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METHYLMERCURY IN FISHES AND SHELLFISHES: A PUBLIC HEALTH CRISIS?

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Abstract: This review covers the toxicity and public health concerns of methylmercury from edible tissues of fishes and shellfishes. Public health risk from methylmercury has been a subject of intense debate among scientific and medical communities. The relevant literature on the aspects of the source, toxicology, toxicokenetics, fetal brain developmental health risks, cancer risks and fish consumption limits has been discussed in detail and control measures of methylmercury has been suggested.

Keywords: Methylmercury, public health crisis, Toxicology, fish consumption, control measures

INTRODUCTION

Mercury is a heavy metal that can be toxic to humans and other organisms. Mercury occurs naturally in the environment, and exists in various forms including elemental or metallic mercury, inorganic, and organic mercury (IARC, 1993; ATSDR, 1999). Once mercury is released into the environment, it cycles through land, air, and water. Many streams and other water bodies in India have sediments containing mercury at relatively high levels.

The deposition of mercury in aquatic ecosystems is a concern for public and environmental health because inorganic mercury can be converted by microorganisms (bacteria and fungi) in the sediments into methylmercury, a particularly toxic form of mercury. The physical, chemical, and biological characteristics of water bodies affect the propensity for mercury to be converted to methylmercury. Methylmercury is taken up by organisms dwelling in the sediments and then subsequently by larger aquatic animals, including fish that feed on the smaller organisms. In this way, methylmercury accumulates or "biomagnifies" in the aquatic food chain, reaching the highest levels in fish and other organisms at the top of the food web. Concentrations of methylmercury in fish tissues can thus be orders of magnitude (*e.g.*, 10, 100, or 1000 times) greater than concentrations in water. Methylmercury accumulation in fish and seafood products is a growing global health concern that poses severe health risk to public (Griesbauer, 2007). Methylmercury is one of the six most serious pollution threats to the planet earth (Toxic Link, 2003).

Consumption of fish is the principal route of exposure to methylmercury. Virtually all fish contain mercury at some level, and most of the mercury in fish is in more toxic organic form, methylmercury. Whether consumption of fish is harmful depends on the concentrations of methylmercury in the fish and the amount of fish consumed. Fish accumulate methylmercury in their tissues, where it becomes strongly bound. Methylmercury is not removed from fish tissue by any practical cooking method (Morgan *et al*., 1997; Chicourel *et al*., 2001).

Human exposure to methylmercury causes a variety of adverse health effects, including developmental delays in children of exposed mother (Cohen *et al*., 2005) and deficit in neurocognitive function in adults (Yokoo *et al*., 2003). Blood methylmercury in individuals are strongly correlated with the frequency and types of seafood consumed (Mahaffey *et al*.,

2004). However, even for pregnant women, consuming seafood has a variety of health benefits when dietary methylmercury intake is known to be low (Daniels *et al*., 2004; Mozaffarian and Rimm, 2006). Regulatory agencies rely on information about how individuals are exposed to methylmercury to evaluate trade-offs among health benefits from fish consumption and potential risks to methylmercury exposure.

The World Health Organization (WHO) considers mercury among the top 10 chemicals of "major public health concern" (Sheehan *et al*., 2014). This WHO report also observed that total mercury in hair (THHg) and total mercury in blood (TBHg) are both validated biomarkers of methylmercury intake correlated with seafood consumption in general population.

Methylmercury research was impaired by inappropriate attention to narrow case definitions and uncertain chemical speciation. It also ignored the link between ecotoxicology and human toxicology. As a result serious delays affected the recognition of methylmercury as a cause of serious human poisoning in Minamata, Japan. Development neurotoxicity was first reported in 1952, but despite accumulating evidence, the vulnerability of the developing nervous system was not taken in risk assessment and other forms of uncertainty to cause an underestimation of methylmercury toxicity and repeated led of calls for research than prevention (Grandjean *et al*., 2010).

BACKGROUND

The first global study on mercury says that "India could be one of the dozen hot spots after the rise in mercury emissions over 30 years". Launching the Global Mercury Assessment Report in Nairobi, UNEP executive director Klaus Toepfer said action is essential. The report says that coal-fired power stations and waste incinerators account for about 1,500 tonnes, or 70 per cent of new quantified manmade mercury emissions annually. The biggest share of 860 tonnes is from Asia (Toxic Link, 2003). Despite its toxicity and related hazards, India imports mercury to the tune of 250 MT per year from various countries such as US, UK, Australia, Germany, Spain and Russia. India also faces the threat of becoming the dumping ground of mercury as most of the developed countries are driving mercury out of their system (Toxic Link, 2003).

