

ORIGINAL ARTICLES

Overview of Cadmium Toxicity in Fish

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ABSTRACT

Discharge of heavy metals into aquatic environment from various sources, even below permissible levels, creates health hazards in aquatic organisms. The persistence and ubiquitous nature of these pollutants coupled with their tendency to accumulate in organisms ultimately produce toxic reactions in aquatic biota, especially fish. This document gives a brief overview of the heavy metal, cadmium, considered as one of the most toxic heavy metals. The overview deals among other issues, the toxic effects of cadmium on the aquatic biota with emphasis on fish and the public health implication.

Key words: Cadmium, Toxicity, Fish.

Introduction

The contamination of fresh waters with a wide range of pollutants has become a matter of concern over the last few decades (Vutukuru, 2005; Dirilgen, 2001; Voegborlo *et al.*, 1999; Canli *et al.*, 1998). The natural aquatic systems may extensively be contaminated with heavy metals released from domestic, industrial and other man-made activities (Velez and Montoro, 1998; Conacher, *et al.*, 1993). Heavy metal contamination may have devastating effects on the ecological balance of the recipient environment and a diversity of aquatic organisms (Farombi, *et al.*, 2007; Vosyliene and Jankaite, 2006; Ashraj, 2005).

Cadmium is considered as one of the most toxic heavy metals. It is a nonessential element to all living organisms. Rivers and lake shores are the areas primarily affected by diluted cadmium waste from industrial facilities in big cities (Randi, 1996). The cadmium-related contamination of the aquatic habitat has greatly increased in the last decades, resulting in an increase of cadmium deposits in tissues of aquatic organisms in all food chain systems (Giles, 1988). It is important to note that cadmium is a highly toxic element for all mammals and fish. Cadmium levels have constantly been increasing, and consequently, the research on cadmium has become quite topical and urgent. Accumulation of cadmium in living organisms is a major ecological concern, especially because of its ability to accumulate very quickly. By contrast, the excretion of cadmium from living organisms is a slow process. In fish, cadmium can cause a number of structural and pathomorphological changes in various organs. The highest cadmium levels were detected in the kidneys and liver of fish (Thophon *et al.*, 2003). Cadmium is noted for its tendency to accumulate in the organisms of mammals for a prolonged biological semi- life. It is responsible for increased hypertension, emphysema, kidney tubule damage, impaired liver function, and cancer (Ribelin and Migaki, 1975). The study performed on seabass (*Dicentrarchus labrax*), in the case of acute cadmium poisoning, has shown that the primary site of toxicity are the gills' lamellae and kidney tubules. In the case of subchronic poisoning, the primarily affected organs appear to be kidneys and liver, and to a lesser extent the gills (Thophon *et al.*, 2003).

Among animal species, fishes are the inhabitants that cannot escape from the detrimental effects of these pollutants (Olaifa *et al.*, 2004; Clarkson, 1998; Dickman and Leung, 1998). Fish are widely used to evaluate the health of aquatic ecosystems because pollutants build up in the food chain and are responsible for adverse effects and death in the aquatic systems (Farkas *et al.*, 2002; Yousuf and El-Shahawi, 1999). The studies carried out on various fishes have shown that heavy metals may alter the physiological activities and biochemical parameters both in tissues and in blood (Basa and Rani, 2003; Canli, 1995; Tort and Torres, 1988). The toxic effects of heavy metals have been reviewed, including bioaccumulation (Waqar 2006; Adami *et al.*, 2002; Rasmussen and Anderson, 2000; Rani, 2000; Aucoin *et al.*, 1999).

The organisms developed a protective defense against the deleterious effects of essential and inessential heavy metals and other xenobiotics that produce degenerative changes like oxidative stress in the body (Abou EL-Naga *et al.*, 2005; Filipovic and Raspor, 2003). A variety of contaminants including toxic heavy metals (cadmium, copper, mercury and zinc) are reported to be ubiquitously present in rivers, reservoirs and are disadvantageous for aquatic organisms (Olsson, 1998). In general, they are not biodegraded and therefore, their bioaccumulation in fish, oyster, mussels, sediments and other components of aquatic ecosystems have been reported from all over the world. It appears that problem of heavy metals accumulation in aquatic organisms including fish needs continuous monitoring and surveillance owing to biomagnifying potential of toxic metals in human food chain (Das and Kaviraj 2000; Laxi, 2005; Jayakumar and Paul 2006; Kumar *et al.*, 2007; Kumar *et al.*, 2008; Kumar *et al.*, 2009).

