International Journal of Zoology Studies
ISSN: 2455-7269
Impact Factor: RJIF 5.14
www.zoologyjournals.com
Volume 3; Issue 2; March 2018; Page No. 153-159

A review on contamination, bioaccumulation and toxic effect of cadmium, mercury and lead on freshwater fishes

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Abstract
Heavy metal pollution in fresh water aquatic systems is a threat for ecosystem as well as for human health and economy. Various routes of contamination of Cd, Hg and Pb have been reported. Cd, Hg and Pb are so dangerous and harmful because of their persistent nature. Cadmium among these three culprits is the most dangerous contaminant that adversely affects growth, reproduction and behaviour of fresh water fishes. Mercury is a potent endocrine disrupter that vigorously impacts on fish reproduction and metabolism. Lead is a neurotoxicant that create behavioural disputes in fish population.

Keywords: biomagnification, genotoxicity, metallothioneins, neutotoxicity.

Introduction

The term "heavy metal" refers to any metal or metalloid element that has toxic effect at low concentrations. It includes mercury (Hg), cadmium (Cd), arsenic (As), chromium (Cr), thallium (Tl), zinc (Zn), nickel (Ni), copper (Cu) and lead (Pb). Heavy metals are non-biodegradable pollutants that pose a serious threat to aquatic ecosystems. The presence of heavy metals in fresh water aquatic systems is an increasing eco-environmental and social problem. Some of these metals (Fe, Zn, Cu, Co, Cr, Mn and Ni) are essential for biological functions. Others, such as Pb, Hg, Cd and as are not required for biological functions. Presence of these elements or their compounds results in toxic effect. So these heavy metals are considered as body burden when they somehow enter in organisms body. Heavy metal contamination in water-bodies is a major eco-environmental problem around industrial belts all over the world.

Cadmium is used widely in electroplating industries, batteries, television sets, ceramics, photography, insecticides, electronics, metal-finishing industries and metallurgical activities. It can be introduced into the environment by metal-ore refining, cadmium containing pigments, alloys and electronic compounds, cadmium containing phosphate fertilizers, detergents and refined petroleum products (Hutchinson, 1987) [27]. Rechargeable batteries with nickel–cadmium compounds are also sources of cadmium (ATSDR, 2007) [3]. Cadmium is the most toxic element and has been found to cause of *itai-itai* disease in Japan.

The natural sources of mercury are volcanic eruption, weathering of rocks and soils, whereas anthropogenic mercury comes from the extensive use of the metal in industrial applications (Pacyna *et al.*, 2002) [48], applications in batteries and mercury vapor lamps (Lars, 2003). Methyl mercury is more toxic than any other species of mercury. The toxicity of mercury has been recognized worldwide, such as in Minamata Bay of Japan. Mentally disturbed and physically deformed babies were born to mothers who were exposed to toxic mercury due to consumption of contaminated fish.

Lead has been used in paint pigments because lead-based paints cling well to wood and lead imparts brightness to the colour. Lead can cross the placenta, resulting in miscarriages, stillbirths, and birth defects (Davidson *et al.*, 1998) [19] such as neurological damage.

Toxic effects of these three heavy metals are wide spread in developing countries like India. Cd, Hg and Pb are known to have neurotoxic (Atchison *et al.*, 1987) [10], genotoxic (Al-Sabti, 1986) [5] and endocrinological (Crump and Trudeau, 2009) [16] effects in fish. Long term exposure to these metals can cause a vast change in ecological community and may result in complete destruction of effected fish species. Fish is a chief protein source in daily meal in a large part of our globe. So heavy metal pollution in fresh water aquatic system is now a big concern to human health and economy.

Contamination of Cd, Hg and Pb in freshwater aquatic systems

Cadmium

Cadmium occurs in the earth's crust at an abundance of 0.1–0.5 ppm and is commonly associated with zinc, lead, and copper ores. Natural emissions of cadmium to the environment can result from volcanic eruptions, forest fires, generation of sea salt aerosols, or other natural phenomena. Cadmium is refined and consumed for use in batteries (83%), pigments (8%), coatings and platings (7%), stabilizers for plastics (1.2%), and nonferrous alloys, photovoltaic devices, and other (0.8%) (USGS, 2008) [60]. Nonferrous metal mining and refining, manufacture and application of phosphate fertilizers, fossil fuel combustion, and waste incineration and disposal are the main anthropogenic sources of cadmium in the environment. Water sources near cadmium-emitting industries, both with historic and current operations; have shown a marked elevation of cadmium in water sediments and aquatic organisms (Angelo *et al.*, 2007) [7]. In surface water and groundwater, cadmium can exist as the hydrated ion or as ionic complexes with other inorganic or organic substances. While soluble forms may migrate in water (table 1), cadmium is relatively non-mobile in insoluble complexes or adsorbed to sediments.

