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RESEARCH ARTICLE

IMPACT OF HEAVY METAL NICKEL CHLORIDE ON ENZYME SUCCINATE DEHYDROGENASE OF FRESHWATER FISH *Labeo rohita* (HAMILTON)

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ABSTRACT

Succinate dehydrogenase is the oxidative enzyme which was drastically affected by the action of heavy metals. Succinate dehydrogenase is chosen as a representative of metabolic enzyme. It is a marker enzyme for detecting the presence of TCA cycle in tissues. The aim of the present study is to assess the enzyme succinate dehydrogenase activities in gill, liver, kidney, brain and muscle of the fish *Labeo rohita* exposed to sublethal concentration of nickel chloride 1/5th (high), 1/10th (medium) and 1/15th (low) of the 96 hour LC_{50} values for the period of 10, 20 and 30 days. The fish exposed to nickel chloride showed a decrease the enzyme succinate dehydrogenase activities for 10, 20 and 30 days in gill, liver, kidney, brain and muscle. However, no information is on record concerning the three different sublethal concentration of heavy metal, nickel chloride on the enzyme succinate dehydrogenase of freshwater fish *Labeo rohita*. The objective of the present work was to observe the effect of nickel on succinate dehydrogenase activities in gill, liver and kidney of Indian major carp, *Labeo rohita.*

INTRODUCTION

Environmental pollution due to toxic heavy metals in air, soil and water is a major global problem. Heavy metals cannot be degraded or destroyed; hence they are persistent in all parts of the environment. The reduction amount of these metals from effluents to a permissible limit before discharging them into streams and rivers is very important for human health and environment (Srividya and Mohanty, 2009). Water pollution is thus a cosmopolitan problem that needs urgent attention and prevention (Ali and Soltan, 1996; Handy, 1994; Osman, 2007). It resulted from many sources, e.g. accidental spillage of chemical wastes, discharge of industrial or sewerage effluents, agricultural drainage, domestic wastewater and gasoline from fishery boots (Handy, 1994; Ali and Soltan, 1996). Water pollution is one of the principal environmental and public health problems (Osman and Kloas, 2010). The aquatic habitats are being contaminated with heavy metals due to industrialization and other anthropogenic activities (Muthupriya and Altaff, 2010). Aquatic animals inhabiting polluted water bodies tend to accumulate many chemicals in high concentrations even when the ambient environmental contamination levels are low potentially hazardous situation for the entire food chain. Among several elements of the periodic table, there are 35 metals are associated with community and occupational exposure. Out of these, 23 are described as heavy metals. These elements are generally released in small amounts into the environment by processes like weathering of rocks, volcanic eruptions etc. and their intake/exposure is necessary in trace amounts for good health. But, presently, there is a steady increase in their concentration in all habitats owing to mining, electroplating, paints and dye, battery making industries etc. The release is rapid with the rapidly growing technology and heavy metal application in these industries (Sopha *et al.,* 2007). The contamination of freshwaters with a wide range of pollutants has become a matter of great concern over the last few decades (Al-Weher, 2008). Heavy metals are natural trace components of the aquatic environment, but their levels have increased due to domestic, industrial, mining and agricultural activities (Leland *et al.,* 1978; Mance, 1987; Kalay and Canli, 2000). Aquatic organisms such as fish and shell fish accumulate metals to concentrations many times higher than present in water or sediment (Olaifa *et al.,* 2004, Gumgum *et al.,* 1994). Discharge of heavy metals into river or any aquatic environment can change both aquatic species diversity and ecosystems, due to their toxicity and accumulative behavior (Heath, 1987). Nickel is a very abundant element. In the environment, it is found primarily combined with oxygen (oxides) or sulfur (sulfides). It is found in all soils and is emitted from volcanos. Nickel has properties that make it very desirable for combining with other metals such as iron, copper, chromium and zinc to form alloys. These alloys have important uses such as in the making of metal coins and jewelry and in industry for making items such as valves and heat exchangers. Most nickel is used to make stainless steel. Nickel compounds are used for nickel plating, to colour ceramics, to make some batteries and as substances known as catalysts to increase the rate of chemical reactions. Nickel is released into the atmosphere during nickel mining and by

