Cadmium

A maximum acceptable concentration of 0.005 mg/L (5 µg/L) for cadmium in drinking water has been established on the basis of health considerations. Food is the main source of cadmium intake for individuals who are not occupationally exposed. A joint FAO/WHO expert committee has estimated a provisional tolerable weekly intake of cadmium for an adult to be from 0.4 to 0.5 mg. Because it would be difficult to reduce cadmium intake in food, intake from water should be as low as possible. Daily consumption of water containing the maximum acceptable concentration of cadmium would result in the ingestion of about 12 percent of the above estimated tolerable intake.

General

Cadmium is a silvery-white, lustrous, but tarnishable metal; it is soft and ductile and has a relatively high vapour pressure. Cadmium is nearly always divalent; chemically it closely resembles zinc and occurs by isomorphous replacement in almost all zinc ores. Most commonly it is found as the sulphide, also known as greenockite or cadmium blende, which is often associated with the zinc ore, sphalerite (ZnS); it is economically recoverable only when found in this form or associated with other non-ferrous metal ores, such as those of lead and copper. Canadian zinc ores contain from 0.001 to 0.067 percent recoverable cadmium; zinc concentrates normally include 0.1 to 0.3 percent cadmium, but levels up to 0.7 percent are known.

During the early part of this century, there was little demand for cadmium, and no attempt was made to isolate the element during zinc extraction. It therefore either remained as a contaminant of zinc products or was released to the environment during the processing of the zinc. Since the 1930s, worldwide demand for cadmium has increased steadily to the extent that consumption is now limited essentially by the concentration of cadmium in zinc ores and the supply of refined zinc. It has been estimated that, in 1982, Canada produced 890 tonnes of cadmium and consumed approximately 34 tonnes. Cadmium may also be imported as a component of lead and zinc concentrates, in cadmium commodities of unknown metal content, and as an impurity in other imported metals or alloys.

Cadmium is used primarily for electroplating other metals or alloys to protect them against corrosion. It is employed extensively in the production of low-melting-point alloys, solders, and low-cadmium copper. Cadmium is also used in Canada in the manufacture of stearate stabilizers for plastics, notably polyvinyl chloride, and pigments. Applications consuming lesser amounts of cadmium include the following: fungicides for golf courses, control rods and shields for nuclear reactors, television picture tube phosphors, nickel–cadmium batteries, motor oils, and curing agents for rubber. In one review, it was noted that the use of cadmium products has expanded in recent years at a rate of 5 to 10 percent annually, and the potential for further growth is very high.

Occurrence

Cadmium is a relatively rare element. It is uniformly distributed in the Earth’s crust, where it is generally estimated to be present at an average concentration of between 0.15 and 0.2 mg/kg. The ratio of cadmium to zinc may vary considerably and in most minerals and soils ranges from 1:100 to 1:1000. Cadmium occurs in nature in the form of various inorganic compounds and as complexes with naturally occurring chelating agents; organo-cadmium compounds are extremely unstable and have not been detected in the natural environment.

Although useful applications for cadmium have been recognized only in comparatively recent times, the metals with which it is commonly associated — copper, lead, and zinc — have been employed for several thousand years. Contamination of the environment has therefore been occurring throughout this period. With the discovery of new uses for cadmium, the problem has intensified. In addition to contamination from the known applications of cadmium or its compounds, the environmental burden of this element may be augmented through its unintentional presence in galvanized products, sewage sludge, and fertilizers.
introduction into the environment, cadmium has been designated “the dissipated element”. Industrial and municipal wastes are the main sources of cadmium pollution.

The solubility of cadmium in water is influenced to a large degree by the acidity of the medium. Dissolution of suspended or sediment-bound cadmium may result when there is an increase in acidity. The need to determine cadmium levels in suspended matter and sediments in order to assess the degree of contamination of a water body has been pointed out. The amount of cadmium in solution may be too small to be detected even when large concentrations are present in solids, especially under alkaline or neutral conditions.

The concentration of cadmium in unpolluted fresh waters is generally less than 0.001 mg/L; the concentration of cadmium in seawater averages about 0.00015 mg/L. Surface waters containing in excess of a few micrograms of cadmium per litre have probably been contaminated by industrial wastes from metallurgical plants, plating works, plants manufacturing cadmium pigments, textile operations, cadmium-stabilized plastics, or nickel–cadmium batteries, or by effluents from sewage treatment plants. Data reported for 2569 U.S. surface waters showed the median cadmium concentration to be less than 0.001 mg/L; the highest concentration reported was 0.13 mg/L. Cadmium concentrations at several sampling stations in Lake Erie and Lake Ontario did not exceed 0.002 mg/L and were often well below 0.001 mg/L; a survey of the lower St. Lawrence River estuary showed concentrations in this region to range from 0.0001 to 0.0056 mg/L (median, 0.0005 mg/L). The lower Fraser River and some of its tributaries were found to contain less than 0.001 mg/L except on two occasions when concentrations of 0.008 and 0.005 mg/L were observed.

