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NA^+ K^+ ATPase post exposure recovery from Lead intoxicated freshwater fish *Anabas testudineus*.

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Abstract
NA^+ K^+ ATPase are important amongst the several molecules available in the cells, Carbohydrates play an important role in the cellular process Under extreme stress conditions, carbohydrate membrane bound enzyme such as NA^+ K^+ ATPase have been known to act as the energy supplier in metabolic pathways and biochemical reactions. In the present investigation fish treated with an equitoxic dose of 10 ppm of lead nitrate and lead acetate intoxicated fish After a period of 15 days of exposure a batch from lead nitrate exposed fish and a batch from lead acetate exposed fish were transferred to lead-free water. Fishes were scarified on 1, 4, 8, 12 and 15 days for the analysis of of recovery pattern in tissues viz. liver, muscle, kidney, gill and brain .It is found that lead toxicated fishes were recovered after 15 days depends upon physical condition of the fish.

Keywords: Carbohydrate, lead, *Anabas.*

INTRODUCTION

The modern industries are making use of various heavy metals such as iron, steel, copper, nickel, platinum and lead. Among the different types of pollutions, chemical pollution appears to be the major type which threatens the living systems very extensively. Among the different habitats aquatic environment is the major target of pollution. Most of the heavy metals are natural constituents of the aquatic environment. Some of them are biologically essential, but some metals like cadmium, lead and mercury are highly hazardous to aquatic biota and normally occur in low concentration [11]. It is clearly known the common forms of lead poisoning result from the mining, processing and commercial dissemination of lead [7].The primary source of lead exposure to animals are contaminated soils, lead paints that remain on older structures, water from plumbing systems that contain lead, and lead based products, especially batteries, used crankcase oil, and linoleum [16]. The lead containing gasoline fumes from automobile exhausts constitute the chief and wide spread source of lead contamination in urban environments. A major source of lead to waterfowl and other wildlife is spent lead shot, bullets, cartridge, and lead sinkers used in sport fishing [4].

MATERIALS AND METHODS

Anabas testudineus which is selected as test species in the typical representative of Anabantoid fishes in South India. It is fresh water, euryhaline and eurythermal teleost. Biochemical assays were made in different tissues from both experimental (exposed to toxicant) and Normal (toxicant free) fishes. Fish approximately of same size and weight were selected and grouped into 6 batches. 2 batch of fish served as controls, 2 batches of fish were exposed to lead nitrate and the remaining two batches were exposed to lead acetate for a period of 15 days. After a period of 15 days of exposure a batch from lead nitrate exposed fish and a batch from lead acetate exposed fish were transferred to lead-free water and scarified at the same intervals to observe the recovery responses. In all the experiments, a minimum of six individual observations were made. The values of different parameters were expressed as mean with their standard error. Significance of the values obtained were tested using student 't' test. The NA^+ K^+ ATPase content in the tissues were estimated by the method of Kaplay [10].

RESULTS AND DISCUSSSION

The activity of Na^+-K^+ ATPase was progressively decreased in all the tissues throughout the exposure period. On the 1st day of exposure except brain all tissues showed significant inhibition. Though there was inhibition in brain, it was insignificant (-2.08 lead nitrate -4.17% lead acetate). Kidney exhibited maximum inhibition (-5.6% lead nitrate $P < 0.05$; -8.8% lead acetate $P < 0.01$) followed by muscle (-3.15% for lead nitrate $P < 0.05$, -5.91% for lead acetate, $P < 0.01$), gill (-2.63% for lead nitrate and -5.26%; lead acetate $P < 0.05$) and liver (-4.89% lead nitrate; -5.08 lead acetate; $P < 0.001$).

On the 4th day of exposure, the inhibition in Na^+ K^+ AT Pase activity was noticed in all the tissues. Maximum inhibition was noticed in kidney (-15.38% for lead nitrate, -16.92% for lead acetate $P < 0.01$) and minimum inhibition was noticed in brain (-5.76% for lead nitrate $P < 0.05$; -10.07% for lead acetate $P < 0.01$). The percent depletion ranged from -5.76% to -15.38% for lead nitrate and -10.07% to -16.92% for lead acetate.

