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Mercury Contamination in Fish and Public Health Aspects: A Review

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Abstract: Mercury is a heavy metal that presents in the earth's crust and it is methylated by bacteria in aquatic environments to methyl mercury (MeHg) in anaerobic conditions. It is then concentrated by the food chain so predatory fish and other seafood animals may have the highest levels. Thus, consuming fish and other seafood lead to human exposure. It generally accepts that seafood represents one of the major sources of non-occupational mercury exposure to human. MeHg readily crosses the placenta and the blood-brain barrier and is neurotoxic. The developing fetal nervous system is especially sensitive to its effects, and chronic exposure cause Minamata disease in human. Controlling of methyl mercury in fish and seafood products is important for public health and there are some responsible organizations in the world, which are in charge of monitoring and controlling methyl mercury in fish and seafood products. This article would have a review of materials and sources of mercury, public health concerns and the methods of mercury contamination control in fish and seafood products.

Key words: Mercury, aquatic environments, fish, seafood products, public health concerns

Introduction

At present, a significant portion of the global diet consists of foods of aquatic origin, either fresh or processed and from fresh or salt water. This consumption has had a positive economic impact on commercial fishing and the associated food processing industries, and each year a wide variety of manufactured seafood products are launched on the market. The need to include seafood, particularly fish, in the human diet has been emphasized with regard to its lower levels of saturated fat, cholesterol, and caloric intake compared with meat, poultry, and dairy products (Velez and Montoro, 1998) but we must investigate the attendance of environmental contaminants in fish and other seafood products. Both terrestrial and aquatic food chains are capable of accumulating certain environmental contaminants up to toxic concentrations. In general, few of the thousands of chemicals produced by human industry may be entered to aquatic environment. Some of these chemical are basically considered as a part of seafood's natural environment while others have anthropogenic sources. Chemical contaminants can come from industrial, municipal, or agricultural sources (Conacher *et al*., 1993; Velez and Montoro, 1998). In terms of organic chemicals, the best known examples of bioaccumulation in aquatic food chains are the polychlorinated biphenyls (PCBs), dioxins, and organochlorine pesticides such as dichlorodiphenyl trichloroethane (DDT). There are also some examples on bioaccumulation of metal compounds. Methyl mercury is arguably the most dramatic and best-documented example of high bioaccumulation (Clarkson, 1995). Methyl mercury is an organic material that conforms from inorganic mercury. The contamination of mercury in seafood such as fish, shellfish, oyster and other types of seafood is one of great concern in places suffering from pollution humans (Bortoli *et al*., 1995; Clarkson, 1998; Dickman and Leung, 1998; Stein *et al*., 1996). They have known as a source of non-occupational mercury exposure in fish and seafood products consuming population groups. Neumann *et al*. (1997) showed the levels of mercury (Hg) in fish tissues. They had total Hg levels exceeding the US Environmental Protection Agency's (EPA) health screening value of 0.6 mg/kg. In Japan, industrial discharge of mercury into Minamata Bay raised the concentrations of the metal in fish, resulting in serious human intoxication and deaths following consumption of the contaminated fish. The Hg discharged in Minamata continued to magnify until reaching values of 10-100 μg/g in sediments, 2000 μg/g at the discharge channel of the Chiss-o Corporation that used and dumped it, and 5-40 μg/g in fish and mollusks (Villanueva and Botello, 1998). The Minamata experience has indicated that the most serious sources are discharges from industrial plants and it is the most dramatic example of environmental contamination by mercury involving fish and affecting man (Aizpurua *et al*., 1997).

