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Research Article

BIOCHEMICAL RESPONSE OF HEAVY METAL, SODIUM ARSENATE EXPOSURE IN CATFISH, *CLARIAS BATRACHUS*

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ABSTRACT

The aim of present investigation was to determine the effect of sodium arsenate on biochemical parameter of catfish, *Clarias batrachus*. Biochemical parameter serum glutamate oxaloacetate transaminase activity (SGOT) and serum glutamate pyruvate transaminase activity (SGPT) was increased as compared to control value at 24 hours interval due to effect of sodium arsenate. The alteration of this parameter can be effectively used for evaluate health of catfish exposed to sodium arsenate in the aquatic environment.

INTRODUCTION

Heavy metals are widely used in various industries and considered as common water pollutants. When the amount of heavy metals in a medium, reach to more than a certain limit, it becomes toxic for those animals that live in the environment. Low concentrations of some heavy metals are essential for aquatic animals. However, at high concentration levels, they accumulate in different organs, damage tissues and interfere with the normal growth and proliferation (Asati *et al.,* 2016; Alkarkhi *et al.,* 2009).

Arsenic toxicity depends upon its chemical form and oxidation states. It exists mainly in 4 oxidation states-arsenate (As +5), arsenite (As +3), arsenic (As 0) and arsine (As -3) (Sharma and Sohan, 2009). This element has long been associated with criminal activity and still is an emotionally highly charged topic, as large doses can cause acute poisoning and death. Ingestion of low dose via food or water is the main pathway of this metalloid into the organism, where absorption takes place in the stomach and intestines, followed by release into the bloodstream. In chronic poisoning, arsenic is then converted by the liver to a less toxic form, from where it is eventually largely excreted in the excretion. Only very high exposure can, in fact, lead to appreciable accumulation in the body. Minor alternative pathways of entry are known through skin exposure (Chakraborty *et al.,* 1987). In aquatic environments, arsenic founds as a mixture of arsenate and arsenite, with arsenate usually predominating (Kumari *et al.,* 2017).

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The toxicity of arsenic in animals depends on species, sex, age, exposure dose, exposure time, valence state, organic or inorganic form, etc. (Allen *et al.,* 2004). In natural water, arsenic mostly exists in inorganic and organic form (Luh *et al.,* 1973) and the inorganic form has been found to be more toxic. Among the various arsenical compounds, sodium arsenate (Na_2HAsO_4), arsenic trioxide (As_2O_3), arsenic pentaoxide (As_2O_5) etc. are mostly used in the synthesis of various inorganic and organic compounds and in agricultural chemicals. It is also used as a chemotherapeutic agent for the treatment of haematological malignancies (Liao *et al.,* 2004).

Transaminases like aspartate aminotransferase (AST) and alanine aminotransferase (ALT) may be used to find tissue disorders or tissue damages caused by the contaminants or toxicants and also used for intake water monitoring. AST acts by catalyzing the shifting of amino group of aspartic acid to α–ketoglutaric acid to form oxaloacetic acid and glutamic acid, whereas ALT roles by catalyzing the transfer of amino group from alanine to α – ketoglutaric acid to form pyruvic acid and glutamic acid. All these enzymes are the strategically associate between carbohydrate and protein metabolism and plays main role in the uses of amino acids for the gluconeogenesis and/or for oxidation (Rodwell, 1988 and Nemcsok and Benedecky, 1990).

Under stable environmental situations, susceptibility of living beings to toxicant varies because of intrinsic factors (species, gender, age, development, maturation). Metabolic enzymes like alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are found mainly in the liver, but also found in muscle, red blood cells, heart and kidneys. Aspartate aminotransferase or alanine aminotransferase levels are a

constructive and valuable support predominantly in the study and analysis of liver disease (Huang, 2006).

