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Original Research Paper

Zoology

EFFCETS OF ZINC ON GLYCOGEN CONTENT OF FRESHWATER FISH, *TILAPIA MOSSAMBICUS*

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ABSTRACT The effect of lethal and sub lethal concentration of zinc was investigated in the different vital tissues of *Tilapia mossambicus*. The fishes were exposed in both concentration of zinc for different time interval to study of lethal and sub- lethal toxicity of zinc at two concentration grade. for lethal concentration prepare stock solution of (2mg/L & 4mg/L,) and for sub lethal con. Selected 1/10 con. (0.2mg, 0.4mg/Lit) for 24h, 48h 72h and 96h. The LC50 value was 1.679ppm for 96h for 4mg/lit. Similarly in sub lethal concentration the LC50 Value was 0.194ppm at 72h for 0.4mg/lit. The biochemical parameter was changes in all vital tissues in both concentration of zinc & showed significant increased & decreased was found in glycogen level. These results indicate that exposure of zinc can alter the glycogen contents of *Tilapia mossambicus*.

KEYWORDS : Lethal, Sub-lethal, Zinc, Biochemical, *Tilapia mossambicus*

INTRODUCTION:

Zinc (Zn) is the second most abundant trace element after Fe and is an essential trace element and micronutrient in living organisms, found almost in every cell and being involved in nucleic acid synthesis and occurs in many enzymes Sfakianakis *et al.*, (2015). Additionally, Zn is involved in more complicated functions, such as the immune system, neurotransmission and cell signaling Celik &Oehlenschläger (2004) ; Hogstrand , (2011).It may occur in water as a free cation as soluble zinc complexes, or can be adsorbed on suspended matter. Zinc wastes can have a direct toxicity to fish at increased waterborne levels Niyogi & Wood (2006), and fisheries can be affected by either zinc alone or more often together with copper and other metals Sorensen, (1991).The other endpoints of toxicity vary amongst freshwater and marine fish with the most common being survival, growth, reproduction, and hatching Hogstrand (2011). Also, fish kidney is considered as a target organ for Zn accumulation Omar *et al.*, (2014). The clinical symptoms and patho-anatomical picture of zinc poisoning in fish are similar to those found for copper Svobodová, (1993);Farombi *et al.*, (2007).Zinc causes mortality, growth retardation, respiratory and cardiac changes, inhibition of spawning, and a multitude of additional detrimental effects which threaten survival of fish. Gill, liver, kidney, and skeletal muscle are damaged Sorensen, (1991).In fish, zinc significantly increases the activity of serum transaminases in some freshwater fishes Nemcsók *et al.*, (1981). Gill proliferation and stimulation of mucous cells and an increase in mucus production generally occur in response to zinc exposure Mallat (1985). Zinc exhibits accumulation in the tissues and organs of freshwater fish Cicik, (2003); Murugan *et al.*, (2008) and may cause disorder in osmoregulation Nussey *et al.*, 2002), cardiac respiratory rhythm Hughes and Adeney, (1977), changes in the blood gases and acid-alkaline status Spry and Wood, (1984), tissue hypoxia Tort *et al.*, (1982).

Zinc (Zn) is one of the most important essential trace elements involved in animal growth and the most widely used metal cofactor of many enzymes involved in protein, nucleic acid, carbohydrate, and lipid metabolism (Carpene *et al.* 2003; Sun *et al.* 2005). Zinc in certain concentration is desirable for fish growth but it's over accumulation is hazardous to exposed fish (Senthil Murugan *et al.* 2008). Zinc is one of the most common contaminants in aquatic systems and is associated with urban runoff, soil erosion, industrial discharges, pharmaceuticals, pesticides and a variety of other activities and sources (Schmitt 2004; Bowen *et al.* 2006). The danger of Zn is aggravated by its almost indefinite persistence in the environment because it cannot be destroyed biologically and is only transformed from one oxidation state or organic complex to another (Everall *et al.* 1989). it is necessary to monitor its potential impact on fish performance and health.

