

## Effect of Aluminum Toxicity on Plasma Electrolytes of a Fresh Water Teleost Fish, *Cyprinus Carpio Var. Communis*



### Biotechnology

**KEYWORDS:** Cyprinus, Aluminium sulphate, Plasma electrolytes.

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### ABSTRACT

The present paper deals with the changes in plasma electrolytes levels ( $Na^+$ ,  $K^+$  &  $Cl^-$ ) of fish *Cyprinus Carpio* when exposed to acute and sublethal concentrations of Aluminium sulphate. In acute treatments, sodium and potassium level decreased significantly during sublethal treatment, sodium and potassium level was gradually increased and chloride level was decreased. The probable reasons governing the facts are interpreted and discussed in the light of earlier literature.

### INTRODUCTION

The industrial effluents contaminating aquatic bodies contain a number of toxic metals, which in turn exercise their effects on fish and pose threat to aquatic life because of their persistence [sen *et al.*, 1992]. Aluminium has come to be regarded as the major factor in the loss of fishers in soft acid waters [cumminis, 1994].

The effect of a pollutant on a target organism may be either acute or chronic; acute effects occur rapidly are clearly defined, often fatal and rarely reversible; chronic effects develop after long exposure to low doses or long after exposure and may ultimately cause death [Saunders, 1976]. The present study was conducted to ascertain the impact of Aluminium sulphate toxicity on the plasma electrolyte of fresh water teleost fish, *Cyprinus Carpio var communis* during acute and sublethal exposure periods.

### MATERIALS AND METHODOLOGY

Fish were acclimatized to laboratory conditions for about 15 days. Before the commencement of the experiment. During this period, Fish were fed, *ad libitum* with rice bran and groundnut oil cake once in a day. After feeding, water was changed daily in order to maintain clear environment and [to avoid any accumulated metabolic waste] and aerated to ensure sufficient, oxygen supply.

The tap water was analyzed for physico-chemical features as per APHA [1971]. The healthy fish with an average weight of 5.0g and length of about 7.5 cm were selected for the experimental purpose. The medial lethal concentration [LC<sub>50</sub>] of Aluminium Sulphate was determined for 24<sup>th</sup> by probit analysis method of Finney [1978].

Fish were exposed to acute concentration for a period of 24 h and sublethal concentration for a period of 28 days. A common control was maintained. After 24 h fish from acute treatment, and at an interval of 7 days, the fish from sublethal and control group were sacrificed for plasma electrolyte estimation. Sodium was estimated by Trinder [1955] and Maruna [1958] method. Potassium was estimated according to Sunderman and Sunderman [1959] and Tietz [1970]. Chloride was estimated by Schoenfeld and Lawellens [1964] method. The data was subjected to statistical analysis.

### RESULTS

During the study period, the physico-chemical features were maintained at constant level. The parameters of the water used for the experiment are pH 7.2 ± 0.1; temperature 25.0 ± 2.0<sup>o</sup> C; dissolved oxygen 6.2 ± 0.01 ppt; total hardness 18 ± 0.5 mg/l; alkalinity 17.5 [170 – 180] mg/l. The LC50 of Aluminium Sulphate determined by probit analysis was 32.96 ppm. For this present study 1/10 of the 24 h LC50 value [3.29 ppm] was taken. Changes in the plasma electrolyte levels of *Cyprinus Carpio* to acute and sublethal concentration of Aluminium Sulphate are presented in Table 1. During acute treatment, plasma sodium and chloride level increased at the rate of 26.70 and 81.12 % respectively. On the other hand, plasma potassium level decreased by 16.34 % at the end of 24 h period.

During sublethal treatment, plasma sodium level slightly increased by throughout the experiment period and it was directly proportional to the exposure period reaching a minimum increase of 6.52 at the end of 14<sup>th</sup> day and 17.22 at the end of 28<sup>th</sup> day, respectively. In sublethal treated fish, plasma potassium level was gradually increased as the exposure period was extended showing a percent increase of 6.27, 12.84 and 15.69 at the end of the 7<sup>th</sup>, 14<sup>th</sup> and 21<sup>th</sup> day respectively. After 21<sup>st</sup> day, potassium level recovered showing an increase level of 35.53% at the end of 28<sup>th</sup> day. In sublethal treatment, a maximum elevation of 72.15 % of plasma chloride level noticed at the end of 7<sup>th</sup> day followed by [14.76%]. After 21<sup>st</sup> day, plasma chloride level was declined. Showing a 54.40 % at the end of 28<sup>th</sup> day.

Statistical analysis indicated that all the values were significant at 5 % level.

