Bioscience Discovery, 8(4): 671-678, October - 2017

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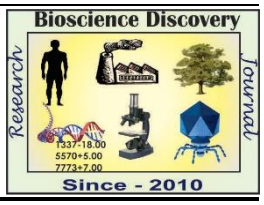
Print & Online, Open Access, Research Journal Available on http://jbsd.in
ISSN: 2229-3469 (Print); ISSN: 2231-024X (Online)
Research Article



Assessment of Iron toxicity by evaluating enzymological markers in liver and kidney of major carp *Labeo rohita* (Ham.)

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Article Info

Received: 09-06-2017,
Revised: 06-09-2017,
Accepted: 22-09-2017

Keywords:
Ferrous sulphate; *Labeo rohita*; Alkaline phosphatase; Acid phosphatase

Abstract

Laboratory experiments were done to determine the enzymological variation in fresh water fish *Labeo rohita* under sub lethal concentration that is 1/4$^{th\ (high)}$, 1/8$^{th\ (medium)}$ and 1/12$^{th\ (low)}$ of 96hr-LC_{50} of Iron (Ferrous sulphate) for exposure period of 15 and 30 days. The results showed significant alteration in the activity of both alkaline and acid phosphatase in liver and kidney of intoxicated fish and reduction were more pronounced in liver. These finding signifies that variation are biomarker for iron induced hepatic and nephrotoxicity in fish and these enzymological indices can be used as indicator of environmental pollution.

INTRODUCTION

The rapid elevations in population growth, development in industrial and agricultural sectors have formed a serious risk for all forms of life in the form of contamination which has now turned out to be a universal problem. Growth in industrialization is one of the most threats to mankind, domestic animals, fishes and wildlife through its effluents. While on one hand technological development has improved the quality of life and on the other hand it has produced a number of health hazards.

Heavy metals are of great concern because they reach the aquatic bodies and deteriorate the life nourishing quality of water and causes impairment to both flora and fauna (Verma *et al.*, 2005; Samanta *et al.*, 2005; Pardeshi and Gapat, 2012; Bhalerao, 2016).

The necessitate to identify and assess the impact of pollutants, particularly a low, sub lethal concentrations on environmental quality had led to development of a range of biological responses measured in number of different species (Fent, 2004). The abrupt death of a fish indicates heavy pollution; the effects of introduction to sub-lethal dose of pollutants can be calculated in terms of biochemical, physiological or behavioral responses of the fish.

Iron Fe is a vital for the organism, because it plays an active part in oxidative/reduction reactions and electron transport associated with cellular respiration. This vital element has essential role in animal's body as part of metalo-proteins like haemoglobin or myoglobin, enzymes, neurotransmitters; they are also in energetic reactions (Dorea, 2000).

Iron can be potentially lethal at high concentrations to fishes especially to *Labeo rohita* (Mohmad *et al.*, 2017). Iron's ability to donate and accept electrons means that if iron is free within the cell, it can catalyze the exchange of hydrogen peroxide into free radicals. Free radicals can cause damage to wide variety of cellular structure, and ultimately kill the cell (Crichton *et al.*, 2002).

Mechanism of free radical production is the Fenton reaction, by which ferrous iron (II) is oxidized by hydrogen peroxide to ferric iron (III), a hydroxyl radical and a hydroxyl anion (Valko *et al.*, 2005).

Phosphatase is a hydrolytic enzyme, leading to liberate of ortho-phosphate from phosphorus compound and based on the optimum pH of action environment, classified into acid phosphatase (ACP, optimum pH≤6.0) and alkaline phosphatase (ALP, optimum pH≥8.0) (Jansson *et al.*, 1988). Acid phosphatase is recognized as a indicator enzyme for the recognition of lysosomes in cell fraction (Cajaraville *et al.*, 2000; Barnhorn and Van Varen, 2004) and alkaline phosphatase is a intrinsic plasma membrane enzyme found in almost all animal cells (Mazorra *et al.*, 2002). Both enzymes are metalloenzyme, concerned in various metabolic processes, such as permeability, development and cell differentiation, protein synthesis, absorption and transfer of nutrients, and gonadal maturation (Ram and Sathayanesan, 1985). In aquaculture sciences, variation in phosphatase level have been regarded as sign of growth, illness of fish (Goldemberg *et al.*, 1987; Matusiewicz and Dabrowski, 1996) and in the evaluation of ecotoxicology, these enzymes have also been acts as bio-indicators of heavy metals pollution because of their sensitivity to metal pollution (Anan *et al.*, 2002; Mora *et al.*, 2004).