History of Cadmium:

Cadmium (Latin *cadmia*, Greek *καδμεία* meaning "calamine", a cadmium-bearing mixture of minerals, which was named after the Greek mythological character, Κάδμος Cadmus, the founder of Thebes) was discovered simultaneously by Friedrich Stromeyer and Karl Samuel Leberecht Hermann, both in Germany, as an impurity in zinc carbonate Hermann (1818). Stromeyer found the new element as an impurity in zinc carbonate (calamine), and, for 100 years, Germany remained the only important producer of the metal. The metal was named after the Latin word for calamine, since the metal was found in this zinc compound. Stromeyer noted that some impure samples of calamine changed color when heated but pure calamine did not. He was persistent in studying these results and eventually isolated cadmium metal by roasting and reduction of the sulfide. Even though cadmium and its compounds may be toxic in certain forms and concentrations, the British Pharmaceutical Codex from 1907 states that cadmium iodide was used as a medication to treat "enlarged joints, scrofulous glands, and chilblains" Dunglison (1866).

In 1927, the International Conference on Weights and Measures redefined the meter in terms of a red cadmium spectral line ($1 \text{ m} = 1,553,164.13 \text{ wavelengths}$) Burdun, G. D. (1958).

Environmental Chemistry and Background Residues:

Cadmium is a silver-white, blue-tinged, lustrous metal that melts at 321°C and boils at 765°C . This divalent element has an atomic weight of 112.4 and an atomic number of 48. It is insoluble in water, although its chloride and sulphate salts are freely soluble (Windholz *et al.* 1976). The availability of Cd to living organisms from their immediate physical and chemical environs depends on numerous factors, including adsorption and desorption rates of cadmium from terrigenous materials, pH, Eh, chemical speciation, and many other modifiers. The few selected examples that follow demonstrate the complex behavior of Cd in freshwater systems.

Adsorption and desorption processes are likely to be major factors in controlling the concentration of cadmium in natural waters and tend to counteract changes in the concentration of cadmium ions in solution (Gardiner 1974). Adsorption and desorption rates of cadmium are rapid on mud solids and particles of clay, silica, humic material, and other naturally occurring solids. Concentration factors for river muds varied between 5,000 and 500,000 and depended mainly on the type of solid, the particle size, the concentration of cadmium present, the duration of contact, and the concentration of complexing ligands; humic material appeared to be the main component of river mud responsible for adsorption (Gardiner 1974). Changes in physicochemical conditions, especially pH and redox potential, that occur during dredging and disposal of Cd-polluted sediments may increase chemical mobility and, hence, bioavailability of sediment-bound Cd (Khalid *et al.* 1981). For example, cadmium in Mississippi River sediments spiked with radiocadmium was transformed from potentially available organic forms to more mobile and readily available dissolved and exchangeable forms (i.e., increased bioavailability) under regimens of comparatively acidic pH and high oxidation (Khalid *et al.* 1981). The role of dissolved oxygen and aquatic plants on Cd cycling was studied in Palestine lake, a 92-ha eutrophic lake in Kosciusko County, Indiana, a long-term recipient of cadmium and other waste metals from an electroplating plant. The maximum recorded concentration of dissolved Cd in the water column was 17.3 ppb; for suspended particulates, it was 30.3 ppb (Shephard *et al.* 1980). During anaerobic conditions in the lake's hypolimnion, a marked decrease in the dissolved fraction and a corresponding increase in the suspended fraction were noted. The dominant form of cadmium was free, readily bioavailable, cadmium ion, Cd^{2+} ; however, organic complexes of Cd, which are comparatively nonbioavailable, made up a significant portion of the total dissolved Cd. Cadmium levels in sediments of Palestine Lake ranged from 1.5 ppm in an uncontaminated area of the lake to 805 ppm near the outlet of a metal-bearing ditch that entered the lake (McIntosh *et al.* 1978).

