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Studies on Aluminium Toxicity to a Fresh Water Fish, *Channa punctatus*

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ABSTRACT

Aluminium toxicity to an edible fresh water fish, Channa punctatus has been studied in aquarium model. 10 fish in each case were exposed to 50 ppm and 25 ppm concentration of Al^{3+} in the form of aluminium sulphate for 50 days or till total mortality, whichever was earlier. Similar number of fish were also studied in the Control set. Mortality of the fish was recorded. At the end, the fish that died last in case of Experimental sets and five surviving fish of Control set were sacrificed. Aluminium uptake by the tissue of the fish in different parts viz., head, middle and tail part was studied. Results revealed that the exposure of fish to aluminium concentration of 50 ppm is acutely toxic. By 96 hours, all the fish died. In case of 25 ppm Al^{3+} exposure, the toxicity was found to be chronic. 50% of the fish died gradually in 50 days. The upper part of fish (brain and gills) were found to be the major sites of aluminium accumulation. The lower part (tail) showed relatively low aluminium accumulation. The total aluminium uptake by fish tissue was found to be 4.47 mg/g by 96 hours, in case of 50 ppm Al^{3+} exposure, and 4.80 mg/g by 50 days, in case of 25 ppm Al^{3+} exposure.

Key Words: Aluminium toxicology, Aluminium ecotoxicology, Aluminium toxicity to fishes, Aluminium uptake.

INTRODUCTION

Aluminium toxicity to fauna and flora has recently been finding much interest. This is because aluminium exposure has been reported to be neurotoxic [1-14]. Bioavailability of aluminium is limited, despite its heavy content in the soil. This is because aluminium in the soil is present as complex alumino silicates, which are quite stable. However, in the event of acid rain and other pH lowering factors, there may be leaching of aluminium to the ground water in the soluble form, resulting in toxicity to the living kingdom. Aluminium toxicity to the fish has been reported earlier [15-21]. Studies on aluminium toxicity in the fish would serve as effective models for studying aluminium neurotoxicity in humans.

With the above view in mind, we have presently studied on the aluminium toxicity to an edible fresh water fish, *Channa punctatus*.

MATERIALS AND METHODS

Channa punctatus fish were procured from the local market and were reared in an aquarium in fresh water under laboratory conditions. The fish were allowed to acclimatize to the aquarium condition for one week. After one week, 10 fish each were placed in three aquarium of similar dimension and capacity. 40 L water was taken in each of the aquarium. Calculated quantity of aluminium sulphate [$Al_2(SO_4)_3.16H_2O$] was weighed out and added to two of the aquarium so that the Al^{3+} concentration in the aquarium became 50 ppm and 25 ppm. These aquariums were labeled as Experimental sets. The aquarium with 50 ppm Al^{3+} concentration was designated as Experimental set no.1 and the one with 25 ppm Al^{3+} concentration was designated as Experimental set No. 2. The third aquarium with ten fish and 40 L water was left as such and designated as Control set. The fish in the aquarium were fed with fish food (procured from the market) at stipulated time during the day. Almost equivalent quantity of food was given to all the

three sets. Water in the aquarium was well aerated throughout. The health as well as mortality (if any) of the fish were noted at a stipulated time, every 24 hours till 50 days or total mortality time, whichever was earlier. Dead fish were immediately removed out from the aquarium. pH of the aquarium water was also noted from time to time. The pH of aluminium exposed water (Experimental sets) was found vary in the range of 6.0 to 6.5 during experimentation.

In the Experimental set (50 ppm Al^{3+}) all the 10 fish died gradually within 96 hours. On the other hand, in the Experimental set No. 2 (25 ppm Al^{3+}), the onset of toxicity seemed to be slow, because the death of fish started only after 30 days. The Al^{3+} solution in this set was changed every 10 days to maintain the concentration (25 ppm). The observation was continued upto 50 days, by which time 50% of the fish died. In the Control set, none of the fish died at 50 days.