Rarer literature is found on the intoxication of Iron in the form of ferrous sulphate on the ACP and ALP in *Labeo rohita*, a key biological element inhabiting local fresh water ecosystems of India. The present work is on the general use of enzymological measurements that can be used as biomarkers as diagnostic and prognostic tools for fresh water monitoring taking the fish *Labeo rohita* as a bioindicator species (Authman *et al.*, 2015, Whitfield and Elliott, 2002).

MATERIALS AND METHODS

The fresh water fish species of *Labeo rohita* (30±2.13 gm*)* was collected from the Narmada River at Hoshangabad. The fish were transported in polythene bags containing sufficient amount of oxygen in order to reduce stress. Laboratory aquariums and equipments were cleaned and rinsed completely before filling with water. Potassium Permanganate was used as a disinfectant. Short term test of acute toxicity over period of 96 hrs was performed on the fishes following the renewal of bioassay. LC_{50} values were determined by EPA Probit Analysis Program (Finney, 1971). After determination of 96hrLC_{50} of heavy metal iron, different sub lethal concentration of iron were carried out in 80 liter glass aquaria filled with 40 liter of tap water. The three sub lethal concentration constitute of $1/4^{th}$, $1/8^{th}$ and $1/12^{th}$ i.e., (17.69 mg/l, 8.84 mg/l and 5.89 mg/l) of 96hr LC_{50} exposed for the duration of 15 and 30 days. After the stipulated time period (15 and 30days) fish were sacrificed and homogenate of liver and kidney was prepared by the process of homogenization and clear supernatants were used as sources for the determination of alkaline and acid phosphatase Acid and alkaline phosphatase activities were assayed spectrophotometrically as per Bergymer (1956) using diagnostic reagent kit from SPAN. The mean values of the various biological parameters for the control and experimental fish were analyzed for statistical significance of differences using the ANOVA. $p<0.05$ was taken as the level of significance.

RESULTS AND DISCUSSION

The level of Alkaline and Acid phosphatase in liver and kidney of the major carp, *Labeo rohita* during sub lethal exposure to iron toxicity recorded overall decline in its activity level over that of the control. The normal level of ALP in liver of control fish was 121.20±3.89 (IU/L). When fish was treated with $1/4^{th}$ of 96LC_{50} for 15 and 30 days, the ALP shows a significant inhibition with value of 101.73±2.37 and 85.55±3.10 respectively. In case of kidney the normal level of ALP was 82.71±2.48, after 15 days and 30 days exposure to $1/4^{th}$ of LC_{50} of Iron, the ALP shows a significant decrease with a value of 71.78±2.07 and 63.00±2.24. (Table1) (Graph 1, 3).

The level of ACP in liver of control fish was 72.76±2.09. When treated with a high concentration of iron for 15 days and 30 days the ACP recorded a significant decline with a mean of 62.88±2.15 and 49.05±2.25. Similarly in case of kidney the level of ACP in control fish was 33.23±2.28. After 15 and 30 days exposure to $1/4^{th}$ of LC_{50} of iron, the highly significant decline was observed with a mean of 27.39±1.90 and 23.73±1.72 (Table1) (Graph 2, 4). When fish was treated with $1/8^{th}$ of LC_{50} for 15 and 30 days the level of ALP in liver recorded a significant inhibition with a value of 106.95±2.93 and 94.82±4.24 as compared to control fish with a mean value of 116.38±3.02. In case of kidney the ALP activity expressed in IU/L was 78.04±3.19 in control fish.

Table 1: Alterations in biochemical parameters in Liver and kidney of *Labeo rohita* intoxicated with 1/4th of LC_{50} of iron (Ferrous sulphate)