MATERIAL AND METHODS

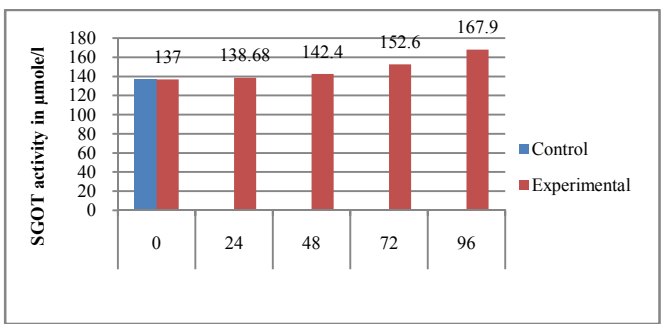
The catfish *Clarias batrachus* were used as an experimental animal and it was collected from local fish market of Indore and acclimatized in the laboratory for one week. The analytical grade sodium arsenate ($Na_2HAsO_4 \cdot 7H_2O$) (CAS No.: 10048-95-2) (Heptahydrate) was ordered from Spectrum chemical mfg. corp., Mumbai, India and used without further purification for the experiment. To determine the lethal concentration (LC_{50}) of sodium arsenate, catfish (*Clarias batrachus*) were selected from the stock and exposed to different concentrations of sodium arsenate in different tanks. Ten fish were kept in each tank and water was replaced daily with fresh sodium arsenate mixed water to maintain a constant level of sodium arsenate during the exposure period. The mortality or survival of fish was observed at the end of 24 hours and the concentration at which 50% mortality of fish occurred was taken as the lethal concentration (LC_{50}) (Kumari *et al.,* 2017). The blood collected by disposable syringe and needles from cardiac puncture of *Clarias batrachus* and kept in sterilized appropriate vials then processed for various biocheical analyses (Dacie and Lewis, 1975). In the present investigation experimental fishes were divided into two groups control and sodium arsenate treated group. Ten (10) fishes were kept in control group and exposed to normal water and in experimental group forty (40) fishes were exposed to concentration of sodium arsenate at different time intervals. In both control and experimental group fishes were exposed to maximum 96 hours. The activity of serum glutamate oxaloacetate transaminase (SGOT) and serum glutamate pyruvate transaminase (SGPT) were determined by adopting the method of Boyer (2000).

RESULTS

In the present investigation biochemical estimation of control and sodium arsenate (LC_{50} value- 42 mg/l) treated fishes were completed. The biochemical parameters were SGOT (serum glutamate oxaloacetate transaminase) and SGPT (serum glutamate pyruvate transaminase) of control fish were 137.00 µmole/l and 42.42 µmole/l respectively.

Serum Glutamate Oxaloacetate Transaminase Activity (SGOT) of Sodium Arsenate Treated Group

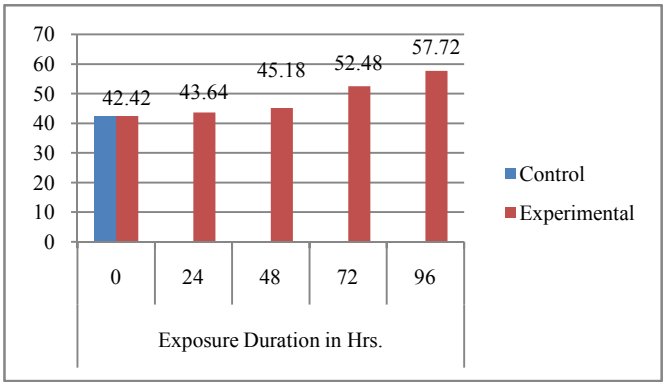
The SGOT activity was found increased as the duration of exposure of sodium arsenate increased. The increased SGOT activity after 24, 48, 72 and 96 hrs. of exposure of sodium arsenate was 138.68, 142.40, 152.60 and 167.90 µmole/l respectively.



Graph 1 Showing SGOT activity of *C. batrachus* exposed to sodium arsenate (42 mg/l) for different duration.

Serum Glutamate Pyruvate Transaminase Activity (SGPT) of Sodium Arsenate Treated Group

The SGPT activity was found increased as the duration of exposure of sodium arsenate increased. The increased SGPT activity after 24, 48, 72 and 96 hrs. of exposure of sodium arsenate was 43.64, 45.18, 52.48 and 57.72 µmole/l respectively.



