ejpmr, 2016,3(11), 700-703

SJIF Impact Factor 3.628

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Research Article

ISSN 2394-3211

EJPMR

RECOVERY IN SUCCINATE DEHYDROGENASE FROM MERCURY EXPOSED FRESHWATER FISH *CHANNA PUNCTATUS.*

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Article Received on 22/09/2016 **Article Revised on 12/10/2016** **Article Accepted on 02/11/2016**

ABSTRACT

Succinate dehydrogenase is important amongst the several molecules available in the cells and carbohydrates plays an important role in the cellular process. In the present investigation, fish, *Channa punctatus* treated with an equitoxic dose of 11 ppm of Mercuric nitrate and Mercuric acetate were scarified on 1, 4, 8, 12 and 15 days for recovery patterns in liver, muscle, kidney, gill and brain . Mercury toxicated fishes recovered after 15 days which depends on the physical condition of the fish.

KEYWORDS: *Carbohydrate, Mercury, Channa.*

1. INTRODUCTION

Pollution of the aquatic environment by heavy metals is a subject of great concern. These substances are generally discharged into the environment as a result of industrial processes and pose a problem because they are toxic and tend to accumulate in organisms. Minimata disease caused by the consumption of mercury contaminated shellfish and finfish taken from Minimata Bay in Japan, and Itai-Iatai disease, caused by the consumption of food contaminated by cadmium in Japan (Ui 1972) have increased the awareness of toxic effects of the heavy metals on human beings and that has Prompted to take steps for their control. The modern industries are making use of various heavy metals such as iron, copper, nickel, platinum and Mercuric. Chemical pollution threatens the living systems and aquatic environment. Some of these metals are biologically essential, but others like cadmium, Mercuric and mercury are highly hazardous to aquatic biota and normally occur in low concentrations. It is known that common forms of Mercuric poisoning results from mining, processing and commercial dissemination of Mercuric (Hammond, 1969).

The natural emissions of mercury, mainly result from the degassing of the Earth's crust and evaporation from water bodies, are two to four times larger than those from anthropogenic sources (Hutchinson and Meema 1987). The heavy metals are not only hazardous to aquatic animals but also alternatively to mankind; as human beings use most of the freshwater animals as a source of food material. The disposal of industrial and agricultural waste directly or indirectly into aquatic system burdens the ecosystem. (Bela Turkey Kaushal & Abha Mishra, 2013).

2. MATERIALS AND METHODS

Channa punctatus selected as test species is a representative of ray finned fishes in South India. They are well known for their air breathing ability and can survive out of water in moist air for six days. It is selected as the test animal because of its euryhaline and eurythermal nature and unique position in food chain. They are quite sturdy and ideally suited for experimentation in laboratory for longer periods.

Biochemical assays were done in different tissues from both experimental and control fishes. Fish, approximately of same size and weight were grouped into 6 batches. 2 batch of fish served as controls, 2 exposed to Mercuric nitrate and the remaining two exposed to Merucric acetate for a period of 15 days. After a period of 15 days of exposure, a fish from each batch were transferred to Mercury-free water and scarified at the same intervals to observe the recovery. The values of different parameters were expressed as mean with standard error. Significance of the values obtained was tested using student 't' test .Succinate dehydrogense was estimated by modified method of Nachalas et.al, (1960).

3. RESULTS AND DISCUSSSION

3.1 RESULTS

The results of the present investigation report the changes in the metabolite levels and enzyme profiles concerned with the carbohydrate metabolism in the

tissues of fish during exposure and recovery periods after Mercuric nitrate and Mercuric acetate intoxication. Analyses of the data performed at different exposure periods in the tissues such as liver, muscle, kidney, gill and brain – are as follows:

1) Succinate Dehydrogenase

Succinate dehydrogenase activity was found progressively inhibited in all the tissues from 4th day of exposure, however, on the first day exposure a statistically significant enhancement in the activity was recorded in all the tissues. Maximum elevation was recorded in kidney (+13.93% for Mercuric nitrate $P < 0.001$; +15.76% for Mercuric acetate $P < 0.05$ followed by muscle (+12.85% for Mercuric nitrate, +14.25% for Mercuric acetate; $P<0.05$), liver(+13.25% for Mercuric nitrate,+13.44% for Mercuric acetate, $P < 0.001$), gill (+10.16% for Mercuric nitrate, +13.11% for Mercuric acetate, $P < 0.01$) and brain (+7.93% for Mercuric nitrate $P < 0.05$ and +7.61% for Mercuric acetate $P<0.01$).

