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Bioaccumulation of cadmium in fish and human health risk assessment

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Abstract
Cadmium is a toxic heavy metal and can cause harm to both aquatic organisms and human being. Several anthropogenic activities result in the release of Cd in the environment. It eventually goes to the water bodies and exposed to aquatic organisms including fish. In fish, it accumulates in different tissues and causes toxic reactions. When these fishes are consumed by a human being, it can cause serious health hazard to them. So, the necessary steps should be taken so that the ecosystem, as well as the human being, can be saved from the harmful effects of Cd.

Keywords: Cadmium, fish, toxicity, bioaccumulation, biomagnification

1. Introduction
With the progression of human civilization environmental pollution has also increased drastically. As a result, we are being continuously exposed to noxious pollutants through ingestion, inhalation and many other routes. The food we consume every day may contain several toxins including pesticides, heavy metals etc. The threats, especially from heavy metals, have emerged as a great problem to the ecosystem as well as human being due to its persistence and non-biodegradable nature. Among these, Cadmium (Cd) is one of the most toxic heavy metals in the periodic table. By nature, it is a relatively rare, silvery grey, soft solid metal. It is hardly found in elemental form in nature but mostly occurs as compound form with other elements, i.e., cadmium oxide, cadmium chloride, cadmium sulphide, cadmium cyanide etc. One of the major routes of entry of this metal in human is through fish, a daily food item for a large population in India. Cd is highly toxic to some aquatic life, though very less present in surface waters. But, with increased anthropogenic activity ecosystems are continuously polluted with industrial waste products which contain a high amount of Cd. This Cd eventually contaminates the water bodies. As a consequence, aquatic organisms are being continuously exposed to Cd [1]. This metal not only accumulates in the tissue of organisms but also increases in the higher trophic level i.e. biomagnifications. So, the consumption of these fish by a human being can lead to serious health risk. Already different researchers have reported a high level of Cd in several fish species in different parts of India [2, 3]. The objective of this review is to discuss briefly the bioaccumulation of Cd in fish and its toxicity; so that necessary steps can be taken to reduce the human health risk.

2. Sources
Natural and anthropogenic activities can be identified as an important source of Cd to the biosphere. Natural emissions are mainly from the mobilization of naturally occurring Cd from the earth's crust and mantle, e.g. volcanic eruptions and weathering of rocks. Anthropogenic sources are mainly from the mobilization of Cd impurities in raw materials (e.g., phosphate minerals, fossil fuels) and emissions from manufacturing, use, disposal, recycling, reclamation or incineration of products intentionally [4].

3. Routes of entry
There are different routes of entry of Cadmium in the body of a living organism. The main pathway of entry of Cadmium in the body of living tissue is gastro-intestinal absorption, absorption via gill and pulmonary absorption**.** Gastrointestinal (GI) absorption of ingested Cadmium is about 2% to 6% under normal conditions. In the case of aquatic organisms, the major absorption occurs through diet or gill. The great portion of the metal precipitates and resides in the floor of sediments when Cd is added to freshwaters.

So, bottom sediment may be an important source for Cd introduced to the aquatic systems. The bottom-feeding fauna and sediment-rooted flora can take up Cd in sediments. Significant amounts of cadmium may also be absorbed via lung due to inhalation of tobacco smoke or occupational exposure to atmospheric cadmium dust. This route of absorption occurs only in terrestrial animals. After absorption via the GI tract or the lung, cadmium is transported to the liver where the production of a cadmium-binding low molecular weight protein metallothionein takes place.

4. Uptake of Cadmium by fish

Direct uptake of Cd by fish mainly occurs from exposure to a contaminated medium or by consumption of food containing the chemicals. Direct uptake from water by fish is mainly in its free ionic form (Cd^{2+}) and the indirect exposure occur mainly as dietary means. Dietary exposure is possible especially when organisms ingest metals accumulated in lower trophic level organisms with potential for bioaccumulation. Metal intake of fish differs from that of terrestrial animals as gills of fish are being constantly submerged in a metal ions solution. So gills are the main point of entry for dissolved metals. A small portion of Cd present in dissolved form in water might be ingested through the skin by fish [5]. The entrance of Cd in the chloride cell of the gills occurred through the cadmium channels. Absorption of cadmium across the gills or intestinal walls is distributed through the circulation, bond to transport proteins and distributed to different tissues of the body [4].

