

Research Article

IMPACT OF CADMIUM CHLORIDE'S TOXICITY ON OXYGEN CONSUMPTION IN AN AIR BREATHING TELEOST *HETEROPNEUSTES FOSSILIS* (BLOCH)

S. N. Mishra

Department of Zoology Maharani Kalyani College, Laheriasarai, Darbhanga, India

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ABSTRACT

Metabolic demand of oxygen maintained by the utilization of dissolved oxygen from aquatic medium by respiratory organs in piscian fauna. *Heteropneustes fossilis* is an air breathing teleost are able to use dissolved oxygen in water through gills and atmospheric oxygen through its accessory respiratory organs known as suprabranchial chamber and air sacs. Contamination of heavy metal, cadmium chloride through industrial discharge in water body, produce histological alteration and toxicological impact on gills and accessory respiratory organs which decrease the efficiency of oxygen consumption and leads serious threat for survival of fish. By acquiring the effect of toxic pollutant in aquatic medium, its discharge into nearby water resources may be regulated to protect aquatic life and its effect on the human consumption.

Keywords: Accessory Respiratory Organs, Oxygen consumption, Heavy metal, Cadmium chloride.

INTRODUCTION

Heteropneustes fossilis(Bloch) are air breathing fish having accessory respiratory organ as suprabranchial chamber and air sacs. Wall of the supra branchial chamber is formed by the fused gill lamellae of the gill arches. The gill lamellae born by their respective hemibranchs fuse to form the fans. The curious disposition of the 3rd and 4th fan in the suprabranchial cavity causes it to be divided into an anterior smaller and posterior larger chamber. The air sacs which extend on either side of the body right upto the middle of the tail region and provide: the main air-breathing organ of the fish, is the product of gill mass and as such can be looked upon as the modified 5th gill.

The gill mass remains present at the posterior extremity of the air sacs even in the adult condition and adds to its length. The pillar cells of the ARO differing from those of the gills and retaining certain embryonic features is due to the facts that they are not derived from the gill lamellae once formed at the gills but from the gill mass which persist in the embryonic form at the summit of the air sacs even in the adult condition. In the present work the effect of toxicity on gills and ARO has been

investigated by using common heavy metal cadmium chloride. Since, the gills are initially associated with the toxicant which distributed in other vital organs of body via blood circulation including in supra branchial chamber and air sacs. Keeping this in mind, the gills have widely been used as bio-indicator not only to detect lead and cadmium toxicity (Parashar and Banerjee2002) but also for analysis of several other pollutants (Munshi and Singh, 1992, Chandra and Banerjee, 2004). Many of the fishes in Indian subcontinent have developed a bimodal respiratory of toxic pollutant in aquatic medium, its discharge into water body and aquatic resources may be regulated to protect aquatic life. Several reports are available on the toxicity of various insecticides to fishes (Eisler and Edmonds, 1971, Joshi, 2002, Singh *et al.*, 1990; Banerjee, 2004). Further, it is also well known from the works of Jones (1964), Verma *et al* (1993). The susceptibility of fish to pesticides is also frequently influenced by pH and /or temperature of water several reports have also appeared in recent years suggesting that various concentrations of different heavy metals may cause significant changes in metabolic process of the fish. Metabolic activity of an organism is increased by its oxygen utilization. Various environmental factors

*Corresponding Author: Dr. S. N. Mishra, Department of Zoology Maharani Kalyani College, Laheriasarai, Darbhanga, India Email: snmishra6@gmail.com.

