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Impact of Sodium Arsenate on Histological Changes in Liver and Kidney of Fresh Water Catfish, *Clarias batrachus*

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ABSTRACT

Background: In new age, Arsenic is a major environmental pollutant and exposure occurs through agricultural, environmental, medicinal and occupational sources. The toxicity of sodium arsenate has been shown in catfish, *Clarias batrachus* and the histology report suggested that the sodium arsenate may adversely effect on the catfish, *Clarias batrachus*.

Methods: The histological analysis carried out during the period of 2018-19 for sodium arsenate with *Clarias batrachus* as test animal for 96 hour as per ideal method and determine the lethal concentration (LC_{50}). The liver and kidney tissues were collected from the fishes exposed to sodium arsenate and standard histology procedure were followed to investigate the histological alterations.

Result: Exposure of sodium arsenate causes severe histological changes in liver like nucleus blabbing, infiltration, necrosis of hepatocytes, disruption of normal architecture, shrunkeri hepatocytes, lytic hepatocytes *etc.* In kidney sodium arsenate exposure causes hemorrhage, inflammation and tubular atrophy in renal tubules, disruption of tubular linings, dense chronic inflammation, hemorrhage and vacoulation in Bowman's capsule *etc*. The present investigation suggests that the inorganic forms of arsenic showing the highest toxicity level.

Key words: *Clarias batrachus*, Heavy metals, Histology, Kidney, Liver, Sodium arsenate.

INTRODUCTION

Environmentally heavy metals are defined as total condition surrounding an organism or group of organism especially, the combination of external and physical conditions that affect and influence the development, growth and survival of the organisms (Asati *et al.* 2016). The toxicity in animals varies with animal species, specific metal, concentration, chemical form and pH, as many heavy metals are considered to be essential for animal growth (Katare *et al.* 2015; Pichhode and Kumar, 2015; Pichhode and Gaherwal, 2020a). The arsenate form is less toxic than arsenite in both *in vivo* and *in vitro* condition. In water, arsenic is usually found in the form of arsenate or arsenite (Cervantes *et al.* 1994). The most significant commercial compound, As (III) oxide, is produced as a byproduct in the smelting of copper and lead ores (Abernathy *et al.* 1999). The channel catfish (*Ictalurus punctatus*) were treated with sodium arsenate and sodium arsenite for one week resulted in dose dependent induction of hepatic metallothionein, with significant induction finding in fish exposed to monosodium methyl arsonate and sodium arsenite (Kovendan *et al.* 2013). Arsenic induced significant expression with metallothionein activity can be a beneficial biomarker in environmental biomonitoring of arsenic contamination (Kapila and Ragothaman, 1999). In rainbow trout (*Oncorhynchus mykiss*), cause arsenic-induced fibrosis in the kidney and in lake white fish (*Coregonus clupeaformis*), observed histological lesions on kidney with dietary arsenic toxicant exposure (Kovendan *et al.* 2013).

The histopathological abnormalities showed in the kidneys of the studied fish are similar to those observed, in plaice (*Pleuronectes plaiessa*) affect to crude oil (Haensly *et al.*1982), in European eels (*Anguilla anguilla*) contaminated and/or infected with the parasite *Myxidium giardi* (Cepede) (Ventura and Paperna, 1984), in *Clarias batrachus* and *Tilapia nilotica* affected to arsenic, sulphur and fluorine pass off (Aly *et al.* 1992; Pichhode and Gaherwal, 2019a,b). The direct toxic impact of the arsenic pollutants and some other contaminants are ahead to necrosis, tissue degeneration and the cellular hyperplasia to consider with stress (Pichhode and Gaherwal, 2019c; Pichhode and Gaherwal, 2020b). In the present study has been focused in examination of histology of liver and kidney of catfish, *Clarias batrachus* due to exposure of sodium arsenate.

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MATERIALS AND METHODS

Experimental animal

The healthy fresh water 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 more than one week.

Test chemical

The analytical grade sodium arsenate ($Na_2HAsO_4 \cdot 7H_2O$) (CAS No.: 10048-95-2) (Heptahydrate) was taken from Spectrum chemical mfg. corp., Mumbai, India and used without further purification for the experiment.

Determination of LC_{50} value of sodium arsenate

To determine the lethal concentration (LC_{50}) of sodium arsenate, fish (*Clarias batrachus*) were randomly 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 hour and the concentration at which 50% mortality of fish occurred was taken as the lethal concentration (LC_{50}) (Kumari *et al.* 2017; Pichhode and Gaherwal, 2019d).