5. Interaction with other components

Once Cd enters into the cells, it involves in interactions with cytoplasmic components such as enzymes which causes toxicity and metallothioneins (MTs). Metallothioneins (MTs) are low-molecular-weight metal binding proteins are known to play important roles, especially in the metabolisms and protection against heavy metal toxicity [6]. The MTs transport, detoxify and store Cd. Following the absorption, Cd is bound to albumin and transported to the liver. After that MT-bound Cd enters the plasma and in the glomerular filtrate it appears. From the glomerular filtrate, it is re-released intracellularly by renal tubule cells [7].

6. Factors affecting the absorption of cadmium in fish

Under the conditions of low pH, low hardness, low suspended matter levels, high redox potential and low salinity enhanced the mobility and bioavailability of Cd in aquatic environments. Water acidification, directly and indirectly, affects the metal accumulation rates by the fish. Water acidification affects indirectly bioaccumulation of metals by changing the solubility of metal compounds or directly by the damage of epithelia which become more permeable to metals. With reducing water hardness and concentrations of dissolved organic matter, the toxicity of Cd generally increases [8]. Water temperature also causes a difference in metal accumulation in different organs of freshwater fish [9]. Humus content of water also affects the concentration Cd in fish [10]. Cadmium is less toxic in saltwater than freshwater. Less toxicity of Cd is observed in saltwater because of its combination with chlorides in saltwater to form a less available molecule [11].

7. Bioaccumulation of cadmium in fish

Bioconcentration of heavy metal is generally inversely correlated with the trophic level and accumulation of Cd in tissues of carnivorous fish is low [12]. Toxic effects of Cd are likely to increase with increasing trophic levels [13]. Knowledge regarding the effects of Cd on genetic and biochemical adaptive responses of aquatic species [14]. On the basis of cellular level toxicological studies, it is evident that Cd inhibits the mitochondrial electron transfer chain and induce relatively oxygen species production [15].

8. Toxicity of cadmium in freshwater fish

Cadmium is a toxic as well as stress inducing agent for fish. Cadmium exposure can influence some physiological damages including growth rate reduction in fish. Cd induces both hepatic and renal injuries in mammals and fish which has the capacity to cause Oxidative stress. Cd is also known to interfere with many protein and carbohydrate metabolism by enzyme inhibition process [16]. It can disturb the ion balance in teleost fishes [17]. Cd can influence the calcium metabolism and causes low cadmium level (Hypocalcaemia) probably by calcium uptake inhibition process. Due to the long period exposure of the animal to a lower concentration, it would be chronically toxic. Effects of long-term exposure can include larval mortality and temporary growth reduction [18]. Chronic exposure can lead to mortality. Acute toxicity of Cadmium generally results from exposure to a high concentration over a short period of time. Accumulation Cadmium in aquatic biota may cause chronical exposure to sub-lethal concentration [10]. Common sub-lethal effects are reduced growth and reproductive failure. The most bioavailable form of Cadmium of Cd is also the divalent ion (Cd^{2+}). When organisms are exposed to this form of Cadmium, it induces the metallothionein synthesis which binds with Cd and decreases its toxicity [19] and this generally occurs in the liver of the fish. Impaired ability of the fish to find food and avoid predators is considered as the result of skeletal deformities in fish which cause this sub-lethal effect as a lethal effect. The elevated frequencies of micro-nucleated and bi-nucleated erythrocytes in peripheral blood, gill epithelial cells and liver cells providing evidence of cytotoxic and genotoxic effects of cadmium [20].

9. Trends of accumulation of cadmium in fish tissue

Several studies show that chronic exposure causes tissue-specific accumulation of cadmium in fish. Different tissues have different capacity for heavy metal accumulation [21]. According to many studies, Cd is primarily accumulated in the kidney and liver but may reach a high concentration in the gill, alimentary canal and muscles as well. Cd concentration is generally higher in gills and kidney in case of waterborne exposure whereas Cd concentrations are generally higher in the gastrointestinal tract than in the gills. Researchers have also demonstrated that Cd triggered the liver cell apoptosis as a result caspase3A activity was markedly increased [22]. So it indicates that Cd can trigger liver cell apoptosis through the activation of caspase3A as it plays an essential role in Cd-induced apoptosis.