and stresses alter metabolic rate of animals which would be indicated by oxygen utilization rate. Fry (1957) has considered the rate of oxygen consumption as an index to denote the intensity of metabolism. A change in respiratory parameter has been taken up as an index for the harmful effect of pollutant (Sastry *et al.*, 1982). Several biologists have measured the oxygen uptake rate in water breathing fishes taking into account the different variables such as temperature, body size, different stages of life cycle, respiratory surface area, nutrition and other factors causing changes in energy requirement and consequently the oxygen consumption by the fish (Munshi and Singh 1992). In air breathing fishes, however the gas exchange is bimodal with water via buccopharynx, gill, and skin and with atmospheric air through accessory respiratory organs, but the relative role of gill and air breathing organ in respiration varies from species to species. Hence any change in normal respiratory epithelium would ultimately affect the rate of oxygen uptake. Therefore direct contact with respiratory surface area with the polluted water lead to alteration in the normal respiration as well as diffusion capacity of gills and accessory respiratory organs (Brown *et al.*, 1986; Munshi and Singh, 1992, Murphy *et al.*, 1987;). Recently changes in respiratory behaviour and metabolic rate of heavy metals induced fishes have drawn the attention of several biologists (Wilson *et al.*, 1993) ; Haider and Indraj (1986) have stated that the changes produced by various pollutants look alike superficially their harmful effect on different genera of fish is not of the same magnitude. Further it is also well known from the work of Munshi and Singh (1968) that mucous secretion is known to have an important function in the maintenance of respiratory activity of the gills in fishes. Thus, the available investigation related with the effect of heavy metals on the respiratory biology of fishes with special reference to histopathological changes in the accessory respiratory organ .Oxygen Consumption and biochemical changes are insufficient to establish the exact toxicological effects of heavy metals on suprabranchial chamber in *H. fossilis*. Larsson *et al.*, (1981) investigated the histopathological changes due to heavy metals in association with disturbance in osmotic and ionic balance. They also elucidated the chronic exposure to sublethal concentrations of cadmium resulting the damage of organs of respiration and excretion like kidney through impaired metabolism of ions like sodium, potassium, calcium, magnesium, chloride and inorganic phosphate in the flounder, *Platichthys flesus*. Pool (1981) proposed that like other heavy metals, Cd also possesses strong affinity for sulphur, dithiol and other S-groups in the biological materials Under laboratory conditions, hyperglycaemia is a typical symptom of cadmium poisoning, as reported in an Indian fresh water cat fish *H. fossilis* Sastry and Subhadra, (1982). Histological alteration in liver and intestine of *Sartherodon mossambicus* in response to Hg toxicity was depicted by Naidu *et al.*, (1983). They recorded engorged blood sinusoid, vacuolation, rupture, granular degeneration of hepatocytes, edema focal necrosis and proliferation in

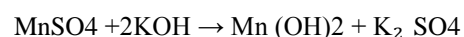
fibroblast in liver. Jhingaran (1985) narrated air breathing fish, *Clarias batrachus* to be more refractive to cadmium toxicity than the non-air breathing ones, viz., *Labeo rohita*. Dubale and shah (1979) exposed the *Channa punctatus* to different concentration of cadmium nitrate (0.01 to 0.05 ppm) and observed hepatopathic effects such as necrosis of hepatocytes. The reason behind the observation of Dubale and Shah (1979) were envisaged that these might happen due to the effect of cadmium on sulphur.

MATERIALS AND METHODS

Dissolved oxygen: Assessment of dissolved oxygen concentration of an aquatic medium is important because it reflects the physical and biological nature prevailing in water body. Low amount of oxygen is always harmful for the fishes. Oxidizable substances and inorganic reductant have a tendency for decrease the dissolved oxygen in water .Therefore, for the sustenance of life, especially in higher forms; in polluted water aeration is allowed preferably Winkler's iodometric method was applied for the estimation of dissolved oxygen. Winkler's Iodometric Method:

Principle

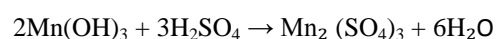
The magnous sulphate reacts with the alkali, potassium or sodium hydroxide, to form a white precipitate of magnous hydroxide. This may be represented by chemical equation.