Experimental design

In the present investigation experimental fishes were divided into two groups. Ten (10) fishes were kept in the 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 and these work carried out in Department of Zoology, Govt. Holkar Science College, Indore, M.P. during 2018-19.

Experimental duration

In both control and experimental group fishes were exposed to maximum 96 hour.

Histological examination

For histological analysis sacrifice the catfish to remove liver and kidney. The liver and kidney were used for paraffin sectioning and the most widely used method for doing histological analysis. The liver was placed in specific fixatives. After 48-72 hour, bouin's preserve tissue pieces and washed over night in running tap water, dehydrated in ascending grades of alcohol, cleared in benzene and embedded in paraffin wax (60-62°C melting point), section of 4-6 micron thickness were cut though as Spencer's rotary microtome and stained with haematoxylin and eosin as per the standard procedure (Lillie, 1954).

RESULTS AND DISCUSSION

In the present investigation histopathological examination of liver (Fig 1) and kidney (Fig 6) of control fishes and Sodium arsenate exposed fishes (*Clarias batrachus*) were observed. The histological investigations of the liver of fishes due to exposure of sodium arsenate at different time interval (24, 48,72 and 96 hour) were compared to control fishes. Exposure of sodium arsenate causes severe histopathological changes in liver like nucleus blabbing (Fig 2), infiltration (Fig 3), necrosis of hepatocytes, disruption of normal architecture (Fig 4), shrunkeri hepatocytes, lytic hepatocyte cells (Fig 5) *etc*. In kidney sodium arsenate exposure causes hemorrhage (Fig 7), inflammation and tubular atrophy in renal tubules (Fig 8), disruption of tubular linings, dense chronic inflammation (Fig 9), hemorrhage and vacoulation in Bowman's capsule (Fig 10).

The liver is an important vital organ through which most of the metabolic functions are occurring and the entry of

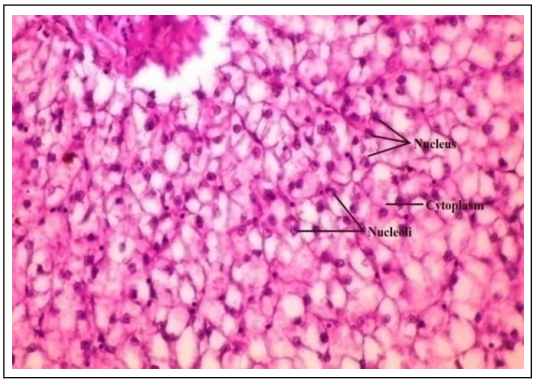

Fig 1: Histopathology of normal (Control) liver of *Clarias batrachus* (10x40 at the normal compound microscope).

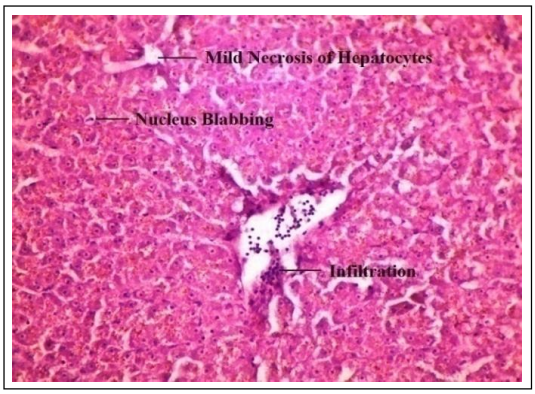

Fig 2: Histopathological changes in liver of *Clarias batrachus* at 24 hrs. exposure of Sodium arsenate.

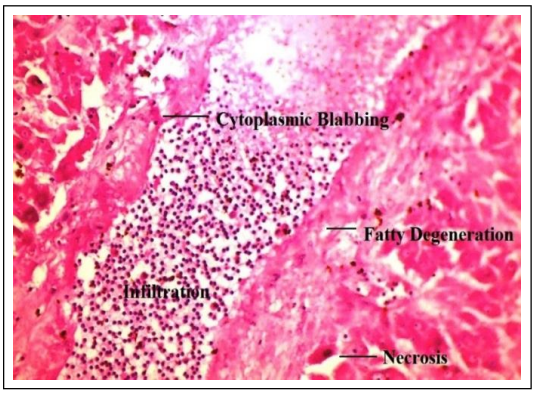

Fig 3: Histopathological changes in liver of *Clarias batrachus* at 48 hrs. exposure of Sodium arsenate.

