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Research Article

OXIDATIVE STRESS BIOMARKERS AND HISTOLOGICAL STUDY IN KIDNEY OF *HETEROPNEUSTES FOSSILIS* (BLOCH) TREATED WITH SODIUM FLUORIDE EXPOSURE

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ABSTRACT

The present study is carried out for the assessment of oxidative stress induced by fluoride which in aquatic ecosystems like other pollutants. Study has been carried out in cat fish (*Heteropneustis fossilis*) for investigation of definite biomarkers in kidney. Biomarkers selected for stress monitoring were malondialdehyde (MDA), an index of lipid peroxidation and antioxidant defence system enzymes, mainly reduced glutathione (GSH), Superoxide dismutase (SOD), Catalase (CAT), Glutathione peroxidise (GPx), Gluthione S transferase (GST) and Protein activities. Fish were exposed to 35 mg F /L and 70 mg F/L of water for 45 & 90 days. After experiment kidney was collected; a control experiment was also run for comparison .There was decrease activity of GSH, SOD, CAT, GPx and Protein in kidney tissues and increase activity of LPO and GST. Histopatholgical study of kidney revealed renal degeneration and hypertrophy of renal tubules epithelial cells and dilation in the glomeruli. These observations indicate that sub chronic exposure to the fluoride is capable of inducing oxidative stress in fish.

INTRODUCTION

The superoxide, hydrogen peroxide, hydroxyl radicals and other reactive oxygen species (ROS) to damage tissues and cellular components, called oxidative stress, in biological systems. Oxidative stress occurs when the critical balance between oxidants and antioxidants is disrupted due to the depletion of antioxidants or excessive accumulation of the reactive oxygen species (ROS), or both, leading to damage of cellular structure (Scandalios, 2005).

The higher concentration of toxicants causes adverse changes in the tissues like kidney (Karaoz *et al.,* 2004). In higher concentration, fluoride can interfere with carbohydrates, lipids, protein, enzymes and mineral metabolism (Bajpai *et al.,* 2010; Yadav *et al.,* 2015a).

The water serves as base of life to the all living organism on earth. Unfortunately polluted water is common throughout the world (European Public Health Alliance, 2009). Water pollution is the most important factor affecting the life on earth. Water resources are decreasing day by day due to degradation in water quality. Water pollution is most important as all the pollutants get their way to aquatic system (Verger *et al.,* 2007). Contaminants change water quality and may cause diseases and several adverse effects in fish and other edible organism (Chang *et al.,* 1998). Increasing awareness about the effects of anthropogenic activities and pollution on aquatic environment has focused interest on health of fish populations and possibilities to utilize health parameters for assessment of the quality of aquatic environment (Henry *et al.,* 2004). Water may be contaminated by natural sources or industrial effluents. Fluoride pollution in the aquatic ecosystems is mainly due to industrial activities. Fluoride toxicity to aquatic organism including fishes increases with increasing fluoride concentration in the aquatic medium, its exposure time and water temperature (Camargo and Tarazona, 1991; Camargo, 2003).

There are several routes of fluoride intake such as water, food, industrial exposure, air, medicaments and cosmetic out of these, water is main source of fluoride exposure (Susheela, 1993). The Fluoride ion is able to use powerful effects on various enzymes and endocrine gland functions that affect the status of oxidant and antioxidant systems in living organisms (Burgstahler, 2009; Rao and Bhatt, 2012). Fluoride ion exerts its effects on organisms by combining with calcium ions (Ca^{2+}) which can readily penetrate cell membranes by simple diffusion and cause adverse effects on cell metabolism and function. It has also been reported that fluoride ion interfere

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with hydrogen bonding and inhibit numerous cellular enzymes (Emsley *et al.,* 1981; Zhou *et al.,* 2013a).