In India, some of the major rivers tested for heavy metals by the Industrial Toxicological Research Centre (ITRC), Lucknow, were found to contain mercury in alarming levels (ITRC, 1998). Testing of seawater by the National Institute of Oceanography, Goa found increased concentrations in the Arabian Sea (Kaladharan *et al.*, 1999). Several studies on fish and prawns in Mumbai, Kolkata, and Orissa have reported alarming rates of mercury concentrations (Thejam and Haldar, 1975). Mercury content in drinking water in various parts of the country is above the international permissible limits.

The total mercury pollution potential from coal in India is estimated to be 77.91 tonnes per annum, if average concentration of mercury in coal is assumed to be 0.272 ppm. About 59.29 tonnes of mercury per annum is mobilized from coal-fired thermal power plants alone (CPCB, 2001). The five super thermal power plants in the Singrauli area, which supply 10 per cent of India's power, are responsible for 16.85 per cent or 10 tonnes per annum of total mercury pollution through power generation (Toxic Link, 2003).

SOURCES OF METHYL MERCURY

Mercury that is released into the air is mercury vapor or inorganic mercury. Once in the atmosphere, as a gas ultimately it is redeposited on the earth with precipitation. Once on the earth or in the waterways, it is incorporated into sludges or sediments, where it is methylated by microbial or abiotic processes into methylmercury. The plant and sedimentary materials containing methylmercury are consumed by small fish that are consumed progressively by larger fish and finally by humans. During the course of this progression a great increase in concentration occurs—known as bioaccumulation. This increase can result in concentrations of methylmercury in fish tissues that are hundreds of thousands of times higher than the levels of inorganic mercury in the water (Mahaffey, 1999). In general, more than 90 % of the mercury in fish is found as methyl mercury, but contents of methyl mercury can vary considerably between species. Predatory species that are at the top of the food chain and live a long time, may

accumulate higher levels of methyl mercury (Mania *et al*., 2012).

The form of mercury that typically bioaccumulates in fish is monomethyl mercury, which can constitute 85% of the total tissue mercury. The balance is the soluble, ionic form of mercury, Hg^{+2} which is commonly found in fish gut lining. However, in edible muscle tissue (fillet), the portion normally consumed, virtually all of the incorporated mercury is in the monomethyl form (Jones and Slotton, 1996). Bivalves appear to accumulate mercury in a manner different from fish. Mercury in these organisms accumulates principally as Hg^{+2} and only 15–20% of the total mercury is methylmercury. Consequently, a doubling of the most toxic form of mercury, monomethyl mercury, can occur in bivalves without producing a statistically significant change in concentration of total tissue mercury.

Methylmercury is bound to the amino acids in fish muscle and cannot be removed by food preparation (e.g., skinning the fish) or cooking techniques including removing the visible fat (Morgan *et al.*, 1997). Fish and shellfish are the dietary source of methylmercury, although trace amounts of total mercury may be detected in other dietary components [e.g., eggs, organ meats such as kidney (Larsen *et al*., 2002), or offal (Ysert *et al*., 2000)]. The methylmercury concentration in fish is determined by the feeding habits of the fish, the mercury concentration in the tissues of its prey, the fish's age, and place in the food chain. The concentration of methylmercury in fish and shellfish species ranges from <0.1 ppm for shellfish species to >1 ppm for high-end predatory fish including ocean fish [such as marlin (Schultz *et al*., 1976), sharks (Penedo de Pinho *et a*l., 2002) and tuna (Storelli *et al*., 2002), and certain freshwater fish e.g., walleye and northern pike (Gilmour and Riedel, 2000; Jewett *et al*., 2003). Consequently a person's mercury intake depends on the species of fish consumed, as well as the quantity of fish eaten.

Dietary intake is by far the dominant source of exposure to mercury for the general population. Fish and other seafood products are the main source of methylmercury in the diet. Methylmercury concentrations in fish and shellfish are approximately 1,000 to 10,000 times greater than in other foods, including cereals, potatoes, vegetables, fruits, meats, poultry, eggs and milk (U.S. EPA, 2001).

Individuals who may be exposed higher than average levels of methylmercury include those who consume large amounts of locally caught fish where mercury contamination is frequent and those routinely consume the meat and organ tissues of marine mammals (U.S. EPA, 2001).