On the 4th day the enzyme activity was inhibited in all the tissues. The inhibition was more in muscle (-16.16% Mercuric nitrate; $P < 0.05$ and -17.26% Mercuric acetate $P < 0.01$) followed by brain (-12.00% Mercuric nitrate, -14.29% Mercuric acetate, $P < 0.001$) kidney (12.62% Mercuric nitrate, -13.85% Mercuric acetate, $P < 0.05$), liver (-12.07% Mercuric nitrate, -13.85% Mercuric acetate, $P < 0.05$), liver (-12.07% Mercuric nitrate $P < 0.001$; -13.75% for Mercuric Mercuric acetate $P < 0.01$) and gill (-8.47% for Mercuric nitrate $P < 0.05$ -10.42% for Mercuric acetate $P < 0.01$).

On 8th day the SDH activity was decreased in all the tissues. The present decrease in the SDH activity was statistically significant at $P < 0.001$; $P < 0.01$; $P < 0.05$. Maximum depletion was record in liver (-26.56% for Mercuric nitrate, -27.64% for Mercuric acetate) and minimum was recorded in gill (27.64% for Mercuric nitrate, -16.61% for Mercuric acetate). The percent depletion ranged from -17.07% to -26.56% for Mercuric nitrate and -16.61% to -27.64% for Mercuric acetate. On 12th day of exposure maximum inhibition was noticed in liver (-39.36% for Mercuric nitrate, -41.09% for Mercuric acetate and minimum in brain (-27.42% for Mercuric nitrate, -24.73% for Mercuric acetate). The percent inhibition ranged between -26.33% to -39.36% for Mercuric nitrate and -24.73% to -41.09 for Mercuric acetate.

On 15th day maximum enzyme inhibition was noticed in all the tissues. Liver exhibitied maximum drop in the activity (-40.59% Mercuric nitrate: -42.03% Mercuric acetate, $P < 0.001$) followed by kidney (-35.28% Mercuric nitrate $P < 0.01$ and -37.86% for Mercuric acetate $P < 0.001$), muscle (-34.62% for Mercuric nitrate, -35.50% for Mercuric acetate; $P < 0.001$), brain (-29.60% Mercuric nitrate and -31.73% Mercuric acetate, $P < 0.001$) and gill (-30.74% for Mercuric nitrate and -29.29% for Mercuric acetate, $P < 0.001$).

During the 1st day of recovery period the inhibition in SDH activity was continued, but the magnitude of inhibition was slightly less than 15th day of exposure. However, the depletion was narrowed down over the rest of the recovery periods indicating maximum recovery in SDH activity.

The recovery was found to be tissue-specific and time dependent. Earliest recovery was found on 8th day of recovery period in the brain followed by gills and muscle. Liver and kidney exhibited a recovery on the 15th day of recovery period. On the 15th day of recovery all the tissues witnessed a statistically insignificant percent variations between control and experimental fishes. (Fig.1)

3.2 DISCUSSION

The present investigation is aimed to understand the alterations in enzyme of carbohydrate and energy metabolism during exposure and recovery periods after mercuric nitrate and mercuric acetate intoxication. Two heavy metal salts of Mercury i.e. mercuric nitrate and Mercuric acetate were selected in order to understand the relative toxicities of these salts. The alterations observed enzyme appear to be tissue-specific and time-dependent. The differential responses of tissues during exposure to mercuric salts can be attributed to absorption, distribution and elimination kinetics of mercuric nitrate and mercuric acetate and also on the characteristics of tissues like vascularity, perfusion and residual blood volume (Villarreal and Villegas, 1987).

On the 1st day of exposure all the tissues recorded an elevation and on the subsequent exposures, a progressive inhibition was recorded in all the tissues. The responses in general were more in the organic form of mercury in comparison to the inorganic form. The maximum amount of responses was recorded in the liver followed by kidney, muscle, gill and brain. The tissue-specific and time-dependent variations in the responses could be attributed to the kinetic variations of mercury ions in different tissues. The initial elevation of SDH activity suggests the increased operation of kreb's cycle to provide higher amounts of energy during the early stages of toxic manifestations. The enhancement also indicates the higher metabolic rate in the initial stages of toxicosis. The activities of SDH showed a decrease in all the tissues of the mussels exposed to zinc and cadmium, and enzyme activities were decreased more in zinc than compared to cadmium (Laxmi Prasad et. al., 2009).

Inhibition in the SDH activity during subsequent exposures may be due to the increased concentrations of toxicant in the tissues. The progressive reduction in SDH activity indicates the cumulative action of Heavy metal (Sastry and Agrawal 1979).

Succinate dehydrogenase are the key respiratory enzymes in glycolysis and TCA cycle and their alteration under toxic condition could be considered as an

indication of the fish showing a shift towards anaerobic metabolism resulted in the depressed oxidation of mitochondria (Shobha Rani et. al., 2000). Further the inhibition could also be attributed to the reduction in the mitochondrial number.