Table 1: Cadmium concentrations in Indian Rivers

Location	Conc. (mg/l)	References
Bellandur Lake, Bangalore	0.70	Lokeshwari and Chandrappa, 2006
Matla River, West Bengal	0.68	COMAPS, 2007
Saptamukhi River, West Bengal	0.85	COMAPS, 2007
Hugli River, West Bengal	0.59	COMAPS, 2007
Subernarekha River, West Bengal	0.47	COMAPS, 2007
Yamuna River, Delhi	0.02	CPCB, 2006

Mercury

Mercury (Hg) is released to the environment from a multitude of anthropogenic sources (UNEP, 2002) [61]. On the other hand, mercury can also be naturally present in Earth crust, especially in globally distributed Hg mineral belts, and in areas of altered rock that contain elevated concentrations of Hg. During its transport in the environment there are several ways in which Hg can enter aquatic environments through both diffuse and point sources. Mercury is used in a variety of products worldwide because of its many unique properties. In addition, elemental Hg is used in artisanal and small-scale mining of gold and silver (Pai *et al.,* 2000) [49]; and in products such as chlor-alkali, vinyl chloride monomers, manometers for pressure measurement, thermometers, electrical switches, fluorescent lamp bulbs, and dental amalgam fillings. Moreover, Hg compounds are used in some batteries, pharmaceuticals, paints, and as laboratory reagents and industrial catalysts. Production, uses and disposal of Hg-containing products and wastes can therefore result in the release of mercury to the nearby aquatic systems (Zhou *et al.*, 1998) [67]. Indirect pathways are associated with Hg previously deposited and/or accumulated due to the anthropogenic discharges in the terrestrial ecosystems via soil erosion and surface runoff (Pacyna, 2002) [48].

Inorganic mercuric compounds include mercuric sulphide, mercuric oxide and mercuric chloride. The most common organic mercury compound that micro-organisms and natural processes generate from other forms is methyl-mercury. Methyl-mercury is of particular concern because it can build up (bio-accumulate and bio-magnify) in many edible freshwater and saltwater fish and marine mammals to levels that are many thousands of times greater than levels in the surrounding water. New research has shown that methyl-mercury can be released directly from municipal waste landfills (Lindberg *et al.*, 2001) [34] and sewage treatment plants.

Lead

In fresh water, lead may partially exist as the divalent cation at pH below 7.5, but complexes with dissolved carbonate to form insoluble $PbCO_3$ under alkaline conditions (ATSDR, 2005) [4]. Even small amounts of carbonate ions formed in the dissolution of atmospheric CO_2 are sufficient to keep lead concentrations in rivers at the 500 µg/L solubility limit (ATSDR, 2005) [4]. Lead is known to form strong complexes with humic acid and other organic matter (Guibaud *et al.,* 2003) [25]. Lead-organic matter complexes are stable to a pH of 3, with the affinity increasing with increasing pH but decreasing with increased water hardness (Callahan *et al.*, 1979) [12]. Chief sources of lead release in aquatic systems are Domestic wastewater, smelting and refining: Manufacturing processes: like Metals, Pulp and paper, Petroleum products, and Dumping of sewage sludge. (Nriagu and Pacyna, 1988) [45]. Electricity supply, extraction and manufacturing, Metal ore mining, Coal mining, Non-ferrous base-metal industry, Iron and steel industry, Metal industry, Chemical industry, Ceramics, stone and clay products, Paper manufacturing, Petroleum industry are some another important sources of lead contamination in fresh water aquatic systems (Jacobs, 2002) [28]. In table 2, tolarence standard of cadmium, mercury and lead have shown.