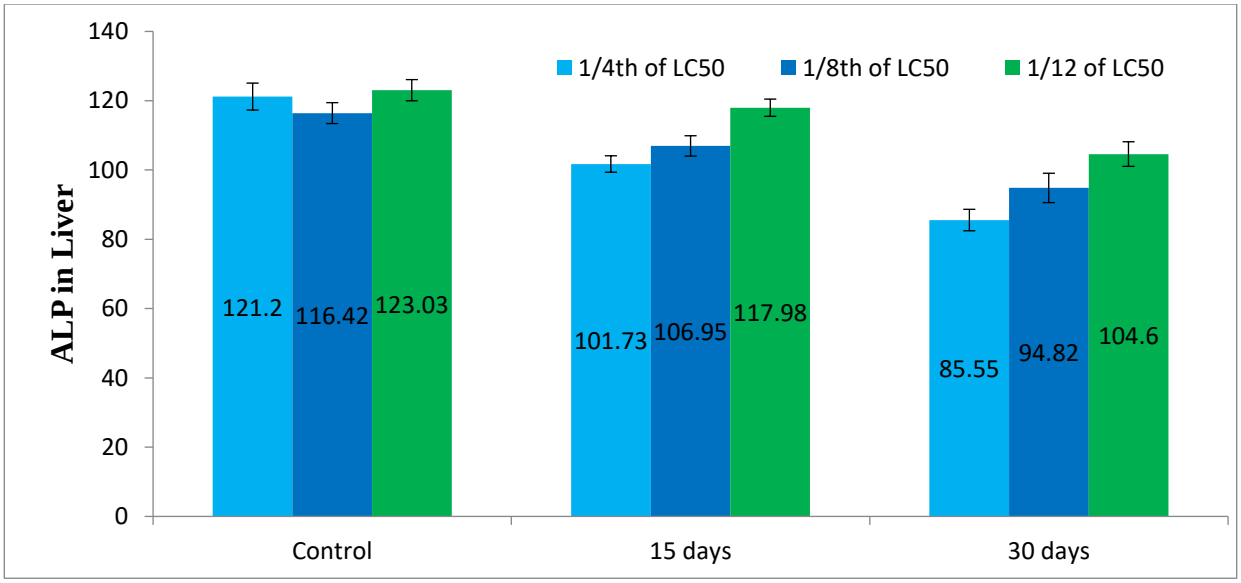
Parameters (Liver)	Control	15 days	% change	30 days	% change
Alkaline Phosphatase	121.20±3.89	101.73±2.37**	-16.06	85.55±3.10**	-29.41
Acid Phosphatase	72.76±2.09	62.88±2.15*	-13.57	49.05±2.25**	-32.58
Parameters (Kidney)					
Alkaline phosphatase	82.71±2.48	71.78±2.07*	-13.21	63.00±2.24**	-23.83
Acid Phosphatase	33.23±2.28	27.39±1.90**	-17.57	23.73±1.72**	-28.58

Table 2: Alterations in biochemical parameters in Liver and kidney of *Labeo rohita* intoxicated with 1/8th of LC_{50} of iron (Ferrous sulphate)

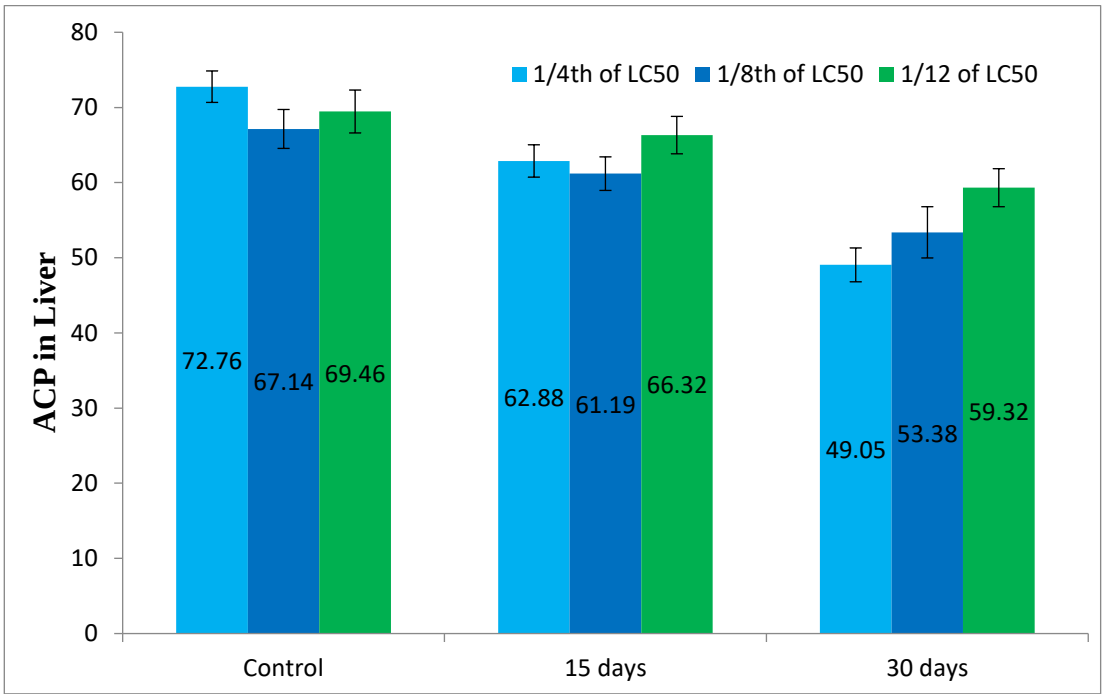
Parameters(Liver)	Control	15 days	% change	30 days	% change
Alkaline phosphatase	116.42±3.02	106.95±2.93*	-8.13	94.82±4.24*	-18.55
Acid Phosphatase	67.14±2.59	61.19±2.23*	-8.86	53.38±3.41*	-20.49
Parameters(Kidney)					
Alkaline phosphatase	78.04±3.19	70.85±3.19*	-9.21	64.71±3.52**	-17.08
Acid Phosphatase	34.24±2.21	31.25±2.36*	-8.73	28.34±1.83**	-17.23

