Available online at www.pharmaresearchlibrary.com/jpbr

Research Article

Journal of Pharmaceutical and Biological Research
www.pharmaresearchlibrary.com/jpbr
JPBR, 2013: Vol.1(1): 19-21

Effect on the Biomarker of Lebistes reticulates due to sublethal dose of Cadmium

Sabiha Khan*
Department of Zoology
*E-mail: dr.sabihakhan786@gmail.com, Available Online: **27 November 2013**

ABSTRACT

The fish Lebistes reticulatus was exposed to sub-lethal concentration of $9x10^{-3}$ µg/ l cadmium for 30th days (probit method). Autopsy of liver and kidney was done after 30th days and biomarkers SDH, GDH, MDH and LDH levels were estimated by using the method of Nachlas et al. (1960), Srikanthan and Krishnamoorthy (1955), Lee and Lardy(1965) and Burns and Bergmeyer (1965) respectively. A decrease in SDH activity levels is due to accumulation of the succinate. Decreased GDH activity in both tissues is an indication of decreased oxidation of glutamate. Reduction in GDH activity may also be due to low utilization of amino acids and low oxidation of amino acids. Marked drop in MDH levels in both tissues might be due to restricted supply of substrate, since it was observed that SDH activity decreased. Decrease in MDH may be due to diversion of aerobic metabolism at the level of malic acid, leading to the production of pyruvic acid. The elevation in the activity levels of LDH in both tissues indicates an increased production of pyruvate (or) greater breakdown of lactate. Increase in LDH activity might be due to high energy demand for survival in anaerobic conditions and to overcome cadmium stress in polluted environment.

Keywords: Sublethal, Biomarker, Substrate, Cadmium.

INTRODUCTION

Most of industrial effluents often contain pollutants toxic to the biota. Cadmium is released in considerable amounts through industrial effluents into soil, surface and ground water system. Cadmium was found to interfere with many protein and carbohydrate metabolisms by inhibiting the enzymes involved in the processes. Cadmium bioaccumulation can affect humans through biomagnification. Short term tests of acute toxicity were performed over a period of 96 hours using cadmium chloride. The toxicity experiments were then conducted using the chosen concentration of $CdCl_2$ on the fingerlings (Wt. 6±1 grams) in triplicate and the LC_{50} was determined using simple graphic (% Mortality Vs. Log Concentration), probit graphic (Probit value Vs. Log concentration) and unweighted regression analysis methods. Cadmium is a well known heavy metal toxicant with a specific gravity 8.65 times greater than water. Heavy metals become toxic when they are not metabolized by the body and accumulate in the soft tissues. The target organs for cadmium have been identified as liver, placenta, kidneys, lungs, brains and bones. Roberts (1999). Cadmium (Cd), one of the non-essential heavy metals, known for its non-corrosive nature is widely used in paints and dyes, cement and phosphate fertilizers. Jarrup (2003). In the present study the fish Lebistes reticulatus was exposed to sublethal concentration of cadmium for 30 days. To understand the fate of cadmium and its effect on fish, SDH, LDH, MDH and GDH activity levels were estimated in liver and kidney.

MATERIAL AND METHOD

Lebistes reticulatus weighing 0.4±1g, length 2.6±3.0 cm. were collected from local ponds and acclimatized to laboratory conditions. The sub-lethal concentration was determined through probit analysis. The fish were exposed to $9x10^{-3}$ μgl^{-1} concentration for 30 days. Constant temperature (28 ± 1^0 C) and pH (6.5) were maintained throughout the period of experimentation. The other physicochemical parameters maintained throughout the period of experimentation were Dissolved oxygen 5.8±0.2 mgl^{-1}, Alkalinity 46 mgl^{-1} as $CaCo_3$, Chlorinity 2.6 mgl^{-1}, Co_2 1.4 mgl^{-1}. The fish were fed with fish food. Two glass aquaria containing ten fishes in each. One served as control and other as experimental. After 30 days autopsy of liver and kidney was done from both control and experimental group

and homogenate was used for the estimation of SDH levels (Nachlas et al. 1960), GDH levels (Srikanthan and Krishnamoorthy, 1955), MDH levels (Lee and Lardy, 1965) and LDH levels (Burns and Bergmeyer, 1965).