Drinking water supplies contain low concentrations of cadmium (<0.001 mg/L) when they are drawn from unpolluted water sources. A survey of Canadian drinking water supplies published in 1979 stated that the maximum cadmium concentration in raw water was 0.00113 mg/L, and in distributed waters 0.00027 mg/L; the median concentration in each case was 0.00001 mg/L or less. More recently, a NAQUADAT survey of 3067 samples of raw water taken across Canada found only four samples containing cadmium at concentrations higher than the detection limit (0.01 mg/L), the highest concentration found being 0.061 mg/L.

In the course of a survey of Canadian diets, concentrations of cadmium in drinking water in five Canadian cities were all found to be less than 0.0001 mg/L, with a mean of 0.000044 mg/L. Dietary studies in the United States have found the mean concentration of cadmium in drinking water to be 0.0017 mg/L.

Data obtained for drinking water, either at the source or immediately after treatment at the municipal plant, may not be indicative of the cadmium concentration at consumers’ taps. Cadmium present as an impurity in galvanized pipes or as a constituent of solders used in fitting water heaters and water coolers may contaminate supplies during their distribution. This possibility will be enhanced where water supplies are soft and slightly acidic. Cadmium may also be extracted from black polyethylene pipes in which cadmium compounds are incorporated as stabilizers. Extraction from these sources is likely to be appreciable only when water has been standing for extended periods, and extracted cadmium would therefore be expected to be present in the first-drawn samples.

The vapour pressure of cadmium at a given temperature is greater than that of zinc and other metals with which it is commonly associated. Consequently, losses to the atmosphere during industrial processing of these metals are to be expected. Of the 560 tons of cadmium emitted to the atmosphere from various Canadian sources in 1972, it is estimated that 78 percent originated during primary copper and nickel production. The remainder came from other metal-processing industries (3.6 percent), fossil fuel combustion (18 percent), solid waste incineration, pesticide application, and wear of products containing cadmium.

High concentrations of cadmium in air are associated with heavily industrialized cities, notably those having refinery and smelting activities, where levels may be several hundred times those found in non-contaminated areas. The cadmium concentration of air in non-industrial areas is about 0.000001 mg/m³. Few data were found on cadmium levels in the air of Canadian communities. In a study conducted during August to September 1971, in the vicinity of the Surrert Battery Plant, Springhill, Nova Scotia, mean concentrations (averaged over a 24-hour period) at four stations ranged from 0.000007 to 0.000023 mg/m³. According to the 1969 data from the U.S. National Air Sampling Network, annual average concentrations at 29 non-urban stations were all less than 0.000003 mg/m³; those for the 20 largest cities ranged from 0.000006 to 0.000036 mg/m³. Levels averaged over shorter time periods may be higher in certain urban environments.

Data on the cadmium content of foodstuffs are plentiful, but the need for caution in interpreting some of the results generated by atomic absorption spectrophotometry has been emphasized by a number of
authors. Some reported levels were erroneously high owing to failure to remove the interfering element, sodium.

The presence of cadmium in vegetation may arise from deposition of cadmium-containing aerosols directly on plant surfaces and by absorption of cadmium through the roots. Plants vary in their tolerance to cadmium in soil and in the amounts they are able to accumulate; available data demonstrate that the normal concentrations of cadmium in foods are much lower than those found in vegetation grown on cadmium-contaminated soils or near cadmium-emitting industries. Certain shellfish, such as crabs and oysters, may concentrate cadmium to extremely high levels in certain tissues, even if they inhabit waters containing low levels of cadmium. Cadmium may also concentrate in the kidney and liver of swine, sheep, and cattle. The intentional use of cadmium in the manufacture or production of containers employed for storing or packaging of foods and beverages is prohibited in most countries, including Canada. The possibility for accidental contamination still exists, however, and cases have been documented where cadmium-plated or galvanized vessels have been improperly used for dispensing, storing, or processing of foods.

