

Mercury Poisoning

Signs and Symptoms

Common symptoms of mercury poisoning include [peripheral neuropathy](#) (presenting as [paresthesia](#) or [itching](#), burning or [pain](#)), skin discoloration (pink cheeks, fingertips and toes), swelling, and [desquamation](#) (shedding of skin).

Because mercury blocks the degradation pathway of [catecholamines](#), [epinephrine](#) excess causes profuse sweating, [tachycardia](#) (persistently faster-than-normal heart beat), increased salivation, and [hypertension](#) (high blood pressure). Mercury is thought to inactivate [S-adenosyl-methionine](#), which is necessary for catecholamine [catabolism](#) by [catechol-o-methyl transferase](#).

Affected children may show red [cheeks](#), [nose](#) and lips, loss of [hair](#), [teeth](#), and [nails](#), transient rashes, [hypotonia](#) (muscle weakness), and increased sensitivity to light. Other symptoms may include [kidney](#) dysfunction (e.g. [Fanconi syndrome](#)) or neuropsychiatric symptoms such as emotional [lability](#), [memory](#) impairment, or [insomnia](#).

Thus, the clinical presentation may resemble [pheochromocytoma](#) or [Kawasaki disease](#).

An example of [desquamation](#) of the hand of a child with severe mercury poisoning acquired by handling elemental mercury is [this photograph](#) in Horowitz, *et al.* (2002)

Causes

The [consumption of fish](#) is by far the most significant source of ingestion-related mercury exposure in humans and animals, although plants and livestock also contain mercury due to [bioaccumulation](#) of mercury from soil, water and atmosphere, and due to [biomagnification](#) by ingesting other mercury-containing organisms.^[4] Exposure to mercury can occur from breathing contaminated air;^[5] from eating foods containing mercury residues from processing, such as can occur with [high-fructose corn syrup](#);^[6] from exposure to mercury vapor in mercury amalgam dental restorations;^[7] and from improper use or disposal of mercury and mercury-containing objects, for example, after spills of elemental mercury or improper disposal of [fluorescent lamps](#).^[8]

Consumption of whale and dolphin meat, as is the practice in [Japan](#), is a source of high-levels of mercury poisoning. Tetsuya Endo, a professor at the Health Sciences [University of Hokkaido](#), has tested whale meat purchased in the whaling town of Taiji and found mercury levels that are more than 20 times acceptable Japanese standards.^[9]

Human-generated sources such as coal plants emit approximately half of atmospheric mercury, with natural sources such as [volcanoes](#) responsible for the remainder. An estimated two-thirds of

human-generated mercury comes from stationary combustion, mostly of [coal](#). Other important human-generated sources include [gold production](#), [non-ferrous metal](#) production, [cement](#) production, [waste disposal](#), human [crematoria](#), [caustic soda](#) production, [pig iron](#) and [steel](#) production, mercury production (mostly for batteries), and biomass burning.^[10]

Small independent gold mining operation workers are at higher risk of mercury poisoning because of crude processing methods. Such is the danger for the [galamsey](#) in Ghana and similar workers known as *orpailleurs* in neighboring [francophone](#) countries. While there are no official government estimates of the labor force, observers believe twenty thousand to fifty thousand work as galamseys in Ghana, a figure that includes many women, who work as porters.

Mercury and many of its chemical compounds, especially [organomercury](#) compounds, can also be readily absorbed through direct contact with bare, or in some cases (such as dimethylmercury) insufficiently protected, skin. Mercury and its compounds are commonly used in chemical laboratories, hospitals, dental clinics, and facilities involved in the production of items such as fluorescent light bulbs, batteries, and explosives.^[11]

Mechanism

Mercury is such a highly reactive toxic agent that it is difficult to identify its specific mechanism of damage, and much remains unknown about the mechanism.^[12] It damages the [central nervous system](#), [endocrine system](#), [kidneys](#), and other organs, and adversely affects the mouth, gums, and teeth. Exposure over long periods of time or heavy exposure to mercury vapor can result in brain damage and ultimately death. Mercury and its compounds are particularly toxic to [fetuses](#) and infants. Women who have been exposed to mercury in pregnancy have sometimes given birth to children with serious birth defects (see [Minamata disease](#)).

Mercury exposure in young children can have severe neurological consequences, preventing nerve sheaths from forming properly. Mercury inhibits the formation of [myelin](#).

There is some evidence that mercury poisoning may predispose to [Young's syndrome](#) (men with [bronchiectasis](#) and [low sperm count](#)).^[13]

Mercury poisoning's effects partially depend on whether it has been caused by exposure to elemental mercury, inorganic mercury compounds (as salts), or organomercury compound

Elemental mercury

[Quicksilver](#) (liquid metallic mercury) is poorly absorbed by ingestion and skin contact. It is hazardous due to its potential to release mercury vapour. Animal data indicate that less than