The dominant form of Cd in sediments was a carbonate. Levels of Cd in water varied over time and between sites, but usually ranged from 0.5 to 2.5 ppb. It is possible that significant amounts of cadmium are transferred from the sediments into rooted aquatic macrophytes and later released into the water after macrophyte death (natural or herbicide-induced), particularly in heavily contaminated systems. In Palestine Lake, Cd levels in pondweed (*Potamogeton crispus*), a rooted aquatic macrophyte, were about 90 ppm dry weights; a maximum burden of 1.5 kg was retained by the population of *P. crispus* in the lake (McIntosh *et al.* 1978). Release of the total amount could raise water concentrations by a maximum of 1 ppb. This amount was considered negligible in terms of the overall lake Cd budgets; however, it might have limited local effects. As judged by these and other complexities regarding Cd bioavailability, it appears that the organism remains the ultimate arbiter of its environment, regardless of the source of cadmium and its geophysical surroundings.

Background levels of cadmium in uncontaminated, nonbiological compartments extended over several orders of magnitude (Korte 1983). Concentrations (ppb) of cadmium reported ranged from 0.05 to 0.2 in freshwater, up to 0.05 in coastal seawater, from 0.01 to 0.1 in open ocean seawater, up to 5,000 in riverine and lake sediments, 30 to 1,000 in marine sediments, 10 to 1,000 in soils of nonvolcanic origin, up to 4,500 in soils of volcanic origin, 1 to 600 in igneous rock, up to 100,000 in phosphatic rock, and 0.001 to 0.005 µg/m³ in air (Korte 1983). Where Cd is comparatively bioavailable, these values are very near those that have been shown to produce harmful effects in sensitive biological species, as will be discussed later.

Cadmium, unlike synthetic compounds, is a naturally occurring element, and its presence has been detected in more than 1,000 species of aquatic and terrestrial flora and fauna. Concentrations of cadmium in a few selected species of aquatic biota are shown in Table 1;

Table 1: Cadmium concentrations in field collections of selected aquatic species. Values shown are in mg Cd/kg fresh weight (FW), dry weight (DW), or ash weight (AW).

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration	Reference ^a
Marine		
Fish		
Flounder, <i>Platichthys flesus</i>		
Whole		
Barnstaple Bay, U.K.	Age III 1.1DW	Hardisty <i>et al.</i> 1974
Age III	1.4DW	
Age IV	1.6DW	
Age V	1.7DW	
Oldbury on Severn, U.K. (metals-contaminated area)		
Age	II 4.0DW	
Age III	4.5DW	
Age IV	5.1DW	
Age V	5.2DW	
Yellowtail flounder, <i>Limanda limanda</i>		
Liver	0.4DW	Wesernhagen <i>et al.</i> 1980
Skin	0.2DW	
Otoliths	0.2DW	
Gills	0.2DW	
Fin	0.2DW	
Muscle	0.1DW	
Backbone	0.05DW	
Blue Marlin, <i>Makaira indica</i>		
Muscle	0.1-0.4FW	Mackay <i>et al.</i> 1976
Liver	0.2-83.0FW	
Striped bass, <i>Morone saxatilis</i>		
Muscle	0.03FW	Heit 1979
Liver	0.3FW	
Atlantic cod, <i>Gadus morrhua</i>		
Roe	0.0-0.5DW	Julsman and Braekkan 1978
Muscle	0.02DW	Julsman and Braekkan 1978
Gonad	0.0-0.07DW	
Liver	0.09DW	
Bluefish, <i>Pomatomus saltatrix</i>		
Muscle	up to 0.08DW	Bebbington <i>et al.</i> 1977
Shorthorn sculpin, <i>Myoxocephalus scorpius</i>		
Muscle	1.4DW	Bohn and Fallis 1978
Liver	4.1DW	
Freshwater		
Fish		
United States, Nationwide, 1976-1977		
Whole	0.07FW(0.01-1.04)	May and McKinney 1981

Table 1: Continue.