Reported concentrations of cadmium in foodstuffs vary widely; concentrations in most foods average about 0.05 mg/kg on a wet-weight basis. Concentrations in beef livers, kidneys, and brown crab meat can reach 0.2, 1.6, and 21.0 mg/kg, respectively. Other fresh meats generally contain less than 0.05 mg/kg; cadmium concentrations in fish are usually less than 0.02 mg/kg. In cadmium-polluted areas, cadmium levels may be significantly elevated; rice and wheat from contaminated areas of Japan have been found to contain concentrations near 1 mg/kg, at least a factor of 10 higher than those found in vegetation grown on cadmium-contaminated soils or near cadmium-emitting industries. A preliminary survey of Canadian freshwater fish indicated that, in general, the cadmium concentrations in species found near industrialized areas do not differ significantly from those in non-industrialized areas; concentrations found were always 0.06 mg/kg or less. The concentrations of cadmium in fruit juices, carbonated beverages, and wines available on the Canadian market were found to be less than 0.01 mg/L.

Canadian Exposure

The main source of cadmium intake is food. Estimates of the mean daily intake from this source have been made in a number of countries and range from 0.02 to 0.06 mg. A survey of Canadian diets found that the mean daily intake of cadmium was approximately 0.014 mg, with a range of 0.007 to 0.034 mg. Less than 4 percent of the mean intake was from beverages. Mean daily intakes in the United States have been found in the range of 0.010 to 0.040 mg.

Consumption of drinking water obtained from unpolluted sources results in only a small contribution to the total dietary intake of cadmium. Contamination of the source water or dissolution of cadmium from the piping material or other plumbing fixtures could result in a more appreciable contribution. The use of first-drawn water for infant feeding could result in a significant cadmium exposure for babies. From the limited data available, it appears that, in the majority of cases, water contributes less than 0.01 mg/day, assuming a maximum cadmium concentration of 0.005 mg/L and an adult daily consumption of 1.5 L. For some Canadians, however, it may provide in excess of 0.02 mg/day. Further studies on cadmium levels in drinking water as received by the consumer at the tap are required before an accurate assessment of exposure to cadmium in drinking water can be made.

Assuming a daily respiratory volume of 20 m³, airborne cadmium will probably contribute no more than 0.0006 mg to the total daily intake of persons residing in non-urban environments. Based on U.S. data, urban dwellers may inhale between 0.0001 and 0.0007 mg/day; those living in the vicinity of cadmium-emitting factories could be subjected to as much as 0.002 to 0.01 mg/day.

The daily exposure from food, water, and air for a Canadian adult that is not occupationally exposed therefore ranges from less than 0.01 mg to about 0.05 mg; levels in food depend to a large degree on cadmium levels in air and water, which in turn are a
function of geographical location. Smokers may be exposed to additional amounts. One cigarette contains approximately 0.001 mg of cadmium, of which 10 to 20 percent may be inhaled; smoking 20 cigarettes per day therefore results in an additional daily exposure of 0.002 to 0.004 mg. The total body burden and level of cadmium in the kidney have been estimated to be approximately twice as high in heavy smokers as in non-smokers. High cadmium levels detected in dust samples collected from houses in a small U.S. urban community were traced to the rubber mats or rubber-backed carpeting now used in many residential and other buildings. It was postulated that a child might ingest 0.002 mg of cadmium daily by placing dust-contaminated fingers in its mouth.

**Treatment Technology**

Cadmium can be efficiently removed from source waters by lime softening and coagulation with ferric sulphate. Lime softening removed over 98 percent of an initial cadmium concentration of 0.03 mg/L in the pH range 8.5 to 11.3; ferric sulphate coagulation removed more than 90 percent above pH 8, but only 30 percent at pH 7. Alum coagulation removed less than 50 percent in the pH range 6.5 to 8.3. Ion exchange is used industrially to remove cadmium from waste waters, and it has been reported that a home ion-exchange water softener removed 99 percent of the cadmium present in drinking water. Reverse osmosis has also been reported capable of removing 90 percent or more of the cadmium present in drinking water.

**Health Considerations**

Cadmium is not at present believed to be an essential nutrient for animals or man. Some preliminary studies, however, suggest that the presence of low levels of cadmium in diet may stimulate growth in mammals.

**Absorption**

Several studies on human subjects indicate that 4 to 7 percent of a single dose of ingested cadmium is absorbed from the intestine. The absorption of cadmium nitrate or cadmium chloride in animal studies ranged from 0.5 to 3 percent. In one study, cadmium was administered to rats in drinking water for several months, and less than 1 percent of the ingested amount was retained. Results of animal experiments also indicate that intestinal absorption is dependent upon age and diet. The amount absorbed depends on other components of the diet, such as iron, calcium, and protein. The total amount absorbed by humans has been estimated as 0.0002 to 0.005 mg/day. In animal studies, females have been found to absorb more dietary cadmium than males, and it has been reported that iron-deficient women absorbed up to 20 percent of the cadmium ingested.

