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Research Paper

Assessment of toxic effect of fertilizer muriate of potash on SGPT levels of teleost *Clarias batrachus*

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Abstract

The fertilizer Murate of Potash elevated the levels of SGPT in fish *Clarias batrachus* at all concentrations & exposures but most lethal effect of murate of Potash is seen at 6.05g/L at 96 hours of exposure and worst at 7.10 g/L at 48 hours of exposure.

Keywords: Muriate of potash, Fertilizer, *Clarias batrachus*, SGPT

Introduction

Glutamate pyruvate transaminase is an active aminotransferase enzyme widely distributed in almost all tissues of animals. It catalyses the reaction of L-oxoglutarate and L-alanine resulting in the formation of L-glutamate and pyruvate, which is a reversible mechanism[1].Significantly elevated levels of SGPT often suggest the existence of other medical problems such as viral hepatitis, diabetes, congestive heart failure (Myocardial infarction), liver damage, bile duct problems, infectious mononucleosis, or myopathy, so SGPT is commonly used as a way of screening for liver problems[2,3]. Elevated SGPT may also be caused by dietary choline deficiency. However, elevated levels of SGPT do not automatically mean that medical problems exist. Fluctuation of SGPT levels is normal over the course of the day, and they can also increase in response to strenuous physical exercise. The two transaminases commonly measured are alanine transaminase (ALT) and aspartate transaminase (AST). These levels are also called as Serum Glutamate-Pyruvate Transaminase (SGPT) and Serum Glutamate-Oxaloacetate Transaminase (SGOT). Measurement of ALT and AST were used in diagnosing heart attacks, although they have been replaced by newer enzyme and protein tests that are more specific for cardiac damage. The effects of fertilizer muriate of potash on SGPT activity of teleost *C. batrachus* were observed, and results are given in this communication.

Materials and Methods

Live and healthy fishes collected from river Gomti at Lucknow, were transported to the laboratory in plastic containers in natural water. They were treated with 2.5% $KMnO_4$ to remove external infections, the fishes were: allowed to rest for 48 hours, to bring them to their normal, mental and physiological conditions after stress and strain of catch transport. They were also watched for 72 hours for any mortality (even upto 2%) during these tests against diseases etc., and the groups with good mortality rates were rejected. Static Bioassay tests were followed[4].

The fishes were exposed to six different concentration of the fertilizer muriate of potash (5.50-8.65 g/L) found lethal in 24 to 144 hours (5 and 6). After the required interval of exposure, the fishes were taken out of the aquaria, blotted dry with the help of clean turkish towel. Blood was collected from caudal vein in dry test-tube and allowed to clot. Soon after, the contents of the test tube were centrifuged at 3000 rpm and clean serum was transferred to another dry test-tube, and stored in

refrigerator at 0°C. SGPT estimations were done according to the method of Reitman and Frankel[7]. Optical density was determined by Spekol Spectrophotometer at 520 nm.

Results and Discussions

The results obtained on SGPT levels of *C. batrachus*, exposed for 24 to 144 hours, to six concentrations of muriate of potash, are summarised in Table 1.

Table 1: Effect of fertilizer Muriate of Potash on SGPT levels of *C. batrachus*

Fertilizer Concentration g/L	SGPT µ moles pyruvate formed/ml/hour Mean ± S.D. Range in Parentheses Exposure Times in Hours					
	24	48	72	96	120	144
			Control value	3.07±0.19 2.90-3.28		
5.50	3.85±0.26 3.66-4.04	3.71±0.26 3.52-3.90	3.18±0.54 2.80-3.57	3.72±0.58 3.09-4.23	3.95±0.38 3.57-4.23	3.66±0.31 3.33-3.95
6.05	3.39±0.49 2.52-3.66	3.56±0.54 2.80-3.95	4.09±0.53 3.71-4.47	4.20± .58 3.57-4.71	3.76±0.26 3.57-3.85	
6.60	3.36±0.19 3.14-3.52	3.71±0.49 3.14-4.28	3.51±0.64 2.85-4.37	3.99±0.49 3.42-4.57		
7.10	3.52±0.53 3.14-3.80	4.33±0.38 3.95-4.33	3.38±0.48 3.04-2.72			
7.70	3.42±0.38 3.04-3.80	3.85±0.26 3.66-4.04				
8.65	3.33±0.26 3.14-3.52	No. of observation 16 in each case				

Maximum rise of 42.14% above control occurred at 7.10g/L concentration after exposure of 48 hours, while minimum 3.82% was observed at lowest concentration of 5.50 g/L after 72 hours. All the values, except that obtained after 72 hours at 5.50 g/L concentration, were statistically significant ($P < 0.05$).