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industries that convert scrap or new nickel into alloys or nickel compounds or by industries that use nickel and its compounds. These industries may also discharge nickel in wastewater. However, the major sources of nickel exposure are tobacco smoke, auto exhaust, fertilizers, superphosphate, food processing, hydrogenated-fats-oils, industrial waste, stainless steel cookware, testing of nuclear devices, baking powder, combustion of fuel oil, dental work and bridges. High exposure can cause cough, shortness of breath and fluid in the lungs, which is sometimes delayed for 1 to 2 days after exposure. Single high or repeated lower exposures may damage the lungs, with scarring of lung tissues and may cause damage to heart muscle, liver and/or kidney (Al-Attar, 2007). Fish has been the main supply of cheap and healthy protein to a large percentage of the world's population. In most Asian countries, especially those in Southeast Asia, fish is a main protein of the diet. It is particularly valuable for providing proteins of high quality comparable with those of meat, milk or eggs, and is also a good source of omega-3 fatty acids; calcium and phosphorus, iron, trace elements like copper, and a fair proportion of the B-vitamins (Tucker, 1997). Beside good health benefits of fish, there were many reports on contamination of fish by chemical in the environment. The fish, as a bioindicator species, plays an increasingly important role in the monitoring of water pollution because it responds with great sensitivity to changes in the aquatic environment. The sudden death of fish indicates heavy pollution; the effects of exposure to sublethal levels of pollutants can be measured in terms of biochemical, physiological or histological responses of the fish organism (Mondon *et al.,* 2001). Changes in age and species distribution in a stock fish population are general indicators of water pollution, but there are also responses specific to a single pollutant or a group of contaminants. Biochemical markers are biochemical responses induced in the presence of a specific group of contaminants that have the same mechanism of toxic activity (Iroka and Drastichova, 2004). Succinate dehydrogenase is a primary enzyme in the oxidative catabolism of sugars (Lehninger et al., 1993) and as such is used effectively as a marker of mitochondrial abundance and activity to identify any possible physiological disturbance in fishes. Hence, in the present investigation has been made to find out the effect of sublethal concentration of nickel chloride on succinate dehydrogenase activities in gill, liver, kidney, brain and muscle of fresh water fish *Labeo rohita.*

MATERIALS AND METHODS

The fish *Labeo rohita* having mean weight 14-16 gm and length 12 – 14 cm were collected from PSP fish farm, at Puthur and acclimatized to laboratory conditions. They were given the treatment of 0.1%KMNO4 solution and then kept in plastic pools for acclimatization for a period of seven days. They were fed on rice bran and oil cake daily. The nickel chloride was used in this study and stock solutions were prepared. Nickel chloride LC_{50} was found out for 96 h (32.64 ppm) (Sprague, 1971) and $1/5^{th}$ (high), $1/10^{th}$ (medium) and $1/15^{th}$ (low) of the LC_{50} values were 6.528, 3.264 and 2.176 ppm respectively taken as sublethal concentrations for this study. Forty fish were selected and divided into 4 groups of 10 each. The first group was maintained in free from nickel chloride and served as the control. The other 3 groups were exposed to sub lethal concentration of nickel chloride in 10 litter capacity aquaria. The 2nd, 3rd and 4th groups were exposed to nickel chloride for 10, 20 and 30 days respectively. At the end of each exposure period, the fish were sacrificed and the required tissues were collected for succinate dehydrogenase activity estimation. Fishes were exposed to three sublethal concentrations of nickel chloride separately in plastic troughs and control fishes were also maintained separately. They were fed on ad libitum diet of rice bran and oil cake. The medium was renewed daily with sublethal concentration of the nickel chloride. The succinate dehydrogenase activities of the tissues were estimated by the method of Nachales *et al.* (1960). The data were analyzed by applying analysis of variance DMRT one way ANOVA to test the level of significance (Duncan, 1957).

