

# Chronic Mercury Poisoning

## A Brief Summary of the Science

Chronic mercury poisoning is an underdiagnosed condition described in the toxicology literature but not yet recognized by most physicians or institutions.

### Symptoms

Symptoms are nonspecific and varied, and may include chronic fatigue, chemical sensitivities, fibromyalgia, auto-immunity, immune dysfunction (including Lyme and Candida), diabetes, cardiovascular disease, allergies, digestive disorders, thyroid and adrenal problems, stress intolerance, infertility, insomnia, tinnitus, erethism, depression, psychiatric disorders, hearing loss, vision loss, and neurodegenerative problems.

Having multiple health problems suggests mercury as a root cause.

### Mechanisms

Mercury causes general oxidative damage, equivalent to premature cellular aging. Most notably this includes the oxidative chain reaction called lipid peroxidation, which affects cell membranes.

Mercury's other toxic mechanism is its proclivity to bind to sulfur, which is ubiquitous in the body — in membrane transport proteins, in signaling and receptor proteins, in structural proteins, and most importantly, in enzymes, which facilitate nearly every biochemical process in the body. Lastly, mercury binds irreversibly to selenium, which is a cofactor in several key enzymes.

At the cellular level, these mechanisms mean that many key processes like enzyme reactions and mineral assimilation are altered.

In a vicious cycle, mercury blocks detoxification enzymes and their mineral cofactors, thus causing increased retention of mercury itself as well as other toxicants.

Overall, outward symptoms appear non-specific and highly variable — they depend on biochemical individuality and on nutritional status. For example, a nutrient-dense diet and/or nutritional supplements can temporarily alleviate many symptoms of chronic mercury poisoning.

### No reliable diagnostic tests

Standard diagnostic criteria for mercury poisoning usually require a finding of elevated blood or urine mercury. Thus, there is a wide misunderstanding, even within medical institutions, about the meaning of such tests. In fact, elevated blood mercury reveals only recent, not chronic, exposure. Mercury resides only briefly in the blood before migrating to fatty tissues like the brain, where it cannot be measured directly except on autopsy, and where its half-life is estimated in decades.

Urine mercury tests measure excretion but reveal nothing about retention or toxicity.

With low sensitivity and high specificity, a porphyrins test can identify severe chronic mercury poisoning. But since porphyrins are easily destroyed by heat, light, or motion, and since they can also be normalized by antioxidants, the risk of false negatives is high.

A trace mineral analysis of hair can be informative, but there are no standard guidelines for interpretation, thus counter-intuitive results are easily misinterpreted. Specifically, since mercury alters mineral transport, hair mercury may appear low

when the body burden is high. But mercury poisoning can be inferred from a hair test if the results for most “essential minerals” appear abnormally high and/or low instead of average, suggesting altered mineral transport, the only known cause of which is mercury.

In summary, most cases of chronic mercury poisoning can only be diagnosed indirectly, based on symptoms and lab anomalies.

### Exposures

Sources of mercury are numerous. The mother's womb provides toxic mercury along with essential minerals. Dental issues like bruxism, malocclusion, oral acidity, and mixed metals affect the release of mercury vapor from dental amalgam. High-copper amalgams emit more mercury vapor. Improper removal of amalgam can yield severe exposure to this vapor. Combustion of coal and hazardous waste spreads mercury into the food chain — and levels of mercury in fish have increased significantly in recent years. Antibiotics can potentiate mercury's toxicity. Nutritional factors affect detoxification capacity — for example, zinc is required for many detox enzymes, and vitamin D induces certain detox enzymes.

### Genes

At least ten genetic polymorphisms — including the ApoE4 allele implicated in Alzheimer's — are associated with poor heavy-metal detoxification, yielding susceptibility to mercury poisoning. Dozens to hundreds more may exist, since mercury attacks proteins, which are coded by genes that vary among individuals.

### Amalgam illness: a mind-blowing epidemic?

Most human population studies of mercury dental amalgam show no association with health problems. Yet such studies lack the power and design necessary to detect associations between a chronic, low-dose toxicant and diseases that involve long latencies, genetic susceptibilities, and non-specific symptoms. For example, the key input — total mercury exposure and/or body-burden — is virtually impossible to assess. And genetic susceptibility has only recently been identified; it has not been evaluated.

Further, many existing studies naively use blood or urine mercury levels to represent body burden, thus are of little value. Similarly, many existing studies draw conclusions that assume a linear toxicity for mercury, even though mercury's ability to block detoxification pathways means that toxicity increases with time and with dose.

For investigating mercury toxicity, lab science is a better tool. Based on compelling research using lab animals and cell cultures, as well as a small number of human autopsy studies, mercury dental amalgam appears to play a primary role in many chronic diseases, particularly Alzheimer's, MS, and autism. It appears to play a synergistic role with other toxicants in Parkinson's and ALS.

### Treatment

Unlike many other neurodegenerative conditions for which it may be mistaken, chronic mercury poisoning appears to be curable. Some methods are more effective and more economical than others. Unfortunately, some methods can be dangerous, causing redistribution of mercury to the brain.

## References

### Overview:

Berlin M, Zalups RK, Fowler BA. Mercury. In: Nordberg G, Fowler RA, Nordberg M, Friberg LT, eds. Handbook on the toxicology of metals. 3rd ed. Amsterdam, Boston: Academic Press; 2007.

Weiner JA, Nylander. Aspects on health risks of mercury from dental amalgams. In: Chang LW, ed. Toxicology of metals. Boca Raton: Lewis Publishers; 1996.

### Clinical symptoms:

Gerstner HB, Huff JE. Clinical toxicology of mercury. J Toxicol Environ Health 1977 Jan;2(3):491–526.

Trakhtenberg IM. Chronic effects of mercury on organisms [translation]. U.S. Dept. of Health Education and Welfare Public Health Service National Institutes of Health; U.S. Govt. Print. Off. Washington; 1974.

### Problems with the old science

Mutter J, Curth A, Naumann J, Deth R, Walach H. Does inorganic mercury play a role in Alzheimer's Disease? A systematic review and an integrated molecular mechanism. J Alzheimers Dis. 2010.

Mutter J, Naumann J, Guethlin C. Comments on the article "The toxicology of mercury and its chemical compounds" by Clarkson and Magos (2006). Crit. Rev. Toxicol. 2007;37(6):537–549.

Mutter J, Naumann J, Sadaghiani C, Walach H, Drasch G. Amalgam studies: disregarding basic principles of mercury toxicity. Int J Hyg Environ Health. 2004 Sep;207(4):391-7.

Grandjean P, Budtz-Jørgensen E. An ignored risk factor in toxicology: The total imprecision of exposure assessment. Pure Appl. Chem. 2010 Jan;82(2):383–391.

### Relation to other illnesses

Bernard S, Enayati A, Redwood L, Roger H, Binstock T. Autism: a novel form of mercury poisoning. Med. Hypotheses. 2001;56(4):462–471.

Mutter J, Curth A, Naumann J, Deth R, Walach H. Does inorganic mercury play a role in Alzheimer's Disease? A systematic review and an integrated molecular mechanism. J Alzheimers Dis. 2010.

### Treatment

Catsch A, Harmuth-Hoene A-E. Pharmacology and therapeutic applications of agents used on heavy metal poisoning. In: Levine WG, editor. The Chelation of Heavy Metals. Oxford: Pergamon Press; 1979.

Andersen O. Chemical and biological considerations in the treatment of metal intoxications by chelating agents. Mini Rev Med Chem. 2004 Jan;4(1):11-21.