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Mercury Risk Assessment: The Hidden Danger

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1. Introductory Overview

Food is the main source of human exposure to mercury – with much of it originating from atmospheric releases by combustion processes, including burning fossil fuel (especially coal) for energy, municipal solid wastes, and medical wastes. Anthropogenic sources are responsible for introducing an estimated 2,000 to 3,000 tonnes of mercury into the environment. Another 3,000-5,000 tonnes are released naturally from the degassing of the earth's crust and oceans (FDA 2003).

Most mercury released to the atmosphere is inorganic and is found in one of three forms: $Hg°$, $Hg^{2+,}$ and Hg_p (or particulate-bound mercury). Atmospheric mercury can travel hundreds of kilometers before being deposited (Blanton et al. 1975). Ambient levels are often less than 1 mg/kg and typically range from less than 0.02 to about 6 milligrams per kilogram (mg/kg). Mercury leaches somewhat slowly from soil; the concentration associated with soil particles is estimated to be about 10 to 100 times higher than that in the water between the particles. In air, concentrations of mercury can range from about 0.01 to 0.02 microgram per cubic meter ($\mu g/m^3$) (ATSDR 1999).

Upon deposition on terrestrial or aquatic environments, biological processes can transform the inorganic form to the more toxic and readily available organic form, methyl mercury (CH_3Hg). Levels of methyl mercury can increase up the food chain such that it is magnified in the higher trophic levels. Aquatic organisms, especially secondary and tertiary consumers, may contain significant levels.

Although some terrestrial food sources contain mercury, humans are exposed primarily through the consumption of fish and processed fish products. Individuals especially susceptible to mercury exposure include women of childbearing age who might become pregnant, pregnant women, their fetuses, infants, and young children. At high exposure levels, mercury can cause significant neurological damage, especially for the developing brain and central nervous systems of young children. It can also cause renal and gastrointestinal toxicity.

2. Risk Assessment Context

(The following summary highlights information from the mercury fact sheet in J. Peterson et al. [2004], which reflects data from the toxicological profile prepared by the Agency for Toxic Substances and Disease Registry [ATSDR 1999], the Integrated Risk Information System Database [EPA 2004], and other scientific sources.)

Upon ingestion, less than 0.01% of metallic mercury and less than 10% of inorganic mercury are absorbed from the gastrointestinal tract, while absorption of organic mercury can exceed 95%. Unlike organic mercury or inhaled mercury vapor, very little inorganic mercury penetrates the brain following exposure, including to mercuric chloride (Jugo 1976); however, retention is longest in the brain tissue.

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Mercury tends to stay in the body for weeks or months, accumulating in the kidneys and eventually leaving through the urine and feces. A body burden half-life of 1 to 2 months has been estimated for humans. By interrupting intracellular thiol processes, mercury promotes oxidative stress, lipid peroxidation, mitochondrial dysfunction, and changes in heme metabolism; mercuric chloride has been shown to depolarize the mitochondrial inner membrane and increase the formation of hydrogen peroxide (ATSDR 1999).

The nervous system is very sensitive to all forms of mercury, but metallic mercury and methylmercury are of most concern for the brain because they can enter it fairly easily. In addition to causing kidney damage when exposures exceed certain levels, mercury can affect the central nervous system, causing tremors, memory loss, and changes in personality, vision, and hearing. Children and fetuses are particularly sensitive, and effects can range from mild to severe impacts on behavior and learning depending on the exposure level. Although animal studies indicate that mercuric chloride and methylmercury can cause cancer in laboratory animals, these compounds have not been shown to cancer in humans. On the basis of animal data, the Environmental Protection Agency (EPA) has identified mercuric chloride and methylmercury as possible human carcinogens. As a note, mercury was previously used in leather production, and in the book *Alice in Wonderland* the Mad Hatter displays personality changes seen in people who made hats following that process in the 1800s and early 1900s.

The disorder of mercury poisoning in humans is commonly called "Minamata Disease." Various poisonings have been reported, including a tragic incident in Iraq in the early 1970s involving wheat seed that had been coated with methylmercury to limit fungal growth. Intended for planting, the seed was instead ground into flour and baked in bread. More than 50,000 people were exposed to some level of mercury; 6,530 were hospitalized and more than 450 died. Exposure of the fetus *in utero* to the methylmercury was evident in impacts to children born to mothers who had eaten the contaminated bread.

The EPA has developed toxicity values to estimate the risk of developing noncancer effects as a result of ingesting or inhaling mercury. These toxicity values are based on studies of workers exposed to metallic mercury and epidemiological studies of mothers and infants exposed to methylmercury, as well as studies of animals given relatively high doses of mercury compounds over their lifetimes; those values were then adjusted and normalized to a mg/kg-day basis for humans (e.g., for mercuric chloride and phenylmercuric acetate). The value developed for estimating noncancer effects from oral exposure is called a reference dose (RfD), which is an estimate of the highest dose that can be taken in every day without causing an adverse non-cancer effect. The RfD for mercuric chloride as mercury equivalents is 0.0003 mg/kg-day. (A reference concentration for inhalation has also been established.) To illustrate how this RfD can be applied, a 70-kg person could safely ingest 0.0003 mg of mercuric chloride every day for a lifetime without expecting any adverse noncancer effects.

3. Risk Management

Fish and shellfish have long been considered part of safe dietary guidelines and a healthy diet because they contain high-quality and healthy omega-3 fatty acids. The positive health effects of these fatty acids have been known for decades. However, recent findings that indicate mercury contamination of fish and shellfish is increasing have caused officials to reevaluate earlier dietary recommendations. It is interesting to note that fish with generally higher mercury concentrations (those at upper levels of the food chain) do not necessarily have the highest levels of omega-3 fatty acids (Table 1). It has already been recommended that people avoid large predatory fish from the higher trophic levels from certain areas, including swordfish, shark, king mackerel, tilefish, halibut, pollack and all forms of tuna (canned, fresh, and frozen). Fish at the lower trophic levels such as planktivores usually contain much lower concentrations of mercury. Blood levels of mercury correlate with the frequency as well as the timing of exposures to fish or fish products, that is, how recently they have been consumed (Mahaffey et al. 2004).

Fish	Average Level of Mercury *(ppm)*	Level of Omega-3 Fatty Acid *(g/100 g)*
Herring	0.01	2.34
Sockeye salmon	0.03	3.00
Mackerel	0.08	3.61
Albacore tuna	0.26	2.33
Swordfish	0.95	0.58
King mackerel	0.97	0.18
Shark	1.30	0.07
Tilefish	1.60	0.17

Tab. 1: Comparison of Mercury and Omega-3 Fatty Acid Levels in Selected Fish
Source: Modified from Mahaffey (2004)

4. Summary

Potential exposures and health risks associated with mercury are on the rise with the increasing combustion of fossil fuels and conversion of mercury from less available (Hg°, Hg^{2+}) to more available forms (CH_3Hg). As new data are developed to assess exposures and toxicity, previous advisory levels are being reevaluated to ensure public health protection. Important factors include the chemical form and exposure route, the nature of the exposure (ongoing or intermittent) and potential for recovery versus accumulated damage, and the nature of the exposed individuals (e.g., susceptible subgroups) and risk-benefit tradeoffs regarding other dietary benefits of fish and shellfish consumption compared with replacement foods.

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