EFFECT OF COBALT CHLORIDE ON THE OXYGEN CONSUMPTION AND VENTILATION RATE OF A FRESHWATER FISH, CIRRHINUS MRIGALA (HAM)

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Abstract

The fish Cirrhinus mrigala (Ham.) exposed to lethal and sublethal concentrations of cobalt chloride at selected periods showed a decrease in their ventilation rate up to 27.91% in lethal concentration at 240hr of exposure, while, in sublethal concentrations initially increased up to 23.95 & 27.91% at 96 and 240hr of exposure followed by a decline up to 24.70 and 12.94% at 960hr of exposure to 39.45 and 13.10 mg/l concentration respectively. The O_2 uptake rate initially increased followed by a decline up to 54.47% at 240hr of exposure to lethal concentration (92.00 mg/l) & up to 28.80 & 10.65% in sublethal concentration at 960hr of exposure.

Keywords: *O_2 uptake; ventilation rate: Cirrhinus mrigala; Cobalt chloride*

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1. INTRODUCTION

Oxygen consumption rate in fish has been considered as an index for denoting the intensity of metabolism (fry, 1957, 1971). The metabolic activity of an organism is increased by its oxygen utilization and so it becomes possible to determine "no stress" effect of any toxicant on the organism if we know oxygen-utilization rate of that particular organism. Changes in the respiratory behavior and metabolic rate of pollutant induced fish have drawn the attention of several biologists, (Singh & Singh, 1979; Roy & Munshi, 1988; Sastry & Shukla, 1990; and Kumar, 1999), but their results are conflicting as some have reported increased gill ventilation and decreased O_2-uptake rate (Singh & Singh, 1979 and Roy & Munshi, 1988) while others have reported an increase in O_2-uptake rate (Anderson *et. al.*, 1974 and Natrajan & Rajulu, 1983). As far cobalt is concerned, Vitamin B12 (Cobalbamin) contains 4% cobalt and as little as 3μg/day controls pernicious anemia in man. Its toxicity is low but large amount may cause polycythemia. On the whole, cobalt is a potential metal pollutant but its effect on aquatic organisms especially fish is not yet fully known.

Hence, the present study, was conducted with an objective to evaluate the effect of different concentrations of cobalt chloride on ventilation rate & oxygen consumption of a major carp, Cirrhinus mrigala (Ham.) at selected periods.

2. MATERIALS AND METHODS

Healthy and living specimens of Cirrhinus mrigala(Ham.) of 24.6±3.2gm weight groups were procured from local fish-farm and brought to the laboratory in large buckets containing water of the same pond. The fish were bathed for 10-15 minutes in potassium permanganate solution (0.1%) followed by several changes of ground water and then transferred to large aquaria / tubs containing ground water. They were acclimatized in the laboratory for 10-15 days. During this period and also during experiments, they were provided artificial food prepared in the laboratory following the method of Suraj (1995). The food was provided daily at least three hours prior to change of water / toxicant solution. The aquaria / Tub were provided aerator to maintain oxygen level of the water.

Stock solution of cobalt chloride (BDH reagent grade) were prepared by diluting a known quantity of chemical in 500ml distilled water as described in "Standard Method" APHA (1985). One or two drops of HCl was mixed in the water containing the metallic salt to prevent the formation of precipitate. Same amount of HCL was also mixed in water used for control experiment.

Lc50 value for 24, 48, 72 & 96hr was determined as described by Dandoroff et. al.(1951) and APHA (1985), which were recorded to be 293.22, 219.96, 172.48 and 131.51 mg/l CoCl2. Therefore, to study the effect of lethal and sublethal concentrations, 0.7th, 0.3rd & 0.1st of 96hr Lc50 value were taken, which were 92.00, 39.45 and 13.10 mg/l respectively.

Sufficient number of fish was exposed to each selected concentrations along with control. The oxygen consumption rate of five fish in normal and toxicant induced concentrations was measured in a continuous flow glass respirometer at 8, 96, 240, 480 and 960 hr of exposures. The ventilation rate of the fish were counted by visual observation of the opercular movements with the help of magnifying glass for 5 minutes at the time of O2 consumption reading and average value per fish per minute was calculated. For measuring the Oxygen consumption rate of fish, Winkler's iodemetric method (APHA, 1985) was followed to measure dissolved oxygen content in water.

3. RESULTS

Exposure to cobalt chloride water excites the fish and it started a frantic effort to come out of container and became aggressive for first few hours followed by inactiveness for some time, perhaps due to hyper-activity, difficulties in respiration as indicated by frequent surfacing to engulp air, loss of equilibrium, uncoordinated movements of the body musculature and titanic extension of jaw followed by turned over and laid floating either on the surface or on the bottom of the water with highly decreased opercular frequency depended on concentration and exposure period and abnormal secretion of mucus all over the body surface at the time of death.

The result of the experiments set up i.e. ventilation rate /minute and oxygen consumption rate (VO2:mlO2/hr and mlO2/Kg/hr/fish) are tabulated in Table -1, which contains the average value of five fish exposed to 92.00, 39.45 & 13.10 mg/l cobalt chloride along with control for 8, 24, 96, 240, 480 and 960hr of exposures.

The fish exposed to lethal concentration (92.00 mg/l) showed a gradual decrease in ventilation rate as the decline was significantly decreased at 24 hr onward with maximum decline ($P<0.01$) up to 27.1% at 240hr of exposure, whereas, the fish exposed to sublethal concentrations (39.45 & 13.10 mg/l) a significant increase was observed at 96 & 240hr of exposure respectively, followed by a decline ($P<0.01$ & <0.05) up to 27.40 & 12.94% respectively at 960hr of exposure when compared with that of their normal values.