Table 2: Canadian Water Quality Standards for Metal: Aquatic Life (Ministry of Environment, Canada, 1992)

Metal	Aquatic life protection standard (ppb)
cadmium	0.017
mercury	0.1
Lead	1-7 depending on water hardness

Bioaccumulation and biomagnification of Cd, Hg and Pb

The term bioaccumulation is defined as a process by which the chemicals are taken up by an organism either directly from exposure to a contaminated medium or by consumption of food containing the chemical. Metal bioaccumulation is influenced by multiple routes of exposure (diet and solution) and geochemical effects on bioavailability. As metals are not metabolized, bioaccumulation of metals and metalloids is of particular value as an exposure indicator. Similarly, bioaccumulation is often a good integrative indicator of the chemical exposures of organisms in polluted ecosystems. The metal contamination in aquatic ecosystem is considered to be unsafe not only for aquatic organisms but also for terrestrial organisms including the human. The long-term consumption of fish from the polluted waters may result in bioaccumulation of persistent pollutants in ultimate recipient of the food web. In fish, heavy metals are taken up through different organs because of the affinity of such organs for the accumulation of heavy metals (Karadede *et al.,* 2004) [31]. In this process, many heavy metals are concentrated at different levels in different organs of the body. Bioaccumulation is a process in which a chemical substance reaches higher concentrations in an organism compared to the concentrations of that chemical in its food or in the surrounding environment. Bioaccumulation is affected by elemental speciation, active and passive uptake mechanisms, transport and distribution between tissues, growth dilution and excretion (Arnot and Gobas, 2006) [9]. The uptake of nonessential trace elements (As, Cd, Hg and Pb) by organisms is often related to the competitive uptake of resembling nutrient ions, and is highly dependent on bioavailability (Merian *et al.,* 2004) [39]. The bioavailability of an element refers to the proportion of the element which is available for uptake. In general, only a small fraction of the dissolved pool of any trace metal is available for uptake by organisms. The bioavailability of trace metals is of great importance in regards to biomagnification. Biomagnification is the transport of chemicals along the successive links in food chains, with increasing concentrations at each trophic level.

Cadmium

Bioavailability refers to the availability of a metal to enter and affect a biological system. The most bioavailable and therefore most toxic form of cadmium is the divalent ion. Exposure to this form induces the synthesis of a low molecular weight protein called metallothionen, which can then bind with cadmium and decrease its toxicity. This normally takes place in the liver of fish and humans. But if the cadmium concentration is high, the metallothionen detoxification system can become overwhelmed and the excess cadmium will be available to produce toxic effects (Wright and Welbourn, 2002) [66]. Bioaccumulation of Cd in aquatic media can be direct and indirect exposure. Fish takes in Cd directly from exposure to a contaminated medium or by consumption of food containing the chemical (USEPA, 2008) [59]. Direct uptake of Cd by fish from the water is mainly in its free ionic form (Adedji and Okocha, 2012) [2] and the indirect exposure is possible as dietary means when consumer organisms subsequently ingest metals bio-accumulated in organisms at a lower trophic level. In freshwater fish, gills are the main point of entry for dissolved metals. The gastro-intestinal tract also acts as an important route for metal absorption in fish. Metal ions are usually absorbed through passive diffusion or carrier mediated transport over the gills while metals associated with organic materials are ingested and absorbed by endocytosis through intestine. It has been suggested that Cd ions enter the chloride cells in the gills through calcium channels (Olsson *et al.*, 1998) [47]. Cadmium that absorbs across the gills or the intestinal walls is distributed via the circulation, bond to transport proteins and distributed, to different tissues of the body (Olsson *et al.*, 1998) [47]. Once Cd enters into the cells, the metal is made available for the interaction with cytoplasmatic components such as enzymes causing toxic effects and Metallothioneins (MTs). Metallothioneins are a low-molecular-weight metal binding proteins and are known to play several important roles, especially in the metabolism and protection against heavy metal toxicity (Nordberg and Nordberg, 2000) [44]. The MTs play a role in transport, detoxification and storage of Cd (Thompson and Bannigan, 2008) [58]. After the absorption, Cd is bound to albumin and transported to liver (Nordberg and Nordberg, 2000) [44]. Following release from the liver, MT-bound Cd enters the plasma and appears in the glomerular filtrate, from where it is re-released intra-cellularly by renal tubule cells (Annabi *et al.*, 2011) [8]. At this stage, Cd is cleaved from the MT-Cadmium complex by lysosomal action and Cd ions are re-excreted into the tubular fluid and finally eliminated in the urine. Bioaccumulation is the net result of the interaction of uptake, storage and elimination of a chemical. Nakayama *et al.*, 2013 [43] show that carnivorous fish (*Serranochromis thumbergi*) shows low accumulated levels of Cd in its tissues and heavy metal concentration is generally inversely correlated with the trophic level. However, Croteau *et al.*, 2005 [17] show that toxic effects of Cd are likely to occur with increasing trophic positions.