TOXICOLOGY OF METHYLMERCURY

Methylmercury, is one organic form of mercury, can accumulate up the aquatic food chain and lead to high concentration in predatory fishes (U.S. National Research Council, 2000). The health effects from exposure to methylmercury have been evaluated in several instances of human exposure that occurred under acute and/or high-dose conditions in the 1950's, 1960's, and early 1970's (Gassell, 2000). In Japan, fish that were contaminated with very high levels of methylmercury following the discharge of mercury-containing factory waste were consumed in large quantities. In Iraq, poisoning occurred from the consumption of seed grain that had been treated with methylmercury. These incidents showed the nervous system to be the primary target organ for methylmercury and that the developing fetus is particularly vulnerable. Signs of toxicity experienced by adults included paresthesia (numbness and tingling); ataxia (loss of muscular coordination); visual, auditory, and other sensory disturbances; impairment of speech; and mental disturbances. In each of the poisoning episodes, many people experienced severe effects, and death occurred in adults and children. Subsequent studies documented abnormalities in the brain resulting from methylmercury poisoning. It was also noted that infants born from women who were exposed showed nervous system damage even when the mothers were only slightly affected or showed no signs of toxicity. Infants exposed during fetal development displayed cerebral palsy, altered muscle tone and deep tendon reflexes, and delayed developmental milestones (*e.g.*, walking and talking; IRIS, 1999). However, the amounts of mercury ingested in these episodes were much higher than levels commonly consumed in the U.S., and the more subtle effects from low-dose exposure to methylmercury have had to be

extrapolated from the data from acute high-dose poisonings. Office of Environmental Health Hazard Assessment (OEHHA) has listed methylmercury as a developmental toxicant under Proposition 65 since 1987 (Gassel, 2000).

TOXICOKINETICS OF METHYL MERCURY

According the Griesbauer (2007) methylmercury in fish and other sea foods is readily and almost completely absorbed from the gastrointestinal tract, where it forms a complex with amino acid cysteine. This new complex resembles a large neutral amino acid found in the body methionine, and can more easily gain entry into cells. Once in bloodstream, methyl mercury will accumulate in the brain and cause damage to the central nervous system. Methylmercury is naturally removed from the body over time. Eventually, this methylmercury-cysteine complex is transported to the liver where it is secreted into bile, after which enzymes break the complex down into its amino acid and methylmercury parts. Some of this methylmercury then comes in contact with the bacteria in the intestine and is broken down into inorganic mercury and carbon. The rest of the methylmercury that does not interact with bacteria is reabsorbed by the body and goes through the process again. It takes about 30 to 40 hours for the methylmercury to reach to the tissues of the body (Clarkson and Magos, 2006). This cycle is the reason it takes so long to rid the body of mercury and how it can accumulate in the blood. It can take up to a year for mercury level to drop significantly (CFSAN, 2004).

Methylmercury can pass across the placenta to the fetus and to the nursing children through breast milk. Excretion of methylmercury compounds is mainly in the bile, but also in feces, urine as well as breast milk. In humans, methylmercury compounds have a biological half-life of approximately two months (IARC, 1993).

OTHER HEALTH EFFECTS

Exposure to methylmercury most commonly occurs when people eat the kind of fish and shell fish that show significant levels of methylmercury in their tissues. Methylmercury is a powerful neurotoxin and people exposed to high levels may experience other adverse health effects (US EPA, 1997). In addition to neurologic effects, other health effects from exposure to methylmercury have been shown in studies on experimental animals. Kidney damage was reported in some species and several studies of mice exposed to methylmercury showed an increased incidence of tumors (IARC, 1993). However, health effects differ among species, and these effects resulted from exposure to high doses. It is generally thought that the nervous system is the most sensitive endpoint, particularly in the case of exposure to low doses of methylmercury. Non-neurological risks for adults associated with methylmercury, including the potential for adverse cardiac outcome, have not incorporated into risk assessments (Mahaffey, 2005).

CANCER RISKS

The United States Environmental Protection Agency (U.S. EPA) evaluates a chemical's potential for causing cancer and classifies chemicals into one of five categories depending on the likelihood that it is a human carcinogen. Group A designates those chemicals for which the evidence suggests it is a human carcinogen. Each subsequent group (*e.g.*, B, C) represents decreasing likelihood of carcinogenicity based on the supporting evidence (or lack thereof). U.S. EPA classified methylmercury as a possible human carcinogen (Group C) based on inadequate data in humans and limited evidence of carcinogenicity in animals Gassel (2000).

