

Thallium sulfate

MF: Tl_2SO_4

MW: 504.8

Solubility in water: 48.7 g/l (20°C), 191.4 g/l (100°C) [1].

Tl^+

MW 204.37

Major uses

Thallium sulfate is used as a very effective rodenticide against mice and rats. Since this gift is very dangerous, it is forbidden in many European countries; however, it is still in extensive use in South America and Asia, and also to some degree in the Southern regions of the United States [2].

At one time thallium salts were used to produce depilation in dermatology, but it was abandoned because such use resulted in paralysis and fatalities of 10 to 15% of persons so treated [1, 3].

Thallium and its salts, such as sulfate, acetate and carbonate, are used in several industries, e.g. for manufacture of imitation jewelry pigments; of special alloy anode plates; of fireworks; in semiconductors, scintillation counters, optical lenses, low-temperature thermometers etc.[1].

Human toxicity

Thallium and its salts are extremely toxic in humans; and many cases of suicidal and homicidal poisoning have been recorded. The lethal dose varies considerably from case to case. The average lethal dose for thallium sulfate is approximately 1 g in the adult (8 to 15 mg/kg). In severe cases death does not usually occur earlier than after 8 to 10 days; most frequently between 10 and 12 days [2].

The symptoms of thallium sulfate poisoning are usually delayed 12 to 24 h in acute poisoning; the gastrointestinal tract and nervous system most often show the first signs of poisoning. The most typical symptoms are listed below [2, 4]:

- a) Gastrointestinal: nausea, vomiting, and abdominal pain;
- b) Neurologic: peripheral neuropathy, convulsions, muscle weakness, cranial nerve damage, delirium and coma; generally, the central, peripheral and autonomic nervous system damage;
- c) Cardiovascular: hypertension, dysrhythmias, bradycardia, and tachycardia;
- d) Respiratory: pulmonary edema and pneumonia, respiratory paralysis;
- e) Psychiatric: anxiety, insomnia, depression, dementia, psychosis, and permanent neuropsychiatric sequelae.

The most reliable tests for thallium are both blood and urine concentrations. For example, in 6 adults who survived the accidental or intentional ingestion of thallium sulfate, blood concentrations of 0.08-1.0 mg/l and urine concentrations of 1.4-4.1 mg/l were observed within 1-30 days after admission to the hospital [5].

In other study, the serum levels 12 to 24 h after the intake of 1 g of thallium sulfate were between 0.2 and 0.35 mg/l; and in the urine the levels were up to 12 mg/l, and falling to 1 to 1.2 mg/l after hemodialysis [2].

The average, time-related, clinically monitored lethal blood concentration of thallium in 5 cases was 5.4 mg/l (26.4 μ M) [6].

The threshold limit value for thallium as a time-weighted average (TWA) is 0.1 mg/m³ [4].

Carcinogenicity: No data are available to assess the carcinogenic potential of thallium sulfate (EPA, 2004) [4].

Kinetic data

Absorption: thallium sulfate is rapidly absorbed from the gastrointestinal tract and from the skin. Following ingestion of this salt, thallium can be detected in urine and faeces within one hour [1].

Kinetics is biphasic, at the overdose situation.

Distribution: In animal experiments it was shown that the distribution of thallium can be described by a three-compartment model, which is also applicable to the toxicokinetics in man. In the first phase lasting about 4 h, thallium is distributed through the entire central compartments, such as the blood as well as well-perfused peripheral organs and tissues. The second phase, lasting 4-48 h, involves distribution into the brain. The third phase, which occurs after 24 h, is determined mainly by the elimination of thallium from the body [1, 7].

Volume of distribution (V_d): between 3.6 and 5.6 l/kg [1]; 4.6 l/kg [8].

Time to peak (ingestion) is 2-4 h [8].

Elimination half-life is approximately 2-4 days [8]. According to another author the half-life of thallium in blood is about 8 days [9].

Metabolism and excretion

Thallium is not a normal constituent of animal and human tissues. The most important sources of thallium exposure in the general population are air emissions from coal-burning power plants, and copper, lead and zinc smelters. Humans are exposed through polluted atmosphere, drinking water and food. Thallium is widely distributed in the tissues, with the highest concentration in the kidney. The dietary intake of thallium has been estimated at the level about 2 µg/day [10].

Excretion is primarily via the kidney. Thallium is slowly excreted in the urine; large part is excreted via the feces. Usually thallium is still present in the urine 3 months after ingestion [4, 5].

Toxicological mechanisms

Several hypothetical mechanisms have been proposed: 1) Thallium causes enzyme inhibition by binding to sulfhydryl groups of mitochondrial membranes [8]; 2) Inhibition of mitochondrial oxidative phosphorylation; 4) Competition with the cellular transport mechanisms dependent on the action of Na⁺/K⁺-ATPase [3, 8].

The acute cardiovascular effects of thallium ions probably result from competition with potassium for membrane transport systems [4, 7].

Target organs: histopathological organ lesions in heart, kidney, liver and CNS; other organs: vascular system, peripheral nervous system [8].

References

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Written by Ada Kolman, December 2005; revised March 2007
ada.kolman@telia.com