0.01% of ingested mercury is absorbed through the intact [gastrointestinal tract](#); though it may not be true for individuals suffering from [ileus](#). Cases of systemic toxicity from accidental swallowing are rare, and attempted suicide via intravenous injection does not appear to result in systemic toxicity.^[12] Though not studied quantitatively, the physical properties of liquid elemental mercury limit its absorption through intact skin and in light of its very low absorption rate from the gastrointestinal tract, skin absorption would not be high.^[14] Some mercury vapour is absorbed dermally but uptake by this route is only approximately 1% of that by inhalation.^[15]

In humans, approximately 80% of inhaled mercury vapor is absorbed via the [respiratory tract](#) where it enters the [circulatory system](#) and is distributed throughout the body.^[16] Chronic exposure by inhalation, even at low concentrations in the range 0.7–42 µg/m³, has been shown in [case control studies](#) to cause effects such as tremors, impaired cognitive skills, and sleep disturbance in workers.^{[17][18]} Acute inhalation of high concentrations causes a wide variety of cognitive, personality, sensory, and motor disturbances. The most prominent symptoms include tremors (initially affecting the hands and sometimes spreading to other parts of the body), emotional lability (characterized by irritability, excessive shyness, confidence loss, and nervousness), insomnia, memory loss, neuromuscular changes (weakness, muscle atrophy, muscle twitching), headaches, polyneuropathy (paresthesia, stocking-glove sensory loss, hyperactive tendon reflexes, slowed sensory and motor nerve conduction velocities), and performance deficits in tests of cognitive function.^[14]

Organic mercury compounds

Compounds of mercury tend to be much more toxic than the element itself, and organic compounds of mercury are often extremely toxic and have been implicated in causing [brain](#) and [liver damage](#). The most dangerous mercury compound, [dimethylmercury](#), is so toxic that even a few [microliters](#) spilled on the skin, or even a latex glove, can cause death.^{[24][25]}

[Methylmercury](#) is the major source of organic mercury for all individuals.^[1] It works its way up the [food chain](#) through [bioaccumulation](#) in the environment, reaching high concentrations among populations of some species. Larger species of fish, such as [tuna](#) or [swordfish](#), are usually of greater concern than smaller species. The U.S. [Food and Drug Administration](#) (FDA) and the U.S. [Environmental Protection Agency](#) (EPA) advise women of child-bearing age, nursing mothers, and young children to completely avoid [swordfish](#), [shark](#), [king mackerel](#) and [tilefish](#) from the Gulf of Mexico, (Golden Tilefish from the Mid- and North-Atlantic present no risk), to limit consumption of [albacore \("white"\) tuna](#) to no more than 6 [oz](#) (170 [g](#)) per week, and of all other fish and shellfish to no more than 12 oz (340 g) per week.^[26] A 2006 review, conducted by Dr. Dariush Mozaffarian and Dr. Eric B. Rimm, of the risks and benefits of fish consumption found that for adults the benefits of one to two servings of fish per week outweigh the risks,

even (except for a few fish species) for women of childbearing age, and that avoidance of fish consumption could result in significant excess [coronary heart disease](#) deaths and suboptimal [neural development](#) in children.^[27] (Dr. Rimm has reported in the past that he has received payment or honoraria for presentations about food and diets from both the Culinary Institute of America and the International Chefs Association, among others.)^[27]

There is a long latent period between exposure to methylmercury and the appearance of symptoms in adult poisoning cases. The longest recorded latent period is five months after a single exposure, in the Dartmouth case (see [History](#)); other latent periods in the range of weeks to months have also been reported. No explanation for this long latent period is known. When the first symptom appears, typically [paresthesia](#) (a tingling or numbness in the skin), it is followed rapidly by more severe effects, sometimes ending in [coma](#) and death. The toxic damage appears to be determined by the peak value of mercury, not the length of the exposure.^[12]

[Ethylmercury](#) is a breakdown product of the antibacteriological agent ethylmercurithiosalicylate, which has been used as a topical antiseptic and a vaccine preservative (further discussed under [Thiomersal](#) below). Its characteristics have not been studied as extensively as those of methylmercury. It is cleared from the blood much more rapidly, with a half-life of 7 to 10 days, and it is metabolized much more quickly than methylmercury. It probably does not have methylmercury's ability to cross the [blood-brain barrier](#) via a transporter, but instead relies on simple diffusion to enter the brain.^[1]

Other exposure sources of organic mercury include phenylmercuric acetate and phenylmercuric nitrate. These were used in indoor latex paints for their anti-mildew properties, but were removed in 1990 because of cases of toxicity.^[1]

Diagnosis

Diagnosis of elemental or inorganic mercury poisoning involves determining the history of exposure, physical findings, and an elevated [body burden](#) of mercury. Although whole blood mercury concentrations are typically less than 6 µg/L, diets rich in fish can result in blood mercury concentrations higher than 200 µg/L; it is not that useful to measure these levels for suspected cases of elemental or inorganic poisoning because of mercury's short half-life in the blood. If the exposure is chronic, urine levels can be obtained; 24-hour collections are more reliable than spot collections. It is difficult or impossible to interpret urine samples of patients undergoing [chelation therapy](#), as the therapy itself increases mercury levels in the samples.^[28]

Diagnosis of organic mercury poisoning differs in that whole-blood or hair analysis is more reliable than urinary mercury levels.^[28]

Prevention

Mercury poisoning can be prevented (or minimized) by eliminating or reducing exposure to mercury and mercury compounds. To that end, many governments and private groups have made efforts to regulate the use of mercury heavily, or to issue advisories about its use. For example, the export from the [European Union](#) of mercury and some mercury compounds has been prohibited since 2010-03-15.^[29] The variability among regulations and advisories is at times confusing for the lay person as well as scientists.