Table 3: Alterations in biochemical parameters in Liver and kidney of *Labeo rohita* intoxicated with 1/12th of LC_{50} of iron (Ferrous sulphate)

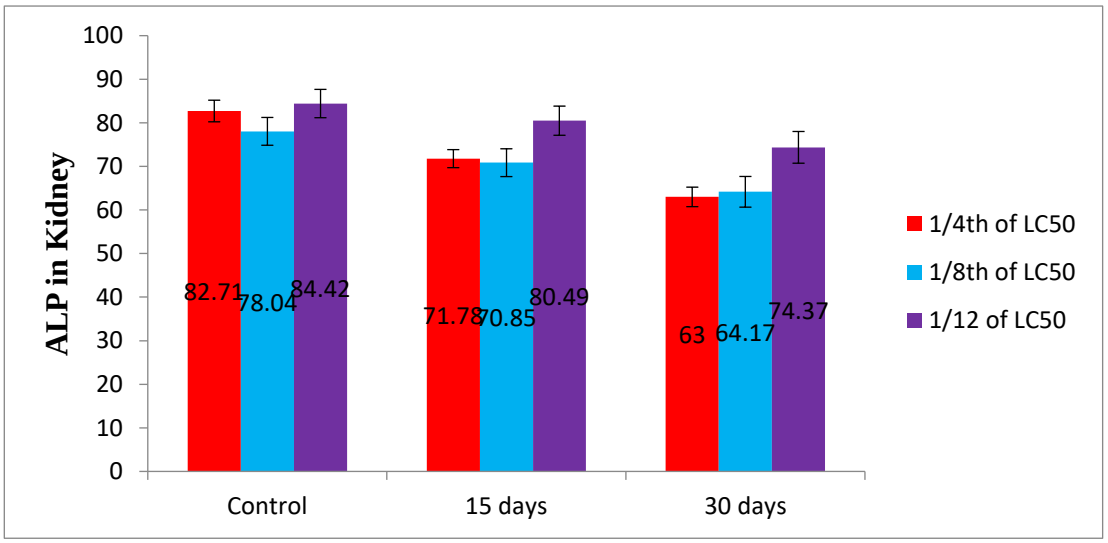
Parameters(Liver)	Control	15 days	% change	30 days	% change
Alkaline Phosphatase	123.03±3.06	$117.98±0.49^{NS}$	-4.10	104.60±3.55*	-14.98
Acid Phosphatase	69.46±2.86	$66.32±2.41^{NS}$	+4.52	59.35±2.53*	-14.55
Parameters(Kidney)					
Alkaline Phosphatase	84.42±3.25	$80.49±3.35^{NS}$	-4.65	74.37±3.64*	-11.90
Acid Phosphatase	31.25±2.73	$29.50±2.58^{NS}$	-5.60	27.12±2.20*	-13.21