Mercury

Fish can accumulate mercury either through food or directly from the water. The efficiency of mercury assimilation from food appears to vary among species. Experimental studies have shown that 68-80% of mercury ingested is assimilated by rainbow trout (Rodgers and Beamish, 1982) [53], about 20% in northern pike, and an efficiency of 80% has been used in uptake models for yellow perch. However, it has also been shown that mercury assimilation across the gut will begin to decrease above certain critical concentrations (Rodgers and Beamish, 1982) [53]. Mercury uptake from the water will be determined by the water concentration, fish metabolic rate and efficiency of uptake (bioavailability) as determined by the ambient water characteristics.

Unlike other trace metals, mercury is “biomagnified” at all levels of aquatic food chains making its bioaccumulation from water more akin to that of hydrophobic organic compounds than inorganic metal ions. Mercury is concentrated roughly a million times between water and piscivorous fish. Mercury bioaccumulation in aquatic organisms is sometimes attributed to the lipophilic character of organic mercury, while the accumulation of inorganic mercury is typically regarded as of secondary importance. Mercury is an interesting metal because its organic form, methyl-mercury, is the most toxic. This compound is formed in aquatic ecosystems when naturally occurring bacteria methylate inorganic mercury. The reaction takes place at the water-sediment interface (Wright and Welbourn, 2002) [66] and is facilitated by low pH and high dissolved organic carbon. Methyl-mercury dissolves well in water, crosses biological membranes, and persists in fatty tissues of organisms. In addition to bio-concentration, methyl-mercury undergoes bio-magnification; each level of the food chain has higher tissue concentrations than its prey. Mercury levels at the top of the food chain are thousands or millions of times higher than in water or sediments (Wright and Welbourn, 2002) [66]. Mercury is well known to biomagnify in aquatic food chains (Merian *et al.*, 2004) [39]. The biomagnification of Hg is related to the high bioavailability of methylmercury. Methylmercury is produced by microbial methylation of Hg under the anoxic conditions found in aquatic sediments, wetlands, and other hydric soils. The assimilation efficiency of MeHg is high and it also has a long biological half-life. As a consequence, top predators in aquatic food chains can acquire high levels of Hg which can result in severe toxicological effects (Merian *et al.*, 2004) [39].

Lead

The lead is known to accumulate the tissues of fish including the bone, gills, kidneys, liver and scales. The accumulation pattern of lead in the selected organs of *Catla catla* is: kidney>liver>gill>brain>muscle. It can be bio-concentrated from water, but does not bioaccumulate and tends to decrease with increasing trophic levels in freshwater habitats (Eisler, 1988) [22].

Effects of Cd, Hg and Pb on fish

Behavioural and neurotoxic effect

Environmental contaminants may result in noticeable changes in fish behavior like attraction/avoidance behavior, swimming activity, reproductive behavior, feeding and prey capture behavior. Cadmium, Lead and Mercurry are well-known neuro-toxicants that can alter the behavior of some fish. Atchison *et al.*, (1987) [10] reviewed many articles dealing with contaminant effects on fish behavior, including locomotion, attraction and avoidance, swimming performance, respiratory behavior, learning, social interactions, reproductive behavior, feeding, and predator avoidance. Shavaji and Asiya Nuzhat (2015) [54] studied Common carp in toxic media exhibited irregular, erratic and darting swimming movements, hyper excitability, capsizing, attaching to the surface. Behavioural alterations like erratic swimming, restlessnees and surfacing observed by Mishra *et al.*, (2012) [40].

Webber *et al.*, (2003) said that mercury exposure at levels currently occurring in northern United States lakes alters fish predator-avoidance behavior in a manner that may increase vulnerability to predation. They examined the effects of dietary mercury exposure at environmental levels in a common forage species, golden shiner (*Notemigonus crysoleucas*). Kania and O'Hara (1974) [30] reported after a 24-hr exposure to sub-lethal mercury concentrations of 0.1, 0.05 and 0.01 ppm Hg++, the ability of mosquito fish to avoid predation by bass was impaired. Crump and Trudeau (2009) [16] hypothesize that mercury in the aquatic environment negatively impacts the reproductive health of fish.