At the end, the fish that died last (96 hours) in case of Experimental set no. 1 (50 ppm), the fish died at the 50^{th} day in case of Experimental set no. 2 (25 ppm) and five fish of the Control set (after sacrificing) were chopped into three parts viz, head part, middle part and the tail part. Each part of the fish was weighed out and treated separately with 10 ml of 1M HNO_3 solution in a conical flask and boiled for 15 minutes; where upon the entire tissue got dissolved. The solution was then cooled to room temperature and quantitatively filtered into 100 ml volumetric flask. The solution was made upto the mark (100 ml) with the help of distilled water. Aluminium content of the solution was estimated Spectrophotometrically using Eriochrome Cyanine-R reagent [22]. The content of aluminium in mg/g of the tissue in the different parts of the fish of the Experimental and Control sets were calculated out separately.

The entire work was carried out in three replicates and mean aluminium uptake by the different parts of the fish was calculated out.

Norms of institutional committee for ethics in animal experimentation were strictly followed during the experimentation.

RESULTS AND DISCUSSION

Mortality rate of the fish exposed to Al toxicity (50 ppm and 25 ppm Al^{3+}) is recorded in Table-1. Aluminium uptake by the fish, exposed to 50 ppm and 25 ppm Al^{3+} concentration are recorded in Table-2 and Table-3 respectively.

Aluminium has been found to be toxic to the fish at an exposure concentration of 50 ppm as well as 25 ppm. As seen from the results, exposure to 50 ppm Al^{3+} proved to be acutely toxic. All the fish in this case died within 96 hours. Exposure to 25 ppm Al^{3+}, on the other hand, proved to be chronically toxic. None of the fish in this case died upto 30 days. Mortality in this case, started only after 30 days. As seen from Table-1, 50% animals have died by 50 days even at chronic toxicity level (25 ppm Al^{3+}). It is seen that there is sudden increase of mortality after 40 days of exposure. It seems that a prolonged exposure upto 40 days slowly builds up aluminium concentration on the cell membranes of vital organs of the fish and the animal, physiologically, fights/resists the toxicity during this period. However, beyond this period (40 days) the built up concentration might be becoming too large to be fought against (to be resisted) and the toxicity at this level proves fatal. In case of acute toxicity level (50 ppm exposure), this lethal toxicity level seems to be building up by 24 hours. This is because, in this case, the majority of the fish died between 24 hours and 48 hours period.

A study of total aluminium uptake by the fish that died last (after putting up maximum resistance) seems to be almost identical (4.47 mg/g to 4.80 mg/g) in both (50 ppm and 25 ppm) the exposures. A study of Tables -2 & 3 (50 ppm and 25 ppm) show that the head part including brain upto gills, absorb relatively higher quantity (1.74 mg/g - 1.80 mg/g) of aluminium as compared to the middle (1.51 mg/g -1.63 mg/g) and the tail (1.22 mg/g -1.37 mg/g) parts. The tail part uptaking the lowest quantity. It seems the brain and gill cells have some special affinity for Al^{3+} ions. Accumulation of Al^{3+} in the gills must be intoxicating the respiratory tract, which results in the death of the fish. Aluminium also seems to be producing neurotoxicity, as the fish became mostly inactive much before their death. The total accumulation of Al^{3+} by the fish tissue in our Experiments were found to be 4.47 mg/g (at 50 ppm) and 4.80 mg/g (at 25 ppm) respectively. The fish in Control set showed no presence of aluminium in their tissue. The ability of aluminium to associate with the cell membrane and thus destroy the properties of membrane might be the factor behind the toxicity of aluminium.

TABLE -1 Mortality rate (%) of the fish (n=10) exposed to 50 ppm and 25 ppm Al^{3+} concentration.

50 ppm Al^{3+} Concentration			25 ppm Al^{3+} Concentration		
Time (Hours)	Total Mortality (Number of fish)	Total Mortality (%)	Time (Days)	Total Mortality (Number of fish)	Total Mortality (%)
0-24	3	30	0-10	0	-
24-48	8	80	10-20	0	-
48-72	9	90	20-30	0	-
72-96	10	100	30-40	1	10
			40-50	5	50

n = number of fish

TABLE -2 Mean aluminium uptake by the fish exposed to 50 ppm Al^{3+} (By 96 hours).