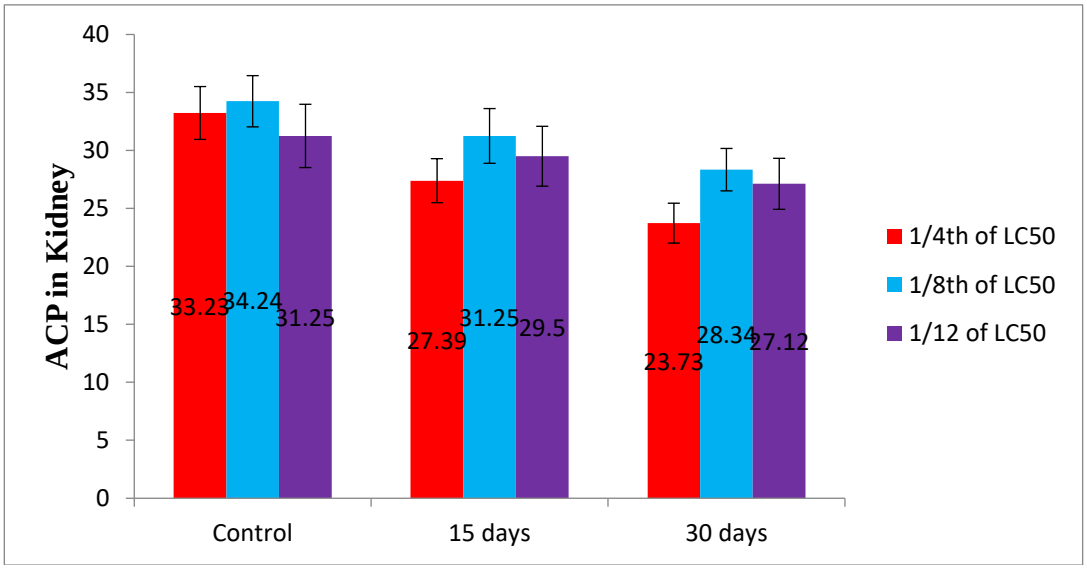
Graph1: Showing variations in ALP activity in liver of *Labeo rohita* exposed to sublethal concentration of Iron intoxication for 15 and 30 days.



Graph2: Showing variations in ACP activity in liver of *Labeo rohita* exposed to sublethal concentration of Iron intoxication for 15 and 30 days.



Graph 3: Showing variations in ALP activity in kidney of *Labeo rohita* exposed to sublethal concentration of Iron intoxication for 15 and 30 days.



Graph 4: Showing variations in ACP activity in kidney of *Labeo rohita* exposed to sublethal concentration of Iron intoxication for 15 and 30 days.

When exposed to 1/8th of LC_{50} for 15 and 30 days, the result shows a significant variation with a value of 70.85±3.21 and 64.71±3.52 (Table 2) (Graph 1, 3).

Similar results are found in ACP level in liver and kidney of *Labeo rohita*. In case of liver the normal level of ACP was 67.17±2.59. However when intoxicated with 1/8th of iron for 15 and 30 days, the ACP activity results a significant alteration with a mean value of 61.19±2.23 and 53.38±3.41. While in kidney the normal level of ACP was 34.24±2.21. When exposed to 1/8th of iron for 15 and 30 days, the ACP level shows a significant alteration with a value of 32.12±0.85 and 28.34±1.82. (Table 2) (Graph 2, 4).

However in case of 1/12th of LC_{50} of iron, the result shows insignificant alteration and the result was dependent on exposure period. The level of ALP in liver of control fish was 123.03±3.06. When treated with 1/12th of LC_{50} for 15 and 30 days the enzyme level was 117.98±2.49 and 104.6±3.55. In case of kidney the ALP activity was 84.42±3.25. After 15 and 30 days exposure of iron intoxication, the kidney ALP level was 80.49±3.35 and 74.37±3.64. (Table 3) (Graph 1, 3).

The ACP level in liver of control fish was 69.46±2.86. When fish was treated with 1/12th of LC_{50} for 15 and 30 days, the ACP level was 66.35±2.41. In case of kidney the normal ACP activity was 31.25±2.73. After 15 and 30 days exposure of iron intoxication, the ACP level was 29.50±2.58 and 27.12±2.20. (Table 3) (Graph 2, 4)

Alkaline phosphatase is a hydrolase enzyme responsible for removing phosphate groups from many types of molecules such as nucleotides, proteins and alkaloids. It is the P-stress marker enzyme most effective in an alkaline environment that catalyzes the hydrolysis of phosphorus compounds and the transfer of phosphoryl groups to an acceptor molecule. The rate of catalytic activity of the enzyme is inversely proportional to the concentration of inorganic phosphate in the ambient environment (Dyhrman and Palanik, 1999). This enzyme could serve as good indicator of intoxication because of its sensitivity to metallic salts (Bage *et al.*, 1992). Acid phosphate on the other hand is a phosphatase which frees attached phosphate groups from other molecules during digestion. It is a lysosomal, hydrolytic enzyme with an acidic pH optimum. It takes part in the dissolution of dead cells and as such serve as a good indicator of stress condition in the biological system (Gupta *et al.*, 1983, Verma *et al.*, 1984)

