

INTERNATIONAL JOURNAL OF INSTITUTIONAL PHARMACY AND LIFE SCIENCES

Life Sciences

Original Article.....!!!

Received: 11-02-2013; Revised; Accepted: 03-11-2013

MERCURY AND ITS TOXIC EFFECTS ON LIVING ORGANISMS

Baby Joseph*, Jeevitha Mohan

Interdisciplinary Research Centre, Department of Biotechnology, Malankara Catholic College,
Mariagiri, Kaliakkavilai, Tamil Nadu, India

Keywords:

Mercury

For Correspondence:

Dr. Fr. Baby Joseph

Interdisciplinary Research
Centre, Department of
Biotechnology, Malankara
Catholic College, Mariagiri,
Kaliakkavilai, Tamil Nadu,
India

E-mail:

petercmiscientist@yahoo.co.in

ABSTRACT

Mercury, one of the most widely diffused and hazardous organ-specific environmental contaminants, exists in a wide variety of physical and chemical states. Nearly 10000 to 15000 tons of mercury and its compounds are produced per year. This element is classified as one of the most toxic metals, which are introduced into the natural environment by human interferences. This review focus on the characteristics of mercury, sources of mercury, Absorption, Distribution and Biotransformation of mercury, Mercury accumulation and the toxic effect of mercury on living organisms.

INTRODUCTION

Heavy metals are ubiquitous in the biosphere, where they occur as part of the natural background of chemicals. Anthropogenic activities have also introduced substantial amounts of them into the environment by mobilization from their natural insoluble deposits or environmental sinks (Chiesa et al., 2006). They represent a significant ecological and public health concern due to their toxicity and their ability to accumulate in living organisms. Mercury pollution in aquatic ecosystems has received a great amount of attention since the discovery of mercury as the cause of Minamata disease in Japan in the 1950s. Mercuric compounds are very toxic and the toxicity of mercury was known as early as the 16th century¹. Mercury is considered one of the most dangerous of the heavy metals because of its high toxicity, bioaccumulative properties and other deleterious effects of biota including genetic alteration or mutagenesis. (WHO 1999) There are many reports indicating its genotoxic potential in a variety of organisms including humans² and aquatic species³. In India, about 200 ton of mercury and its compounds are introduced into the environment annually as effluents from industries⁴.

Characteristics of Mercury

Mercury is a naturally occurring metallic element noted for its occurrence as a liquid at room temperature. Elemental mercury is a heavy, silver-white liquid metal that can be found at trace levels in many minerals with greater concentrations in fossil fuels. There are three forms of mercury in the environment: elemental, inorganic, and organic mercury. All forms of mercury are toxic. Elemental mercury occurs naturally in three valence states: elemental (Hg_0), monovalent-mercurous (Hg^{1+}), and the divalent mercuric (Hg^{2+}). Elemental mercury is the most stable form and is only slightly water-soluble. Both mercuric and mercurous mercury are thermally unstable and readily decompose to elemental mercury. Vapors of elemental mercury can occur at room temperature presenting a hazard if spills occur. Inorganic mercury compounds contain ionic mercury usually in a salt formation (e.g. mercuric chloride). Inorganic mercury compounds continue to be used globally as disinfectants and pesticides. Organic mercury compounds can be chemically synthesized or biologically converted from mercury compounds by bacteria (e.g. methyl mercury). Some organic mercury compounds are water-soluble and cable of transport in the aquatic food chain through the process of bioaccumulation.

SOURCES OF MERCURY

Sources of mercury to the environment are natural and anthropogenic. The natural sources include volcanic emissions, windblown dust from continental areas, and the emission of gaseous Hg from

soils, vegetation, and the ocean. Anthropogenic mercury releases are mainly from industrial processes and combustion sources. Fossil fuel combustion, mining/smelting, and waste incineration are the major anthropogenic sources of mercury^{5, 6}. Anthropogenic emissions of mercury are from use of fossil fuels (especially coal), and other extracted, treated, or recycled mineral materials as well as from mercury used intentionally in products or processes. Mercury has been used in thousands of products and industrial processes including chlorine and caustic soda manufacture; use in laboratories; paint manufactured before 1991; electronic uses such lighting (e.g. fluorescent lamps), wiring devices and switches and batteries; thermometers, thermostats, barometers, and other related instruments; and dental supplies (e.g., dental amalgam fillings) and medical equipment.

SITE FOR MERCURY ABSORPTION IN FISH

In the fish, the possible areas of absorption of dissolved metals are the gills (respiratory tract), the intestine (ingestive intake) and the skin (transcutaneous uptake).