Part of Fish	Aluminium uptake (mg/g)	
	Experimental Set	Control Set
Head	1.74	0.0
Middle	1.51	0.0
Tail	1.22	0.0
Total	4.47	0.0

TABLE -3 Mean aluminium uptake by the fish exposed to 25 ppm Al^{3+} (By 50 days)

Part of Fish	Aluminium uptake (mg/g)	
	Experimental Set	Control Set
Head	1.80	0.0
Middle	1.63	0.0
Tail	1.37	0.0
Total	4.80	0.0

CONCLUSION

Our present studies suggest that exposure of *Channa punctatus* (an edible fresh water fish) to aluminium is definitely toxic. The upper part of the fish i.e., brain and gills are the major sites of aluminium accumulation and hence are the main target organs for toxicity in fishes. Since fishes are the part of food chain, the toxicity might be transmitted onward to other animals and humans through the fishes. In view of this, there should be strict monitoring of the aluminium content of water bodies in which the fishes are cultured. Factors responsible for aluminium leaching from the soil such as low pH, as well as, other pathways leading aluminium to natural waters, should be monitored and checked. The fishes cultured in the vicinity of mines and industries of aluminium should be tested for their aluminium content before consumption by the people. Generally consumption of local fishes in such areas should be discouraged.

REFERENCES

[1] Candy J M, Klinowski J, Perry R H, Perry E K, Fairbairn A, Oakley A E, Carpenter T A, Atack J R, Blessed G and Edwardson J A, *Lancet*, **1986**; 327: 345-356.
[2] Sigel H and Sigel A, Aluminium and its Role in Biology In : Metal Ions in Biological Systems, New York: *Marcel Dekker*, **1988**; 24: 424.
[3] Exley C, Chappell J S and Birchall J D, *J.Theor.Biol*. **1991**; 157: 417-428.
[4] Exley C and Birchall J D, *J.Theor. Biol*., **1992**; 159: 83-98.
[5] Zatta P, *Trace Elem.Med*., **1993;** 10**:** 120.
[6] Alfrey A C, Garg R, Legendra G R and Kochhv W D, *N.Engl. J.Med*., **1994**; 294: 184.
[7] Alfrey A C, *Life Chem. Rep*. (S), **1994**; 11**:** 197.
[8] Harrington C R, Wischik C M, McArthur F K, Taylor G A, Edwardson J A and Candy J M, *Lancet*, **1994**; 343**:** 993.
[9] Zatta P, *Med. Hypoth*., **1995**;44: 169.
[10] Nayak P and Chaterjee A K, *J.Toxicol. Sci*., **1998** ; 23**:** 1-14.
[11] Nayak P and Chaterjee A K, *J.Environ. Biol*., **1999**; 20**:** 77-84.
[12] Nayak P and Chaterjee A K, *Food Chem. Toxical*., **2001** ;39: 587-592.
[13] Nayak P *Environ, Res. Sec. A*, **2002**; 89: 111-115.
[14] Nayak P and Chaterjee A K, *BMC Neuro. Sci*., **2002**; 3**:** 12.
[15] Howells G, Dalziel T R K, Reader J P and Solbe J F, *Chem. Ecol*., **1990**; 4**:** 117-173.
[16] Spry D J and Wiener J G, *Environ. Pollut*., **1991**; 71**:** 243-304.
[17] Karatas M, Seker Y and Sezer M, *Asian J.Chem*., **2007**; 19: 574-578.
[18] Karatas M and Seker Y, *Asian J.Chem*., **2008** ; 20: 3310-3312.

[19] Karatas M, *Asian J. Chem*., **2008**; 20: 5741-5744.
[20] Exley C and Struthers W, *Aquaculture*, **1992**; 100: 323-324.
[21] Rao T V R K, Yadav Archana and Mishra Kumari Kanchan, *Asian J. Chem.,* **2013**; 25: 3497-3498.
[22] Vogel A I, A Text Book of Quantitative Inorganic Analysis, E L B S and Longman, London, **1978**; edn. 4: p 729.