

Chronic Mercury Poisoning

A Brief Summary of the Science

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

Symptoms

Symptoms are nonspecific and varied, and may come and go in the early stages. Common symptoms include chronic fatigue, chemical sensitivities, fibromyalgia, autoimmunity, immune dysfunction (including chronic 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.

Mercury's broad toxic mechanism

The molecular mechanism of mercury toxicity is its high binding affinity for sulfhydryl (S-H). Sulfhydryl, also called thiol, is ubiquitous in the body — in membrane transport proteins, in signaling and receptor proteins, in structural proteins, in peptides like glutathione, and most importantly, in enzymes, which facilitate nearly every biochemical process in the body.

By blocking mitochondrial enzymes, mercury causes mitochondrial dysfunction, generating large amounts of free radicals (oxidative stress).

By blocking transport proteins in cell membranes, mercury alters the cellular uptake of minerals and other nutrients, including enzyme cofactors, thus causing functional nutrient deficiencies.

Mercury also displaces essential metals from their normal binding sites. When mercury displaces reactive essential metals like iron and copper, these can become free radicals.

Free radicals cause lipid peroxidation, a self-propagating chain reaction in which the unsaturated fatty acids in cell membranes are attacked, causing extensive tissue damage.

By promoting free radicals, mercury depletes glutathione, the body's most important antioxidant. Furthermore, mercury blocks several enzymes needed to recycle and use glutathione. Thus, by impairing the glutathione system, mercury causes increased retention of mercury itself (as well as other toxicants) in a vicious cycle of increasing toxicity.

Finally, mercury binds irreversibly to selenium, which is a cofactor in several dozen enzymes including those that

maintain the brain's redox balance. Thus, mercury overactivates brain enzymes, leaving the brain less able to rest and repair.

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

No reliable diagnostic tests

Medical textbooks address mercury poisoning as an acute (e.g., accidental) problem, rather than a chronic condition. Thus standard diagnostic criteria usually require a finding of elevated blood or urine mercury levels. However, blood levels reveal only recent, not chronic, exposure. Mercury resides only briefly in the blood before migrating to 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 essentially measure kidney levels, not body burden. Provoked urine tests (using a challenge agent like DMSA or DMPS) measure mercury that is readily mobilized but reveal little about intracellular retention or toxic effects. Ironically, individuals with severe chronic mercury toxicity may show low levels in blood, urine, feces, hair, and nails, due to poor excretion and high retention of mercury.

A porphyrins panel has adequate sensitivity and specificity to diagnose moderate to 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 may be high.

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

Exposures

Sources of mercury are numerous. The mother's womb concentrates toxic mercury, along with essential minerals, into the fetus. 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. Antibiotics can potentiate mercury's toxicity. Nutritional factors affect detoxification capacity — for example, zinc is required for many detox enzymes, and vitamin D induces protective metallothioneins.

Genes

In the past decade, at least twelve common genetic polymorphisms – including the ApoE4 allele implicated in Alzheimer's – have been shown in human studies to cause increased susceptibility to the toxic effects of mercury. Dozens to hundreds more such polymorphisms that convey susceptibility may exist, since mercury attacks proteins, which are coded by genes that vary among individuals.

Amalgam illness: a mind-blowing epidemic?

Historically, most human population studies of mercury dental amalgam have shown no clear association with health problems, so industry asserts that amalgams are safe. 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 susceptibilities have only recently been identified; they have 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.

Mercury's broad toxicity and ubiquitous presence make it a likely causal agent in the epidemics of chronic illnesses, including neurodegenerative and developmental conditions. But, under the influence of lobbyists, funding of mercury studies has been discouraged, perhaps because much exposure has been through medical or dental products.

Treatment

Unlike many other neurodegenerative conditions for which it may be mistaken, chronic mercury poisoning may 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.

The lipophilic antioxidant, alpha lipoic acid has some ability to chelate (to chemically bind at two or more binding sites) mercury and other toxic metals. Cilantro is chemically similar to alpha lipoic acid. Such chelators should be avoided by persons with dental amalgams lest the chelator equilibrate the dental mercury throughout the body and brain. Furthermore, due to the risks of

immediate or delayed neurological damage caused by redistribution of mercury, alpha lipoic acid and other chelators should not be administered by health practitioners. Even lipophobic chelators like DMSA and DMPS may cause neurological damage when the blood-brain barrier is leaky. Such chelators should only be used by individuals who understand the risks inherent in chelation.

The degree to which recovery is possible may depend on one's genetic ability to excrete mercury — which, unfortunately, is what allowed the toxicity in the first place.

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