,420 lb) (TRI96, 1998).
The total underground injection of "cadmium compounds" (injected as a disposal method) was 109 lb (0.049 Mg) in 1995 (TRI95, 1997). In 1996, the total underground injection of "cadmium compounds" was 82 lb (TRI96, 1998).

Cadmium can leach into ground water from pipes and solder used on pipes or from chemical/hazardous waste sites. It has been estimated that the ground water in New Jersey has a median level of 1 µg Cd/L with a high level of 405 µg/L. Ground water surrounding waste sites was found to have a concentration of 6,000 µg/L (ATSDR, 1997-H010).

Cadmium is taken up and retained by aquatic and terrestrial plants and is then transferred to animals, where the cadmium deposits in the liver and kidneys. Cadmium in soil may exist in soluble form in soil water or in insoluble complexes with inorganic and organic soil constituents. Cadmium concentrations in soil tend to be greater when the pH of the soil is low. Increases in cadmium soil levels have resulted from application of municipal sewage sludge and phosphate fertilizers, which increases human exposures from food chain accumulation in plants and animals (ATSDR, 1997-H010).

Contaminated topsoil may be indirectly responsible for the greatest human exposure to cadmium due to the uptake of cadmium in soil into edible plants and tobacco. Topsoil may be contaminated by the application of phosphate fertilizers or sewage sludge. It has been found that uncontaminated top soil in the Unites States may contain an average of 0.25 ppm cadmium; levels up to 800 ppm have been found for soils in polluted areas (ATSDR, 1997-H010).

Consumer products may contain cadmium. The greatest potential for above-average exposure of the general public to cadmium is from smoking, which may double an individual's intake of cadmium; tobacco smokers are exposed to an estimated 1.7 µg Cd/cigarette (ATSDR, 1997-H010). Cadmium pigments have been used in the production of cosmetics. The European Economic Community (EEC) banned this use in 1990. Cadmium pigments have also been used in plastics and printing inks for paper and textiles. CPSC has investigated the consumer hazard posed by inks used in printed products and by the products themselves but found no cadmium in the inks or in the final consumer products (IARC V.58, 1993). Other consumer products include nickel-cadmium (nicad) batteries and cadmium plated fasteners (Budavari, 1996).

Food is the main source of human exposure for nonsmoking members of the population. In the United States, adult intake is estimated to be about 30 µg/day based on the Total Diet Study, with the largest contribution from grain cereal products, potatoes, and other vegetables; average cadmium levels in U.S. foods range from 2 to 40 ppb (ATSDR, 1997-H010). Some grains have been found to have a higher natural concentration of cadmium than others because of inherent genetic and physiological characteristics. The sunflower kernel, for example, does so, even when grown in uncontaminated soils, and habitual consumption will lead to an increased average intake of dietary cadmium. There is, therefore, potential for an increased body burden of the element (Reeves and Vanderpool, 1997). Fishermen who consume large amounts of fish from contaminated waters may also be exposed to higher levels of the metal, so much so that the EPA has recommended that it be monitored in fish and shellfish tissue samples collected as part of state toxics monitoring programs (ATSDR, 1997-H010). WHO (1989; cited by WHO, 1993) set the provisional tolerable weekly intake (PTWI) of dietary cadmium as 7 µg/kg body weight, which was reconfirmed in 1990 (WHO, 1993).

Cadmium can occur in drinking water, which may be contaminated by a cadmium impurity in galvanized zinc coatings or by cadmium in solders used to join copper pipes. A guideline value of 3 µg/L for all cadmium forms in drinking water and a maximum level of 10 µg/L in drinking and bottled water is recommended (WHO, 1984, 1992; 1992 paper cited by IARC V.58, 1993). Cadmium intake from drinking water is usually less than 2 µg/day (WHO, 1989; cited by WHO, 1993).

REGULATIONS

Under the Clean Water Act (CWA), the water quality criteria published by EPA for cadmium and its compounds for the protection of human health are identical to Safe Drinking Water Act (SDWA) standards of 10 µg/L. EPA's Carcinogen Assessment Group includes cadmium oxide, cadmium sulfide, and cadmium sulfate on its list of potential carcinogens. Under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), reportable quantities (RQs) have been established for cadmium and cadmium chloride. EPA issued a Rebuttable Presumption Against Registration (RPAR) for cadmium-containing pesticides under FIFRA. Also under FIFRA, there are labeling and reporting requirements. By 1997, all cadmium pesticides had undergone voluntary cancellation (OPP, 1997).

Cadmium and cadmium compounds are also regulated under the Resource Conservation and Recovery Act (RCRA) and Superfund Amendments and Reauthorization Act (SARA). Both RCRA and SARA subject cadmium and its compounds to reporting requirements. FDA, under FD&CA, has set a maximum concentration level of 0.005 mg Cd/L in bottled water and limits the amount of cadmium in color additives and direct food additives. In 1984, NIOSH recommended that exposure to cadmium be reduced to the lowest possible level (NIOSH, 1996). OSHA adopted permissible exposure limits (PELs) for toxic effects other than cancer for cadmium: 0.1 mg/m^3 as an 8-hr time-weighted average (TWA) for fumes, 0.3 mg/m^3 as a ceiling for fumes, 0.2 mg/m^3 as an 8-hr TWA for dust, and 0.6 mg/m^3 as a ceiling for dust; the standards were adopted by OSHA. OSHA regulates cadmium and certain cadmium compounds under the Hazard Communication Standard and as chemical hazards in laboratories. Regulations are summarized in Volume II, Table A-13.