The World Health Organizations' International Agency for Research on Cancer (IARC) Working Group on the Evaluation of Carcinogenic Risks to Humans (1993) concluded that there is inadequate evidence in humans for the carcinogenicity of mercury and mercury compounds. The IARC (1993) determined, however, that there is sufficient evidence in experimental animals (based on mouse studies) for the carcinogenicity of methylmercury chloride. The IARC (1993) thus listed methylmercury compounds as possibly carcinogenic to humans (Group 2B classification). OEHHA added methylmercury compounds to the Proposition 65 list of carcinogens in 1996, based on the IARC classification. OEHHA is further evaluating this issue; however, numerical values for the cancer potency of methylmercury have not been developed at this time. The potential for carcinogenic effects

from exposure to methylmercury should be noted, but current understanding of the toxicology of methylmercury supports consideration of neurotoxicity as the principal and appropriate endpoint of concern (Gassel, 2000).

FISH CONSUMPTION LIMITS

United States Environmental Protection Agency in the Mercury Study Report to Congress (U.S. EPA,1997) emphasize that the typical consumer was not in danger of consuming harmful levels of methylmercury from fish and was not advised to limit fish consumption on the basis of mercury content. This advice is appropriate for typical consumers who eat less than 10 grams of fish and shell fish per day with mercury concentrations averaging between 0.1 and 0.15 ppm. At these rates of fish intake, methylmercury exposures are considerably less than the reference dose (RfD) of $1x10^{-4}$ mg/kg/day. However, eating more fish than is typical or eating fish that are more contaminated, can increase the risk to a developing fetus. Karagas *et al.* (2012) reported that there may have adverse effects on neurologic and other body systems at common low levels of exposure to methyl mercury. Impacts of methylmercury could vary by individual susceptibility or be confounded by beneficial nutrients in fish containing methyl mercury. Two large epidemiological studies designed to address the methylmercury at the low levels that typically occur from consumption of seafood has been conducted and reported. The results of these studies from Seychelles and Faroe islands are evaluated by various national and international organizations (Gassel, 2000).

PUBLIC HEALTH CONCERN

Methylmercury exposure results principally from consumption of fish and other sea food contaminated by methyl mercury from anthropogenic (70%) sources (Trasande *et al*., 2005). Fingernails and toenails at parturition are useful biomarkers for prenatal methylmercury exposure for mothers and fetuses, especially during the third-trimester of gestation (Sakamoto *et al*., 2015). Exposure to methylmercury can cause lifelong loss of intelligence in hundreds of Indian babies born each year and that this loss of intelligence exacts a significant economic cost to Indian society, a cost that amounts to at least hundreds and millions of rupees each year. Moreover, these costs will recur each year with each new birth cohort as long as dumpings and emissions of mercury are not controlled. The high costs of *in utero* exposure to methyl mercury are due principally to lifelong consequences of irreversible injury to the developing brains (Trasande *et al*., 2005).

Fetal exposure to large amounts of methylmercury from maternal consumption of fish results in a pattern of severe neurodevelopmental defects and fatalities. Chronic low-dose prenatal methylmercury exposure from maternal consumption of fish has been associated with more subtle decrements in several measures of neurological development, which may resemble a number of learning disabilities present in the overall population of children (Mahaffey, 2000).

CONTROL MEASURES FOR METHYLMERCURY

The methylmercury in fish and other sea foods and its assimilation by humans is a universally recognized potential health hazard. Control measures should be taken by calculating the risk to human health, particularly for fetal and neonatal development, the importance of fish in the riparian diet, the wide intra and inter-species variations in mercury content and seasonal fluctuations in diet (Lebel *et al.*, 1997). Human hair is a useful indicator of mercury exposure. We can determine methyl mercury concentrations by hair sampling in people who consume coastal fishes and other seafoods (Miling Li, 2014).

Methylmercury is controlled by World Health Organization, The U.S. Environmental Protection Agency, FAO/WHO Expert Committee, the U.S. Food and Drug Administration (FDA) in different countries (Yess, 1993). The World Health Organization has adopted the U.S. EPA levels for mercury and recommends that food with mercury concentrations of 0.5 mg/kg or more should not be sold for human consumption and Canadian Federal Consumption Guidelines for Mean methyl mercury levels in fish is also 0.5 mg/kg wet weight (Wagemann *et al*., 1998).

As per Lunder and Sharp (2014) methylmercury contamination levels vary widely among seafood

species. Predatory ocean fish generally accumulate the highest concentrations of mercury. The levels can vary widely within a species based on the age of fish and the region where they are caught. Freshwater fish from polluted regions can have high mercury levels.