^[30]

Country	Regulating agency	Regulated activity	Medium	Type of mercury compound	Type of limit	Limit
US	Occupational Safety and Health Administration	occupational exposure	air	elemental mercury	Ceiling (not to exceed)	0.1 mg/m ³
US	Occupational Safety and Health Administration	occupational exposure	air	organic mercury	Ceiling (not to exceed)	0.05 mg/m ³
US	Food and Drug Administration	drinking	water	inorganic mercury	Maximum allowable concentration	2 ppb (0.002 mg/L)
US	Food and Drug Administration	eating	sea food	methylmercury	Maximum allowable concentration	1 ppm
US	Environmental Protection Agency	drinking	water	inorganic mercury	Maximum contaminant level	2 ppb (0.002 mg/L)

The [United States Environmental Protection Agency](#) (EPA) issued recommendations in 2004 regarding exposure to [mercury in fish](#) and shellfish.^[31] The EPA also developed the "Fish Kids" awareness campaign for children and young adults^[32] on account of the greater impact of mercury exposure to that population.

Treatment

Identifying and removing the source of the mercury is crucial. Decontamination requires removal of clothes, washing skin with soap and water, and flushing the eyes with saline solution as needed. Inorganic ingestion such as mercuric chloride should be approached as the ingestion of any other serious [caustic](#). Immediate [chelation therapy](#) is the [standard of care](#) for a patient showing symptoms of severe mercury poisoning or the laboratory evidence of a large total mercury load.^[1]

[Chelation therapy](#) for acute inorganic mercury poisoning can be done with [DMSA](#), [2,3-dimercapto-1-propanesulfonic acid](#) (DMPS), [D-penicillamine](#) (DPCN), or [dimercaprol](#) (BAL).^[1] Only DMSA is FDA-approved for use in children for treating mercury poisoning. However, several studies found no clear clinical benefit from DMSA treatment for poisoning due to mercury vapor.^[33] No chelator for methylmercury or ethylmercury is approved by the FDA; DMSA is the most frequently used for severe methylmercury poisoning, as it is given orally, has fewer side effects, and has been found to be superior to BAL, DPCN, and DMPS.^[1] [Alpha-lipoic acid](#) (ALA) has been shown to be protective against acute mercury poisoning in several mammalian species when it is given soon after exposure; correct dosage is required, as inappropriate dosages increase toxicity. Although it has been hypothesized that frequent low dosages of ALA may have potential as a mercury chelator, studies in rats have been contradictory.^[34] [Glutathione](#) and [N-acetylcysteine](#) (NAC) are recommended by some physicians, but have been shown to increase mercury concentrations in the kidneys and the brain.^[34] Experimental findings have demonstrated an interaction between [selenium](#) and methylmercury, but epidemiological studies have found little evidence that selenium helps to protect against the adverse effects of methylmercury.^[35]

Even if the patient has no symptoms or documented history of mercury exposure, a minority of physicians (predominantly those in [alternative medicine](#)) use chelation to "rid" the body of mercury, which they believe to cause [neurological](#) and other disorders. A common practice is to challenge the patient's body with a chelation agent, collect urine samples, and then use laboratory reports to diagnose the patient with toxic levels of mercury; often no pre-chelation urine sample is collected for comparison. The patient is then advised to undergo further chelation.^[33] No scientific data supports the claim that the mercury in vaccines causes autism^[36] or its symptoms,^[37] and there is no scientific support for chelation therapy as a treatment for autism.^[38]

Chelation therapy can be hazardous. In August 2005, an incorrect form of EDTA used for chelation therapy resulted in [hypocalcemia](#), causing [cardiac arrest](#) that killed a five-year-old autistic boy.^[39]

Prognosis

Many of the toxic effects of mercury are partially or wholly reversible, either through specific therapy or through natural elimination of the metal after exposure has been discontinued.^[40] However, heavy or prolonged exposure can do irreversible damage, particularly in fetuses, infants, and young children. [Young's syndrome](#) is believed to be a long term consequence of early childhood mercury poisoning.^[41] Mercuric Chloride may cause cancer as it has caused increases in several types of tumors in rats and mice, while methyl mercury has caused kidney tumors in male rats. The EPA has classified mercuric chloride and methyl mercury as possible human carcinogens (ATSDR, EPA)

Detection in biological fluids

Mercury may be measured in blood or urine to confirm a diagnosis of poisoning in hospitalized victims or to assist in the forensic investigation in a case of fatal overdose. Some analytical techniques are capable of distinguishing organic from inorganic forms of the metal. The concentrations in both fluids tend to reach high levels early after exposure to inorganic forms, while lower but very persistent levels are observed following exposure to elemental or organic mercury. Chelation therapy can cause a transient elevation of urine mercury levels.^[42]