Askari Hesni *et al.*, (2011) [37] observed various behavioral disorders resulted from lead exposure. These disorders included loss of balance, respiratory difficulty, slowness of motion capsizing in water, sinking to the bottom of the tank and increased mucus secretion. Behavioural abnormalities were observed in all treatments, but severity of signs increased with high concentrations of lead acetate. Erratic movements and abnormal swimming are triggered by deficiency in nervous and muscular coordination which may occur due to accumulation of acetylcholine in synaptic and neuromuscular junctions (Rao *et al.*, 2005) [51]. Loss of equilibrium follows erratic and darting swimming movements, might be related to the inhibition of brain cytochrome C oxidase activity, causing cytotoxic hypoxia, thus causing brain damage to the region associated with the maintenance of equilibrium.

Endocrinological effect

Gracia (2013) studied the impact of Cd on thyroid function in tilapia (*Oreochromis niloticus*). Her study shows that acute exposures to sub-lethal cadmium concentrations induce changes in endocrine status and carbohydrate metabolism in *O. niloticus*. Chronic exposure to cadmium influences reproductive disruption in fish, inhibiting induction of vitellogenin, delaying oogenesis in brown trout enhancing luteinizing hormone (LH) secretion, and decreasing parameters of gonado-somatic index (GSI) and ovulation in Prussian carp (P Szczerbik *et al.*, 2006) [56].

Mercury in the aquatic environment negatively impacts the reproductive health of fish. There appears to be sufficient evidence from laboratory studies to link exposure to mercury with reproductive impairment in many fish species. Over a prolonged period of mercury exposure, T4 levels would have fallen to an extent that would have ultimately affected serum T3 levels (Thangam and Manju, 2015) [57]. Basal LH and sex steroid levels are significantly reduced in post-spawning female goldfish (*Carassius auratus*) after a 28-d dietary exposure to 0.88 µg/g of methylmercury (Crump and Trudeau, 2009) [16]. Deleterious effects of mercury on gonad's growth, fish spawning, and egg production and development, fertilization, and hatching were observed (Crump and Trudeau, 2009) [16].

Environmental Pb can be a potent endocrine disruptor, affecting ovarian steroid-genesis, gametogenesis, and ovulation, which may lead to adverse impact on fish reproduction and population density and that female Prussian carp (Luszczek-Trojnar *et al.*, 2014) [35]. Ramesh *et al.*, (2009) [38] reported the acute and sublethal toxicity of lead nitrate on plasma cortisol and prolactin level of a freshwater fish, *Cyprinus carpio*. (Ramesh *et al.*, 2009) [38]. the toxic effect of lead on gametes was shown in studies with both male and female animals (Goyer, 1986) [24]. Weber (1993) [64] observed that multiple effects on reproductive behavior and overall reproductive success in adult fathead minnows, where lead suppressed spermatocyte production and retarded ovarian development, decreased the number of eggs oviposited, increased inter-spawn periods, and suppressed embryo development. Lead can have an impact not only directly on LH secretion from the pituitary but also at lower levels of reproductive control because this metal negatively affects not only LH receptors in the ovary but also estradiol receptors in the body (Luszczek-Trojnar *et al.*, 2014) [35]. Ronis *et al.*, (1996) [50] suggested that Pb influences the hypothalamic–pituitary–gonadal axis at multiple action sites.

Genotoxic effect

Fish are good indicators for assessing the genotoxic and mutagenic effects of xenobiotics and physical agents (Al-Sabti, 1986) [5]. Sister chromatid exchange (SCE) tests have been applied to various fish species (Vigfusson *et al.*, 1983) [63] and the clastogenic effects of carcinogenic-mutagenic chemicals on kidney cells of *Cyprinius carpio* have been described (Al-Sabti, 1986) [5]. The comet assay is useful for evaluating genetic alterations and has been used as a rapid method to monitor genotoxicity in bullheads and carp (Monteith and Vanstone, 1995) [42] and to detect the effects of carcinogens, such as aflatoxins, in trout and channel catfish (Abd-Allah *et al.*, 1999) [1]. Common carp (*Cyprinus carpio*), Prussian carp (*Carassius gibelio*) and Peppered cory (*Corydoras paleatus*) were evaluated by Cavas *et al.*, (2005) as target species to perform genotoxicity tests for heavy metals. Their results indicated the formation of micronuclei and binuclei in fish cells caused by their exposure to cadmium.