Graph 2 Showing SGPT activity of *C. batrachus* exposed to sodium arsenate (42 mg/l) for different duration

In the present study due to effect of sodium arsenate Serum glutamate oxaloacetate transaminase activity (SGOT) and Serum glutamate pyruvate transaminase activity (SGPT) was increased as compared to control value at 24, 48, 72 and 96 hours.

FINDING AND DISCUSSION

In the aquatic fauna, fish appears to be particularly susceptible to toxicity of arsenic as they are continually exposed to it through gills and intake of arsenic-contaminated food (Chatterjee *et al.,* 1993). The sensitivity of fish to arsenic is variable in terms of 96 hours of LC_{50} with the range of 10.8 to 105mg/l (Ahmed *et al.,* 2008). In the present study, lethal concentration (LC_{50}) of sodium arsenate was 42 mg/l exposure to 96 hours.

The result obtained may be due to necrosis, which induce increase in permeability of cell membrane resulting in the damage of tissues. Parthiban and Muniyan (2011) reported in their study the level of AST and ALT activities are increased due to sodium arsenate in chronic liver damages. Saravanan *et al.* (2010) observed that the present study also bring out the aspartate amino transferase (AST) and alanine amino transferase (ALT) was significant increase in blood and liver. Such heavy metals, insecticide, etc. induced increase in ALT and AST has been reported by earlier authors.

Liver is the main organ responsible for detoxification of harmful substances which reach it through circulation. Thus the liver is most susceptible to toxicants entering the body of animals. The high degree of liver damage in acute treatment than the chronic exposure with arsenic trioxide on *Clarias batrachus*. Zaki *et al.* (2009) and Abalaka *et al.* (2011) have reported an increase level of activity of ALT in the fingerlings exposed to insecticide. They observed that in fingerlings of *Clarias gariepinus* elevated activity are suggestive of hepatic damages leading to their outflow into circulation (Mousa *et al.,* 2008) and increased the production of enzyme in the liver.

Shakoori *et al.* (1996) reported, suppression of AST referred that oxaloacetate and glutamate are not available to Kreb's

cycle through this route of transamination, but through ALT, which describes for its increasing activity. Transfer amino group of an amino acid by transamination enzymes to a keto acid converting the latter into a newer amino acid and forms a new keto acid, thereby rearrangements of amino group among amino acids forming new amino acids. ALT and AST both enzymes are referred to amino acid metabolism that connect amino acids to intermediates of pathways involved in energy production particularly the TCA cycle. Thus AST and ALT are enzymes often used in diagnosis of damage caused by toxicants in various tissues (De La Torre *et al.,* 2000).

CONCLUSION

In the present study due to effect of sodium arsenate, serum glutamate oxaloacetate transaminase activity (SGOT) and serum glutamate pyruvate transaminase activity (SGPT) was increased as compared to control value at 24 hours interval. Disruption of organ system and resulting deterioration in enzymes activities due to impact of sodium arsenate.