ABSORPTION, DISTRIBUTION AND BIOTRANSFORMATION OF MERCURY TO VITAL ORGANS:

Heavy Metals such as cadmium and mercury are absorbed from polluted water through gills, skin and digestive tract of fish. Liquid metallic Hg is slowly absorbed by the gastrointestinal tract (approximately 0.01%) and is considered to be of no toxicologic consequence⁷. Although Hg is poorly absorbed if indigested, Hg vapor is effectively absorbed through the lungs and quickly passes the blood-brain barrier⁸. Due to its lipophilic nature, Hg has a high affinity for myelin and lipid membranes. Once inside a cell, Hg is oxidized by catalase to the highly reactive Hg²⁺. MeHg derived from fish⁹ and demethylation can also more commonly produce Hg. Once assimilated in the cells, Hg²⁺ and MeHg⁺ form covalent bonds with glutathione and cysteine residues of proteins as well as other sulfur-containing molecules. Gastrointestinal absorption of Hg²⁺ is about 15% while MeHg is about 90%. MeHg has greater affinity for the brain, especially the posterior cortex. Table.1 describes the transport of mercury species throughout the body.

Table 1: Distribution of Mercury Species in the Human Body

| Elemental Hg | Ionic Hg | MeHg |
|---|--|--|
| Absorption results in rapid diffusion across the lungs and entrance into the bloodstream, where it is distributed throughout the body (because it is lipophilic), including the blood-brain barrier and the placenta. | The ingested dose is rapidly distributed from the the GI tract to the blood and organs. Mercuric Hg has a high affinity for sulfhydryl groups in the RBCs and plasma. The highest concentration is in the kidneys. Mercuric mercury induces metallothionein production in the kidneys, which may contribute to the kidney's accumulation of mercuric mercury. It does not cross readily cross the blood-brain barrier or the placenta because of its ionic charge. | The percentage of absorbed MeHg from the GI tract that is distributed to the blood ranges from 1% to 10%. About 5% is absorbed into the bloodstream and is distributed to all tissues within a few days. The concentration in RBCs is roughly 20X the concentration in plasma. Maximum levels (~10%) occur in the brain, in 5-6 days. It is also readily transferred to the fetus and the fetal brain. The high mobility of methyl mercury in the body is not due to lipid solubility. It is present in the body as water-soluble complexes mainly attached to the sulfur atom of thiol ligands. MeHg transport across the blood-brain barrier occurs via a MeHg-L-cysteine complex, which is transported by the L-system (leucine preferring) amino acid carrier. |

BIOTRANSFORMATION

Table 2 delineates the metabolic pathways of mercury species within the body.

Table 2. Biotransformation of Hg in the Human Body.

| Elemental Hg | Ionic Hg | MeHg |
|--|---|--|
| Elemental Hg is oxidized in the red blood cells by catalase and hydrogen peroxide to divalent ionic (mercuric) Hg. | Mercuric Hg is unstable in vivo and has been shown to convert to elemental Hg (rat study). It can also be methylated by intestinal flora, but cannot be methylated in body tissues. | MeHg is stable in the body compared to other species. It is slowly demethylated to mercuric Hg in tissue macrophages, intestinal flora, and the liver. It is metabolized to ionic mercury. The mercuric Hg resides for long periods of time in the CNS, probably in an inert form. |

MERCURY BIOACCUMULATION

Persistent hydrophobic chemicals may accumulate in aquatic organisms through different mechanisms: via the direct uptake from water by gills or skin (bioconcentration), via uptake of suspended particles (ingestion) and via the consumption of contaminated food (biomagnification).

As mercury elimination rates by organisms are very low, its concentration through food chains tends to increase¹⁰. Fish and shellfish tend to contain high concentrations of mercury in relation to other animals, and over 90% is in the form of methyl mercury, principally because fish feed on aquatic organisms that contain this compound¹¹. The amount of mercury in fish is normally correlated with a number of factors including the size and age of the fish, its trophic position, as well as the mercury content in water and sediment and the pH of the water¹¹.

CELLULAR ACTION

Mercury binds to sulfhydryl groups within proteins in the body. Proteins are long chains of amino acids linked together. One of these amino acids, cysteine, contains sulfhydryl groups. The sulfhydryl groups within cysteine function to form “cross-links” or disulfide bridges between two cysteines. These cross-links are what give proteins three-dimensional structure. When mercury binds to the sulfhydryl groups, the disulfide bonds are broken and the protein loses its structure and is rendered non-functional.