10. Cadmium toxicity in human

10.1. Acute toxicity

Inhalation of cadmium compounds at concentrations above 1 mg Cd/m3 in the air for 8 hours or at a higher concentration for shorter periods, may lead to chemical acute toxicity of Cadmium. Symptoms generally occur within 1 to 8 hours after exposure. The more severe symptoms of acute toxicity of Cd may have a latency period up to 24 hours. Death may occur after 4 to 7 days. Symptoms are nausea, vomiting, abdominal pains and sometimes diarrhea.

10.2. Chronic toxicity

Chronic toxicity has been reported after prolonged exposure to cadmium oxide fumes, cadmium oxide dust and cadmium stearates. Chronic cadmium toxicity may be local, where they involve the respiratory tract or system, resulting from absorption of cadmium. Systemic changes include kidney damage with proteinuria and anemia. Obstructive lung disease in the form of emphysema is the main symptom at heavy exposure to Cadmium in air, where as kidney dysfunction and damage are the most prominent findings after long-term exposure to low levels of cadmium in work room air or via cadmium contaminated food. Hypochromic anemia is also frequently found among workers exposed to high levels of Cadmium. This may be due to both increased destruction of red blood cells and iron deficiency. Yellow discoloration of the necks of teeth and anosmia may also be seen in cases of exposure to very high Cadmium concentrations. Pulmonary emphysema is considered a possible effect of prolonged exposure to Cadmium in the air at a concentration exceeding 0.1 mg Cd/m^3. Cadmium-induced pulmonary emphysema can decrease working capacity and may be the cause of invalidity of life-shortening. In the case of long-term low-level cadmium exposure, the kidney is the critical organ, which is 1st affected. This causes tubular proteinuria with increase excretion of low molecular protein such as β2-micro globin. As the kidney dysfunction progresses, amino acids, glucose and minerals, such as Cadmium and phosphorus are also lost into the urine. Increased excretion of Cadmium and Phosphorus may disturb the bone metabolism and kidney stones are frequently found in exposed persons. Osteomalacia has been found in cases of severe chronic cadmium poisoning. Excessive Cd exposure has occurred in the general population through ingestion of contaminated rice of other foodstuff and possible drinking water. The disease itai-itai is a painful type of osteomalacia, with multiple fractures appearing together with kidney dysfunction has occurred in Japan in the areas with high Cd exposure.

11. Control of anthropogenic release of cadmium

The controlling measures of the anthropogenic release of cadmium are varied which include control of uses and applications Cd-containing products. Guidelines for Cd content in effluents, fertilizers and sewage sludge, standards for air, soil, sediment, water have been provided by UNEP (United Nations Environment Programme) [23]. The kidney is the critical organ with long-term cadmium exposure via air or food. The critical concentration is about 200µg Cd /g wet weight. In order to keep the kidney cortex concentration below this level, the average concentration in the air should not exceed 0.01mg Cd $/m^3$. The intake of Cadmium should not cross for the purpose via food 70µg Cd /day [24–26]. Work processes and operations which may release cadmium fumes or dust into the atmosphere should be designed to keep concentration levels to a minimum and if practicable be enclose and fitted with exhaust ventilation. In case of the area with hazards of flying particles, chemical splashes, radiant heat etc.e.g., electroplating tanks and furnaces, the worker should wear appropriate safety equipment, such as eye, face, hand and arm protection clothing. Adequate sanitary facilities should be supplied and workers should encourage to wash before meals and to wash thoroughly and change clothes before leaving work. Smoking, eating, drinking in work areas should be strictly prohibited. Contaminated exhaust should be filtered and persons in a change of dust collectors and filters should wear respirators while working on the equipment. To ensure the excessive accumulation of cadmium in the kidney does not occur, Cadmium level in blood and urine should be checked regularly. We have also seen that Cd mainly accumulate in the internal organs of the fish. So, during preparation, the offal portion of the fish can be discarded. It can reduce a significant amount of Cd from the diet [2].

12. Conclusion

It is clear from the above discussion that Cd toxicity has become a serious threat to the ecosystem as well as human health. With increased anthropogenic activity environmental pollution has also increased. So, first, we should try to reduce the level of Cd in the environment. It can be done by controlling the emission of Cd containing wastes from the industries and other sources. Secondly, we should also try to reduce uptake of Cd through proper diet selection and following proper fish preparation method. Unless and until we take the necessary steps to save our environment, we could not offer a better World for our next generations.

13. References

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