The conditions governing pulmonary deposition, clearance, and absorption of cadmium from the lung are unknown. Although few quantitative data have been published, it has been estimated that approximately 25 percent of inhaled soluble cadmium compounds are absorbed. This value can vary considerably depending on the particle size and solubilities of the cadmium compounds. It has been suggested that absorption of cadmium inhaled in cigarette smoke is substantial. After deposition in the respiratory tract, some cadmium is moved by mucociliary action to the gastrointestinal tract.

**Distribution**

Absorbed cadmium accumulates mainly in the renal cortex and liver. The pancreas, thyroid, gall-bladder, and testes can also contain relatively high concentrations. Several studies suggest that accumulation of cadmium in the human body is a function of age; one author claims that there is a 200-fold increase in the cadmium content of the body in the first 3 years of life, and that in this early period humans accumulate almost one-third of their total body burden. The human placenta is an effective barrier to cadmium, and the body burden of the newborn is estimated to be less than 0.001 mg compared with 15 to 30 mg in an adult. Cadmium accumulates with age until a maximum level is reached at about age 50; the total body burden of a person of 50 years of age ranges from 5 to 40 mg. About half the body burden is found in the kidneys and liver; the cadmium concentration of the cortex of the kidneys ranges from 0.005 to 0.1 mg/g. Concentrations of cadmium in the renal cortex are normally 5 to 20 times those in the liver.

Within minutes of cadmium exposure in animals, the metal is present in the plasma of the blood, from which it is readily taken up by the liver and kidneys. Twenty-four hours after exposure, most of the cadmium is distributed in blood cells, probably bound to metallothionein, a metal-binding protein that is rich in cadmium and thionein. Metallothionein has been identified in the liver, kidneys, duodenum, urine, and blood of animals. It has been postulated that metallothionein passes through the red cell membrane and is transported to the kidney. Cadmium in the red blood cells is also released into the plasma when haemolysis occurs. Metallothionein may play an important role in the detoxification of cadmium; toxic effects probably result when the amount of metallothionein present in the liver is insufficient to bind with absorbed cadmium. An in vitro study has shown that human serum alpha-2 macroglobulin is also a cadmium-binding protein.
Excretion

Only a small proportion of absorbed cadmium (less than 10 percent in animal experiments) is eliminated, mainly in the urine and faeces. Negligible amounts are eliminated through hair, nails, and sweat. Daily excretion of cadmium for the “normal” adult has been considered to be 0.002 mg. For those who are occupationally exposed to cadmium, levels in the urine can be a few hundred times this value. Studies indicate that the excretion of cadmium occurs in three stages and that the half-life for the slow component of excretion is approximately 20 to 30 years.

Toxic Effects

The acute oral lethal dose of cadmium for man has not been established; it has been estimated to be several hundred milligrams. Doses as low as 15 to 30 mg from acidic foodstuffs stored in cadmium-lined containers have resulted in acute gastroenteritis. Consumption by humans of fluids containing 13 to 15 mg of cadmium per litre has caused vomiting and gastrointestinal cramps.

Acute cadmium poisoning has occurred following exposure to fumes during the melting or pouring of cadmium metal. Fatalities have resulted from a 5-hour exposure to 8 mg/m³, although some individuals have recovered after exposure to 11 mg/m³ for 2 hours. Acute pneumonitis resulted from inhalation of concentrations between 0.5 and 2.5 mg/m³ for 3 days. Symptoms of acute poisoning include pulmonary oedema, headaches, nausea, vomiting, chills, weakness, and diarrhoea.

A syndrome called Itai-itai, first described in Japan, has been associated with chronic ingestion of cadmium. The role that cadmium plays in the etiology of Itai-itai is not clear, and the dose of cadmium required to cause symptoms has not been determined. Symptoms of the disease, which occurred most often among elderly women who had many children, are the same as those of osteomalacia (softening of the bone); the syndrome is characterized by lumbar pain, myalgia, and spontaneous fractures with skeletal deformation. It is accompanied by the classical renal effects of industrial cadmium poisoning: proteinuria and often glucosuria and aminoaciduria. Daily cadmium intake by ingestion in the endemic area has been estimated to be 0.6 mg, but it would have been considerably greater before 1955, when pollution control measures were instituted in the nearby mine. Epidemiological studies carried out in other parts of Japan indicate that the incidence of proteinuria is significant in cadmium-polluted areas.