Upper Clark Fork River, Western Montana		
Muscles, 3spp	0.2-0.6FW	Hammons <i>et al.</i> 1978
Liver, 7spp.	0.3-0.8FW	
Great Lakes		
Whole, 3spp.	0.0-0.6FW	
Liver, 7spp	0.3-0.8FW	
Great Lakes		
Whole, 3spp	0.0-0.14FW	
Liver, 10spp	0.1-1.4FW	
Illinois River		
Whole, 10spp	<0.08FW	
New York State, various locations		
Whole		
Adirondacks region	0.02-0.05FW	Lovett <i>et al.</i> 1972
Hudson River	up to 0.14FW	
47 other areas	<0.02FW	
Rainbow trout, <i>Salmo gairdneri</i>		
Alaska		
Whole	<0.07FW	Jenkins 1980.
Arizona		
Whole	<0.05FW	
White crappie, <i>Pomoxis annularis</i>		
Whole	0.0-0.3FW	
Sauger, <i>Stizostedion canadense</i>		
Whole	<0.05FW	
Walleye, <i>Stizostedion vitreum vitreum</i>		
Liver	0.2FW	
Whole	up to 0.16FW	

*Each reference applies to the values in the same row and in the rows that follow for which no other reference is indicated.

Source of Exposure:

Cadmium is released to the biosphere from both natural and anthropogenic sources. It is an element that occurs naturally in the earth's crust and got rank 7 of ASTDR's "Top 20 list" (ASTDR, 1999). Percentage of cadmium in the upper soil has been increasing because it is found in insecticides, fungicides, sludge, and commercial fertilizers which are routinely used in agriculture. Dental alloys, electroplating, motor oil, and exhaust are other sources of Cd pollution. Hence, anthropogenic activities have increased Cd magnification in the environment. 10% of total Cd in the environment is derived from natural sources, whereas remaining 90% is derived from anthropogenic activity.

(Okada *et al.*, 1997). Volcanic activity contributes about 62% of natural emissions and other natural sources include decaying of vegetation (25%) airborne soil particles (12%) and forest fire (2%). Its non-corrosive and cumulative nature has made it very important due to its applications in electroplating or galvanizing. It is also used as colour pigment for paints, plastics, and as a cathode material for nickel-cadmium batteries. Anthropogenic activities like; smelting operations, use of phosphate fertilizers, pigment, cigarettes smokes, automobiles etc. have contributed to the entry of cadmium into human and animal food chain (WHO, 1992; Okada *et al.*, 1997; Kumar *et al.*, 2007). Presence of cadmium at higher concentration than the maximum allowable limits in water, vegetation and food have been reported by author (Agarwal and Raj, 1978; Khandekar *et al.*, 1980; Allen, 1995; Laxi, 2005; Kumar *et al.*, 2008; Asagba, 2010).

Higher level of Cd has also been detected in sewage sludge (rich in almost all nutrients and hence generally used as plant fertilizer), various vegetables (Roblenbeck *et al.*, 1999), animals feed and their tissues (Kumar *et al.*, 2007). Topsoil enriched in sludge contributes Cd accumulation in the blood, milk, hair, liver and kidney of sheep, goat, cow, buffalo (Brebner *et al.*, 1993; Swarup *et al.*, 2005; Balagangatharathilagar *et al.*, 2006 and Patra *et al.*, 2007).

In India, various levels of cadmium concentration have been reported to be present in aquatic ecosystem which is more than 5ng/ml in the Yamuna river water at Agra, Delhi, Etawah and Mathura (Ajmal *et al.*, 1985) and 0.50-114.8 mg/kg in the Yamuna river sediments at Agra and Delhi but the water around the industrial areas have been found to contain higher levels of cadmium (Singh, 2001 and Kaushik *et al.*, 2003). Similarly, Hindon River (Uttar Pradesh) has also been contaminated with heavy metals including cadmium (Jain and Sharma, 2001 and Sharma, 2003). Moreover, high concentration of cadmium (70-100 ng/ml) has been detected in Bombay city (Agrawal and Raj, 1978), Lalbag pond water of Baroda city (Kannan, 1997) and edible tissues of fish and chicken in western UP market (Kumar *et al.*, 2006 and 2007; Burger, 2008).