Methylmercury in fish and seafood products will continue to be a challenging issue for governmental of all levels. Fortunately, the health benefits of fish in the diet can be attained by buying commercially caught fish or fishing in safe waters. Ultimately, underlying environmental contamination problems must be addressed (Goldman and Fartland, 1998). Therefore, for controlling contamination of fish and seafood products by methyl mercury, the following guidelines are required:

- To avoid harmful accumulation of methyl mercury in human body, the gills, the liver and the intestine should preferably be discarded while processing fish and other seafoods for consumption (Ipinmoroti *et al*., 1997).
- In aquaculture systems, regulatory authorities must intervene to prevent distribution of contaminated stock until the danger is passed. It is difficult to control chronic contamination of methyl mercury in aquaculture facilities which use polluted water supplies, leaching of agricultural or industrial mercury from treated or contaminated soils into surface waters and deposition from the atmosphere.
- A program of shared responsibility between central agencies and state governments should be established, where central agencies develop a set of monitoring and inspection practices while state governments should be responsible for site closures, issuing advisories; research and public education by government agencies and health professionals should be expanded; mandatory labeling should be considered for specific contaminants in fish and other seafood products (Ahmed *et al*., 1993).
- A capable route for monitoring of mercurial contaminant is the determination of methyl mercury in fish and seafood products and scalp hair or blood of seafood consuming human in polluted areas (Feng *et al*., 1998; Kehrig *et al*., 1998; Lipfert *et al*., 1996; Mahaffey and Mergler, 1998; Renzoni *et al*., 1998). Doctor can take samples and send them to a testing laboratory.
- Encouraging health professionals to inform pregnant women and nursing mothers about the potential dangers of consuming methylmercury-contaminated fish, and the potential impact of over-consumption of mercury containing fish on children's health (APHA, 1999).
- A public health intervention using a focused dietary advice combined with a hair-mercury analysis can substantially lower the neurotoxic mercury exposure among pregnant women without decreasing their overall intake of seafood (Kirk *et al.*, 2017).

While danger of methylmercury exposure may seem like a good reason to refrain from consuming fish, the benefits of eating fish and seafood may outweigh many of the risks. Fish is high in protein, low in saturated fats, and contains important nutrients such as heart healthy omega-3 fatty acids. One fatty acid found in fish oils, Docosahexaenoic acid (DHA), is one of the most important fatty acids for normal brain development and function (Sakamoto *et al*., 2004). It is possible that DHA may even counteract the negative effects of mercury though this relationship has not yet proven significantly (Griesbauer, 2007). Eating fish has also found to reduce the risk of heart attacks, lower blood pressure, and improve arterial health (Senkowsky, 2004).

CONCLUSION

It's high time for urgent policy intervention, both at the national and state levels, to correct our mercury consumption pattern. Meanwhile, a nationwide awareness drive with people's movements needs to be launched to reduce the mercury emissions in municipal and industrial waste streams. The ultimate goal is to eliminate mercury use. Several international pressure groups are also working to ban the use of mercury.

Fish and seafood products are the main protein diets. They are very delicious and useful for health and they have many consumers throughout of the world. Therefore, they must be safe for human, but some of chemical contaminants enter aquatic environment and then accumulate in seafood animals. Today, the main source of exposure to chemical contaminants

such as mercury is from methylation of inorganic mercury in bodies of fresh and ocean water, the ensuing bioaccumulation in the aquatic food chain, and the consumption of fish or other sea foods by humans. Mercury is one of the most toxic metals that can readily accumulate in tissues of fish and other seafood animals even if the concentrations in water and aquatic plants are low. The ingestion of seafood animals contaminated with methyl mercury is the leading cause of mercury poisoning in humans. Methyl mercury is taken up predominately from ingested food and in aquaculture systems generally, and in other systems the fish are fed formulated diets. The feeds will, or should, have low mercury contents, and the harvested products will thereby have low concentrations of mercury in their tissues for being harvested at a young stage and would be expected to have less body burden even if their foods contained mercury.

Nonetheless, fish and other seafood should be monitored for methyl mercury contaminant and its human health hazards. Therefore, authorities responsible or sentinels of food and environmental pollution should give more attention to assuring clean and safe sea foods and aquatic environments. So, Mercury contamination affects not only the aquatic ecosystems which are exposed to it, but can also have an impact on human health. Consequently, fish and other sea creatures could be served as alarms regarding to risks for seafood consumers and they need to reduce or eliminate sources of this contamination.

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