In the presence of oxygen, the magnous hydroxide get oxidized to a brown colour compound, the magnic hydroxide .This may be depicted by impirical formula:



In a non oxidising strong acid medium, the manganic ions are reduced by iodide ions and the latter gets converted to iodine equivalent to the original concentration of oxygen in sample Chemical reaction underlying as follows:



$2\text{Mn}_2(\text{SO}_4)_3 + 4\text{KI} \rightarrow 4\text{MnSO}_4 + 2\text{K}_2\text{SO}_4 + 2\text{I}_2$ this iodine is titrated against sodium thiosulphate with starch as an indicator. This involves following chemical reactions:



Reagents

Magnous sulphate solution 4.8 % $\text{MnSO}_4 \cdot 4\text{H}_2\text{O}$

Alkaline Iodide solution: 700 g of KOH and 150g of KI dissolved in distilled water and made up to one liter.

Sulphuric acid concentrated H_2SO_4 , Spgr 1.84,

Sodium thiosulphate: 6.025 g. pure crystalline $\text{Na}_2\text{S}_2\text{O}_3$, was dissolved in freshly boiled distilled water, the volume was made up to 1 litre in order to obtain a 0.025 N solution. 5 ml pure chloroform was added to this solution and stored

in amber coloured bottle. Starch solution 5% solution of potato starch in warm (80°C to 90°C) distilled water was prepared and kept in refrigerator.

Procedure

The stopper of a 250 ml capacity sample bottle containing the sample water was added with 1 ml MnSO₄, by a long graduated volumetric pipette. In the same way 1 ml of KOH. KI Solution was added using a different pipette. The stopper was replaced and the solution was mixed thoroughly by inverting the bottle several times. The dissolved oxygen was then fixed. The precipitate was allowed to settle for a few minutes. After final settling of the precipitate, a clear brown precipitate appeared. In this solution, 2 ml. concentrated H₂SO₄ was introduced and mixed well. The precipitate disappeared and golden yellow colour solution was obtained. This sample was allowed to stand for 10 minutes. 200 ml sample was removed in a conical flask and titrated rapidly against 0.025 N solution of sodium thiosulphate from the burette until the iodine colour of the sample was reduced to a pale yellow-straw colour. A few drops of starch solution was added and the titration continued rapidly until the blue colour just disappeared. This was taken as an end point.

Calculation

As the 200 ml of sample was titrated, the quantity of ml of sodium thiosulphate solution used in the titration is exactly numerically equal to DO in mg/l.

The DO in test water was 7.8 mg/l

Free CO₂ = 2.1 mg/L¹

Total Hardness as CaCO₃, = 145.5 mg/L¹

Total Alkalinity as HCO₃ = 130 mg/L¹

Experimental design

Healthy specimen of *H. fossilis* of either sex belonging to a single population (Body length 12-18 cm and weight (25-

35 gm) were collected from the local fisherman. The selected healthy fish were acclimatized in laboratory aquaria for one month. The fish were fed with tubifex worm, sliced goat liver and commercial fish food (fishbone). No mortality was recorded among the fish during this period. The experimental fish were pretreated with 1% methylene-blue for 15 minutes to avoid disinfection. Dechlorinated tap water was used as test water. 6 test containers (glass battery jar) used as a test container in which the first container contained no heavy metal and acted as control. To observe, five different concentrations were used as 20ppm, 40ppm, 60 ppm, 100 ppm and 200 ppm of cadmium chloride up to different exposure time as 24 hours, 48 hours, 72 hours, 96 hours and 120 hours.

