

Mercury or compounds thereof

Definition of causal agent

Elemental mercury is a silver-grey liquid at room temperature which vaporises slowly. Mercury is produced mainly from the cinnabar ore (HgS). Secondary mercury is recovered from heating scrapped mercury-containing products and industrial waste. It readily forms amalgams with other metals. Its inorganic compounds are numerous and include oxides, sulphates, chlorides and nitrates.

Organic compounds (alkyl- such as (di)methyl mercury and aryl-mercury such as phenylmercury) are not considered today as major occupational risks. The most common compound is methyl mercury; the main source of exposure is non-occupational: biotransformation of inorganic mercury compounds into methyl mercury when in contact with water and soil explains high concentrations of organic mercury in fish and other sea foods. Seafood consumption increases the levels of organic mercury in blood.

Main occupational uses and sources of exposure:

Main forms of mercury at the work place are the elemental metallic mercury and its inorganic compounds. Organic compounds were previously widely used as fungicides, algaecides, insecticides and disinfectants.

Occupational elemental mercury exposure can occur in chemical industry in the production and reuptake of mercury compounds, in chloralkali industry, in the manufacture, maintenance, repair and extinction of measuring instruments, in the manufacture of lamps, in chemical processes using mercury as a catalyst, and in laboratories. Mercury amalgams were widely used in dentistry and mining industry uses mercury to amalgamate gold and silver. Main route of exposure is inhalation.

Inorganic mercury compounds are used as catalysts in plastic industry, as reagents in laboratories, in manufacture of galvanic batteries, and in chloralkali industry and exposure occurs in handling mercury-containing industrial waste (fluorescent lamps). Previously also used in felt hat industry and in the treatment of the fur. Main route of absorption is ingestion.

Toxic effects

Acute poisoning

- Respiratory tract (elemental mercury vapours). Massive exposure can cause cough, dyspnoea, chest pain, chemical bronchitis, bronchiolitis, pneumonitis and pulmonary oedema.

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- Oral cavity and gastrointestinal tract (mercury vapours and inorganic mercury compounds): metallic taste, excessive salivation, gingivitis and stomatitis, nausea, vomiting, abdominal pain and diarrhoea.
 - Skin rashes, non-allergic and allergic reactions (elemental mercury and its divalent inorganic compounds)
See documents on occupationally caused irritant and allergic contact dermatoses
 - Conjunctivitis (elemental mercury vapours).

1. Systemic effects

☐ Nervous system effects

Inhalation of elemental mercury vapours: headache, tremor, myoclonus and fasciculations, hallucinations, irritability, emotional lability, violent behaviour and suicidal tendency.

☐ Renal effects

Transient proteinuria, tubular impairment, and in severe cases, tubular necrosis and renal failure (elemental mercury and inorganic compounds).

Exposure criteria

for elemental mercury and inorganic compounds:

Minimum intensity of exposure:

Occupational exposure confirmed, and if possible assessed, by:

- history and study of the working conditions showing evidence of (sub) acute exposure to mercury,
- and, if available:
 - Biological monitoring (B-Hg preferred to U-Hg in acute exposures):
Inorganic mercury in blood (B-Hg-i) >18 µg/dl
Mercury in urine >500 µg/g creatinine
 - workplace air monitoring: Elemental mercury and inorganic compounds:
1 mg/m³.

Minimum duration of exposure: A few hours to a few days depending on the intensity of exposure.

Maximum latent period: 7 days.

Chronic poisoning

Inhalation of elemental mercury vapour causes predominantly kidney and nervous system toxicity. Inorganic mercury compounds affect mainly the gastrointestinal tract and the kidneys. Alkyl-mercury ((di)methyl mercury) causes nervous system toxicity by ingestion, inhalation or skin contact.

☐ Oral cavity and gastrointestinal tract

See **acute effects**. Dark mercurial line along the gingival margins, loosening or loss of teeth, alveolar destruction on radiographs, digestive disturbances, chronic gastritis, and gastroenterocolitis.

□ Nasal effects

Nasal irritation, epistaxis, disturbances of taste and smell.

□ Nervous system effects

Tremor at rest in eyelids, face, fingers and hands, and it may fluctuate in severity. The tremor is first intentional but in more severe cases postural.

Neuropsychiatric manifestations (erethism): emotional lability, excessive timidity, irritability, mental hyperactivity and outbursts of temper, anxiety, depression.

Cognitive dysfunction: difficulties in concentration, memory deficits, reduced psychomotor speed and precision.

Peripheral nervous system: sensory loss, decreased sensory and motor velocities on electroneuromyography.

General symptoms: insomnia, fatigue and headache.

In alkyl-mercury toxicity the sensory, visual, auditory and cerebellum functions are affected.

□ Renal effects

Renal damage leads to albumin and proteinuria (nephrotic syndrome). Membranous nephropathy, minimal change nephropathy and anti-glomerular basement membrane anti-body mediated renal disease may develop.

□ Dermatological effects

See **acute effects**.

□ Reproductive effects

Maternal exposure to alkyl mercury compounds, in particular methyl mercury during the first trimester of pregnancy may cause severe mental and motor retardation in children.

Exposure criteria: for elemental mercury and inorganic compounds;

Minimum intensity of exposure:

Occupational exposure confirmed, by:

- history and study of the working conditions showing evidence of prolonged/repeated exposure to mercury,
- and, if available:
 - biological monitoring (U-Hg preferred to B-Hg in stable chronic exposures):
Mercury in urine > 50 µg /g creatinine, early effects have been described > 35 µg /g creatinine.
 - workplace air monitoring showing levels well in excess of 0.02 mg/m³ (8 hour TWA)

Minimum duration of exposure: a few months to a few years depending on the intensity of exposure.

Maximum latent period: Late appearance of renal and central nervous system degeneration is possible. A latent period cannot be defined.