Site for 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).

Molecular Mechanisms of Absorption:

There are various mode of Cd uptake in aquatic organism, where it is most readily absorbed by organisms directly from the water in its free ionic form Cd (II) (AMAP 1998). 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 cadmium ions enter the chloride cells in the gills through calcium channels (Olsson, P.E., 1998). Once enter in the cells the metal is made available for the interaction with cytoplasmatic components such as enzymes (causing toxic effects) and Metallothioneine (probably being detoxified). Although Metallothioneine is induced in the gills it does not appear to be as capable of sequestering the vast majority of accumulated Cd²⁺, as it is in the liver (Olsson and Hogstrand, 1987). The reason for this is believed to be due to the high affinity of Cd²⁺ for Ca²⁺ binding sites in the gills (Flick *et al.*, 1987), and it is also believed that Cd²⁺ binds to the active sites on the basolateral Ca²⁺-pump in chloride cells. It thus seems that Cd²⁺ enters the gills through Ca-channel on the apical side and is further translocated to the circulation interactions with Ca²⁺-ATPases on the basolateral side.

Interaction with Other Elements:

Zinc increases the toxicity of cadmium to aquatic invertebrates. However, high calcium concentrations in water protect them from cadmium uptake by competing at uptake sites. It is very rare that only one toxic element, at a time, is released into the aquatic ecosystem. Most of the heavy metals interact with each other and also influenced by other ions (e.g. Ca²⁺, Mg²⁺, Na²⁺, Mn²⁺, Fe²⁺, Pb²⁺, S²⁺, Se²⁺ and Ni²⁺). Calcium has been shown to interact with Cd²⁺ to potentiate or minimize their toxicity. Elevated ingestion Cd²⁺ can produce deficiency states of both Cu²⁺ and Zn²⁺. Exposure of animals to Cd²⁺ results in alteration in the Zn²⁺ metabolism.

Tissue Distribution:

Bioaccumulation of cadmium takes place at tropic level and found to be highest in algae (Ferard *et al.*, 1983; Pinto *et al.*, 2003). It also accumulates in considerable concentrations in various organs of fish (Sindayigaya *et al.*, 1994; Kumar *et al.*, 2006; Kumar *et al.*, 2008). Smet & Blust (2001) reported that cadmium accumulates in tissues of carp *Cyprinus carpio* in following order: kidney> Liver> Gills. Kumar *et al.* (2005) have also reported similar accumulation pattern in *Clarias batrachus* in an experimental study. Some insects can also accumulate high levels of cadmium without showing any adverse effects (Jamil and Hussain, 1992). Kidney is the prime target organ for cadmium. The liver also stores a considerable part of the accumulated cadmium. Cadmium is redistributed to these organs directly following uptake through the gills and intestine, but there may also be redistribution of cadmium from other organs (Olsson and Hogstrand, 1987).

Toxic Effects:

In aquatic systems, cadmium is most readily absorbed by organisms directly from the water in its free ionic form Cd (II) /AMAP 1998/. The acute toxicity of cadmium to aquatic organisms is variable, even between closely related species, and is related to the free ionic concentration of the metal. Cadmium interacts with the calcium metabolism of animals. In fish it causes lack of calcium (hypocalcaemia), probably by inhibiting calcium uptake from the water. However, high calcium concentrations in the water protect fish from cadmium uptake by competing at uptake sites. Effects of long-term exposure can include larval mortality and temporary reduction in growth /AMAP 1998/. Zinc increases the toxicity of cadmium to aquatic invertebrates. Sublethal effects have been reported on the growth and reproduction of aquatic invertebrates; there are structural effects on invertebrate gills. There is evidence of the selection of resistant strains of aquatic invertebrates after exposure to cadmium in the field. The toxicity is variable in fish, salmonoids being particularly susceptible to cadmium. Sublethal effects in fish, notably malformation of the spine, have been reported. The most susceptible life-stages are the embryo and early larva, while eggs are the least susceptible.