In the present study, the alkaline phosphatase (ALP) and acid phosphatase (ACP) activity showed a significant decline in liver and kidney of *Labeo rohita* after iron intoxication. A significant reduction in ovarian and hepatic ACP and ALP has been reported in *Heteropneutes fossils* after Cd treatment for 15, 30 and 60 days (Sastry and Subhadra, 1985). Naidu *et al.*, 1984 also reported a significant decline in liver ACP and ALP of *Sarotherodon mossabicus* after an acute exposure (96hr) to mercury.

According to them impaired oxidative and transphosphorylative activities and utilization of carbohydrates during mercury toxicosis causes this depletion. Supporting the above view, Bhatnagar and Bana 1993 reported a decrease in hepatic acid phosphatase in *Channa gachua* after exposure to thiodan and rogar for 30 days; they suggested that the decline may be due to uncoupling of phosphorylation. In the present study, the decline noted in liver kidney ACP and ALP reflects the effects of iron toxicity. Sarus and Andal (2005) reported a decrease in hepatic ACP and ALP in *Hypophthalmichthys molitrix* and *Catla catla* after exposure to distillery effluent for 30 days. Humtsoe *et al.,* (2007) reported significant reduction in hepatic ACP and ALP after exposure of *Labeo rohita* to heavy metal arsenic for 30 days. Zodape (2010) reported a significant decrease in ALP and ACP activity in liver and muscle of *Labeo rohita* following chromium intoxication. In the present study, the decline in liver and kidney ACP and ALP could be results of increased concentration of iron in the liver and kidney since significant accumulation of iron has been reported in the liver and kidney of fish *Labeo rohita* in earlier studies (Javeed and Azhar, 2010).

Sreekala and Zutshi (2010) reported a significant reduction in the ALP and ACP in the major tissues (Liver, Kidney, Gills) of *Labeo rohita* obtained from polluted lakes. The accumulation of toxicants beyond a tolerable level in the liver might causes such enzymatic changes. According to Parthasarathi and Karuppasamy (1998) alkaline phosphatase in liver is capable of inactivating phosphorylase enzymes, thus promoting glycogen synthesis. Therefore inhibition in alkaline phosphatase activity may cause alteration in glycogen content. In the present research work, glycogen and protein content also is being estimate and the result is very much in correlation to the level of ACP and ALP. According to Shaikila *et al.,* 1993 severe acidosis may be the cause for inhibition of alkaline phosphatase activity in intoxicated liver, which in turn could be adaptive for the fish to meet the energy demand by the anaerobic breakdown of glycogen. They further opined that inhibition of activity of activity could also be due to interaction of toxicant with cofactors and regulators. Similar findings were reported in liver and kidney of *Labeo rohita* on exposure to heavy metal intoxication (Gowher *et al*., 2016) also in *Oreochromis noloticus* when exposed to methyl parathion (Sarabadhikary and Sur, 1992) and in *Cyprinus carpio* exposed to vegetable oil factory effluent (Ramesh *et al*., 1994). The decreased activities of these enzymes indicate disturbances in the structure and integrity of cell organelles, like endoplasmic reticulum and membrane transport system (Humtsoe *et al.,* 2007).

The outcome from present work may be helpful in the assessment of environmental stress in the aquatic ecosystem so that attempts could be made to reduce the outcome of environmental pollutants including heavy metals in fresh water ecosystem by pretreatment prior to discharging into the aquatic ecosystem.

Acknowledgment

The authors are thankful to the Principal, Dr. O. N Choubey, Dr. Q. J Shammi and PBRI for encouragement and providing laboratory facilities.

REFERENCES

Anan Y, Kunito T, Ikemoto T, Kubota R, Wanatabe I, Tanabe S, Miyazaki N, Petrov EA and Petrov EA, 2002. Elevated concentrations of trace elements in Caspian seals (*Phoca caspica*) found stranded during the mass mortality events in 2000. *Arch Environ Contam Toxicol* ., **42:** 354-362.