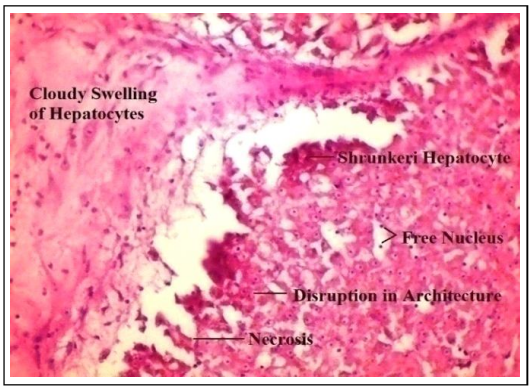

Fig 4: Histopathological changes in liver of *Clarias batrachus* at 72 hrs. exposure of Sodium arsenate.

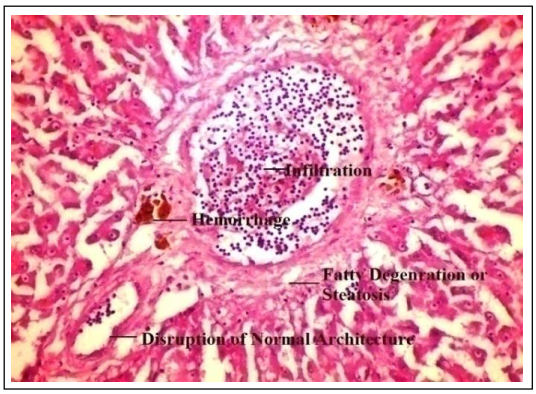

Fig 5: Histopathological changes in liver of *Clarias batrachus* at 96 hrs. exposure of Sodium arsenate.

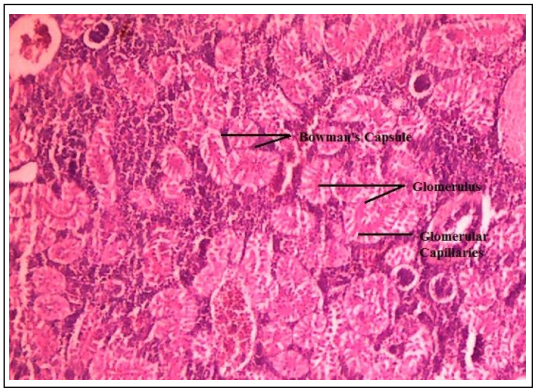

Fig 6: Histopathology of normal (Control) kidney of *Clarias batrachus* (10x40 at the normal compound microscope).

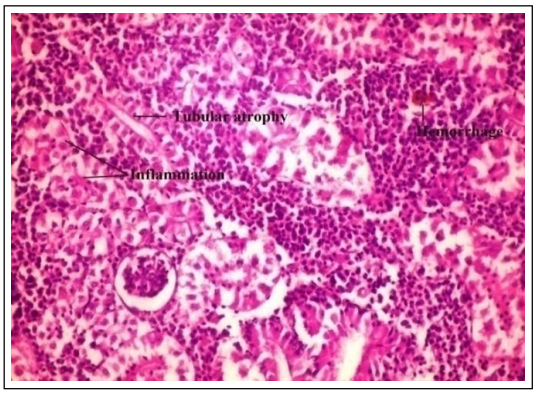

Fig 7: Histopathological changes in kidney of *Clarias batrachus* at 24 hrs. exposure of Sodium arsenate.

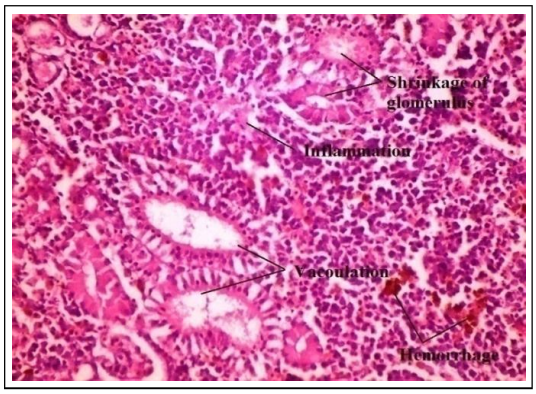

Fig 8: Histopathological changes in kidney of *Clarias batrachus* at 48 hrs. exposure of Sodium arsenate.