It has been suggested that there is a relationship between chronic ingestion of cadmium and hypertension, however, data available at the present time are inconclusive. The results of several studies suggest that persons with hypertension have more cadmium and a higher cadmium to zinc ratio in their kidneys than those without hypertension. In contrast to these findings, no relationship between cardiovascular disease and cadmium levels in the kidney was found in a study of 80 individuals at postmortem. In addition, no relationship between hypertension and urinary cadmium excretion has been observed.

Oral administration of cadmium has produced hypertension in animals; the dose–response curve, however, is not monotonic. The greatest effects are observed with oral doses of 0.01 mg/day or intraperitoneal injections of 0.0001 to 0.001 mg/kg. Doses an order of magnitude higher have little effect.

Chronic exposure to airborne cadmium results in a number of toxic effects; the two main symptoms are lung emphysema and proteinuria. Emphysema appears after approximately 20 years of exposure; levels of exposure that result in disability have not been systematically determined. In one study, exposure to cadmium concentrations of 3 to 15 mg/m³ produced emphysema. A renal disturbance that includes the excretion of low-molecular-weight proteins in the urine and an increase in amino acids, calcium, and glucose accompanies the emphysema. Study at autopsy has revealed that the principal renal effects of chronic cadmium poisoning are seen in the tubules but are pronounced only in the most severe cases. It has been proposed that the minimum critical level of cadmium in the kidney required to produce renal tubular damage is approximately 0.2 mg/g. There is some evidence of an increase in the incidence of renal stones in those with prolonged exposure.

In chronic cadmium exposure, bones appear to be secondarily affected or may be directly impaired before renal tubular damage develops. Liver function can be lowered by severe exposure; however, there have been few reports on liver diseases resulting from occupational exposure to cadmium.

Sex hormones, intake of calcium, proteins, or vitamin D, zinc, and selenium may modify the toxicological effects of cadmium. Zinc is antagonistic to cadmium, and it has been shown to prevent or reduce various experimental effects of cadmium administration, such as hypertension. foetal abnormalities, damage to ovaries, and testicular injury.

Teratogenic and Reproductive Effects

No report on teratogenic effects in populations exposed to cadmium is known, but cadmium compounds have been shown to be teratogenic in rats at doses of 1.25 mg/kg given parenterally; cadmium inhibits placental transport of zinc. Oral doses of 10 mg/kg per day for six weeks have produced birth defects in rats.
Mutagenicity

Both in vitro and in vivo studies have given conflicting results on the mutagenic properties of cadmium. On balance, the evidence suggests that some cadmium compounds are weakly mutagenic.(97)

Carcinogenicity

In animal studies, cadmium compounds have produced sarcoma at the site of injection and also interstitial cell tumours in the testes of rats.(98) Inhalation of cadmium chloride aerosol has produced a high incidence of primary lung cancer in rats.(98) However, the administration of cadmium salts by ingestion has not given rise to cancer.(97)

Epidemiological studies of workers occupationally exposed to cadmium have provided only inconclusive evidence that such exposure increases the risk of lung, prostate, and renal cancers. The results are difficult to interpret because of such confounding factors as the incidence of smoking and exposure to other possible carcinogens.(38,97) The U.S. Environmental Protection Agency has concluded that cadmium inhalation is dose-related to lung cancer in exposed workers but that there is no evidence that cadmium is carcinogenic by ingestion.(99)

Acceptable Daily Intake

The World Health Organization has recommended that the provisional permissible intake of cadmium not exceed 0.4 to 0.5 mg per week or 0.057 to 0.071 mg/day.(87)

On the basis of a multicompartmental model for cadmium distribution in the body and the estimated deviation of cadmium levels in the renal cortices of human populations, it has been estimated that a daily intake of 0.04 to 0.05 mg would lead to only 0.1 percent of the population reaching the "critical" cadmium concentration of 0.2 mg/g in the renal cortex after 50 years.(41,100)

Rationale

1. Cadmium is not considered to be an essential element in human nutrition. Food is the main source of cadmium intake for humans that are not occupationally exposed. Because it is difficult to reduce cadmium intake from food, the intake from water should be as low as possible.

2. A joint FAO/WHO expert committee has estimated a provisional tolerable weekly intake of cadmium to be between 0.4 and 0.5 mg. Daily consumption of 1.5 L of water containing cadmium at a concentration of 0.005 mg/L would result in the ingestion of about 12 percent of the provisional permissible intake.

3. The maximum acceptable concentration of cadmium in drinking water is therefore 0.005 mg/L.

References


Cadmium (09/86)