Present study has been undertaken to observe the effect of fluoride on biochemical and histopathological observations were recorded on the locally available economically important fish *H. fossilis*. Histological technique has been used to observe the structure of kidney to evaluate after fluoride exposure. Various constituents such as LPO, GSH, SOD, CAT, GPx, GST and protein has been observed.

MATERIALS AND METHODS

Experimental animals and chemicals

The fresh water cat fish *H. fossilis* (Mean weight 26.74±0.18 g and length 17.32±0.19 cm) were purchased from the local market in Lucknow and used for the chronic toxicity. They were acclimated to laboratory conditions for 30 Days prior to the experiments. The specimens were given prophylactic treatment by bathing them twice in 0.05% $KMnO_4$ solution for 2 minutes to avoid any dermal infections. The NaF (AR grade) obtained from Qualigens Fine Chemicals Limited, Mumbai, India.

Experimental plan

Fishes were divided into three groups with 12 fish in each group. Group A served as the control (without F added to the water) and Group B was exposed to a concentration of fluoride 35 mg F ion/L (one-tenth of 96-h LC_{50} value). The Group C was exposed to a concentration of fluoride 70 mg F ion/L (one-fifth of 96-h LC_{50} value). At the end of the experiment, the fish were sacrificed. The kidney of both control and treated fish were dissected. The organs were washed in ice-cold physiological saline solution and stored at 80°C until analysis

Preparation of Homogenate

The kidney was homogenized in10% (w / v) ice cold chilled potassium phosphate buffer (0.1 M, pH 7.4) using a Potter-Elvehjem Homogenizer. A part of this homogenate was used for biochemical estimations such as LPO, GSH and other part was centrifuged at 9,000 rpm for 30 minutes at 4^0C to using a Sigma refrigerated centrifuge to obtain the supernatant which was further used for SOD, CAT, GPx, GST and protein estimations.

Lipid peroxidation (LPO)

Tissue LPO was measured using the method of Ohkawa *et al.* (1979). Absorbance was recorded at 530 nm and the results were expressed as n moles MDA/hr/mg tissue.

Reduced glutathione (GSH)

GSH concentration was measured in kidney tissue using the method of Ellman (1959). The absorbance of GSH-DNTB conjugate was determined at 412 nm and the concentration (nM GSH/mg protein) was calculated using standard calibration.

Superoxide dismutase (SOD)

SOD activity was analyzed using the method of Kakkar *et al.* (1984). Colour intensity of the chromogen was measured at 560 nm. The result was expressed as µmoles /min/mg of protein.

Catalase (CAT)

The activity of CAT was measured according to the method of Sinha, (1972). The mixture was cooled and absorbance was read at 570 nm. The CAT activity was calculated in terms of µ moles/min/mg protein.

Glutathione peroxidase (GPx)

The GPx was measured using the procedure of Rotruck *et al.,* (1973). Absorbance was read at 420 nm. The results were expressed as n moles/min/mg protein.

Glutathione S-transferase (GST)

GST was determined spectrophotometrically at 25^0C by following the formation of GSH conjugate with 1-chloro-2,4-dinitrobenzene (CDNB) at 340 nm using extinction coefficient of 9.6×10^3 m^{-1} cm^{-1} (Habig *et al*. 1974). GST activity was expressed as n mole /min/mg of protein.

Protein estimation

Protein was estimated by (Lowry *et al*. 1951).

Histopathological examination of tissues

Samples of kidney washed with 0.9% saline, were taken for histopathological examinations. Samples were immediately fixed in 10% formalin, drained and embedded in paraffin. Sections was made of the paraffin blocks and stained with haematoxylin-eosin (HE).

Statistical analysis

Results were expressed as Mean ± SE. Data was subjected to one way analysis of variance (ANOVA).The treatment groups were compared with a control group using Dunnett's test. All the statistics were carried out in Graph Pad In Stat Software Inc., v. 3.06, San Diageo, USA.