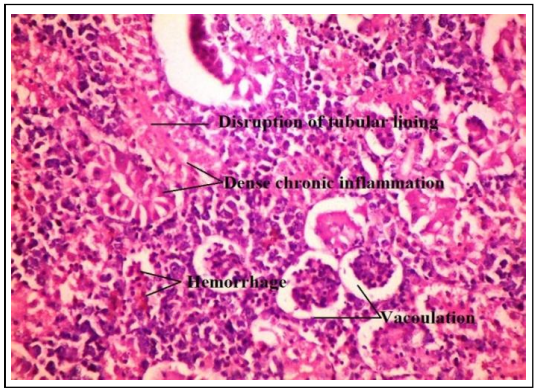

Fig 9: Histopathological changes in kidney of *Clarias batrachus* at 72 hrs. exposure of Sodium arsenate.

contaminants primarily affects the liver (Pichhode *et al.* 2020a). In the liver, arsenate is reduced in arsenite, so that the adverse effects may be caused by both compounds (Chandra and Sajda, 2015; Pichhode and Gaherwal, 2020a). By sodium arsenate, alteration in the architecture and structure of liver could be a significant in the evaluation of health of fish and exhibit the effects of environmental toxicants. The liver tissue shows necrosis, cell wall rupture, parenchymal cells leading to appear smaller in size, cytoplasm become granulated and vacuolated and damaged vacuolar degeneration of hepatocytes (Fig 2-5) are associated with inhibition of energy depletion, protein synthesis and disaggregation of microtubules (Lam *et al.* 2006; Pichhode and Gaherwal, 2020b).

Tubular degeneration and necrosis in the kidney had suffered detrimental damage induced by the exposure of heavy metal such as cadmium (Reimschuessel *et al.*1990). The glomeruli shrinkage, vacuolization, cytolysis of the epithelial cells of tubules and complete necrosis of a few renal tubules (Fig 7-10) were reported and exposed to sub

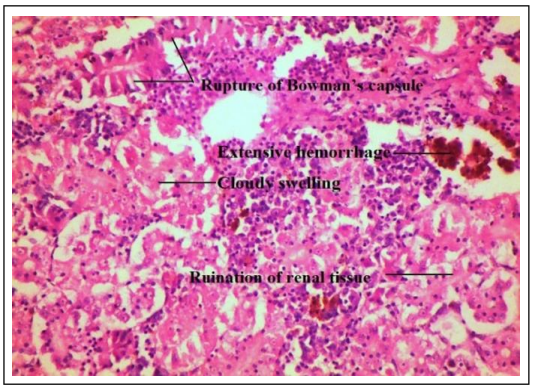

Fig 10: Histopathological changes in kidney of *Clarias batrachus* at 96 hrs. exposure of Sodium arsenate.

lethal concentration of lead under long term exposure. The disintegration of haemopoietic tissue, degeneration of renal tubules, formation of vacuoles around glomeruli and tubular atrophy were found in arsenic, cadmium, copper and mercury treated fish (Pichhode *et al.* 2020b). Vacuolated cytoplasm and dilation of nuclear envelop were observed in the kidney of catfish, *Clarias batrachus* (Shalaby and Abbassa, 2009; Pichhode and Gaherwal, 2020b). In the present investigation liver and kidney of *Clarias batrachus* were affected by exposure of sodium arsenate. Thus, the results of the present study corroborate with the above mentioned authors.

CONCLUSION

In the present investigation, moderate and severe damage in liver and kidney of catfish, *Clarias batrachus* respectively due to exposure of sodium arsenate. These adverse effects of sodium arsenate in liver and kidney were simultaneously correlated with severe biochemical, physiological changes. These histological changes can alter with various physiological activities of the catfish.

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Conflict of interest: None.

REFERENCES

Abernathy, C.O., Liu, Y.P., Longfellow, D., Aposhian, H.V., Beck, B., Fowler, B. and Waalkes, M. (1999). Arsenic: health effects, mechanisms of actions and research issues. Environmental Health Perspectives. 107(7): 593-597.

Aly, S.R., Ibrahim, Th.A., Mahmoud, A.Z. (1992). hazardous effect of some industrial pollutant on *Tilapia nilotica* (*Oreochromis niloticus*). Assiut. Vet. Med. J. 27(54): 189-200.

Asati, A., Pichhode, M., Nikhil, K. (2016). Effect of heavy metals on plants: An overview. Int. J. Appl. Innov. Eng. Manage. 5: 2319-4847.

Cervantes, C., Ji, G., Ramírez, J.L., Silver, S. (1994). Resistance to arsenic compounds in microorganisms. FEMS Microbiology Reviews. 15(4): 355-367.

Chandra, J.H., Sajda, S. (2015). Acute toxicity studies on the fresh water fish Clarias batrachus exposed to pesticide rogorin. Journal of Chemical and Pharmaceutical Sciences. 8(1): 34-39.