Material and sources of mercurial contaminant: Fish and seafood can be exposed to a range of mercury contamination from the water to the table (Kurtzweil,

1997). Mercury has a widespread occurrence in nature albeit, in trace quantities. Its principal mineral in the earth's crust, the region of gold mining, is cinnabar (HgS) which occurs in a few places in the world. Mercury or its compounds have been used in industry as catalysts (inorganic form) in the synthesis of important industrial compounds (e.g. acetaldehyde, vinyl chloride), as fungicides in agriculture and horticulture, as antis liming agents in the paper and pulp industries, and as antifouling agents in paints. At the present, in trace quantities of mercury in crude oil and coal, it can release to the environment during combustion such as coal burning, trash incineration, and industrial emissions. Mercury can enter aquatic environment and then it can enter body of aquatic animals such as fish, shellfish, shrimp, oyster and other types of seafood by absorption through the gills or by absorption through food by fed plankton, but it appears that the second is the more important, and mercury accumulate to organic form in tissues of fish and other seafood animals (Biddinger and Gloss, 1984; Clarkson, 1995; Hall *et al*., 1997). The measurement of mercury concentration among species of fish, cephalopods, crustaceans and molluscs in marine environment showed that fish and crustaceans accumulated the highest levels of this element (Schuhmacher *et al*., 1994) as the descending order of mercury concentration in fish organs are: gills, intestines, head and muscles (Ipinmoroti *et al*., 1997; Amundsen *et al*., 1997). The mercury concentrations are usually lowest in muscle and highest in liver or the gills (Amundsen *et al*., 1997). Inorganic mercury can be methylated and formed organic mercury compounds covalently bound to carbon, such as methyl mercury (Hg CH_3) (Hrudey *et al*., 1996). This function is done by biological, predominantly microbiological, and chemical processes in the aquatic environment, and this organic form is taken up by aquatic organisms such that the mercury concentrations in tissues can be greater than in the ambient water. The methyl mercury bioaccumulates up the trophic chain so that the highest concentrations are found in predatory fish. There is more than 95% of the total mercury in the edible portions of fish (Andersen and Depledge, 1997; Bloom, 1992; Concon, 1988; Gutleb *et al*., 1997; Howgate, 1998; Kehrig *et al*., 1998; Lodenius and Malm, 1998; Nakagawa *et al*., 1997) and other seafood is in the form of methyl mercury (Bloom, 1992; Nakagawa *et al*., 1997). Methyl mercury is more toxic, very persistent, and readily bioaccumulated, especially in aquatic food chains. It is lipid soluble, stable in pH variations and biological factors (Lodenius and Malm, 1998). Under certain conditions the mercury accumulated in fish and seafood products and its levels reach to toxically unacceptable levels. The metal is fixed to the sulphydryl group of the protein, accumulating especially in one of its most toxic forms, methyl mercury (Biddinger and Gloss, 1984; WHO, 1990). Carnivorous fish consumption is the most significant source of exposure to organic mercury for the general population (Hrudey *et al*., 1996). In salt water fish great differences in mercury concentrations can be found. The variations served are attributed to factors such as species, geographical location, size, sex and period of catch. In most species the mercury is less than 0.5 µg/g. The higher levels of mercury are found in predator fish at the top of the food chain, such as sword fish, tuna and shark. They generally show concentrations between 0.2 and 1.5 µg/g, which can reach up to 5 µg/g (Biddinger and Gloss, 1984; WHO, 1990). The maximum limit for mercury contents in seafood products for human consumption is 0.5 mg/kg (Guerrin *et al*., 1990; Wagemann *et al*., 1998).

Methyl mercury is usually enriched in aquatic food chains, affecting fish and trace amounts are found in nearly all fish, and for most fish this ranges from 0.01 ppm to 0.5 ppm with an average of less than 0.3 ppm for commercially important marine species (Kehrig *et al*., 1998; Swan, 1998).