RESULTS

Depletion of succinate 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 $1/5^{th}$, $1/10^{th}$ and $1/15^{th}$ of the LC_{50} values of sublethal concentrations were estimated. Among these, the maximum depletion of succinate dehydrogenase was observed in liver during 30 days. Generally, depletion in succinate dehydrogenase activities is 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

Pollution by heavy metals is an important problem due to the metals' persistence in the environment. Since the aquatic environment is the ultimate recipient of the pollutants produced by natural and anthropogenic sources, accumulation, and persistence of heavy metals in the aquatic environment constitute a formidable threat to biological life (George, 1989; Gagne et al., 1996; Fleeger et al., 2003; Aramphongphan et al., 2009). Heavy metals are some of the most-active polluting substances as they can cause serious impairment to circulatory, metabolic, physiological, and even structural systems when high concentrations are present in aquatic ecosystems (Shugart et al., 1992). Although heavy metals are often referred to as a common group of pollutants, individual metals pose different problems in freshwater environments, and therefore they have to be considered separately (Lloyd, 1992). Much more extensive biochemical toxicological research has been conducted in mammals than in fish. However, it is not surprising that many biochemical similarities exist among vertebrate species (Hochachka and Mommsen, 1995).

Occurrence of some heavy metals in all the environmental compartments including food chain of aquatic medium, despite their declining trend as the distance from the point of source increased and remained within the permissible limit, was responsible for the heavy metal toxicity that perhaps affected the succinate dehydrogenase enzyme activity of fish (Mukherjee and Jana, 2007). The use of biochemical approaches have been advocated to provide an early warning of potentially damaging changes in stressed fish. In toxicological studies of acute exposure, changes in concentrations and enzymes activities often directly reflect cell damage in specific organs (Casillas *et al.,* 1983). The

Table 1. Succinate dehydrogenase activity (µ mole formazone formed /mg protein/min) in gill, liver, kidney, brain and muscle of *Labeo rohita* exposed to sublethal concentration of nickel chloride

Treatments	10 days	20 days	30 days
Gill control	0.056 ± 0.004^{b}	0.057 ± 0.004^{c}	0.055 ± 0.004^{d}
Low concentration	0.054 ± 0.004^{b}	0.050 ± 0.003^{b}	0.046 ± 0.003^{c}
Medium concentration	0.052 ± 0.004^{ab}	0.045 ± 0.003^{a}	0.039 ± 0.003^{b}
High Concentration	0.049 ± 0.003^{a}	0.041 ± 0.003^{a}	0.030 ± 0.002^{a}
Liver control	0.047 ± 0.003^{c}	0.048 ± 0.003^{d}	0.047 ± 0.003^{d}
Low concentration	0.044 ± 0.003^{c}	0.039 ± 0.003^{c}	0.032 ± 0.002^{c}
Medium concentration	0.040 ± 0.003^{b}	0.032 ± 0.002^{b}	0.026 ± 0.001^{b}
High Concentration	0.033 ± 0.002^{a}	0.024 ± 0.001^{a}	0.018 ± 0.001^{a}
Kidney control	0.040 ± 0.003^{c}	0.039 ± 0.003^{d}	0.038 ± 0.002^{d}
Low concentration	0.037 ± 0.002^{bc}	0.035 ± 0.002^{c}	0.031 ± 0.002^{c}
Medium concentration	0.036 ± 0.002^{b}	0.030 ± 0.002^{b}	0.028 ± 0.002^{b}
High Concentration	0.031 ± 0.002^{a}	0.023 ± 0.001^{a}	0.016 ± 0.008^{a}
Brain control	0.052 ± 0.004^{c}	0.051 ± 0.004^{d}	0.050 ± 0.003^{d}
Low concentration	0.048 ± 0.003^{c}	0.043 ± 0.003^{c}	0.038 ± 0.002^{c}
Medium concentration	0.045 ± 0.003^{b}	0.039 ± 0.003^{b}	0.032 ± 0.002^{b}
High Concentration	0.040 ± 0.003^{a}	0.031 ± 0.002^{a}	0.019 ± 0.001^{a}
Muscle control	0.059 ± 0.004^{c}	0.058 ± 0.004^{c}	0.059 ± 0.004^{d}
Low concentration	0.057 ± 0.004^{c}	0.050 ± 0.003^{b}	0.043 ± 0.003^{c}
Medium concentration	0.053 ± 0.004^{b}	0.046 ± 0.003^{b}	0.037 ± 0.002^{b}
High Concentration	0.047 ± 0.003^{a}	0.038 ± 0.002^{a}	0.024 ± 0.001^{a}

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

succinate dehydrogenase is an important enzyme of kreb's cycle whose qualitatine changes are significant during certain pathological conditions (Harper *et al.,* 1978). Sucinate dehydrogenase is the oxidative enzyme which was drastically affected by the action of heavy metals. Succinic acid dehydrogenase is chosen as a representative of metabolic enzyme. It is a marker enzyme for detecting the presence of TCA cycle in tissues (Natarajan, 1979).

