Eco. Env. & Cons. 26 (2) : 2020; pp. (643-647)
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ISSN 0971–765X

Effect of Nickel chloride on lactate dehydrogenase activity in selected tissues of Fish *Labeo rohita* (Hamilton)

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(Received 4 November, 2019; accepted 23 December, 2019)

ABSTRACT

Lactate dehydrogenase (LDH) is the glycolytic enzyme affected by the toxicity of heavy metals. The aim of the present study is to determine the activity of lactate dehydrogenase in gill, liver, kidney, brain and muscle of the fish, *Labeo rohita* exposed to sublethal concentrations of nickel chloride $1/15^{th}$ (low) $1/10^{th}$ (medium) and $1/5^{th}$ (high) of the 96 hours values for the period of 10, 20 and 30 days. The activity of lactate dehydrogenase was significantly ($P<0.05$) decreased in gill, liver, kidney, brain and muscle of the fish, *Labeo rohita*. Therefore, the effect of nickel chloride have deleterious effect on the activity of lactate dehydrogenase in selected tissues of *Labeo rohita* as revealed in this investigation.

Key words : *Toxicity, Heavy metal, Nickel chloride, Deleterious, Labeo rohita.*

Introduction

Heavy metals are one of the most usual pollutants in water ecosystems and may have its sources from natural and anthropogenic origins, such as industrial, agricultural and domestic loads (Davultuoglu *et al.,* 2011). The environment and its compartments have been severely polluted by heavy metals (Masindi and Muedi, 2018). Heavy metals are high density elements occurring naturally in the environment in a minute quantity (Lu *et al.,* 2017). Heavy metals discharged into aquatic ecosystems, can be absorbed by suspended solids, and then strongly accumulated in sediments and biomagnified along aquatic food chains (Yi *et al.,* 2011). The contamination of aquatic systems by heavy metals have become one of the most challenging pollution issues owing to the toxicity, abundance, persistence and subsequent bioaccumulation of these materials (Fu, 2014). Heavy metals influences the quality of the atmosphere as well as water bodies and threatens the health and life of aquatic organisms, humans and microorganisms (Li *et al.,* 2013). Heavy metals in the aquatic ecosystem even at very low levels may accumulate within the body of aquatic species by various mechanisms to the extent that they exert toxic effect and to affect cellular organelles (Vahya *et al.,* 2018). Nickel is often found in the environment as a result of industrial discharges from electroplating, smelting, mining and refining operations and other industrial emissions (Vijayavel *et al.* ,2009; Attig *et al.,* 2010). The nickel is an essential element at low concentrations for many organisms; it is toxic at higher concentrations (Glark and Keasling, 2002).

Fish found in polluted water exposed to nickel, primarily through the ingestion of contaminated food and sediments (Dallinger and Kautzky, 1985). The heavy metals in the tissue of fish may cause various physiological defects and mortality (Torres *et al.,* 1987). During stress conditions, fish change and adapt their metabolic functions (Malarvizhi *et al.,* 2012). Hence, the present study has been carried out to study the effect of sublethal concentrations of nickel chloride on lactate dehydrogenase activities in gill, liver, kidney, brain and muscle of Indian major carp *Labeo rohita*.

Materials and Methods

Studies were conducted on fingerlings of fresh water fish *Labeo rohita*, obtained from fish farm located in Puthur, 7 Km. away from Chidambaram, Tamil Nadu, India. Fishes were acclimatized to the experimental conditions for 2 weeks. The fishes were fed twice a day. The feeding was stopped for two days before the start of experiment. Stock solution of nickel chloride was prepared from 1g of standard Analar grade granules in 1 liter of deionized water to form 100% concentration. From the stock solution, various concentrations used in the investigation were prepared by dilution. Apparently healthy live specimens of *Labeo rohita*, mean weight (14-16 gm) and mean length (11-13 cm) were used for the experiment irrespective of their sex. 96 hour of the LC_{50} value obtained 32.64 ppm of nickel chloride (Sparague, 1971). The experimental fishes were divided into four groups. The fishes belonged to the first group were maintained in a medium free from nickel chloride was served as control. The second group exposed to $1/15^{th}$ (2.176 ppm) of the LC_{50} of low sublethal concentration of nickel chloride for the period of 10, 20 and 30 days. The third group exposed to $1/10^{th}$ (3.264 ppm) of the LC_{50} of medium sublethal concentration of nickel chloride for the period of 10, 20 and 30 days. The fourth group exposed to $1/5^{th}$ (6.528 ppm) of the LC_{50} of high sublethal concentration of nickel chloride for the period of 10, 20 and 30 days respectively. During the experimental period, the control and the experimental groups were fed on ad libitum diet of rice bran and oilcake. The medium was renewed daily with sublethal concentration of the nickel chloride. At the end of the experiment, the control and experimental fishes were sacrificed. The gill, liver, kidney, brain and muscle were removed from both control and treated fishes for enzymatic assays. The lactate dehydrogenase activity of tissues were estimated by the method of Govindappa and Swami (1965). The data were determined by Duncan's Multiple Range Test (DMRT) using SPSS (Version 16.0). Values represent Mean ± SD of three biological replicates. One way ANOVA used to test the level of significance at 5% level (Duncan, 1957).