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References

1. Abalaka, S. E., K. A. N. Esievo and S. V. O. Shoyinka. 2011. Evaluation of biochemical changes in *Clarias gariepinus* adults exposed to aqueous and ethanolic extracts of Parkia biglobosa pods. *African Journal of Biotechnology*, 10: 234 - 240.
2. Ahmed, K., Akhand, A. A., Hasan, M., Islam, M. and Hasan, A. 2008. Toxicity of arsenic (sodium arsenite) to fresh water spotted snakehead *Channa punctatus* (Bloch) on cellular death and DNA content. Am-Euras J Agric Environ Sci, 4(1):18-22.
3. Alkarkhi, A. F., Ismail, N., Ahmed, A. and Mat Easa, A. 2009. Analysis of heavy metal concentrations in sediments of selected estuaries of Malaysia-a statistical assessment. Environmental monitoring and assessment, 153(1-4):179.
4. Allen, T., Singhal, R. and Rana, S. V. S. 2004. Resistance to oxidative stress in a freshwater fish *Channa punctatus* after exposure to inorganic arsenic. Biological trace element research, 98(1):63-72.
5. Asati, A., Pichhode, M. and Nikhil, K. 2016. Effect of heavy metals on plants: an overview. *Int. J. Appl. Innov. Eng. Manage*, 5, 2319-4847.
6. Boyer, R. 2000. Modern experimental biochemistry, 3rd ed.; Addison Wesley Longman, Inc.: California.
7. Chakraborty, A. K., and Saha, K. C. 1987. Arsenical dermatosis from tube-well water in West Bengal. Indian J. Med. Res., 85:326-334.
8. Chatterjee, A., Das, D. and Chakraborti, D. 1993. A study of ground water contamination by arsenic in the residential area of Behala, Calcutta due to industrial pollution. Environmental Pollution, 80(1):57-65.
9. Dacie V and Lewis SM 1975. In: practical haematology, ELBS publishers, Longman, Singapore.
10. De La Torre, F. R., A. Salibian and L. Ferrari. 2000. Biomarkers assessment in juvenile *Cyprinus carpio* exposed to waterborne cadmium. Environmental Pollution, 109: 277–282.
11. Huang, X. J., Choi, Y. K., Im, H. S., Yarimaga, O., Yoon, E. and Kim, H. S. 2006. Aspartate aminotransferase (AST/GOT) and alanine aminotransferase (ALT/GPT) detection techniques. Sensors, 6(7):756-782.
12. Kumari, B., Kumar, V., Sinha, A. K., Ahsan, J., Ghosh, A. K., Wang, H. and DeBoeck, G. 2017. Toxicology of arsenic in fish and aquatic systems. *Environmental chemistry letters*, 15(1):43-64.
13. Kumari, B., Kumar, V., Sinha, A. K., Ahsan, J., Ghosh, A. K., Wang, H. and DeBoeck, G. 2017. Toxicology of arsenic in fish and aquatic systems. Environmental chemistry letters, 15(1), 43-64.
14. Liao, C. M., Tsai, J. W., Ling, M. P., Liang, H. M., Chou, Y. H. and Yang, P. T. 2004. Organ-specific toxico kinetics and dose response of arsenic in tilapia *Oreochromis mossambicus*. Archives of environmental contamination and toxicology, 47(4):502-510.
15. Luh, M. D., Baker, R. A. and Henley, D. E. 1973. Arsenic analysis and toxicity—a review. Science of the total environment, 2(1):1-12.
16. Mousa, M.M.A., A. M. M. El-Ashram and M. Hamed. 2008. Effects of Neem leaf extract on freshwater fishes and zooplankton community. 8th International symposium on tilapia in aquaculture. The Central Laboratory for Aquaculture Research, Cairo, Egypt. Oct. 12-14.
17. Nemcsok, J. and Benedeczky, I. 1990. Effect of sublethal concentrations of phenol on some enzyme activities and blood sugar level of carp (*Cyprinus carpio* L.). Environmental monitoring and assessment, 14(2-3), 377-383.
18. Parthiban, P and M. Muniyan. 2011. Effect of heavy metal nickel on amino transferase activities in liver tissue of *Cirrhinus mrigala* (ham.). *International Journal of Current Research*, 2 (1): 055 - 060.
19. Rodwell, V.W. 1988. Metabolism of proteins and aminoacids. In: Mayes, P.A., Rodwell, V.W. (Eds.), Review of Biochemistry. Lange Medical Publications, California, 265–319.
20. Saravanan, T. S., P. Rajesh and M. Sundaramoorthy. 2010. Studies on effects of chronic exposure of endosulfan to *Labeo rohita*. *Journal of Environmental Biology*, 31 (5): 755 -758.
21. Shakoori, A. R., A. L. Mughal and M. J. Iqbal. 1996. Effects of sub-lethal doses of Fenvalerate (a synthetic pyrethroid) administered continuously for four weeks on the blood, liver and muscles of a freshwater fish, *Ctenopharyngodon idella*. *Bulletin Environmental Contamination and Toxicology*, 57: 487- 494.
22. Sharma, V. K. and Sohn, M. 2009. Aquatic arsenic: toxicity, speciation, transformations, and remediation. Environment international, 35(4):743-759.
23. Zaki, M. S., S. O. Mostafa, S. Nasr, A. I. Noor El-Deen, N. S. Ata and I. M. Awad. 2009. Biochemical, clinicopathological and microbial changes in *Clarias gariepinus* exposed to pesticide malathion and climate changes. Reports Opinion, 6 - 11.

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