

# Mercury

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## INTRODUCTION

Mercury (Hg) occurs in nature as the mineral cinnabar (red mercuric sulfide) and has found widespread use in industry. The commercial importance of mercury is based on its unusual combination of physico-chemical properties, such as high specific gravity, fluidity at normal temperatures, electrical conductivity, uniform volume expansion, toxicity giving pesticidal activity, and ability to alloy with other metals.

Mercury has three valency states and is found in the environment in the form of various inorganic and organic complexes and as the elemental metal.

## USES

Major uses are in electrical and electronic applications, in measuring and controlling instruments, dental amalgams and paint, and in the manufacture of chlorine and caustic soda. It was formerly used in the extraction of gold and in some pesticides/herbicides.

## OCCUPATIONAL EXPOSURE

Workplace environments presenting the largest potential sources of occupational exposure to mercury include chlorine-alkali production facilities, cinnabar mining and processing operations, and the manufacture and use of instruments containing liquid mercury. Occupational exposure is mainly via inhalation of vapour.

The occupational exposure standards set by Worksafe Australia (May 1995) are:

## Elemental (as Hg)

0.05 TWA ( $\text{mg}/\text{m}^3$ )  
- STEL ( $\text{mg}/\text{m}^3$ )

## Alkyl compounds (as Hg)

0.01 TWA ( $\text{mg}/\text{m}^3$ )  
0.03 STEL ( $\text{mg}/\text{m}^3$ )

## Aryl & inorganic compounds (as Hg)

0.1 TWA ( $\text{mg}/\text{m}^3$ )  
- STEL ( $\text{mg}/\text{m}^3$ )

## ENVIRONMENTAL EXPOSURE

Potential sources of mercury exposure for the general population include inhalation from ambient air, ingestion in water and foodstuffs, and dental and medical treatments. Of these, dietary exposure is the major source of non-occupational exposure. Average daily intake of mercury from food has been estimated to be 2 to 20  $\mu\text{g}$  in the USA.

The Food Standards Code prescribes limits for total mercury in foods of 0.5 ppm for fish and shellfish, and 0.03 ppm for all other foods. The Australian Market Basket Surveys have shown that the highest levels of mercury occur in seafood, mostly as methylmercury. Most other foods contain only very low levels of mercury almost entirely in an inorganic form.

From the 1994 survey results it was estimated that weekly intakes ranged from < 2.5  $\mu\text{g}/\text{kg}$  bw (body weight) in adults to < 4.8  $\mu\text{g}/\text{kg}$  bw in children. There has been little difference between Australian Market Basket Surveys done in 1984, 1985, 1990, 1992 and 1994.

The NHMRC Drinking Water Guideline (1996) value for mercury in drinking water is 0.001 mg/L. It is set on the basis of toxicity of methylmercury, as this is the most toxic form. It is likely that methylmercury would be less than 10% of the total mercury concentration. Levels of mercury in major Australian reticulated water supplies are usually less than 0.0001 mg/L but may range up to 0.001 mg/L.

The average atmospheric concentration of mercury worldwide has been estimated to be about 20 ng/m<sup>3</sup>, mostly present as mercury vapour.

The background range for mercury in soils is 0.001 - 0.1 mg/kg. The former use of mercury in processing of gold bearing ores has resulted in mercury contamination of soils around some gold mining areas and much higher levels of mercury may be encountered in these situations.

Dental amalgams are 50% mercury by weight. Increased breath levels of mercury ranging from 0.1 to 16.2 ng/L (mean 8.2) were detected in 167 people with dental restoration as compared to 0.008 to 0.1 ng/L (mean 0.06) in five people with no amalgams. It is very difficult to estimate exposure to mercury from amalgams as it depends on the number of fillings, surface areas, chewing, teeth grinding etc. However, in persons with a large number of dental amalgams it is possible this could represent a significant source of exposure to mercury.

Persons using skin lightening creams and soaps containing mercury are exposed to higher levels of mercury than the general population.

The Provisional Tolerable Weekly Intake (PTWI) for mercury has been set at 200µg for methylmercury in adults, which equates to 3.3 µg/kg bw/day (Joint FAO/WHO Committee on Food Additives, 1988). The committee noted that pregnant women and nursing mothers were likely to be at greater risk, although the available data were insufficient to recommend a specific mercury intake for these population groups.

There is transfer of inorganic mercury from blood to breast milk and a study in Sweden suggested that exposure of infants to mercury from this source would correspond to about half of the PTWI. The main source of mercury in milk was the mother's amalgam fillings (Oskarsson et al., 1996).

#### DOMESTIC EXPOSURE

Metallic mercury should generally not be used or stored in the home; however, exposure to metallic mercury in the home may result from the accidental breakage of glass thermometers. The volume of mercury in glass thermometers is usually small and providing the mercury is picked up and disposed of appropriately such breakage should not constitute a significant hazard to health. If a thermometer is broken, the mercury should be picked up using an eye dropper and then both the mercury and the eye dropper stored in an air tight container. Given the small amount of mercury involved, such material should be able to be disposed with household refuse.

If a larger volume of metallic mercury from another source is accidentally spilt, the area should be evacuated and well ventilated, and technical assistance sought to decontaminate the area and to dispose of the mercury. If poisoning is suspected, biological and environmental monitoring may be necessary.

#### TOXICITY

Toxicity of mercury depends on the form with alkyl > aryl or alkoxy > inorganic.

#### Inorganic mercury

Human and animal studies indicate about 80% of inhaled mercury vapour is retained by the body, whereas liquid metallic mercury is poorly absorbed by the gastrointestinal tract (<1%). Ingested inorganic mercury compounds are absorbed <10% on average but there is considerable variation.