In conclusion the above observation during exposure to mercuric nitrate and mercuric acetate indicate a significant alteration in enzyme related to carbohydrate metabolism. The alterations were more marked in mercuric acetate treated fish. However, the fish is able to recover the altered enzyme after transferring them to clean water. The recovery studies are of considerable importance because post-exposure recovery undoubtedly promotes their chances of survival and offers a solution to the aquatic fauna from contaminated area. Recovery studies also suggest that when the pollution is detected in the correct time and when appropriate measures are taken the aquatic fauna may be protected.

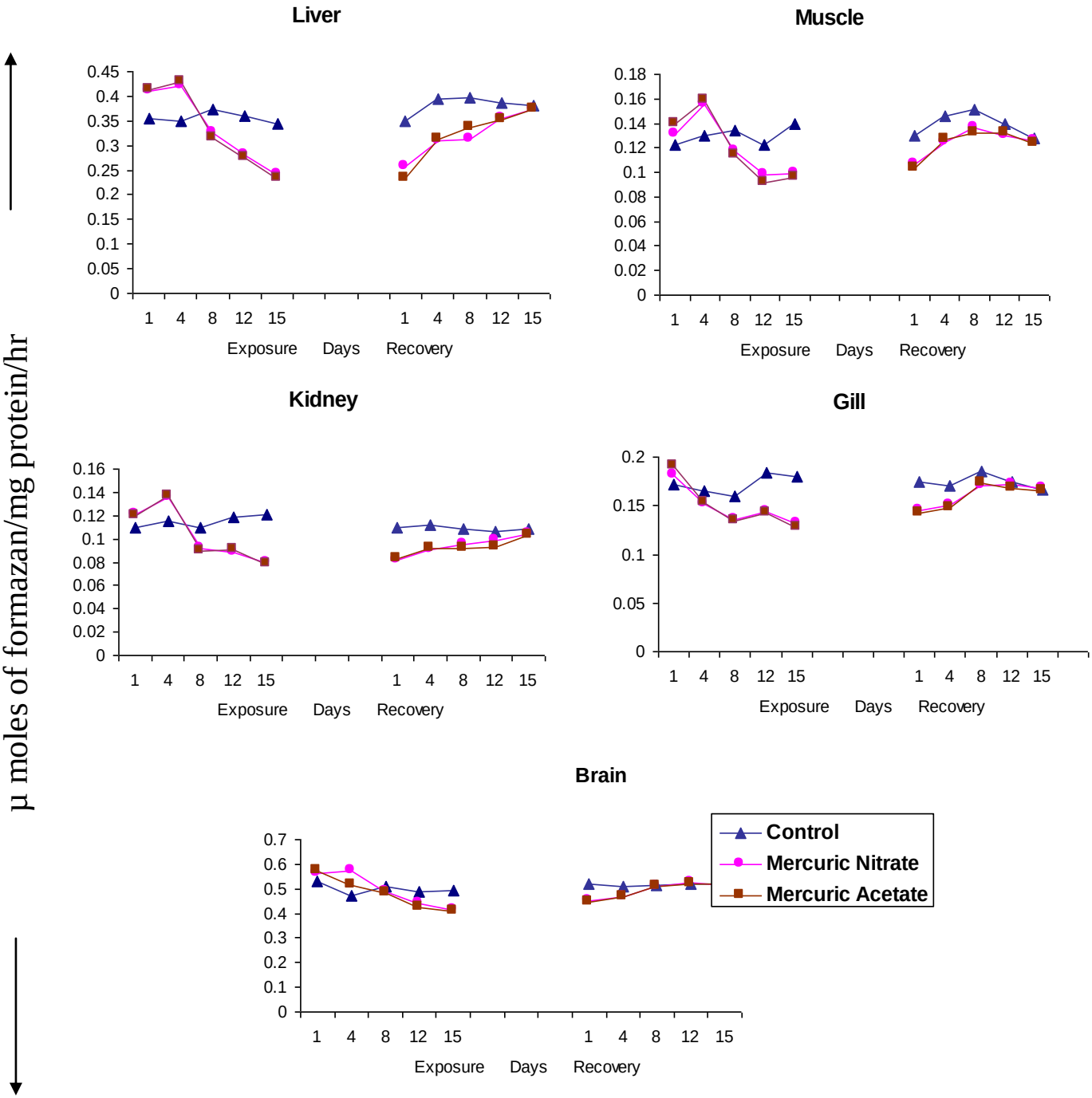

Figure – 1: Succinate dehydrogenase activity in the tissues of *Channa punctatus* after Mercuric nitrate and Mercuric acetate intoxication

ACKNOWLEDGEMENTS

I Thanks to U.G.C for funding MRP,I also thank our Management, Principal, Head Dept of zoology, V.V. College and my Ph.D guide R.P. Dr. Mali of Y.M.N, For there constant encouragement and timely support.

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