The fish exposed to 92.00 mg/l cobalt chloride, initially showed an increased oxygen consumption rate up to 12.65% at 8hr of exposure, followed by a gradual decrease which was found statistically significant ($P<0.05$) at 96hr of exposure with a maximum decline up to 54.47% at 240hr of exposure, whereas, in sublethal concentrations an initial increase up to 96 & 240hr of exposure followed by a decline from 480hr onwards. However, the decline (28.80%) was found statistically significant ($P<0.05$) at 960hr of exposure to 39.45 mg/l concentrations only when compared with that of their normal values.

4. DISCUSSION

Increase in opercular beat & restlessness in toxic environment is characteristic of the fish exposed to hypoxic conditions (Randall & Shelton, 1963; Shidmore, 1970). Jones (1938) observed a decrease on oxygen uptake and correlated with increase in opercular beat in Gasterosteus aculatus exposed to lethal concentrations of few metallic salts and stated that it was mainly due to reduced efficiency of the gills. Singh & Singh (1979) in Mystus vittatus exposed to Zinc and copper, Kumari (1990) in H. fossilis exposed to Zinc and Suraj (1998) in Anabas testudineus exposed to cadmium & cobalt observed an increase in opercular frequency during initial hour of exposures followed by a decrease, more in lethal than sublethal concentrations depended on exposure hours.

In the present study, there is decrease in ventilation rate at higher concentration, but an increase at lower concentrations initially followed by a decrease in later stages of intoxication. The increase in ventilation rate is probably due to an effort to extract more oxygen to fulfill its energy demands to fight the stressful conditions, while decrease in ventilation rate may be due to titanic effect on opercular and branchial muscle caused by cobalt and / or might be due to gill injury or due to disturbances in gas diffusion pathway by abnormal secretion of mucus all over the body surface including gills.

Several workers have reported that metallic salts decreased the oxygen consumption rate of the fish with an increase in concentration and exposure period (David & Ray, 1966; Calabrese et. al., 1975; Sastry & Shukla, 1990 and Suraj, 1998) and suggested that such metallic salt causes asphyxiation in fish resulting in reduced O2 uptake rate. Crandall and Goodnight (1963) have suggested that prolonged exposure of fish to low concentration of heavy metals subjects them to stress which causes hormonal imbalance ultimately leading to a variety of internal pathological changes.

In the present study, an initial increase in oxygen uptake rate up to 8 hours in lethal and 240 hours of exposure in sublethal concentrations followed by a gradual and significant decrease ($P<0.05$) at 96hr with maximum of 54.47 decrease at 240 hr of exposure in lethal and 28.80 & 10.65% decrease at 960 hour of exposure in sublethal concentrations observed, indicates that the initial increase on O2-uptake rate might be due to over activeness of the fish or due to some internal factors (Crandall & Goodnight, 1969) and the subsequent decrease may be due to injury of the gills and other vital organs and / or also might be due to abnormal secretion of mucus all over the body and gills, obstructing gas diffusion pathway.

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Table -1: Ventilation rate /minute and oxygen consumption (VO2: MlO2/hr and MlO2/Kg/hr/fish in Cirrhinus mrigala (Ham.) in relation to lethal and sublethal concentrations of cobalt chloride exposed for different periods at 29.5±2.70C water temperature

Concentration (mg/l)	Exposure Period (hr)	Ventilation rate Per Minute		Oxygen Consumption rate (VO2)		
		Value	% Change	MlO2/hr/Fish	MlO2/Kg/hr/fish	% Change
Control	8	89±2		1.88±0.13	76.50±5.34	
92.00		83±3	-6.74	2.12±0.13	86.17±5.08	12.65
39.45		96±2	7.86	1.96±0.13	79.84±4.94	4.36
13.10		94±3	5.62	1.85±0.12	75.20±4.65	-1.70
Control	24	87±1		2.05±0.12	83.17±5.20	
92.00		79±2*	-9.19	1.68±0.13	68.25±4.66	-17.94
39.45		104±3**	19.54	2.12±0.12	86.18±4.91	3.62
13.10		94±2*	8.04	2.16±0.11	87.97±5.23	5.77
Control	96	89±2		1.97±0.12	79.88±4.85	
92.00		70±2**	-21.35	1.21±0.11*	49.27±4.54	-38.32
39.45		110±2**	23.59	2.16±0.13	87.84±5.23	9.27
13.10		113±3**	26.97	2.02±0.13	82.03±5.06	2.70
Control	240	86±3		1.90±0.12	77.24±4.69	
92.00		62±3**	-27.91	0.87±0.12*	35.16±4.90	-54.47
39.45		99±2*	15.12	1.98±0.13	80.49±5.32	4.21
13.10		110±2**	27.91	2.20±0.13	89.51±5.14	5.89
Control	480	88±2		1.95±0.13	79.06±5.33	
92.00		---	---	---	---	----
39.45		79±2*	-10.23	1.76±0.12	71.50±4.70	-9.56
13.10		82±3	-6.82	1.99±0.13	81.29±5.02	2.52
Control	960	85±3		1.90±0.13	77.07±5.10	
92.00		---	---	---	---	----
39.45		64±3**	-24.70	1.35±0.11*	54.88±4.63	-28.80
13.10		74±2*	-12.94	1.69±0.14	68.85±5.25	-10.65

± is standard error of 5 observations "*" = P<0.05 "" =P<0.01**