The investigations of Marx and Brunner in North Sea in 1998 showed that the mercury content in shrimp was 0.033-0.016 µg/g wet weight. It is lower amounts of the investigated elements than the levels of concern of the Federal Institute for Health Protection of Consumers and Veterinary Medicine, i.e. 0.5 µg Hg/g wet weights. Results showed significantly lower levels of contamination. The main reasons for the overall low levels of contamination might be the short period of feeding of the shrimp and an active mechanism of secretion. Besides this, the mercury load of the shrimp depended on the fishing season (November or May). Voegborlo *et al* were analyzed fifty samples of canned tuna fish from Misurata canning factory for mercury in 1999. They reported that 20 samples of 50 samples were contaminated to mercury. The concentration of mercury in the tuna fish samples varied from 0.2 to 0.66 µg/g. Apart from two samples which have concentrations of 0.55 and 0.66 µg/g mercury, all the samples had concentrations below the 0.5 µg/g limit recommended by the FAO/WHO.

Public health concerns: The consumption of fish, sea foods and their derived products is the main pathway of human exposure to mercury, especially methyl mercury (MeHg). Methyl mercury levels vary widely in fish and other sea foods, depending on age, size, the position of the species in the food chain, and most importantly, on pollution level (Petruccioli and Turillazzi, 1991). Methyl mercury is a neurotoxin present in both fresh and saltwater fish throughout the world. Increased levels of methyl mercury can be found in individuals who regularly consume fish and seafood products (Myers and Davidson, 1998). The first methyl mercury poisoning by consumption of fish arose in Minamata, Japan, in 1953.

Methyl mercury dispersed from Minamata to the Shiranui Sea until 1968. Minamata disease is methyl mercury poisoning that occurred in people who ingested fish and shellfish contaminated by methyl mercury discharged in waste water from a chemical plant (Chisso Co. Ltd.). It was in May 1956, that Minamata disease was first officially "discovered" in Minamata City, south-west region of Japan's Kyushu Island. The marine products in Minamata Bay displayed high levels of Hg contamination (5.61 to 35.7 ppm). The Hg content in hair of patients, their family and inhabitants of the Shiranui Sea coastline were also detected at high levels of Hg (Max. 705 ppm). Mercury, like many heavy metals, has a high affinity for sulfhydryl groups. Thus, as expected, its principal target should be sulfhydryl-containing proteins. However, the symptoms of methyl mercury poisoning strongly indicate that the tissues most severely affected are in the nervous system, specifically the brain tissues (Davidson *et al.*, 1998; Hurdey *et al.*, 1996; Myers and Davidson, 1998; Rice, 1995). Methyl mercury accumulates in kidney, liver, and the nervous system, causing damage and poisoning symptoms. The neurological symptoms observed are: tremor, hyposensitivity, double vision (Petruccioli and Turillazzi, 1991, ASTDR, 2003), confusion, moodiness, and depression (Stine and Brown, 1996). In adult humans, brain damages are focal, affecting specific cell types in certain anatomical areas such as the visual cortex and the cerebellum. Prenatal exposure disrupts the normal developmental processes of the fetal brain (Clarkson, 1998). Typical symptoms of Minamata disease are as follows: sensory disturbances (glove and stocking type), ataxia, dysarthria, constriction of the visual field, auditory disturbances and tremor were also seen (Harada, 1995; Harada *et al.*, 1998; Ninomiya *et al.*, 1995) and also it may cause steroidogenic impairment, reduced sperm counts, and fertility problems in humans (Friedmann *et al.*, 1998). Methyl mercury readily crosses the placenta and the blood-brain barrier and is neurotoxic. The developing fetal nervous system is especially sensitive to its effects, and prenatal exposure can occur when the mother has a diet high in fish. If the level of methyl mercury exposure achieved by eating fish adversely affects the fetus or child's neurological development it could have far reaching public health implications (Myers and Davidson, 1998). Prenatal poisoning with high dose methyl mercury causes mental retardation, brain damage, incoordination, blindness, seizures, and inability to speak (ASTDR, 2003; EnviroTools, 2002). Lower level exposures from maternal consumption of a fish diet have not been consistently associated with adverse neurodevelopmental outcomes (Myers and Davidson, 1998). Further, the fetus was poisoned by methyl mercury when their mothers ingested contaminated fish and seafoods (named congenital Minamata disease). The symptoms of patients were serious, and extensive lesions of the brain were observed. Children poisoned by mercury may develop problems of their nervous and digestive systems, and kidney damage (ASTDR, 2003). While the number of grave cases with acute Minamata disease in the initial stage was decreasing, the numbers of chronic Minamata disease patients who manifested symptoms gradually over an extended period of time was on the increase. For the past 36 years, of the 2252 patients who have been officially recognized as having Minamata disease, 1043 have died (Harada, 1995). Methyl mercury has caused kidney tumors in male mice. The EPA has determined that Methyl mercury is possible human carcinogens (ASTDR, 2003).