TOXIC EFFECT OF MERCURY

Mercury is toxic to all organ systems, particularly the nervous system and is also a mutagen, a teratogen and possibly a carcinogen that can also cause growth deficits locomotory and coordination impairments, loss of appetite, lower reproductive success and ultimately death^{12, 13}. The brain is the primary target tissue for mercury toxicity. Kidney is the critical organ following the ingestion of mercury salts¹⁴. Mercury can cause biochemical damage to tissues and genes through diverse mechanisms, such as interrupting intracellular calcium homeostasis, disrupting membrane potential, altering protein synthesis, and interrupting excitatory amino acid pathways in the central nervous system¹⁵. Mitochondrial damage, lipid peroxidation, microtubule destruction,¹⁵ and the neurotoxic accumulation of serotonin, aspartate, and glutamate are all mechanisms of methylmercury neurotoxicity¹⁶.

IONIC MERCURY

- Induction of C-mitosis with inactivation of mitotic spindle resulting in Aneuploidy and polyploidy.
- Chromosomal Aberrations and micronuclei in peripheral lymphocytes
- Increase in sister chromatid exchanges

Methyl Mercury

- Abnormal mitosis
- Single-strand breaks in cellular deoxyribonucleic acid.
- Spindle disturbances

CONCLUSION

Mercury pollution in aquatic ecosystem has received a great amount of attention since the discovery of Minamata disease in Japan in 1950s. Large quantities of mercury are released into the aquatic environment. Mercury is considered as one of the most dangerous of the heavy metals because of its high toxicity, bioaccumulative properties. From this review it is understood that how the heavy metal mercury reach the environment and how it is absorbed, distributed and biotransformed in the living organism and the toxic effects of heavy metal mercury on living organisms.

REFERENCES

1. Ramalingam U., Paneerdoss S., Girija M., Ilango, S., Mercuric chloride induced changes in the histology of the testis and serum testosterone in adult albino rats, *Pollut. Res*, 2001; 20: 439–442.
2. Betti C., Davini T., Barale R., Genotoxic activity of methyl mercuric chloride and dimethylmercury in human lymphocytes, *Mutat. Res*, 1992; 281: 255–260.
3. Bolognesi C., Landini E., Roggieri P., Fabri R., Viarengo A., Genotoxicity biomarkers in the assessment of heavy metal effects in mussels: experimental studies, *Environ. Mol. Mutagen*, 1999; 33: 287–292.
4. Saffi S.A., Mercury toxicity: biochemical and physiological alterations in nine freshwater teleosts, *J. Toxicol. Lett*, 1981; 8: 187.
5. National Academy of Sciences (NAS)., Toxicological effects of methylmercury, Washington, D.C: National Academy Press; 2000.
6. U.S. EPA., Mercury study report to congress; 1997. <http://www.epa.gov/mercury/report.htm>.
7. Goyer R.A., Clarkson T.W., Toxic Effects of Metals, Klaussen CD, editor. Casarett and Dull's toxicology: the basic science of poisons, New York: McGraw-Hill, 2001; 811–67.
8. Pamphlett R., Cotte P., Entry of low doses of mercury vapor into the nervous system, *Neurotoxicology*, 1998; 19 (1): 39–48.
9. Bloom N.S., on the chemical form of mercury in edible fish and marine invertebrate tissue, *Can J Fish Aquatic Sci*, 1992; 49:1010–7.
10. Pinho A., Guimaraães J., Costa A., Olavo A., Valentin J., Total Mercury in muscle tissue of five shark species from Brazilia offshore waters: effects of feeding habit, sex, and length, *Environ Res Sect, A*, 2002; 89 : 250 – 8.
11. WHO. International Programme on Chemical Safety, Environmental Health Criteria for Methylmercury, Geneva7 World Health Organization, 2003; 1 –120.

12. Eisler R., Mercury Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review, U.S. Fish and Wildlife Service Rep, 85(1.10), Washington, DC, 1987.
13. Wiener J.G., Spry D., Toxicological significance of mercury in freshwater fish, Environmental Contaminants in Wildlife: Interpreting Tissues Concentrations, 1996; 297-339.
14. Jonnalagadda, S.B., Rao, P.V., 1993. Toxicity, bioavailability and metal speciation. Comp. Biochem. Physiol. C 106, 585–595.
15. Yee S, Choi B.H., Oxidative stress in neurotoxic effects of methylmercury poisoning, Neurotoxicology, 1996; 17: 17-26.
16. National Research Council, Toxicological Effects of Methylmercury, Washington, DC: National Academy Press, 2000; 54-55.