Haensly, W.E., Neff, J.M., Sharp, J.R., Morris, A.C., Bedgood, M.F., Boem, P.D. (1982). Histopathology of *Pleuronectes platessa* L. from Aber Wrac'h, Aber Benoit, Brittany, France: longterm effects of the Amoco Cadiz crude oil spill. Journal of Fish Diseases. 5(5): 365-391.

Kapila, M., Ragothaman, G. (1999). Effect of sublethal concentrations of cadmium on the gills of an estuarine edible fish *Boleopthalmus dussumieri* (Curv.). Poll. Res. 18(2): 145-8.

Katare, J., Pichhode, M. and Kumar, N. (2015). Effect of Different Mining Dust on the Vegetation of District Balaghat, MP-A Critical Review. International Journal of Science and Research. 4(8): 603-607.

Kovendan, K., Vincent, S., Janarthanan, S., Saravanan, M. (2013). Expression of metallothionein in liver and kidney of freshwater fish *Cyprinus carpio* var. *communis* (Linn) exposed to arsenic trioxide. Am. J. Sci. Ind. Res. 4(1): 1-10.

Kumari, B., Kumar, V., Sinha, A.K., Ahsan, J., Ghosh, A.K., Wang, H., DeBoeck, G. (2017). Toxicology of arsenic in fish and aquatic systems. Environmental Chemistry Letters. 15(1): 43-64.

Lam, S.H., Winata, C.L., Tong, Y., Korzh, S., Lim, W.S., Korzh, V.,Gong, Z. (2006). Transcriptome kinetics of arsenic-induced adaptive response in zebra fish liver. Physiological Genomics. 27(3): 351-361.

Lillie, R.D. (1954). Histopathological Technique and Practical Histochemistry, The Blackistone Comp. Inc., New York, Toronto, 1: 501.

Pichhode, M., Gaherwal, S. (2019a). Catfish, *Clarias batrachus*. Int. J. Cur. Res. Rev. 11(16): 9-12.

Pichhode, M., Gaherwal, S. (2019b). Study of sodium arsenate induced haematological changes in catfish, *Clarias batrachus*. Int. J. Biol. Med. Res. 10(4): 6882-6885.

Pichhode, M., Gaherwal, S. (2019c). Toxic effect of arsenic trioxide on biochemical response in catfish, *Clarias batrachus*. International Journal of Recent Scientific Research. 10(08): 34033-34036.

Pichhode, M., Gaherwal, S. (2019d). Biochemical response of heavy metal, sodium arsenate exposure in catfish, *Clarias batrachus*. International Journal of Current Advanced Research. 8(08): 19676-19678.

Pichhode, M., Gaherwal, S. (2020a). Effect of heavy metal toxicity, arsenic trioxide on the biochemical parameter of fresh water fish, *Clarias batrachus*. Poll. Res. 39(Suppl.): 123-125.

Pichhode, M., Gaherwal, S. (2020b). Histopathology of liver and kidney of teleost, *Clarias batrachus* of arsenic contaminated Chhilpura pond water. Uttar Pradesh Journal of Zoology. 41(9): 83-92.

Pichhode, M., Kumar, N. (2015). Effect of copper mining dust on the soil and vegetation in India: Acritical review. International Journal of Modern Sciences and Engineering Technology. 2(2): 73-76.

Pichhode, M., Asati, A., Katare, J., Gaherwal, S. (2020a). Assessment of heavy metal, arsenic in chhilpura pond water and its effect on haematological and biochemical Parameters of Catfish, *Clarias batrachus*. Nature Environment and Pollution Technology. 19(5 Suppl): 1879-1886.

Pichhode, M., Gaur, P., Khan, H.R., Dudwe J., Gaherwal, S. (2020b). Histological alteration caused by arsenic trioxide, *Clarias batrachus*. Journal of Xidian University. 14(3): 124-137.

Reimschuessel, R., Bennett, R.O., May, E.B., Lipsky, M.M. (1990). Development of newly formed nephrons in the goldfish kidney following hexachlorobutadiene- induced nephrotoxicity. Toxicologic Pathology. 18(1): 32-38.

Shalaby, A.M., Abbassa, A.H. (2009). The opposing effect of ascorbic acid (vitamin C) on ochratoxin toxicity in Nile tilapia (*Oreochromis niloticus*). Acta Polenica. 2: 18-22.

Ventura, M.T. , Paperna, I. (1984). Histopathology of *Myxidium giardi* (cepede), 1906 infection in European eels, *Anguilla anguilla L., in Portugal*. Aquaculture. 43(4): 357-368.