Methods of mercury pollution control: The incorporation of mercury into the food chain and its assimilation by humans is a universally recognized potential health hazard. Preventive actions should be taken into determination of 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).The primary mechanisms controlling the accumulation of methyl mercury and inorganic mercury in aquatic food chains is very complicate. Differences in lipid solubility alone cannot account for the predominance of methyl mercury in fish because inorganic mercury complexes (e.g., $HgCl_2$), which are not bioaccumulated in fish, are as lipid soluble as their methyl mercury analogs (e.g., CH_3HgCl). Mercury concentrations in fish are ultimately determined by methyl mercury accumulation at the base of the seafood chain. It is governed by water chemistry, primarily pH and chloride concentration (Mason *et al.*, 1996). Human hair is a useful indicator of mercury exposure. We can determine methyl mercury concentration in polluted areas by hair sampling in people who consume fish and other seafood.

As a result of several decades of industrialization, heavy metals, including mercury, have become the focus of ecological and human health concerns in some places of the world such as Newark Bay (New Jersey, USA) [(Mean concentrations of total mercury were elevated above estimated background levels (0.1 mg/kg) to 9.8 mg/kg (dry weight)]. In mercury polluted places, quality criteria and guidelines suggest that the presence of mercury poses a toxic hazard to aquatic biota. These data suggest that further investigation of sources is needed to reduce or eliminate this contamination (Gillis *et al.*, 1993).

Methyl mercury is controlled by the 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 Guideline for Mean MeHg levels in fish is also 0.5 µgHg/g wet wt (Wagemann *et al*., 1998).

In an effort to reduce industrial emissions of Hg, the U.S.EPA developed a new reference dose (RfD) for MeHg in 1996. This RfD (the daily dose of MeHg that can safely consumed over a life time) is used by states to develop their fish consumption advisories. The EPA RfD is 0.1 µg/kg/day. It is one fifth (0.47 µg/kg/day) of the intake guide lines set by the world health organization (WHO) (Egeland and Middangh, 1997).

Contamination of fish and seafood products by methyl mercury will continue to be a challenging issue for governments at 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 Farland, 1998). Therefore, for controlling of contamination fish and seafood products by methyl mercury, we require some of guidelines:

- To avoid harmful accumulation of this metal in the human system, the gills, the liver and the intestine should preferably be discarded while processing fish for consumption (Ipinmoroti *et al*., 1997).
- In aquaculture systems, regulatory authorities might intervene to prevent distribution of contaminated stock until the danger is passed. It is difficult to control chronic contamination of mercury in aquaculture systems which are use of polluted water supplies, leaching of agricultural or industrial mercury from treated or contaminated soils into surface waters and deposition from the atmosphere.
- Mercury accumulates in fish at during its life time, and mercury body burdens in fish in a particular environment are greater in the older, and hence larger fish has more mercury (Jahed Khaniki, 1992). Farmed fish are usually harvested at a young stage and would be expected to have low body burden even if their food contained mercury.
- Mercury exposure is reduced through restricting harvest of aquatic animals from certain sites and excluding certain species (Ahmed *et al*., 1993). Therefore, learn about wildlife and fish advisories in your area from your public health or natural resources department.
- 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 (Clarkson, 1995; 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.
- The limitation of coastal waters pollution, improved surveillance, the development of more sensitive indicators, the responsabilisation of the industry and the information of the public on the health hazards associated with shellfish consumption are the key issues for the improvement of shellfish-borne disease prevention (Desenclos, 1996).

