

## Mercury (II) chloride

**CAS 7487-94-7**

MF: C<sub>12</sub>Hg

MW: 271.52

log Kow= 0.22

**Solubility:** 6.9 g/100 ml water at 20°, and 48 g/100 ml water at 100°.

Soluble in acetic acid.

### Major use

Preserving wood and anatomical specimens; embalming; browning and etching steel and iron; intensifier in photography; white reserve in fabric printing; tanning leather; depolarizer for dry batteries; electroplating aluminum; mordant for rabbit and beaver furs; manufacturing of ink for mercurography; reagent in analytical chemistry; manufacturing of other Hg compounds; for freeing gold from lead; in magic photograms; staining wood and vegetable ivory pink; medication: topical antiseptic, disinfectant [1, 2].

### Human toxicity

Mercury chloride is one of the most toxic inorganic mercury salts; it is corrosive and nephrotoxic following ingestion. Salivation, metallic taste, abdominal pain, bloody diarrhea, proteinuria, and acute renal failure may occur. At higher doses, potentially fatal hypovolemic shock may occur [3].

Death from inorganic mercury salts ingestion is usually due to severe corrosive injury to the gastro-intestinal system, shock, cardiovascular collapse, and/or acute renal failure [3].

The estimated lethal dose of mercury chloride in a 70 kg adult is 1 to 2 g, or 10 to 42 mg mercury/kg. Fatalities have occurred from exposures as low as 0.5 g [3, 4].

*The concentrations of mercury chloride in blood* that have been reported to cause death in humans are in the range of 0.4-22 mg/l [5]. The mean lethal concentration, based on the data from several handbooks, is 2.6 mg/l [4].

*Carcinogenicity:* not classified as human carcinogen.

### Kinetic data

*Absorption:* Inorganic mercury compounds can be absorbed by any route, with the relative degrees of absorption being ingestion > inhalation >

dermal absorption. Absorption following ingestion of inorganic mercury is estimated as 2 to 38%. Other sources estimate absorption as <10%. The age of individual, diet and local effects (e.g. gastrointestinal corrosion) of the compound may also influence absorption [3].

*Volume of distribution:* >1 l/kg [4].

*Distribution:*

Mercury (II) chloride accumulates in blood, kidney, liver and heart [4]. Inorganic mercury does not cross the blood brain barrier or placenta as readily [3].

*The plasma half-life:* The elimination of mercury (II) chloride is biphasic. At overdose, 2 days (first phase) and 24-50 days (second phase) [4].

*Time to peak blood concentration:* not reported [4].

*Protein binding:* not reported [4].

*Passage across blood-brain barrier:* restricted [4].

**Metabolism and excretion**

Metabolites more toxic than mercury (II) chloride: none [4].

*Excretion:* inorganic mercury is excreted in the urine and feces [3].

**Toxicological mechanisms**

Mercury ions bind to sulfhydryl groups and also have an affinity to carboxyl-, amide- and amine-groups. The structure and function of key proteins and enzymes may be disturbed, receptor affinities altered, and cellular metabolism impaired, among other effects. Nonspecific cell injury or death may result [3]. There is limited human data and more extensive animal studies which suggest that toxicity of inorganic mercury is associated with renal toxicity, and that it is due to mercury-induced immunological effects [3].

**Target organs:** kidney (histopathological organ lesions), vascular system, gastrointestinal tract (histopathological organ lesions).

**References**

1. HSDB, TOXNET (2005).
2. ChemIDplus, TOXNET (2005).
3. Poisindex, Thomson Micromedex (2005).

4. Ekwall, B., Clemedson, C., Crafoord, B., Ekwall, B., Hallander, S., Walum, E. & Bondesson, I. (1998) MEIC Evaluation of Acute Systemic Toxicity: Part V. Rodent and Human Toxicity Data for the 50 Reference Chemicals, ATLA 26, 571-616.
5. Winek, C.L. (1994) Drug and chemical blood-level data. Winek's Toxicological Annual, Pittsburgh, Allegheny County Department Laboratories.

*Written by Cecilia Clemedson, August 2005; revised February 2007  
cecilia@stifud.se*