Authman MMN, Zaki MS, Khallaf EA and Abbas HH, 2015. Use of Fish as Bio-indicator of the Effects of Heavy Metals Pollution. *J Aquac Res Development.*, **6**: 328. doi:10.4172/2155-9546.1000328

Barnhorn IEJ and Van Vuren JHJ, 2004. The use of different enzymes in feral freshwater fish as a tool for the assessment of water pollution in South Africa. *Ecotoxicology and Environmental Safety.*, **59:** 180–185.

Bergmeyar HU, 1956. Methods of enzymatic analysis. Second printing revised. *Verlag chemie GMBH Wein Hemi/Bergster Academic press, New York and London* . 779-787 and 837-853.

Bhalerao SN, 2016. Mercury toxicity; behavioral changes and recovery in a freshwater teleost *Heteropneustes fossilis* (Bloch). *Bioscience Discovery.,* **8**: 61-63.

Bhatnagar MC and Bana AK, 1993. Pesticides induced histophysiological alterations in liver of *Channa gachua*. *Proc. Acad. Environ. Biol.,* **2:** 115-118.

Boge G, Leydet M and Houvet V, 1992. The effects of hexavalent chromium on the activity of alkaline phosphatase in the intestine of Rainbow trout *Oncorhynchus mykiss*. *Aquatic.Toxicol*., **23:** 247.

Cajaraville MP, Bebianno MJ, Blasco J, Porte C, Sarasquete C and Viarengo A, 2000. The use of biomarkers to assess the impact of pollution in coastal environment of the Iberian Peninsula: A practical approach. *Sci. Total Environ.,* **247**: 295–311.

Crichton RR, Wilmet S, Legssyer R and Ward RJ, 2002. Molecular and cellular mechanisms of iron homeostasis and toxicity in mammalian cells. *J Inorg Biochem.,* **91**: 9–18.

Dorea JG, 2000. Iron and copper in human milk. *Nutrition.,* **16**(3): 209-220.

Dyhrman ST and Palanik B, 1999. Phosphatase stress in *Labeo rohita* fingerlings: Biochemical enzymatic and haematological consequence. *Comp. Biochem. Physiol.Toxicol. Phamacol*., **137**: 325-333.

Fent K, 2004. Ecotoxicological effects at contaminated sites. *Toxicology.*, 205 (3): 223-240.

Finney DJ, 1971. Probit analysis. London Cambridge university press., 333.

Goldemberg AL, Paron L and Crupkin M, 1987. Acid phosphatase activity in pre- and post-spawning hake (*Merluccius hubbsi*). Comp Biochem Physiol., **87:** 845–849.

Gowher HM, Mahesh T, Altaf H, Younus A and Abid R, 2016. Variations in acid phosphatase (ACP) & alkaline phosphatase (ALP) activities in liver & kidney of a fresh water fish *Labeo rohita* exposed to heavy metal concentrations. *Ejpmr.,* **3**(3): 398-401.

Gupta S, Dalela RC and Saxena PK, 1983. Effect of phenolic compounds on in vivo activity of transaminase in certain tissues of the fish *Notopterus notopterus*. *Environ Res.,* **32:** 813.

Humtsoe N, Davoodi R, Kulkarni BG and Chavan B, 2007. Effect of arsenic on the enzymes of the Rohu Carp, *Labeo rohita*. *Raffles Bul. Zool.,* **14:** 17-19.

Jansson M, Olsson H, and Pettersson K, 1988. Phosphatase: origin, characteristics and function in lakes. *Hydrobiologia.,* **170**: 157–175.

Javeed M and Azhar MS, 2010. Growth and bioaccumulation of Iron in the body organs of *Catla catla*, *Labeo rohita* and *Cirrhina mrigala* during chronic exposures. *Int. J. Agric. Biol.,* **12**: 881–886.

Matusiewicz M and Dabrowski K, 1996. Utilization of the bone/liver alkaline phosphatase activity ratio in blood plasma as an indicator of ascorbate deficiency in salmonid fish. Proc Soc Exp Biol Med., **212**: 44–51.

Mazorra MT, Rubio JA and Blasco J, 2002. Acid and alkaline phosphatase activities in the clam *Scrobicularia plana*: kinetic characteristics and effects of heavy metals. *Comp Biochem Physiol B.,* **131:** 241–249.