In animal studies, the kidney and the central nervous system are the principal target organs for inorganic mercury. After exposure to mercury vapour or inorganic compounds, 50 - 90% of the body burden is found in the kidneys. The faecal and urinary routes are the main pathways for elimination of inorganic mercury in humans.

Acute mercury poisoning is usually by the soluble inorganic salts. Early signs and symptoms include pharyngitis, dysphagia, pain, nausea and vomiting, bloody diarrhoea and shock. Later, swelling of the salivary glands, loosening of the teeth, nephritis, anuria and hepatitis occur. Death results from effects on the gastrointestinal tract and/or kidney.

High levels of mercury vapour are extremely irritating to the lung and may cause erosive bronchitis, bronchiolitis or pneumonia. Subacute exposure to mercury vapour has given rise to psychotic reactions characterised by delirium, hallucinations and suicidal tendency.

Chronic mercury toxicity is more frequently seen than acute toxicity, due to its cumulative nature. Tremors, mental disturbances and gingivitis (inflammation of the mucous membranes surrounding the teeth) have been reported following occupational exposure. Both metallic mercury and mercury compounds have given rise to contact dermatitis.

#### Organic Mercury

Organic forms of mercury are subject to bioaccumulation and bioconcentration in the food chain and higher aquatic organisms may contain significant amounts of mercury.

Organic mercury compounds are more toxic than inorganic mercury compounds. Methylmercury compounds are almost completely absorbed by the gastrointestinal tract. Their greater lipid solubility means methylmercury compounds can cross biological membranes, especially in the brain, spinal cord, peripheral nerves and the placenta. In every animal species studied, the nervous system is a target of methylmercury, fetuses appearing to be at higher risk than adults.

The main effects of methylmercury poisoning are severe, irreversible neurological disorder and mental disability. In Japan, Minamata disease was caused by industrial pollution of Minamata Bay by

methylmercury and other mercury compounds, which accumulated in the fish which were subsequently eaten by humans.

The earliest signs were gradual decreases in the senses of touch, vision, hearing and taste. Numbness in fingers, toes, lips and tongue interfered with normal activities such as walking, eating, drinking and speaking. There was progressive loss of balance, tremors and incoordination through effects on the motor system. Behavioural changes and neurological disturbances were seen.

Methylmercury is fetotoxic in mice, teratogenic in rats and adversely affects behaviour of monkey offspring when mothers were exposed before and during pregnancy. In humans, prenatal exposure affects development of the nervous system. Delayed achievement of developmental milestones, and a history of seizures and abnormal reflexes were seen in infants born to women affected in the Iraqi poisoning episode where methylmercury treated wheat was eaten.

The clinical picture is dose dependent. At higher levels of maternal exposure the effect is of cerebral palsy. Microcephaly, hyperreflexia, and gross motor and mental impairment, sometimes associated with blindness or deafness, is the main pattern.

#### BIOLOGICAL MONITORING

Biological samples that can be monitored as evidence of exposure to mercury include urine, blood and hair. Urine is most frequently monitored to follow chronic exposure to inorganic mercury vapour.

In individuals with no occupational exposure, urine levels are reported to be in the range 0.004 - 0.12  $\mu\text{mol/L}$  (1.0 - 25.0  $\mu\text{g/L}$ ). Occupational exposure during the manufacturing of scientific glassware resulted in urine levels ranging from 0.04 - 3.49  $\mu\text{mol/L}$  (9 to 700  $\mu\text{g/L}$ ). Elevated urine and blood mercury levels have been reported among dentists. In occupationally exposed workers, exposure should be controlled to ensure urine levels are below 50  $\mu\text{g/g}$  creatinine (approximately 0.25  $\mu\text{mol/L}$ ).

Blood and hair are most commonly monitored to assess body burden following exposure to methylmercury compounds. Fish consumption will markedly affect these levels. Hair concentrations of methylmercury are proportional to blood concentrations at the time of formation of the hair strand. In general, the concentration in hair is 250 times the simultaneous concentration in blood.

Total mercury in blood and hair in individuals with no known exposure was reported to be 0.1 - 5.0  $\mu\text{g}/100\text{mL}$  and 1.0 - 5.0  $\mu\text{g/g}$ , respectively. A nearly linear relationship exists between methylmercury intake (40 - 230  $\mu\text{g/day}$ ) and blood mercury concentration. At equilibrium, for each 1  $\mu\text{g}$  methylmercury ingested per day, the blood mercury concentration increased 0.004  $\mu\text{mol/L}$  (0.8  $\mu\text{g/L}$ ).

Persons chronically exposed to alkylmercury exhibit paraesthesia as the earliest sign of poisoning at blood mercury levels greater than 1  $\mu\text{mol/L}$  (200  $\mu\text{g/L}$ ). Corresponding hair levels are greater than 50  $\mu\text{g/g}$ .

**LEGISLATION**

Mercury compounds for human and animal therapeutic uses are in Schedules 2, 4 or 6 of the Standard for the Uniform Scheduling of Drugs and Poisons. Mercury is in Schedule 7 **except**

- (a) when separately specified in this Schedule;
- (b) when included in Schedule 2, 4 or 6;
- (c) in preparations containing 0.01% or less of mercury in organic form as a preservative;
- (d) mercury (metallic) in scientific instruments;
- (e) dental amalgams; or
- (f) in a sealed device, for therapeutic use, which prevents access to the mercury.

Mercuric chloride when prepared for use for agricultural, industrial, pastoral or horticultural purposes is Schedule 7.

**REFERENCES**

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