In studies of lake trout exposed to different levels of cadmium, researchers found that cadmium affected foraging behavior, resulting in lower success at catching prey. Decreased thyroid function as a result of cadmium exposure has also been documented. Both responses indicate a low response threshold for cadmium caused behavioural changes. /AMAP 2002/. Cadmium has been reported to exert deleterious effects in terms of nephrotoxic, cytotoxic, genotoxic, immunotoxic and carcinogenic (ASTDR, 1999; Lippmann, 2000 and Rissode- faverney, 2001).

Nephrotoxicity:

Cadmium is heavy metal and poses high toxicity at very low level of exposure and has acute and chronic effects on aquatic animal health and environment. Long exposure of cadmium produces a wide variety of acute and chronic effects in aquatic animals. It's prime site is kidney (Thomas *et al.*, 1983 and Kuroshima, 1992). According to the current knowledge kidney damage (renal tubular damage) is probably the critical health effect (Jarup *et al.*, 1998). Not only this it also creates disturbances of calcium metabolism, hypercalciuria and takes part in the formation of stones in the kidney. The toxicity is variable in fish, salmonoids being particularly susceptible to cadmium. Sublethal effects in fish, notably malformation of the spine, have been reported.

Induction of Oxidative Stress:

Free radicals and other reactive oxygen species (ROS) have been recently incriminated in the pathogenesis of various metal toxicities (Yiin *et al.*, 1999a and 1999b; Senapati *et al.*, 2001; Basha and Rani, 2003; Rahman, 2003; Suresh, 2009). There are many reports suggesting alterations in free radicals production and antioxidant defense system of the body after cadmium exposure. Administration of Cadmium chloride at the dose rate of 15µg/ml in drinking water for 30 days revealed significant increase in lipid peroxidation (LPO) in cortical region of kidney (Oner *et al.*, 1995). Treatment of rats with Cd²⁺ significantly increases in LPO in heart within 3 hours of Cd²⁺ injection and kidney and liver within 6-12 hours. Superoxide dismutase (SOD) activity increased in heart, liver and kidney within 24 hours of Cd²⁺ intoxication. Catalase activities were also increased significantly in heart after 9 hours of Cd²⁺ injection without any significant change in liver and kidney (Sarkar *et al.*, 1995).

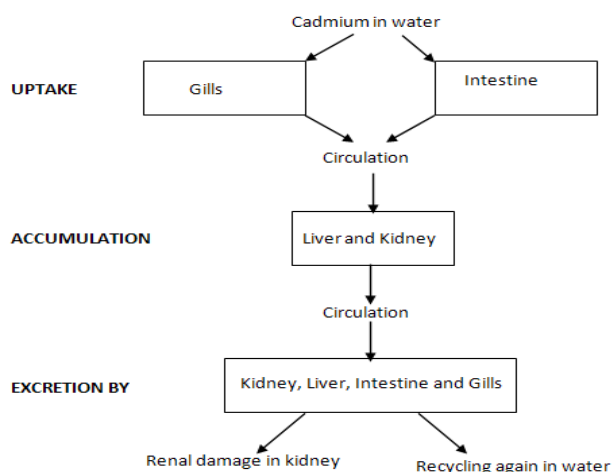


Fig. 1: The cycle of Cadmium in aquatic ecosystem

Source: Puneet K and Anu S (2010).

Immunotoxicity:

A number of investigations have suggested that cadmium may exert immunosuppressive effects of cadmium exposure in both fishes and mammals (Zellkoff *et al.*, 1995; Kim *et al.*, 2000 and Giari *et al.*, 2007). Recent reports suggest that cell mediated immunity is most affected (Kumar *et al.*, 2008) and phagocytosis, natural killer cell activity and host resistance towards experimental infections are markedly impaired in cadmium toxicity. Sovenyl and Szakolczal (1993) also reported marked immunosuppressive effects of cadmium exposure on common carp in terms of lowered antibody response, lysozyme level and microcidal capacity of phagocytes. Some reports suggest that cadmium enhances humoral immune response at low level of exposure (Descotes, 1992; Krumschnabel *et al.*, 2010).