OBSERVATION:

Table: 1 Changes in the activity levels of SDH, LDH, MDH and GDH in the Liver of fish Lebistes reticulatus exposed to concentration of $9x10^{-3}$ µg/ l Cadmium for 30 days.

Values are expressed as µ moles formazan formed, mg $protein^{-1}$ $hour^{-1}$

Name of the Tissue	SDH		LDH		MDH		GDH	
Liver	**C**	**E**	**C**	**E**	**C**	**E**	**C**	**E**
SD	0.650± 0.031	0.542±0.02 43	0.581±0.02 2	0.957±0.078	0.354±0.012	0.239±0.01 1	1.548±0.0 69	0.621±0.0 27
t-Test		P<0.001		P<0.001		P<0.001		P<0.001
% Change		(-20.5)		(+60.2)		(-32.8)		(-60.7)

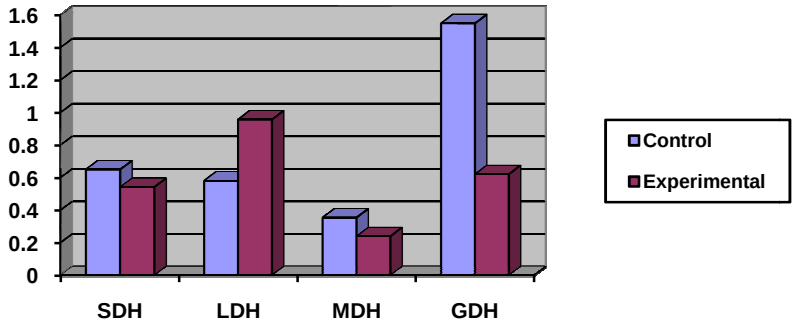

Fig: Changes in the activity levels of SDH, LDH, MDH and GDH in the Liver of fish Lebistes reticulatus exposed to concentration of $9x10^{-3}$ µg/ l Cadmium for 30 days.

Table: 2 Changes in the activity levels of SDH, LDH, MDH and GDH in the Kidney of fish Lebistes reticulatus exposed to concentration of $9x10^{-3}$ µg/ l Cadmium for 30 days.

Values are expressed as µ moles formazan formed, mg $protein^{-1}$ $hour^{-1}$

Name of the Tissue	SDH		LDH		MDH		GDH	
Kidney	**C**	**E**	**C**	**E**	**C**	**E**	**C**	**E**
SD	1.82±0.0 91	0.957±0.0 78	0.341±0.0 10	0.547±0.01 5	0.079±0.0 36	0.697±0.0 26	0.576±0. 27	0.415±0.0 123
t-Test		P<0.001		P<0.001		P<0.001		P<0.001
% Change		(-48.6)		(+56.5)		(-35.6)		(-29.5)

Each value is mean ± SD of six observations. C= Control; E= Experimental

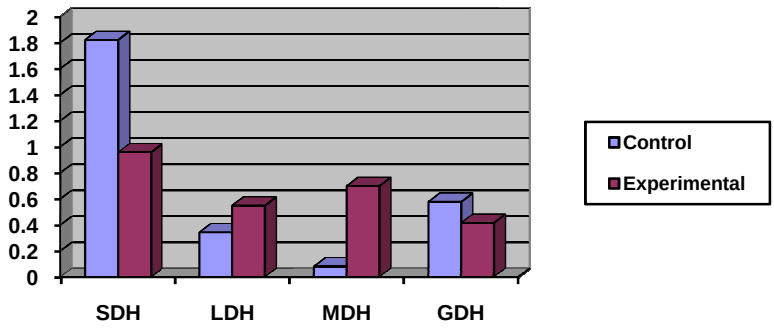

Fig: Changes in the activity levels of SDH, LDH, MDH and GDH in the Kidney of fish Lebistes reticulatus exposed to concentration of $8x10^{-3}$ µg/ l Cadmium for 30 days.
Values are significant at P<0.001
Values in the parentheses indicate % stimulation