The impact of contaminants on aquatic ecosystem can be assessed by measurement of biochemical parameters in fish that respond specifically to the degree and type of contamination (Petrivalsky et al., 1997). Gills are the vital organs in fish which have direct contact with the medium through which pollutants enter into the body (Edwards, 1973). The succinate dehydrogenase enzyme is concentrated in chloride cells within the fish gills and has been used as an indicator of osmoregulatory activity (Langdon and Thorpe, 1984). Liver is one of the most multi faceted and active organs in higher animals. In a vertebrate body, the liver is most important target organ as it is the chief metabolic and detoxification center (Bhattacharya and Mukherjee, 1976). The kidney which is an important organ of excretion and osmoregulation is indirectly affected by pollution through blood circulation (Newman and MacLean, 1974). Fish muscles are edible and economically important. A reduction in the succinate dehydrogenase enzyme activities in the present investigation in *Labeo rohita* suggests that the fish is not in a healthy condition. Many investigators have also recorded such a reduction in succinate dehydrogenase enzyme activities in fishes exposed to different toxicants (Sastry and Sharma, 1980; Natarajan, 1984).

In the present study the activity of succinate dehydrogenase decreased in gill, liver, kidney, brain and muscle of *Labeo rohita* exposed to sublethal concentration of nickel chloride. This suggests that an inhibited mitochondrial oxidation of succinate which may lead to drop in energy production and the suppression of succinate dehydrogenase activity indicates the impairment of oxidative metabolic cycle and hence relies on anaerobic glycolysis may be increased to meet its energy demands. There are evidences that the succinate dehydrogenase enzyme activities in the liver and muscle tissues of tilapia decreased when they were exposed to pesticide thiodon. The inhibition of succinate dehydrogenase enzyme activities indicated the impairment of aerobic metabolism (Rajeswari et al., 1989). Also there was a decrease in the activity of succinate and lactate dehydrogenase in the gill, liver and muscle tissues of fish *Oreochromis mossambicus* when exposed to pesticide methyl parathion, which was caused by binding of endosulfan and methyl parathion with enzyme molecule and/or by blocking enzyme synthesis (Shukla, 1997). Similarly, Sastry and Subhadra, (1982) have reported a decrease in the succinate dehydrogenase activity in the liver tissue of *Channa punctatus* exposed to cadmium and copper. Mary chandravathy and Reddy, (1994) have reported that a decreased in succinate dehydrogenase activity in the gill and liver tissues of *Anabas scandens* exposed to lead nitrate. James *et al.* (1992) have observed that the level of succinate dehydrogenase activity decreased in the liver tissue of animals exposed to metal. They reported a metabolic shift from aerobiosis to an anerobiosis due to metal actions. Alam, (1989) observed alterations in oxidative metabolism of *Viviparous bengalensis* after exposure to heavy metal. More *et al.* (2005) have observed that the level of succinate dehydrogenase activity decreased in *Lamellidens marginalis* exposed to heavy metal. They also reported that the anaerobic activity of the cells due to pollution stress has reversed physiological and biochemical adaptation. This decrease in succinate dehydrogenase activity might be suggestive of the weakening of biochemical differences which in turn could be the results of tissue damage. Rajamanickam, (1992) has observed a reduction in succinate dehydrogenase activity in the liver tissue of *Mystus vittatus* exposed to copper. Radhakrishnaiah *et al.* (1992) have reported that the suppression in succinate dehydrogenase activities in liver tissues of *Labeo rohita* exposed to copper. The results of the present study clearly show significant alterations in succinate

dehydrogenase enzyme due to intoxication of nickel chloride stress in *Labeo rohita.* The decrease in succinate dehydrogenase enzyme activity may reflect decreased dependence on anaerobic carbohydrate metabolism by the gill, liver, kidney, brain and muscle of fish *Labeo rohita* that were exposed to nickel chloride.

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