Conclusion: 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 Marin culture 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.

References

Ahmed, F.E., D. Hattis, R.E. Wolke and D. Steinman, 1993. Human health risks due to consumption of chemically contaminated fishery products. [Review] Environmental Health Perspectives, 101 Suppl., 3: 297-302.

Amundsen, P.A., F.J. Staldvik, A.A. Lukin, N.A. Kashulin, O.A. Popova and Y.S. Reshetnikov, 1997. Heavy metal contamination in freshwater fish from the border region between Norway and Russia. Sci. Total Environ., 201: 211-224.

Andersen, J.L. and M.H. Depledge, 1997. A survey of total mercury and methyl mercury in edible fish and invertebrates from Azorean waters. Marine Environ. Res., 44: 331-350.

Aizpurua, I.C.M., A. Tenuta-Filho, A.M. Skuma and O. Zenebon, 1997. Int. J. Food Sci. Tec., 32: 333-337.

ASTDR, 2003. Mercury. Agency for toxic substances and disease registry. GA: US. Department of health and human services, public health services, Atlanta. ASTDRIC@cdc.gov. http://www.Astdr.cdc.gov/tfacts46.html.

Biddinger, G.R. and S.P. Gloss, 1984. The importance of trophic transfer in the bioaccumulation of chemical contaminants in aquatic ecosystems. Residue Rev., 91: 103-145.

Bloom, N.S., 1992. On the chemical form of mercury in edible fish and marine invertebrate tissue. Canadian J. Fisheries and Aquatic Sci., 49: 1010-1017.

Bortoli, A., M. Gerotto, M. Marchiori, M. Palonta and A. Troncon, 1995. Analytical problems in mercury analysis of seafood. Annali dell Istituto Superiore di Sanita, 31: 359-62.

Clarkson, T.W., 1998. Human toxicology of mercury. J. Trace Ele. Exp. Med., 11: 303-317.

Clarkson, T.W., 1995. Environmental contaminants in the food chain. [Review]. Am. J. Clin. Nutr., 61: 682S-686S.

Conacher, H.B, B.D. Page and J.J. Ryan, 1993. Industrial chemical contamination of foods. [Review] Food Additives and Contaminants, 10: 129-43.

Concon, J.M., 1988. Food Toxicology (Part B): Contaminants and Additives. Marcel Dekker, Inc. USA.

Davidson, P.W, G.J. Myers, C. Cox, C. Axtell, C.Shamlaye, J. Sloane Reeves, N.L. Elsacernichiari, A. Choi, Y. Wang, M. Berlin and T.W. Clarkson, 1998. Effects of prenatal and postnatal methyl mercury exposure from fish consumption on neurodevelopment: Outcomes at 66 months of age in the Seychelles Child Development Study. J. Am. Med. Assoc., 280: 701-707.

Desenclos, J.C., 1996. Epidemiology of toxic and infectious risk related to shellfish consumption. [Review]. Revue d Epidemiologie et de Sante Publique., 44: 437-54.

Dickman, M.D. and K.M. Leung, 1998. Mercury and organochlorine exposure from fish consumption in Hong Kong. Chemosphere, 37: 991-1015.

Egeland, G.M. and J.P. Middangh, 1997. Balancing fish consumption benefits with mercury exposure. Sci., 278: 1904-1905.

EnviroTools, 2002. Mercury. Hazardous Substances Research Center, Michigan State University. National Institute of Environmental Health Sciences. http://www.envirotools.org/factsheets/contaminants/mercury.shtml.