MATERIALS & METHODS:

Tilapia mossembicus, ranging between weighing about 150gm.were collected from a nursery pond at Sawargaon in Umri Tahsil of Nanded district. It is at a distance of about 50km.from Nanded.The animals were brought to the laboratory and were acclimatized to lab condition for one week, where they fade with rice cake and groundnut cake. Feeding was stopped one day prior to acute toxicity test. All the precautions recommended by APHA toxicity test of aquatic organisms (APHA 1998, 2005 and 2012) were followed. The fishes were exposed in both concentration of zinc for different time interval to study of lethal and sub- lethal toxicity of zinc at four concentration grade. for lethal concentration prepare stock solution of (2mg/L, 4mg/L, 6mg/L & 8mg/L) and for sub lethal con. Selected 1/10 con. (0.2mg, 0.4mg, 0.6mg &0.8mg/Lit) for 24h, 48h 72h and 96h. the LC50 value was 29.287ppm for 96h for 8mg/lit. The LC50 calculation &graph was done with MedCal statistical software. Sacrifice all the fishes for each hour. Isolate the tissues like muscle, stomach, intestine, kidney & liver stored in distilled water for the determination of toxic effects of zinc on vital tissues.

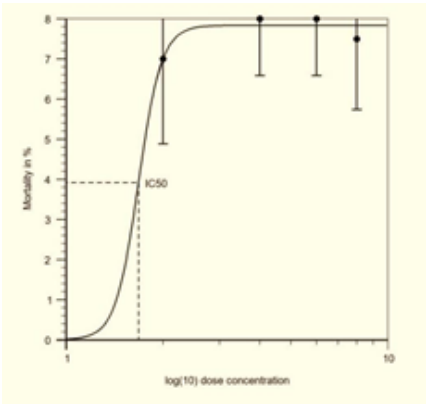
Collection of Blood:

The blood was collected by direct heart puncturing using sterile disposable plastic syringe with a 22-gauge needle (Molnar, 1960). The blood sample was taken in a tube rinsed with separated into two sets and stored in a refrigerator at 4°C. One set of preserved blood samples was used for hematological studies, while another set was used WBC, RBC, Hb, HCT, MCV, and MCH & MCHC were determined by fully automated Cell Counter (Haematology Analyze Vision AD 690 Vet).Both the tests were compared with control test. The total proteins were estimated by the method of Biuret (1951), the glucose by using Anthrone method (Oser, 1965).Total Fats were estimated by using Ethanol- ether method (Folch *et al.*, 1957)

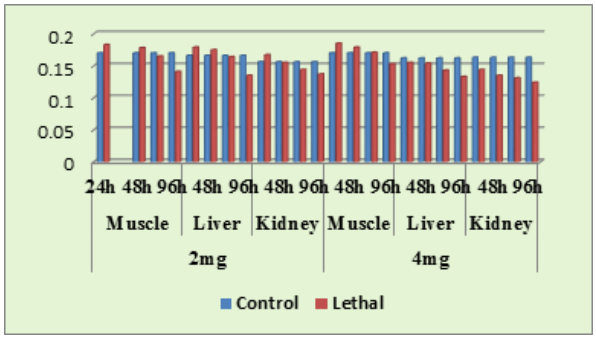
RESULTS AND DISCUSSION:

When fresh water fish *Tilapia mossambicus* exposed in different lethal concentration of zinc for different time intervals the LC50value was 1.679 ppm at 96h for 2mg/L similarly for sub-lethal con. The LC50 value was 0.189 ppm at 96h for 0.8mg/L and it is represented as Graph no1.When the fishes was exposed in 2mg of zinc for 24h & 96h muscle the glycogen content was increased significantly ($p>0.01$) while it was increased in 48h &72h insignificantly .In liver it was highly

increased significantly ($p>0.001$) in all the duration. In kidney the glycogen content was increased significantly in 24h ($p>0.01$),while it was decreased significantly in 72h ($p<0.002$) &in 9 While the fishes was exposed in 4mg of zinc for 24h & 96h muscle the glycogen content was increased significantly ($p>0.05$) & ($p<0.02$) while in liver it was decreased significantly ($p>0.001$), ($p<0.01$), ($p>0.001$) & ($p<0.002$) in all the duration. In kidney it was decreased significantly ($p>0.01$) in all the duration.



A number of studies on the accumulation of zinc in aquatic organisms were conducted both under laboratory conditions and in the field (Golovina 1996; Shaw *et al*. 2006; Fountoulaki *et al*. 2010). Zn absorbed by fish is transported through the bloodstream to various tissues where it causes tissue damage, changes in blood parameters and endocrine and metabolic activity (Tuncosoya *et al*. 2016). Biochemical parameters are used as an index for detecting physiological changes in several species of fish (Adhikari *et al* . 2004; Suvetha *et al* . 2010), because they are sensitive to potential adverse effects of accumulations of metals.