Results

Reduction of lactate dehydrogenase activities of the gill, liver, kidney brain and muscle of *Labeo rohita* exposed to the nickel chloride for 10, 20 and 30 days in $1/15^{th}$ (low), $1/10^{th}$ (medium) and $1/5^{th}$ (high) of the LC_{50} values of sublethal concentrations were estimated. The Lactate dehydrogenase activity was gradually reduced in all sublethal concentrations during the exposure period of 10, 20 and 30 days. The lactate dehydrogenase activities of the gill, liver, kidney brain and muscle are shown in Table 1. Among these, the more remarkable reduction of lactate dehydrogenase activity was noticed in liver during the period of 30 days. The depletion of lactate dehydrogenase activity was more pronounced in gill, liver, kidney and muscle than brain. Generally, reduction in lactate dehydrogenase was directly proportional to the exposure period of the toxicant. The obtained biochemical estimation values of the gill, liver, kidney, brain and muscle were subjected to statistical analysis and showed significant values at $P<0.05$ (Table 1).

Discussion

Heavy metals are accepted as one of the dangerous aquatic pollutants and are highly toxic to many aquatic organisms (Sontakke and Jadhav, 1997). Nickel chloride is known to exert their action on tissues of aquatic organism (Palaniappan and Karthikeyan, 2009). Metal ions are present in the water bodies are absorbed into the body capable of binding and to exert their actions with a variety of active binding sites and then changing the normal physiology of the organism which may lead to the death of organism (Harper *et al.,* 1978). The heavy metal toxicity may cause injury to various tissues of aquatic organisms and changed enzyme activity (More *et al.,* 2005). Lactate dehydrogenase enzyme plays an important role in carbohydrate metabolism and present in most of the animal tissues involved

Table 1. Lactate dehydrogenase activity in gill, liver, kidney, brain and muscle of *Labeo rohita* exposed to sublethal concentration of nickel chloride.

Treatments	10 days	20 days	30 days
LDH activity in Gill (µmoles formazone formed/mg proteins/min)			
Control	0.038 ± 0.002 [c]	0.039 ± 0.003 [d]	0.038 ± 0.002 [d]
Low concentration	0.036 ± 0.002 [c]	0.036 ± 0.002 [c]	0.030 ± 0.002 [c]
Medium concentration	0.030 ± 0.002 [b]	0.027 ± 0.001 [b]	0.022 ± 0.001[b]
High concentration	0.022 ± 0.001 [a]	0.018 ± 0.001 [a]	0.009 ± 0.008 [a]
LDH activity in Liver (µmoles formazone formed/mg proteins/min)			
Control	0.040 ± 0.003 [d]	0.041 ± 0.003 [d]	0.040 ± 0.003 d
Low concentration	0.035 ± 0.002 [b]	0.035 ± 0.002 [c]	0.033 ± 0.002 [c]
Medium concentration	0.032 ± 0.002 [b]	0.030 ± 0.002 [b]	0.024 ± 0.001 [b]
High concentration	0.025 ± 0.001 [a]	0.023 ± 0.001 [a]	0.013 ± 0.008 [a]
LDH activity in Kidney (µmoles formazone formed/mg proteins/min)			
Control	0.032± 0.002 [c]	0.031 ± 0.002 [d]	0.038 ± 0.002 d
Low concentration	0.030 ± 0.002 [c]	0.026 ± 0.001 [c]	0.020 ± 0.001 [c]
Medium concentration	0.027 ± 0.001 [b]	0.021 ± 0.001 [b]	0.012 ± 0.008 [b]
High concentration	0.022 ± 0.001 [a]	0.014 ± 0.008 [a]	0.005 ± 0.000 [a]
LDH activity in Brain (µmoles formazone formed/mg proteins/min)			
Control	0.043 ± 0.003 [b]	0.044 ± 0.003 [d]	0.042 ± 0.003 d
Low concentration	0.042 ± 0.003 [b]	0.040 ± 0.003 [c]	0.037 ± 0.002 [c]
Medium concentration	0.037 ± 0.002 [a]	0.033 ± 0.002 [b]	0.031 ± 0.002 [b]
High concentration	0.036 ± 0.002 [a]	0.026 ± 0.001 [a]	0.019 ± 0.001 [a]
LDH activity in Muscle(µmoles formazone formed/mg proteins/min)			
Control	0.046 ± 0.003 [c]	0.045 ± 0.003 [d]	0.046 ± 0.003 d
Low concentration	0.043 ± 0.003 [c]	0.039 ± 0.003 [c]	0.035 ± 0.002 [c]
Medium concentration	0.038 ± 0.002 [b]	0.032 ± 0.002 [b]	0.021 ± 0.001 [b]
High concentration	0.031 ± 0.002 [a]	0.024 ± 0.001 [a]	0.008 ± 0.008 [a]