Feng, Q., Y. Suzuki and A. Hisashige, 1998. Hair mercury levels of residents in China, Indonesia, and Japan. Arch. Environ. Health, 53: 36-43.

Friedmann, A.S., H. Chen, L.D. Rabuck and B.R. Zirkin, 1998. Accumulation of dietary methyl mercury in the testes of the adult Brown Norway rat: Impaired testicular and epididymal function. Environ. Toxicol. Chem., 17: 867-871.

Gillis, C.A., N.L. Bonnevie and R.J. Wenning, 1993. Mercury contamination in the Newark Bay estuary. Ecotoxicol. Environ. Safety, 25: 214-26.

Goldman, L.R. and W.H. Farland, 1998. Methylmercury risks. Sci., 279: 641.

Guerrin, F., V. Burgat-Sacaze and P. de Saqui-Sannes, 1990. Levels of heavy metals and organochlorine pesticides of cyprinid fish reared four years in a waste water treatment pond. Bull. Environ. Contamination Toxicol., 44: 461- 467.

Gutleb, A.C., C. Schenck and E. Staib, 1997. Giant otter (Pteronura brasiliensis) at risk. Total mercury and methylmercury levels in fish and otter scats, Peru. Ambio, 26: 511-514.

Hall, B.D., R.A. Bodaly, R.J.P. Fudge, J.W.M. Rudd and D.M. Rosenberg, 1997. Food as the dominant pathway of methylmercury uptake by fish. Water, Air, and Soil Pollution, 100: 13-24.

Harada, M., 1995. Minamata disease: methylmercury poisoning in Japan caused by environmental pollution. A review. Cr. Rev. Toxicol., 25: 1-24.

Harada, M., J. Nakanishi, S. Konuma, K. Ohno, T. Kimura, H. Yamaguchi, K. Tsuruta, T. Kizaki, T. Ookawara and H. Ohno, 1998.The present mercury contents of scalp hair and clinical symptoms in inhabitants of the minamata Area. Environ. Res., 77: 160-164.

Howgate, P., 1998. Review of the public health safety of products from aquaculture. Int. J. Food Sci. Tec., 33: 99-125.

Hrudey, S.E., W. Chen and C.G. Roussex, 1996. Bioavailability in environmental risk assessment. CRC Press, Inc, Lewis publishers, USA. pp: 125-133.

Ipinmoroti, K.P., A.A. Oshodi and R.A. Owolabi, 1997. Comparative studies of metals in fish organs, sediment and water from Nigerian fresh water fish ponds. Pak. J. Sci. Ind. Res., 40: 70-74.

Jahed Khaniki, Gh. R ., 1992. Public health aspects of fish and shrimp products. No. 2135. DVM Thesis, Faculty of Veterinary Medicine, Tehran University, Iran.

Kehrig, H.D.A., O. Malm, H. Akagi, J.R.D. Guimaraes and J.P.M. Torres, 1998. Methylmercury in fish and hair samples from the Balbina Reservoir, Brazilian Amazon. Environ. Res., 77: 84-90.

Kurtzweil, P., 1997. Critical steps toward safer seafood. FDA consumer, 31: 10-15.

Lebel, J., M. Roulet, D. Mergler, M. Lucotte and F. Larribe, 1997. Fish diet and mercury exposure in a riparian Amazonian population. Water, Air, and Soil Pollution, 97: 31-44.

Lipfert, W., P.D. Moskowitz, V. Fthenakis and L. Saroff, 1996. Probabilistic assessment of health risks of methylmercury from burning coal. Neurotoxicology. 17: 197-211.

Lodenius, M. and O. Malm, 1998. Mercury in the Amazon. Reviews of Environmental contamination and Toxicology, 157: 25-52.

Mahaffey, K.R. and D. Mergler, 1998. Blood levels of total and organic mercury in residents of the upper St. Lawrence River basin, Quebec: association with age, gender, and fish consumption. Environ. Res., 77: 104-14.

