Cadmium (II) chloride

Synonym: cadmium dichloride
CAS: 10108-64-2
MF: $CdCl_2$
FW: 183.32
Density 4.05 g/cm^3. Odourless.
Soluble in water (1.4 g/l at 20°C); freely soluble in acetone; slightly soluble in methanol and ethanol (ATSDR*, 1993).

Major uses and occurrence

Cadmium is a heavy metal used mainly in nickel-cadmium batteries (79% of cadmium consumption), in cadmium-containing pigments of deep red color for e.g. plastic, ceramics and glass, in dental amalgam, in electronic compounds, such as cadmium telluride (CdTe), and also in metal industries.

Cadmium presents as an impurity in other metals, such as zinc, lead, copper and iron, as well as in fossil fuels (coal, oil, gas, peat and wood). Cadmium is found in air, water, soil, food (the main sources are plants - about 98%, and about 1% is from fish and shellfish), and in cigarette smoke [1].

Cadmium (II) chloride is widely used in industry, e.g. in the manufacture of cadmium yellow pigments, in galvanoplasty, and in electroplating. Workers can be exposed to cadmium (II) chloride dusts and aerosols in battery manufacturing, electroplating, alloy and solder production, ceramics and vapor lamps production, and in welding. Furthermore, this cadmium salt is used in pesticides and fungicides.

Human toxicity

Cadmium and its salts (e.g. chloride, sulfate, oxide, and sulfide) are severe lung and gastrointestinal irritants that can be fatal by inhalation and ingestion. Acute ingestion of as little as 10 mg of cadmium chloride can cause pulmonary toxicity, such as pulmonary edema, emphysema, and bronchitis. Ingestion of more than 100 mg of soluble salt or inhalation of 4 mg of cadmium dust may be fatal [1].

Cadmium is a nephrotoxic agent; its effect on a proximal renal tubular function is manifested by increased cadmium in the urine, proteinuria, aminoaciduria and other kidney diseases. The nephropathy (renal damage) occurs at the cadmium concentrations above 200 μg/g kidney weight [2]. The minimum lethal dose (MDL) of cadmium metal is 1 g/70 kg person [3].

Cadmium chloride can cause the kidney dysfunction and liver injury (necrosis). Also cardiovascular toxicity, e.g. elevated blood pressure, arteriosclerosis, cardiac arrhythmia and coronary ischemia were reported.

Ingestion of cadmium chloride or inhalation of cadmium fumes can cause nausea, vomiting, weakness, headache, abdominal cramps, and diarrhea. Cadmium chloride is irritating to skin, eyes, digestive tract and respiratory passages [4].

Normal blood plasma cadmium concentrations in adults without excessive exposure are generally 0.001- 0.003 mg/l [2, 3]. Concentrations above 0.005 mg/l are warrant careful investigation [4]. The approximate lethal blood level is 0.5 mg/l [3].

Permissible exposure limit (PEL) from the OSHA** standards for cadmium is 5 μg/m^3 (8 hours TWA).

Threshold Limit Values (TLVs) for cadmium salts are 10 μg/m^3 (8 hours TWA) and 2 μg/m^3 (8 hours TWA) for respirable fraction (ACGIH***, 1996).

Carcinogenicity: category B1 (EPA****, 2004), probable human carcinogen, i.e. limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals. Increased incidences of lung, prostate, pancreas and bladder cancers in humans have been reported.

Kinetic data
Data about cadmium (II) chloride are restricted; only data about cadmium are available.

Absorption: average oral absorption of cadmium is 3 to 7%. Symptoms occur within 30 min to 3 h after ingestion. At the inhalation of cadmium/cadmium salt dust, absorption may be as high as 30 – 64 % [3]. Zinc decreases cadmium absorption probably by stimulating production of metallothionein (MT) [2]. MT is a cystein-based protein that transports metals such as copper, zinc, and cadmium in the body.

The biological half-life of cadmium in humans is estimated to be 10 to 30 years.

Plasma protein binding: cadmium is transported in blood bound to red blood cells and large-molecular weight proteins in plasma, particularly albumin. Most tissue cadmium is bound to MT [2].

The placenta provides a barrier for cadmium, at least during the last trimester [4].

Metabolism and excretion
There is no known physiologic need for cadmium in humans. Cadmium is not an essential nutrient, in contrast to copper and zinc.

Excretion: cadmium excretion occurs mainly in the urine, normally, 1 to 2 μg/day. Orally ingested or inhaled cadmium/cadmium salt is transported to the liver where it induces MT, which binds and detoxifies cadmium [2].

Toxicological mechanisms
Toxic effect of cadmium and its salts is not fully understood. Some of toxicological mechanisms are listed here: a) cadmium interferes with the uptake, distribution and action of zinc, which is an essential micronutrient; b) cadmium bound to the plasma protein, is transported and accumulated in the kidney. The kidney accumulates cadmium over a lifetime; c) cadmium may impair glucose transport in the kidney [5]; d) cadmium and its salts (chloride, sulfate) produces DNA-strand breaks, DNA-protein cross-links, oxidative DNA damage and chromosomal aberrations; e) cadmium can induce apoptosis (programmed cell death) in human T cells [6].

Target organs: kidney (especially at the acute poisoning), liver, and lung.

References
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2. *Casarett and Doull's Toxicology* (The Basis Science of Poisons) (1986) Klaassen, C.D., Amdur, M.O., Doull, J., eds., Macmillan Publishing Company, New York, USA.
3. Kaye, S. (1980) *Handbook of Emergency Toxicology: A Guide for the Identification, Diagnosis and Treatment of Poisoning*, 4th edn., pp. 139, also 232-235, Springfield, IL, USA.
4. Poisindex, Thomson Micromedex (2005).
5. Blumenthal, S., Lewand, D., Sochanik, A. (1994) Inhibition of Na(+)-glucose cotransport in kidney cortical cells by cadmium and copper: protection by zinc. Toxicol Appl Pharmacol 129, 177-187.
6. El Azzouri, B., Tsangaris, G., Pellegrini, O. (1994) Cadmium induces apoptosis in a human T cell line. Toxicology 88, 127-139.

* Agency for Toxic Substances and Disease Registry/US Department of Health and Human Services
** Occupational Safety and Health Administration/US
*** American Conference of Governmental Industrial Hygienists.
**** US Environmental Protection Agency

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