Effect on Organ Structure and Function:

Cadmium in high doses induce structural and function alterations in various vital organs including liver, kidney, gill and intestine of fishes.

Liver:

Cadmium accumulates in liver of fishes in high concentrations (Smet and Blust, 2001; Rangsayatorn *et al.*, 2004). It also induces various pathological changes in liver tissues including engorgement of blood vessels, congestion, vacuolar degeneration of hepatocytes, necrosis of pancreatic cells and fatty changes in the peripancreatic hepatocytes (Rani and Ramamurthi, 1989; Dangre *et al.*, 2010).

Kidney:

Cadmium accumulates in kidney of fishes in maximum concentration. Cadmium has been reported to possess nephrotoxic action in man and various animals. In fact, kidney is the principle target organ of cadmium toxicity and chronic cadmium exposure in almost all animal species is characterized by varying degree of renal damage (Roméo *et al.*, 2000; Shukla and Gautam, 2004; Kumar *et al.*, 2006; Kumar *et al.*, 2009; Vesey, 2010).

Gills:

Gills are also reported to act as storehouse of cadmium in experimental studies (Allen, 1995; Tao *et al.*, 2000; Fafioye *et al.*, 2004; Ramesh and Nagaranjan, 2007). Wong and Wong (2000) studied morphological and biochemical changes in the gills of Tilapia (*Oreochromis mossambicus*) after experimental cadmium exposure. In scanning electron microscopic studies, they found an augmentation of microbridges in pavement cells and an increase in the apical membrane of chloride cells. They further reported chloride cells as a prime target of cadmium toxicity, resulting into fish hypocalcemia. Other organs like intestine and gonads of fishes also appear susceptible for ill effects of cadmium toxicity (Taylor, 1983; Kumari and Ram, 1997; Singh *et al.*, 2007; Kumar, 2007).

Public Health Implication:

One of the routes of exposure to cadmium in humans is via food which could be through the consumption of cadmium contaminated fish. Cadmium accumulates in the human body and especially in the kidneys. According to the current knowledge kidney damage (renal tubular damage) is probably the critical health effect, both in the general population and in occupational exposed workers (Järup *et al.* 1998). The accumulation of cadmium in the kidney (in the renal cortex) leads to dysfunction of the kidney with impaired reabsorption of, for instance, proteins, glucose, and amino acids. Both human and animal studies indicate that skeletal damage (osteoporosis) may be a critical effect of cadmium exposure, but the significance of the effect in the Swedish population is according to Järup *et al.* 1998/ still unclear. Lung changes primarily characterised by chronic obstructive airway disease may follow high occupational exposure (WHO 1992a). Early minor changes in ventilatory function tests may progress, with continued cadmium exposure, to respiratory insufficiency. An increased mortality rate from obstructive lung disease has been seen in workers with high exposure in the past. Other effects of cadmium exposure are disturbances in calcium metabolism, hypercalciuria and formation of stones in the kidney. The International Agency for Research on Cancer (IARC) classifies cadmium in Class 1 'The agent (mixture) is carcinogenic to humans. The exposure circumstance entails exposures that are carcinogenic to humans.' (IARC 1993). Occupational exposure is linked to lung cancer and prostate cancer. According to a recent review, the epidemiological data linking cadmium and lung cancer are much stronger than for prostate cancer, whereas links between cadmium and cancer in liver, kidney and stomach is considered equivocal (Waalkes 2000).

Recommendations:

Proposed limits for cadmium in water for protection of human health and aquatic life are shown in Table 2. It is noteworthy that the current upper limit of 10.0 ppb of cadmium in drinking water for human health protection is not sufficient to protect many species of freshwater biota against the biocidal properties of cadmium or against sublethal effects, such as reduced growth and inhibited reproduction.