Mohmad AW, Shammi QJ and Sigh J, 2017. Evaluation of toxicity of heavy metal iron on a fresh water fish *Labeo rohita* and its behavioural impacts**.** *RJPBCS.,* **8l**: 197-203

Mora SD, Sheikholeslami MR, Wyse E, Azemard S and Cassi R, 2004. An assessment of metal contamination in coastal sediments of the Caspian Sea. *Mar Pollut Bull.,* **48**: 61-77.

Naidu KA, Naidu KA and Ramamurthi R, 1984. Acute effect of mercury toxicity on some enzymes in liver of teleost *Sarotherodon mossambicus***.** *Ecotoxicol Environ Saf.,* **8**(3): 215-8.

Pardeshi A and Gapat M, 2012. Ascorbate effect on protein content during nickel intoxication in the freshwater bivalve, *Lamellidens corrianus*. *Bioscience Discovery.,* **32**: 270 -274.

Parthasarathi K and Karuppasamy R, 1998. Fenvalerate impact on tissue acid and alkaline phosphatase activity of the fish, *Channa punctatus* (Bloch). *Poll. Res.,* **17**(3): 281-285.

Ram R and Sathayanesan AG, 1985. Mercuric chloride, cythion and ammonium sulfate induced changes in the brain, liver and ovarian alkaline phosphatase content in the fish *Channa punctatus*. *Environment and Ecology.,* **3**: 263–268.

Ramesh M, Manavalaramanujam R and Sivakumari K, 1994. Effect of vegetable factory effluent on alkaline phosphatase activity in a fresh water teleost fish *Cyprinus carpio*. *Indian. J. Environ. Hlth.,* **36**(3): 192-196.

Samanta S, Mitra K, Chandra K, Saha K, Bandopadhaya S and Ghosh A, 2005. Heavy metals in water of the rivers Hoogley and Haldi and their impact on fish. *J. Environ. Biol., **26***(3): 517-523

Sarabadhikary A and Sur Rk, 1992. Effect of short duration exposure to methyl parathion followed by recovery of activities of some enzymes of the fish *Oreochromis niloticus* (Smith). Environ. Ecol. **10**: 333-340.

Sarus C and Andal S, 2005. Effects of distillery effluent on tissue phosphatase activity of freshwater teleost *Hypohthmichthys molitrix* (Val.) and *Catla catla* (Ham.). *Fish Biology.,* 145-149.

Sastry KV, and Subhadra K, 1985. In vivo effects of cadmium on some enzyme activities in tissues of the freshwater catfish, *Heteropneustes fossilis*. *Environ. Res.,* **36**(1): 3245.

Shaikila, BI, Thangavel P and Ramaswamy M, 1993. Adaptive trends in tissue acid and alkaline phosphatases of *Sarotherodon mossambica* (Peters) under sevin toxicity. *Indian. J. Environ. Hlth.* **35**(1): 36-39.

Sharma RK and Agarwal M, 2005. Biological effects of heavy metals. An overview. *J. Environ. Biol.,* **26**: 301-313.

Sreekala G and Zutshi B, 2010. Acid and Alkaline phosphatase activity in the tissue of *Labeo rohita* from freshwater lakes of Banglore. *Journal of Life Sciences*, **2**: 365-372.

Valko M, Morris H and Cronin MTD, 2005. Metals toxicity and oxidative stress. *Curr. Med. Chem.,***12**: 1161-1208.

Verma RS, Khan MA, Tripathi R, Shukla S and Sharma UD, 2005. Heavy metal toxicity to fresh water prawn *Macrobrachium dayanum* (Crustacea Decapoda). *Aquacult.,* **6**(1): 57-62.

Verma S, Saxena M and Tonk I, 1984. The influence of Ider 20 on the biochemical and enzymes in the liver of *Clarias batrachus*. *Environ. Pollu.,* **33**: 245 255.

Whitfield AK and Elliott M, 2002. Fishes as indicators of environmental and ecological changes within estuaries: a review of progress and some suggestions for the future. *J Fish Biol.,* **61**: 229-250

Zodape GV, 2010. Effect of Aloe vera juice on toxicity induced by arsenic in *Labeo rohita* (Hamilton). *Journal of Applied and Natural Science*., **2**(2): 300-304.

How to cite this article

Mohmad Amin Wani, Q. J. Shammi, Jasdeep Singh, 2017. Assessment of Iron toxicity by evaluating enzymological markers in liver and kidney of major carp *Labeo rohita* (Ham.). *Bioscience Discovery*, **8**(4): 671-678.