When elevated ALT levels are found in the blood, the possible underlying causes can be further narrowed down by measuring other enzymes. For example, elevated ALT levels due to heaptocyte damage can be distinguished from bile duct problems by measuring alkaline phosphatise[9]. Also, myopathy-related elevations in ALT should be suspected when the aspartate transaminase (AST) is greater than ALT, the possibility of muscle disease causing elevations in liver tests can be further explored by measuring muscle enzymes, including creatine kinase.

While reviewing the regulation of serum and tissue phosphate levels discussed the prophylaxis and therapy of phosphate depletion[10]. Higher levels of urate, glucose, LDH and cholesterol were positively associated with weight of humans, while Na, K, Cl, urea and alkaline phosphatase were not weight dependent[11].

Muriate of potash elevated SGPT levels of the fish *C.batrachus* at all concentrations and exposures. Alkaline phosphates, acid phosphates and lipase activities were inhibited by molybdenum in rats[12].Under different stress; significant hepatotoxicity was observed[13-20].

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References

1. Varley H., Practical Clinical Biochemistry, Arnold Heinemann Pub., India **(1975)**

2. Wroblewski F., Advances in Clinical Chemistry, Vol. I. (Eds. Sobotka, H. and Stewart, C. P.) Academic Press, New York **(1958)**

3. Karmen A., Wroblewski F. and Ladue J.S., Transaminase activity in human blood, The Journal of Clinical Investigation, 34(1): 126–31 **(1955)**

4. APHA, Safe Methods for the Examination of Water and Waste Waters, 14th Ed. Amer. Pub. Health Assoc., New York **(1976)**

5. Singh R.K., Ecophysiological Studies on Some Fresh Water Fishes, Ph. D. Thesis, University of Lucknow, Lucknow **(1982)**

6. Naqvi M.S., Effect of Environment Pollution on Physiology of Fresh Water Fishes. Ph.D. thesis, University of Lucknow **(1983)**

7. Reitman S. and Frankel S., Determination of SGPT activity in serum, Amer. J. Clin. Path., 28: 56 **(1957)**

8. Ghouri N. Preiss and D. Sattar N., Liver enzymes, nonalcoholic fatty liver disease, and incident cardiovascular disease : a narrative review and clinical perspective of prospective data, Hepatology, 52(3): 1156–61 **(2010)**

9. Kreusser W., Ritz E. and Boland R., Phosphate depletion. Khin. Wochenschr, 58: 1-15 **(1980)**

10. Munan L., Kelly A., Petitclere C. and Billon B., Associations with Body Weight of Selected Chemical Constituents in Blood, Clin. chem., 24: 772-777 **(1978)**

11. Rana S.V.S. and Kumar A., Lipid deposition in the liver and kidney of rats due to toxicity of molybdelum, Toxicol. Lett., 6: 163-166 **(1980)**

12. Kabir M.A. and OvieKori-S., Effect of sub lethal concentrations of Tobacco (*Nicotiana tobaccum*) leaf dust on some biochemical parameters of Hybrid catfish (*Clarias gariepinus* and *Heterobranchus bidorsalis*), Braz. Arch. Biol. Technol., 54(1): 58-62 **(2011)**

13. Singh R.N., Pandey R.K., Singh N.N. and Das V.K., Acute Toxicity and Behavioural Responses of Common Carp *Cyprinus carpio* (Linn.) to an Organophosphate (Dimethoate), World J. Zool., 4(2): 70-75 **(2009)**

14. Randall D.J. and Tsui T.K.N., Ammonia toxicity in fish, Marine pollution Bull., 45: 17-23 **(2002)**

15. Yadav A., Neraliya S. and Gopesh A., Acute toxicity levels and ethological responses of *Channa striatus* to fertilizer industrial wastewater, J. Environ. Biol., 28(2): 159-162 **(2007)**

16. Ganga rao B., Jaya raju N., Investigation of hepatoprotective activity of *Spondias pinnata*, Int. J. of Pharma Sc. and Res., I(3): 193-198 **(2010)**

17. Pinar T., Atli A.K. and Alacam H., The effect of noise on oxidative and antioxidative balance in human erythrocytes, Inter. J. Hematol, Oncol., 21(1): 27-37 **(2011)**

18. Altnok I. and Capjub E., Histopathology of rainbow trout exposed to sub lethal concentrations of methiocrab or endosulfan, Toxicol. Pathol., 35: 405-410 **(2007)**

19. Chukwu L.O. and Okpe H.A., Differential responses of *Tilapia guinensis* fingerlings in organic fertilizer under various salinity regimes, J. Environ. Bio., 27: 687-690 **(2006)**

20. Erdogan O., Hisar O., Koroglu G. and Iltas A.C., Sub lethal ammonia and urea concentrations inhibit rainbow trout *Oncorhynchus mykiss* erythrocyte glucose-6-phosphate dehydrogenase, Comparative Biochem. & Physiol., 141:145-150 **(2005)**