Graph no.1
Effects of zinc (diff. lethal con.) on various types of tissues (100 mg/wet. of tissues) in freshwater fish *Tilapia mossambicus* for diff. time intervals

In The present investigation highest increase in glucose concentration was in comparison with the control value. Similarly, in *Oncorhynchus mykiss*.it was exposed to zinc for 7 days. McLeay (1977) reported that sera glucose levels increased. With the exception of 24 hours. Sera glucose levels of *C.gariepinus* exposed to 1.0 and 5.0 ppm Zn increased with exposure time. This may result from stimulation of glycogenolysis in the liver and muscle depending on the demand for energy under the zinc effect (Ln and Vosyliene 1999). However, the level of glucose produced in the current study for the next 30 days. The higher level of glucose established during the first days of exposure may result from glycogenolysis (the release of glucose into the blood from energy resources stored in the muscles and liver as glycogen). Initiated by hormones (cortisol and catecholamines) when the organism was in unfavorable condition. The reduced glucose level at the end of exposure probably reflected the exhaustion of the organism's energy reserves and the impairment of the fish's capacity to restore them and the acclimatized condition (Welker *et al*. 2016). However, Welker *et al*., (2016) found no significant differences in rainbow trout between the level of glucose and Zn exposure. The level of glycogen was significantly increased in this investigation while it was reported same in another study, that increase in the glucose level of the tissue while decrement in tissue glycogen in exposed fish makes it clear that the glycogen reserves are being used to meet the stress caused. Increase in serum glucose levels in fish under stress was reported by Bedii and Kenan (2005), Chowdhury *et al*. (2004), Almeida *et al*. (2001). This can be attributed to several factors and one of them is the decrease in the specific activity of some enzymes like phosphofructokinase, lactate dehydrogenase and citrate kinase that decrease the capacity of glycolysis (Almeida *et al*., 2001). Glycogen levels are found to be highest in liver, as it is the chief organ of carbohydrate metabolism in animals, followed by muscle. Liver glycogen is concerned with storage and export of hexose units for maintenance of blood glucose and that of muscle glycogen is to act as a readily available source of hexose units for glycolysis within the muscle itself. A fall in the glycogen level clearly indicates its rapid utilization to meet the enhanced energy demands in fish exposed to toxicant through glycolysis or Hexose Monophosphate pathway. It is assumed that decrease in glycogen content may be due to the inhibition of hormones which contribute to glycogen synthesis. Decrease in liver and muscle glycogen levels is in corroboration with the reports of earlier workers (Bedii and Kenan, 2005; Dubale and Punita shah, 1981; Sastry and Subhadra, 1984).in the present investigation the glycogen was significantly decreased in all the tissues & this could be because the utilize glycogen reserves rapidly to meet the respiratory stress when exposed to the lethal concentration. It can be inferred from several investigations that biochemical parameters could be effectively used to detect the effect of pollutants (Magendran 1990). Carbohydrate is an important energy source of all vital activities of an organism. It supplies a major portion of the energy required to the living system. Sastry & Gupta (1978) have pointed out the fluctuation in glycogen level in *Channa punctatus* when exposed to different concentrations of mercuric chloride. Ramakrishna & Sivakumar (1993) have reported the significant decline in glycogen content of liver tissues in Oreochromis mossambicus under toxic stress of Quinolphos. The fish *Tilapia mossambica* exposed to arsenic toxicity has resulted in the depletion of carbohydrate (Shobha *et al*., Rani 2000). The present study has been undertaken to investigate the changes in the glycogen content of the tissues of liver and kidney at different time intervals in the freshwater fish, *Labeo rohita* exposed to lower and higher sub-lethal concentrations of arsenic. While when it was exposed in 4mg of zinc the level of glycogen was found significantly decreased in the present study. Increased glycogen level in liver in above lethal concentration is due to disturbance of carbohydrate metabolism as it was observed to affect the enzymes of glycolytic pathway and the krebs cycle leading to depletion and disturbance in cell membrane potential and ATP depletion (*Schuliga et.al. 2012).* The depletion of glycogen content in liver and muscle of *C.mrigala* was reported by *Anitha (2010).* The depletion was also observed in fish *Labeo rohita* when exposed to both lethal and sub-lethal concentration of fenvalerate (*Anitha 2010*) whereas glycogen in muscle remains unchanged in *Labeo rohita*.

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