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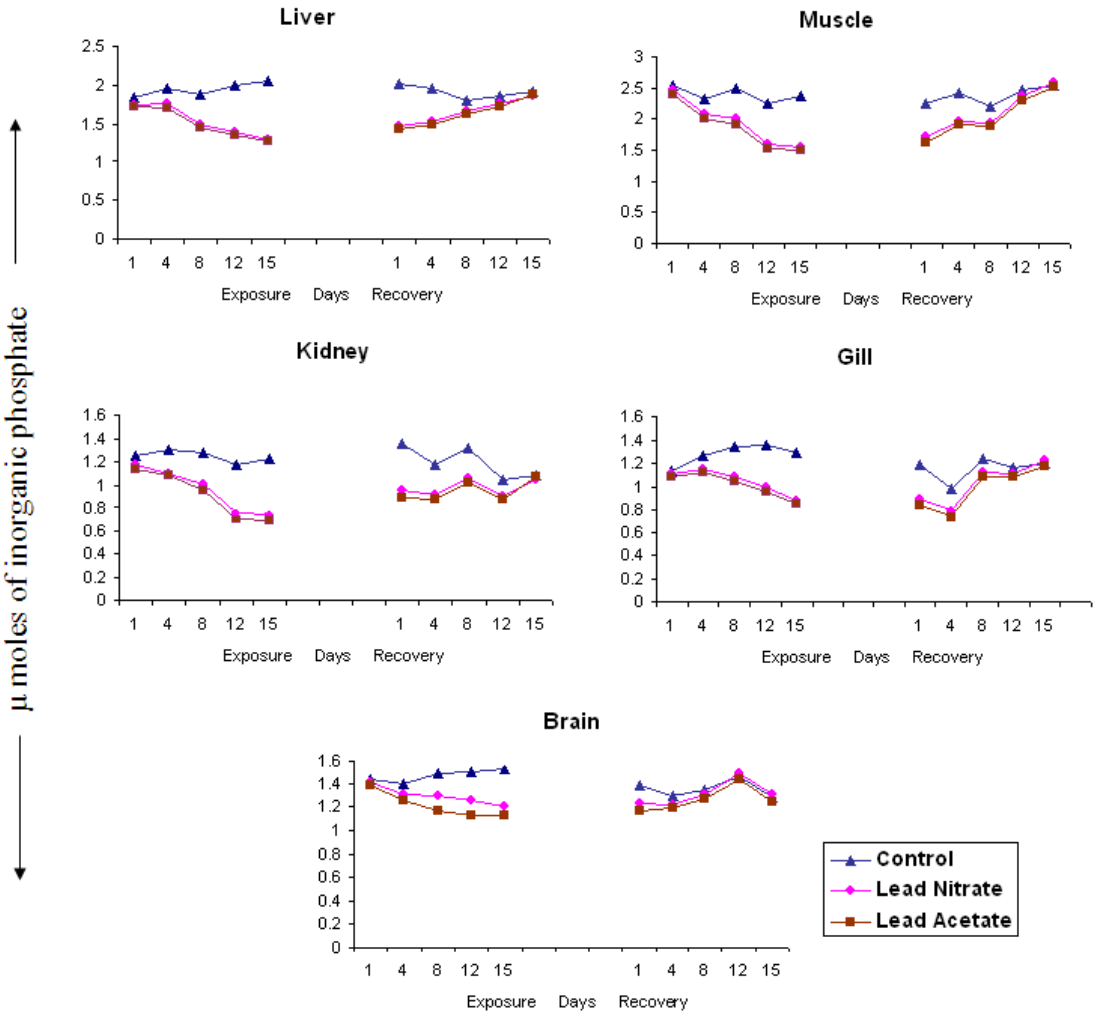

Fig 1. Activity of Na+ k+ ATPase in the tissues of *Anabas testudineus* during exposure and recovery period after Lead nitrate and Lead acetate intoxication.