All the values mean ± SD of six observations.
Values which are not sharing common superscript differ significantly at 5% ($p<0.05$).
Duncan multiple range test (DMRT).

in cellular metabolic activity (Sandhya, 2017). LDH is an anaerobic enzyme located at a strategic point between glycolysis and citric acid cycle, which catalyzes the reversible oxidation of lactate to pyruvate, serving in the terminal step of glycolysis (Reddy *et al.,* 2011). Changes in the activity of LDH enzyme resulting from toxicant or contaminant effects in various organs of fish have been reported (Ruas *et al.,* 2008). Such alterations in fish are aimed at maintaining equilibrium in the presence toxicants which are known to disrupt physiological and biochemical process (Winkaler *et al.,* 2007).

Sankar Samipillai and Uma (2018) reported that increased activity of LDH in brain, gill, liver and kidney tissue of *Catla catla* exposed to sublethal concentration of copper sulphate . Justinraj and Joseph (2015) observed that chronic exposure of Acetamiprid showed increased activity of LDH in liver, brain and gill tissues during all the exposure periods. Janardanreddy (2012) demonstrated that LDH activity was increased in gill, liver and muscle of *Cirrhinus mrigala* exposed to cadmium chloride and mercuric chloride indicating that it may favor anaerobic respiration and also suggested a significant increase in the conversion of pyruvate to lactic acid, thereby leading to the accumulation of lactic acid for the production of glucose during stress induced by toxicity of heavy metals. Ray and Cremer (1979) reported that the increased lactate levels are due to increased muscular activity. Sharma and Jain (2008) observed that under hypoxic condition there is augmented release of lactate dehydrogenase in *Cyprinus carpig* exposed to pesticide. Similar reports were given by Choudhurya *et al.,* (2017) in *Oreochromis mossambicus* and *Channa punctatus* exposed to fungicide.

In the present study, decreased activity of LDH in gill, liver, kidney, brain and muscle of *Labeo rohita* were observed in sublethal concentrations of nickel chloride exposed for the period of 10, 20 and 30

days. The present work agreed with the findings of Pazhanisamy (2002) in liver, muscle and gill tissues of *Labeo rohita* exposed to arsenic trioxide. Similar results were given by Sastry and Rao (1981) who reported decrease in LDH level of brain in *Channa punctatus* exposed to mercuric chloride. Rajamanickam and Muthusamy (2008) reported that reduction in LDH activity in liver of common carp exposed to heavy metals. In the present study, the significant decrease in LDH activity in all tissues suggest a reduction in the conversion of lactate to pyruvate, there by leading to the accumulation of lactic acid. The present finding agrees with the suggestion of Arya *et al.,* (2013) who found that decrease in the activity of LDH in liver, gills and muscle of fish *Cirrhina mrigala* exposed to lead indicates decreased rate of glycolysis. It is inferred from the present study that the reduce in LDH activity suggests there appeared to be a shift in the carbohydrate metabolism from aeropic type to anaerolic type due to nickel ions. The change in the level of activity of LDH enzyme extensively used as a stress in fish (Rani *et al.,* 2015). LDH is a tetrameric glycolytic enzyme and recognized as a potential marker of tissue damage (Diamantino *et al.,* 2011). The present study suggests LDH is a cytoplasmic enzyme might be released into the plasma after the cellular damage caused by nickel toxicity and this might be the real reason for the significant depletion of LDH in selective tissues of *Labeo rohita*. Alexander *et al.* (2013) observed that LDH activity was depleted in liver, kidney and gill of fish *Clarius batrachus* due to increased tissue damage caused by exposure of phenolic compounds. Rashed (2001) reported that any alteration in LDH activity which intern increased permeability and cell necrosis in tissues.

Acknowledgement

Authors would like to thank authorities of Annamalai University for providing the facilities to carry out the study.

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