RESULTS AND DISCUSSION

Oxygen consumption in fish *H. fossilis* related with the respiratory organs, gills and accessory respiratory organ which enable them to use dissolved and ambient oxygen for their metabolic needs. When the fish is exposed to various concentrations of cadmium chloride (20,40,60,100,200 ppm for 24hr,48hr,72hr,96hr,120hr), gradual decline in the percentage rate of oxygen uptake was recorded (Table A). It was recorded that the percentage rate of oxygen consumption was 95% at 20 ppm concentration after 24 hrs exposure where it was 91% after 120 hrs. More prominent decline in percentage of oxygen uptake was recorded at 200 ppm after 120 hrs exposure which was 58%. The decrease in dissolved oxygen uptake through respiratory organ, gills, in a particular concentration was not of the same magnitude but different according to the metabolic activity of the fish. In the present experiment the formation of thick mucous film over the gill surface is due to the toxic effect of heavy metal, cadmium chloride resulting in intense decline in oxygen consumption which leads to ultimate cause of asphyxiation. Such explanation is in conformity with that of the other authors such as Aronson 1971, Skidmore & Tovell 1972, Nagbhusanam and Kulkarni, 1981.

Table 1. Percentage utilization of Oxygen dissolved in test water of experimental fish *Heteropneustes fossilis* treated with various concentrations of cadmium chloride up to different exposure period of experiment. (Control: 98%).

Concentration (in ppm) of cadmium chloride	Exposure period of cadmium chloride treated fish					
	24hr	48hr	72hr	96hr	120hr	
20	95	93	92	91	91	
40	93	92	89	86	85	
60	90	88	82	76	71	
100	86	85	71	68	65	
200	80	76	64	60	58	

In fish, gills are primary site of toxic action of many waterborne pollutants (Mallatt, 1985), with the gill lamellae serving as interface for ion and gas exchange between blood and the external medium (Munshi, 1962). So it faces the contact stress of the environmental hazards. Characteristic changes have been reported in the gills epithelium of different fishes after exposure to various concentrations of many heavy metals (Kim, 1979), low pH and oxygen uptake (Munshi & Singh, 1992) pesticides (Munshi & Singh, 1971). Shrinkage of the respiratory surface area and enlargement of the water/blood barrier of the gill in sublethal concentration of cadmium salt has also been reported by Randi *et al.*, (1996). Decrease oxygen uptake and increased opercular movement in case of *Punctius sophore* is also supported from the finding of Jones, (1964) who also correlated increased opercular movement with decreased oxygen uptake in stockle back, *Gasterosteus aculeatus* exposed to various concentrations of Pb, Cu and Hg. He assumed that decreased oxygen consumption was mainly due to reduced efficiency of the gills death occurred because of suffocation and coagulated mucous film spreading over the gills.

In the present study the percentage oxygen uptake rate in *H.fossilis* exposed to various concentrations of cadmium chloride showed gradual decline from 24 hrs to 120 hrs exposures. Low concentration showed slight decline in oxygen consumption whereas at high concentration (200 ppm cadmium chloride), the fish showed remarkable decrease in the percentage oxygen consumption rate according to increased exposure periods. Increased duration of cadmium exposure is not only result as damage of gill epithelium and accessory respiratory organ but produce its effect on the whole physiology of fish including low oxygen uptake (Playle *et al.*, (1993). Since the heavy metal, cadmium chloride toxicity in present study, normally cause respiratory distress, the decrease in oxygen consumption in the cadmium exposed *Heteropneustes fossilis* observed in this investigation may be due to absorbance of more heavy metal through the respiratory surface (gills and suprabranchial chamber) result in the deformity in the secondary lamellae and associated decline in the oxygen uptake capacity of respiratory organs.

CONCLUSION

After exposure to the different concentrations of Cadmium Chloride, the respiratory surface of gills and suprabranchial chamber were adversely affected by this heavy metal which ultimately hampered the efficiency of respiratory organ of the fish that minimize the oxygen consumption. The result obtained through this investigation has revealed that the Cadmium which was released into water bodies from various industries is toxic to the fish. The toxicity of the heavy metal not only affect the fish itself but also exert negative impact on the health of those people who consume the fish from the stock of polluted water. Future Aspects: To maintain healthy stock of fish, it is necessary to treat the industrial effluents containing heavy metals before draining them out into the water system to free the fish for human consumption from heavy metal contamination.