On 8th day of exposure similar trend in response was noticed in all the tissues. In kidney Na^+-K^+ ATPase activity was dropped maximum (-21.09% lead nitrate, -25.00% lead acetate; $P < 0.001$) followed by liver (-20.74%, lead nitrate, $P < 0.01$, -22.34% lead acetate $P < 0.001$), muscle(-19.35% lead nitrate, -22.58% lead acetate, $P< 0.001$), gill (-18.66% lead nitrate $P < 0.01$, -21.64% lead acetate $P < 0.001$) and brain (-12.84% lead nitrate $P < 0.01$ -21.62% lead acetate $P < 0.001$).

On 12th day of exposure similar response was noticed with higher magnitude of inhibition in activity. The percent variation over control was from -16.00% -40.68% kidney exhibited maximum drop (-36.44% for lead nitrate, -40.68% for lead acetate $P < 0.001$) and brain exhibited minimum depletion (-16.00% for lead nitrate, $P < 0.01$ and -25.33% for lead acetate $P < 0.001$).

On 15th day of exposure minimum inhibition in the activity was recorded in the kidney (-40.16% lead nitrate, -43.44% lead acetate, $P < 0.001$) followed by liver(-36.59% lead nitrate, -37.56% lead acetate, $P<0.001$), muscle (-33.90% lead nitrate,-36.02% lead acetate, $P< 0.001$), gill (-31.78% lead nitrate, -34.11% lead acetate,$P <0.001$) and brain(-21.05% lead nitrate: -26.32% lead acetate $P < 0.001$).

During the recovery period the inhibitory trend in the Na^+ - K^+ ATPase activity was maintained. However, the inhibitory trend was gradually reduced during later stages recovery periods. Liver tissue exhibited near normal activity with insignificant variation (-3.13% lead nitrate, -2.08% lead acetate), on 15th day of recovery period. Even kidney tissue recovered on 15th day (-3.67% lead nitrate, -1.83% lead acetate) with insignificant variation. Muscle from lead nitrate exposed fish showed near normal levels on 12th day, and lead acetate exposed on 15th day. Gill tissue witnessed recovery on 12th day with insignificant variation (-4.31% lead nitrate, -6.9% lead acetate). Brain attained near control levels of activity on 8th day (-2.96% lead nitrate, -5.93% lead acetate) (Fig.1).

ATPases are the enzymes concerned with the immediate release of energy useful for the maintenance for physiological functions. ATPases are also involved in the intracellular ionic regulation and also help in the osmoregulation of the whole animal. These enzymes are very sensitive to trace metal toxicity [8]. Na^+ K^+ ATPases is a biochemical expression of the active transport of Na & K of the cells [14]. It was present in all most all the cell membranes that carry some type of active transport, [13] & known to help in maintaining the intracellular ionic gradient and transport of organic molecules of low molecular weight [5]. Ethanol was found to enhance the toxic effects of lead in terms of decreased cellular energy reserves (ATP levels), Co-exposure to lead and ethanol caused marked decline in the rate of mitochondrial respiration as compared

to lead alone. Na^+ K^+ ATPase in the present study was found inhibited in all the tissues throughout the exposure period. The inhibition was found progressive and tissue specific. The maximum inhibition was noticed in the kidney followed by liver, indicting the nephrotoxic and hepatotoxic nature of lead. The maximum inhibition in these two organs could be attributed to its concentration particularly through enterohepatic circulation in liver and through glomerular filtration in kidney. Lead is known to accumulate more in liver and Kidney causing the inhibition in the enzyme activity [1].

The lowest amount of inhibition was recorded in the brain probably due to the limiting lead ions into the brain by the presence of blood brain barrier. The progressive inhibition of Na^+ -K^+ ATPase activity indicates the persistent and cumulative nature of lead [12,15]. Inhibition of Na^+ K^+ ATPase was recorded by many workers during lead toxicosis in mammalian models [3,6, 9] and in fishes during heavy metal toxicity ([2,12,15].