Ambient water quality criteria formulated for protection of freshwater aquatic life state that, for total recoverable cadmium, the criterion, in $\mu\text{g/L}$, is the numerical value given by e to the power $(1.05 (\ln(\text{hardness})) - 8.53)$ as a 24-h average and the concentration, in ppb, should never exceed the numerical value given by e to the power $(1.05 (\ln(\text{hardness})) - 3.73)$. Thus, at water hardnesses of 50, 100, and 200 mg/L as CaCO_3 , the criteria are 0.012, 0.025, and 0.051 ppb, respectively, and the concentration of total recoverable cadmium should never exceed 1.5, 3.0, and 6.3 ppb, respectively. Unfortunately, data are accumulating that demonstrate that even these comparatively rigorous criteria are not sufficient to protect the most sensitive species of freshwater insects, plants, crustaceans, and teleosts. It now appears that levels in excess of 3.0 ppb of cadmium in freshwater are potentially hazardous to aquatic biota and that levels near 1.0 ppb are cause for concern in waters of low alkalinity. Not listed in Table 2, but still recognized as proposed criteria (EPA 1973), are the comparatively high levels of 10.0 ppb allowed for agricultural use on all soils (except neutral and alkaline soils, which may be irrigated with water having levels as high as 50.0 ppb) and public water supplies for livestock purposes, which may not exceed 50.0 ppb of cadmium.

The saltwater aquatic life protection criterion of 4.5 ppb seems adequate to prevent death, but will not prevent potentially deleterious physiological effects, including disrupted respiration in crustaceans and teleost. Incidentally, at 5.0 ppb of cadmium, the lowest concentration critically examined, oysters biomagnify ambient levels to concentrations hazardous to human consumers and possibly other animal consumers. The maximum allowable saltwater concentration (MAC) during a 24-h period was recommended as 59.0 ppb (Table 2). However, death of various species of marine crustaceans was reported at 60.0 ppb after exposure for 6 weeks and at 14.8 to 19.5 ppb after 23 to 27 days. Furthermore, a MAC of 59 ppb may be met with daily discharges of 59 ppb for 2 h and no discharge of cadmium for the rest of the day. The effects of exposure of marine life to 59 ppb of cadmium salts for 2 h daily for protracted periods have not yet been investigated. Accordingly, seawater concentrations in excess of 4.5 ppb of total cadmium at any time should be considered as potentially hazardous to marine life, until additional data prove otherwise.

Finally, the issue of the significance of cadmium residues in various body parts requires resolution. At this time, it appears that cadmium residues in the vertebrate kidney or liver that exceed 10.0 mg/kg fresh weight or 2.0 mg/kg in whole body fresh weight should be viewed as evidence of probable cadmium contamination. Elevated levels of 13.0 to 15.0 ppm Cd tissue fresh weight probably represent a significant hazard to animals of the higher trophic levels, and residues of 200 ppm fresh weight kidney or more than 5.0 ppm whole animal fresh weight should be considered life-threatening.

Table 2: Current recommendations for Cadmium in water (adapted from EPA 1980).

Ecosystem, Environmental and other variables	Cadmium concentration ^a $\mu\text{g/l}$
Water	
Freshwater aquatic life protection	
At water hardness, in $\text{mg CaCO}_3/\text{l}$, of	
50	1.5
100	3.0
200	6.3
Saltwater aquatic life protection	
24-h average	4.5
Maximum allowable concentration	59.0
Human health protection	
Best case	0.5
Average case	1.3
Worst case	10.0

^aUnits of concentration shown apply to the ecosystem concerned (water).

^bAssumes consumption of 0.75kg food per day by 70kg adult.

Conclusion:

The toxicity of cadmium in fish cannot be over-emphasised. The knowledge about cadmium toxicity in fish is important both with respect to nature management and human consumption. This document shows that the consumption of cadmium contaminated fish has a very serious implication.

Consequently, very close monitoring of cadmium loads in the aquatic system is recommended in view of the possible risks to health of consumers.

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