Hasan *et al.*, (2010) studied molecular changes caused by a heavy metal pollutant mercuric chloride in fresh water snakehead (*Channa punctatus).*The heavy metal induced chromosomal DNA fragmentation and expression of certain proteins. Cestari *et al.*, (2004) [14] proposed that exposure to lead significantly increased the frequency of chromosomal aberrations and the frequency of tailed cell nuclei, the latter indicating DNA damage. These results show that *H. malabaricu*s is a useful biological model for screening the clastogenic effects of lead and possibly other xenobiotics. The genetic damage seen here illustrates the need to investigate the potential effects of heavy metals on fish species in South America.

Discussion

Heavy metal loaded aquatic systems may affect the food chain of inhabitant fish species. It is reported by many researchers that heavy metal pollution in aquatic ecosystems is often more reflected by high metal levels in sediments, macrophytes and benthic animals than by elevated concentrations in water (Van Hassel *et al.,* 1980) [62]. Bottom-dwelling fish species accumulate heavy metals because of their association with metal-containing sediments (Ney and Van Hassel 1983) [46]. The food chain effect is found in those aquatic environments in which metal loaded food (such as macrophytes) represent a large share of fish diet (Murphy *et al.*, 1978) [41]. A second kind of influence on the food chain effect in fish may be due to the reduction of species diversity. Several authors have shown that heavy metal pollution can lead to the elimination of susceptible species (Roch *et al.*, 1985) [52], thus increasing the dominance of a few tolerant and opportunistic species (Lang and Lang-Dobler, 1979) [32]. As a consequence, trophic relationships are simplified: food chains are shortened and predatory fish are forced to feed more and more on a few, or even only one kind of, metal-tolerant food organisms (Dallinger and Kautzky, 1985) [20].

Begum *et al.*, (2009) [11] showed that the highest concentration of heavy metals is in kidney and liver of ten different fish species. Contaminated sediments can threaten creatures in the benthic environment, exposing worms, crustaceans and insects to hazardous concentrations of toxic chemicals. Some contaminants in the sediment are taken up by benthic organisms. Toxic effects of heavy metals on soil microorganisms in situ were investigated by Jadhav *et al.*, (2010) [29] and a negative influence of the test metals on actinomycetes, mineral nitrogen assimilating and oligo-nitrophilic bacteria was found.

Cadmium exposure resulted in disruption of some important metabolic substrates like glucose, glycogen, lactate, lipid and protein in fish (Almeida *et al.*, 2001) [6]. Cadmium exposure may also disrupt enzymes involved in protein metabolism. De Smet and Blust (2001) [21] reported increased activities of glutamate aminoacyltransferase and alanine aminoacyltransferase in the gills, liver and kidneys of carp exposed to cadmium. Smith *et al.,* (1995) [55] revealed that mercury exposure may result in reduced levels of brain neurotransmitter in several fish species while lead exposure causes increased levels of serotonin (Weber *et al.*, 1991) [65]. The aspects of fish behavior, growth and reproduction are influenced by environmental toxicants and heavy metals like cadmium, mercury and lead have strong toxicity on these aspects. It is obvious that these metals have profound influence on metabolic pathways, hormonal activities, neurotransmitter function and also in molecular pathways.

Conclusion

Heavy metal accumulation and their effects on fish are very much complex to elucidate because of dynamic nature of aquatic ecosystems. Anthropogenic activities contribute significantly to environmental contamination of Cd, Hg and Pb. These metals can induce adverse health effects in fish. These effects were found to be mediated by malfunctioning of specific organs, nervous system and endocrine systems. Impact of cadmium, mercury and lead on freshwater fish is a multifaceted area of research. But surprisingly there is only a few works on the effect of these heavy metals on the upper trophic level of consumers of the aquatic food chain. It is important to record the effects of consumption of fishes contaminated with heavy metals on man. This area also requires an immediate multidisciplinary research to evaluate the losses that already have occurred in the aquatic ecosystem.

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