Marx, H. and B. Brunner, 1998. Heavy metal contamination of North Sea shrimp (*Crangon crangon* L.). Zeitschrift Fuer Lebensmittel-Untersuchung und-Forschung, 207: 273-275.

Mason, R.P, J.R. Reinfelder and F.M.M, Morel, 1996. Uptake, toxicity, and trophic transfer of mercury in a coastal diatom. Environ. Sci. Tec., 30: 1835-1845.

Myers, G.J., and P.W. Davidson, 1998. Prenatal methylmercury exposure and children: neurologic, developmental, and behavioral research. A review. Environmental Health Perspectives, 106 (Suppl 3): 841-847.

Nakagawa, R., Y. Yumita and M. Hiromoto, 1997. Total mercury intake from fish and shellfish by Japanese people. Chemosphere, 35: 2909-13.

Neumann, C.M., K.W. Kauffman and D.J. Gilroy, 1997. Methyl mercury in fish from Owyhee reservoir in southeast Oregon: Scientific uncertainty and fish advisories. Sci. Total Environ., 204: 205-214.

Ninomiya, T., H. Ohmori, K. Hashimoto, K. Tsuruta and S. Ekino, 1995. Expansion of methylmercury poisoning outside of Minamata: an epidemiological study on chronic methylmercury poisoning outside of Minamata. Environ. Res., 70: 47-50.

Petruccioli, L. and P. Turillazzi, 1991. Effects of Methyl mercury on Acetylcholinesterase and Serum cholinesterase Activity in monkeys, Macaca fascicularis. Bull. Environ. Contam. Toxicol., 46: 769-773.

Renzoni, A., F. Zino and E. Franchi, 1998. Mercury levels along the food chain and risk for exposed populations. A review. Environ. Res., 77: 68-72.

Rice, D.C., 1995. Neurotoxicity of lead, methylmercury, and PCBs in relation to the Great Lakes. Environmental Health Perspectives, 103 Suppl 9: 71-87.

Schuhmacher, M.J., Batiste, M.A. Bosque, J.L. Domingo and J. Corbella, 1994. Mercury concentrations in marine species from the coastal area of Tarragona Province, Spain. Dietary intake of mercury through fish and seafood consumption Science of the Total Environment, 156: 269-73.

Stein, E.D. Y. Cohen and A.M. Winer, 1996. Environmental distribution and transformation of mercury compounds. Cr. Rev. Environ. Sci. Tec., 26: 1-43.

Stine, K.E. and T.M. Brown, 1996. Principles of Toxicology. CRC press, Inc. USA., pp: 211-225.

Swan, H.B., 1998. Aqueous phase ethylation atomic emmision spectroscopy for the determination of methyl mercury in fish using permeated dimethyl mercury calibration. Bull. Environ. Contam. Toxicol., 60: 511-518.

Velez, D. and R. Montoro, 1998. Arsenic speciation in manufactured seafood products: a review. J. Food Protec. 61: 1240-1245.

Villanueva, F.S. and A.V. Botello, 1998. Metal pollution in coastal areas of Mexico. Rev. Environ. Contam. Toxicol., 157: 53-94.

Voegborlo, R.B., A.M. El-Methnani and M.Z. Abedin,1999. Mercury, cadmium and lead content of canned tuna fish. Food Chem., 67: 341-345.

Wagemann. R, E. Trebacz, G. Boila and W.L. Lockhart, 1998. Methyl mercury and total mercury in tissues of arctic marine mammals. Science of the Total Environ., 218: 19-31.

WHO, 1990. Methyl mercury. Environmental Health Criteria, World Health Organization, Geneva, 101: 5-76.

Yess, N.J., 1993. U.S. Food and Drug Administration survey of methyl mercury in canned tuna. J. AOAC Int., 76: 36-8.