### DISCUSSION

Fish are particularly sensitive to environmental contamination of water and pollutants may significantly damage certain physiological and biochemical processes when they enter the organs of fishes [Nemcsok *et al.*, 1987]. Toxicity of metals to fish has been reviewed by many workers [Lorsson *et al.*, 1976; Fu *et al.*, 1989; Ramesh, 1994; Shekar and Christy, 1996].

The regulation of cations like  $Na^+$ ,  $K^+$ ,  $Ca^{2+}$  and  $Mg^{3+}$  are disrupted in aquatic animals under toxic conditions [Bruin, 1976]. Alterations in the ionic balance of aquatic organisms can be due to stressor effects on the ion regulatory organs [Spront *et al.*, 1971; Gilles and Requeue, 1983] or internal and external sensory receptors involved with detection of changes in osmotic conditions [Inman and Lockwood, 1977; Rosseland and Staurnes, 1944] or on the endocrine system [Mazeaud *et al.*, 1977; Harman *et al.*, 1980] or on the metabolism [Greenway, 1979].

A significant increase in plasma osmolarity,  $Cl^-$  and  $Na^+$  levels in *Salmo salar* exposed to ammonia toxicity was observed by [Knop and Thorud, 1996]. Significant increase in plasma electrolytes was observed in brook trout *Salvelinus fontinalis* on exposure to methyl mercuric chloride, cadmium chloride and lead nitrate [Christensen *et al.*, 1977] and in Indian cat fish *Heteropneustes fossilis* exposure to copper sulphate.

The increase in plasma electrolyte levels *Aphanius dispar* exposed to mercury may be due to disruption of liver metabolism or damaged intestinal mucosa causing free exchange of ions between gut content and submucosal capillary beds [Hilmy *et al.*, 1982]. The increase of electrolytes may be due to dysfunction of osmoregulatory mechanism [Grant and Mehrle, 1973]. In the present study, elevation of sodium and chloride during acute and sublethal treatment and potassium during sublethal treatment might be due to loss of water from circulation or damaged intestinal mucosa causing free exchange of ions or osmoregulatory dysfunction recalling the observations of the authors cited.

A significant decrease in plasma potassium level was noted in pin fish *Lagodon rhomboids* exposed to chemical contaminants [Folmar *et al.*, 1993]. A significant decrease in plasma potassium in rainbow trout, *Oreochromis mykiss* when exposed to

lead and brook trout, *Salmo trutta* to copper [Mckim *et al.*, 1970; Choistensen *et al.*, 1972].

Sivaprasad Rao *et al.*, [1983] reported significant decrease in plasma electrolyte levels in *Tilapia mossambica*. The depression in cation conservation may be due to reduced influx (or) greater efflux through the tissue membrane [Nimmo and Blackman, 1972] or impairment of membrane permeability [Sivaprasad Rao *et al.*, 1983; Subba Rao, 1987; Ramesh, 1994]. In the present study also, the significant decrease in plasma sodium, potassium may be due to adrenaline stress response of fish or inhibition of ATPase activity as suggested by above authors.

**TABLE 1: Changes in the plasma electrolytes [Na<sup>+</sup>, K<sup>+</sup> & Cl<sup>-</sup>] levels of fish *Cyprinus carpio* var. *communis* exposed to acute and sublethal concentration of Aluminium Sulphate.**

EXPOSURE PERIOD	PLASMA SODIUM		PLASMA POTASSIUM		PLASMA CHLORIDE	
	Con.	Exp.	Con.	Exp.	Con.	Exp.
Acute Treatment [24 Hrs]	94.160 ± 1.349	119.300 ± 1.366* [+26.70]	5.546 ± 0.153	4.640 ± 0.139* [-16.34]	95.952 ± 1.034	173.793 ± 1.258* [+81.12]
DAYS	SUBLETHAL TREATMENT					
7	94.530 ± 0.173	75.480 ± 0.185* [-20.15]	6.094 ± 0.333	6.476 ± 0.572* [+6.27]	85.680 ± 0.206	147.500 ± 0.435* [+72.15]
14	92.498 ± 1.889	98.530 ± 0.159* [+6.52]	6.159 ± 0.335	6.950 ± 0.621* [+12.84]	89.582 ± 0.222	102.800 ± 0.356* [+14.76]

21	90.498 ± 0.162	103.590 ± 0.138* [+14.47]	5.423 ± 0.341	8.660 ± 0.513* [+59.69]	98.699 ± 0.230	101.930 ± 0.353* [+3.27]
28	98.684 ± 0.100	115.680 ± 0.986* [+17.22]	5.368 ± 0.329	7.275 ± 0.017* [+35.53]	94.296 ± 0.327	43.000 ± 0.377* [-54.40]

Values are mean ± SE of five individual observation. Values in parentheses are percent change over control.

- Denotes percent decrease over control. + Denotes percent increase over control. Degrees of freedom at 8t 0.05 = 2.306.

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