RESULT AND DISCUSSION

A decrease in SDH, MDH, GDH and increase in LDH levels was observed in both tissues for 30 days. The decrement in SDH activity levels in liver and kidney might be due to accumulation of the succinate. The Krebs cycle fragment of energy metabolism might be significantly dearranged under cadmium stress. The elevation in the activity levels of LDH in both tissues indicates an increased production of pyruvate (or) greater breakdown of lactate. Marked drop in MDH levels in both tissues might be due to restricted supply of substrate, since it was observed that SDH activity decreased. Decrease in MDH level may be the diversion of aerobic metabolism at the level of malic acid, leading to the production of pyruvic acid. Decreased GDH activity in both tissues is an indication of decreased oxidation of glutamate. Reduction in GDH activity may also be due to low utilization of amino acids and low oxidation of amino acids. Thus stressed fish appears to meet the energy demand through anaerobic oxidation as indicated by the elevated LDH and decreased SDH, MDH, GDH activities.

The present finding is similar with a finding of other scientist. Lactate dehydrogenase catalyse the reversible oxidation of L. lactate to pyruvate. It is also capable of oxidizing a number of α hydroxy acid. The enzyme has been found to be very sensitive to cadmium exposure. The higher level of LDH may be due to elevated rate of enzyme synthesis it is probably reflecting enhanced rate of gluconeogenesis. Similar results have been reported in various studies on pesticide exposure. Story and Freedland (1979). Tilak et al. (2003) reported a decrease in pyruvate levels and increase in lactate levels in Channa punctatus (Bloch) exposed to sublethal and lethal concentrations of fenvalerate. This can be attributed to toxic stress resulting in the inhibition of pyruvate oxidation under hypoxic conditions which indicates the shifting of aerobic respiration to anaerobic respiration. LDH level increase in the tissues due to stress when exposed to toxicant. Lee et al. (2004) expressed that the alachlor apparently interferes with physiological processes including biosynthesis of lipids, proteins and flavanoids. It also causes hepato-toxicity, tumour formation in animals. Similar observations were also reported by USEPA (1986). Khan and Jain (2010) reported significant reduction in glycogen level of fish Lebistes reticulatus when exposed to higher and lower sub-lethal concentration of cadmium chloride for 15, 30 and 45 days.

REFERENCES

1. Bruns, F.H. and Bergmeyer, H.V., 1965. Methods of enzymatic analysis, Academic press, New York, London, pp 724-738.
2. Jarrup, L., 2003, Hazards of heavy metal contamination. *Br Med Bull*, vol. 68, pp 167-182.
3. Khan. S and Jain, S., 2010, Statistical Effect of toxicity of Cadmium Chloride on glycogen content in *Lebistes reticulatus*, *International Journal of Physical Science*, vol. 22(1), pp 25-30.
4. Lee, C., Jisung, R., Young, P.S., Kyunghee, C., Hwan, J.S., Gyun, N.J. and Gil, R.D., 2004, Effects of alachlor on the early development and induction of estrogen-responsive genes in medaka, Oryzias latipes, *Dev. Repro. Toxicity*, vol. 66, pp 2993-2999.
5. Lee, Y.L. and Lardy, H.A., 1965. *J. Biol. Chem.,* vol. 240, pp 1427-1430.
6. Nachlas, M.N., Margulies, S.I. and Saligman, A.M., 1960. *J. Biol. Chem.* vol. 235, pp 2731-2743.
7. Roberts, J.R., 1999, Metal toxicity in Children. Training Manual on Pediatric Environmental Health: Putting It into Practice. Emeryville, CA, *Children's Environmental Health Network.*
8. Srikanthan, T.N. and Krishnamoorthy,C.R., 1955. *J. Sci. Indust. Res.* Vol. 14, pp 206-207.
9. Story, D.L. and Freedland, R.A., 1979, The effect of DDT feeding on gluconeogenesis in isolated hepatocytes from starved rats, *Journal of Toxicology Appl. Pharmacol.*, vol. 43, pp 547-557.
10. Tilak, K.S., Satyavardhan, K. and Thathaji, P.B., 2003, Biochemical changes induced by fenvalerate in the fresh water fish, *Channa punctatus*. *J. Ecotoxicol. Environ. Monit.*, vol. 13, pp 261-270.
11. USEPA: 1986, Alachlor, Special review technical document. Position Document 2/ 3. Office of Pesticides and Toxic Substances, U.S. Environmental Protection Agency, Washington, DC.