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REFERENCES

[1] Agrahari, Shweta and Gopal Krishna, 2007. Fate and toxicity of cadmium and lead accumulation in different tissues (gills, liver, kidney, brain) of a freshwater fish *Channa punctatus .J. of Ecophysio & Occupl Hlth* .7, (3 & 4) 151-155.

[2] Arpan, Kumar Maiti, Nimai Chandra Saha and Goutam Paul. 2010. Effect of Lead on Oxidative Stress, Na+K+ATPase Activity and Mitochondrial Electron Transport Chain Activity of the Brain of *Clarias batrachus. Bull Environ Contam Toxicol* 84 (6) :672–676

[3] Bennet, C, Bettaiya R, Rajanna S, Baker L, Yallapragada PR, Brice J. J., White S.L., Bokara K. K. 2007. Region specific increase in the antioxidant enzymes and lipid peroxidation products in the brain of rats exposed to lead. *Free Radic Res* 41(3) 267–273

[4] De, Francisco N, J.D.Ruiz Troya, and E.I. aguera 2003. Lead and lead toxicity in domestic and free living birds. *Avian pathol,* 32:3-13.

[5] Degani, G. and Warburg, M. R. 1984. Changes in concentrtions of ions and urea in both plasma and muscle tissue in a dehydrated Hylid anura. *Comp. Biochem. Physiol.* 77A : 357-360

[6] Flora, G. J. and Seth P. K. 2000. Alterations in some membrane properties in rat brain following exposure to lead. *Cytobios* 103:103–109

[7] Hammond P. B. 1969. Lead poisoning. "An old problem with a new dimension. In: Blood FR (ed) Essays in Toxicology. *Academic Press New york*. (1): 115-155

[8] Haya, K., Waiwood B. A. 1983. Adenylate energy chargeand ATPase activity. Potential biochemical indicators of sublethsal effects caused by pollutants in aquatic animals in : Nriagu Jo (Ed) *Aquatic Toxicology,* Vol. 13, John Wiley and Sons, New York p.525

[9] Kanwar, K.C. and Shakti Sharma 1987. Fluctuations in serum proteinsand enzymes in mouse following oral lead administration. *Res. Bull. Punjab. Univ. Sci.,* 37 (3/4) 99-104.

[10] Kaplay, S. S. 1978. Erythrocyte membrane Na+ -K+ activated ATPases in protein caloric malnutrition. *Am. J. Clin. Nutr.,* 31: pp.579-584.

[11] Mali, R. P. 2002. Studies on some aspects of physiology of freshwater female crab Brytelphusa guerini with special reference to inorganic pollutants. P.hD Thesis submitted to Swami Ramanand Teerth Marathwada ,University,Nanded ,Maharashtra.

[12] Rogers, J.T., Richards J.G., Wood C.M. 2003. Ionoregulatory disruption as the acute toxic mechanism for lead in the rainbow trout *Oncorhynchus mykiss*. *Aquat Toxicol.* 64:215–234

[13] Schwartz, A., Lindenmayer, G.E., Allen, J.C. 1972. The Na+ K+ ATPase membrane transport system. Importance in cellular function. In: Current topics in membranes and transport. Bonnes, F. and Kleinzea, A. New York, Acad. Press. Pp 1-82.

[14] Skou, J.C. 1975. The (Na+ +K+ activate enzyme system and its relationship to transport of sodium and potassium Q. *Rev. Biophys*. 7 : 401-434.

[15] Spokas, E.G., Crivellone M.D., Kemp F, Bogden J.D., Cohen G.M. 2002.Characterization of Sodium, Potassium, ATPase Activity in the Gills of Pimephales promelas (Fathead Minnow): influence of In Vitro Exposure to Lead. *Bull Environ Contam Toxicol* 69:384–392

[16] Waldner, C.S, Checkley, B, Blakley, C, Pollock, and Mitchell V. 2002. Managing lead exposure and toxicity in cow-calf herds to minimize the potential for food residues. *J. Vet. Diagn. Invest.*14:481-486