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REFERENCES

- Banerjee A.K., et al., (1978). Toxicity of cadmium, a comparative theory in the air breathing fish *C. batrachus* and in the *T. mossambicus*. *Journal of Experimental Biology*. 16, 1274-1277.
- Banerjee T.K.,R. Devi, (2007). Toxicological impact of sublethal concentration of lead nitrate on aerial respiratory organ of murrel *Channa striatus*(Bloch),070-38. *Veterinary Archives*, 74, 37-52.
- Dubale M.S, P.Shah, (1979). Toxic effect of cadmium nitrate on the liver of *Channa punctatus*. *Experimentia*, 35,643-644. <https://doi.org/10.1007/BF01960373>.
- Eisler R. (1971). Cadmium poisoning in *Fundulus heteroclitus* (Pisces :Cyprinodontidae) and other marine organisms *Journal of the Fisheries Research Board of Canada*.28,1225-1234. <https://doi.org/10.1139/f71-188>
- Fry F.E.J. (1957). The aquatic respiration of fish, Physiology of fish,ed. ME. Brown Vol 1, Academic press New York. <https://doi.org/10.1016/B978-1-4832-2817-4.50006-8>
- Jhingran, V.G.(1985). Fish and Fisheries of India (Hindustan Publishing Corporation, New Delhi.
- Joshi P.K. and Bose M. (2002). Toxicity of cadmium, a comparative study in the air breathing fish, *Clarius batrachus* and in non- air breathing fish, *Ctenopharyngodon idella*. Aquatic Toxicology, mechanism and consequences Eds.Chris. Kenedy Alan Kolok, Don Mackinlay, *International Congress of Fish Biology*, 109-118.
- Kim J.M (1979). The toxicity of mercury and cadmium on two fresh water fish, carp and loach, *Bulletin Kordi*, 1, 15-21.
- Munshi J.S.D. and Singh (1992). Scanning electron microscopic evaluation of effect of low pH on gills of *Channa punctatus* (Bloch). *Journal of Fisheries Biology*. 41, 83-89. <https://doi.org/10.1111/j.1095-8649.1992.tb03171.x>
- Nagabhushnam R. and G.K.Kulkarni.(1981). Fresh water palaemonid prawn, *Macrobrachium kistensis* (tiwari), Effect of heavy metal pollutants. *Proceedings Indian Natural Science Academy*. B47, 380-386.
- Pankaj Kumar, Ranjana, Mishra A.P (2004). Efficacy of malation on mortality of a fresh water air breathing cat fish *H.fossilis* (Bloch) during different developmental stages. *Journal of Zoology*, 10(1), 47-52.

- Playle R.C., et al.,(1993). Copper, cadmium binding to fish gills: Modification by dissolved organic carbon and synthetic ligands. *Canadian Journal Fish Aquaculture Science*, 50, 2667-2677. <https://doi.org/10.1139/f93-290>
- Randi A.S, et al.,(1996) :Histopathological effect of cadmium on gills of fresh water fish *Macropsobrycon uruguayanae*. Engenmann (Pisces; Atherinidae). *Journal of Fish Disease*, 19, 311-322. <https://doi.org/10.1046/j.1365-2761.1996.d01-82.x>.
- Sastry K.V and Subhadra,(1982. Effect of cadmium on some aspects of carbohydrate metabolism in fresh water cat fish *H.fossilis*. *Toxicology Letters*, 14, 45-55. [https://doi.org/10.1016/0378-4274\(82\)90008-X](https://doi.org/10.1016/0378-4274(82)90008-X).
- Singh B.R. and Mishra A.P. The development of air breathing organ in *Anabas testudineus*(Bloch), *Zoology Analysis*, 205, 5165, 359-370.
- Verma S.R., et al., (1993). In vivo effect of mercuric chloride on tissue ATPase on *Notopterus notopterus*. *Toxicology Letters*, 16, 305-309. [https://doi.org/10.1016/0378-4274\(83\)90191-1](https://doi.org/10.1016/0378-4274(83)90191-1).