RESULTS

Lipid peroxidation and non-enzymatic antioxidant

The LPO activity was insignificantly increased after low dose exposure (P>0.05) and increase highly significant after high dose exposure (P<0.05) in kidney as compared to control group after 45 & 90 days exposure **[Figure1].**

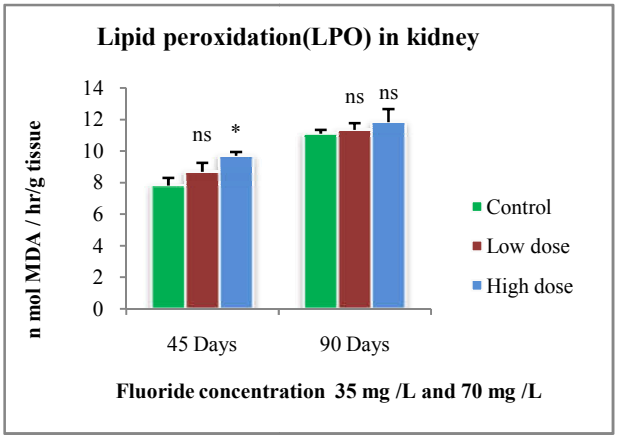

Figure 1 Effects of fluoride exposure on lipid peroxidation after 45 & 90 days. Values as Mean ± SE. Comparison with control group are insignificantly (P > 0.05) and significantly (P<0.05) from each other.

The activity of GSH was decreased significantly after low dose exposure ($P < 0.05$) and significantly after high dose exposure ($P < 0.01$), in kidney as compared to control after 45 & 90 days exposure **[Figure 2].**

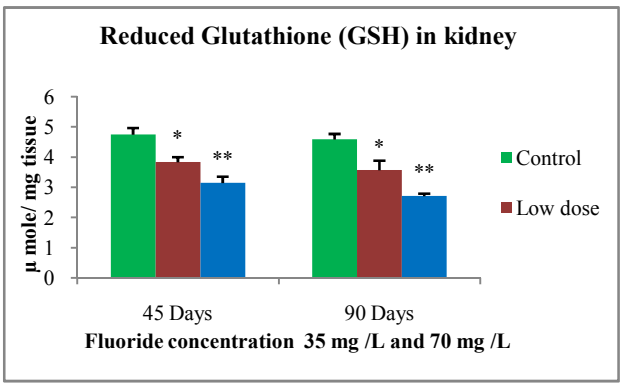

Figure 2 Effects of fluoride exposure on reduced glutathione after 45 & 90 days. Values as Mean ± SE. Comparison with control group are significantly ($P< 0.05$) and high significantly ($P < 0.01$) from each other.

Enzymatic antioxidants

The SOD activity decreased significantly after low dose exposure ($P < 0.05$) as well as high significant at high dose exposure ($P< 0.01$) in kidney as compared to control after 45 & 90 days exposure **[Figure 3].**

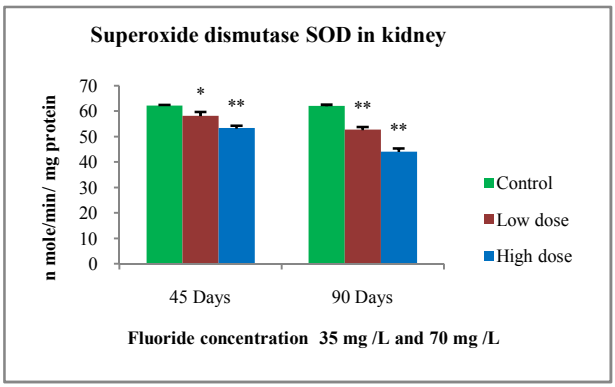

Figure 3 Effects of fluoride exposure on SOD after 45& 90 days. Values as Mean ± SE. Comparison with control group are decreases ($P < 0.05$) and high significantly ($P <0.01$) different from each other.

The CAT activity was decreased significantly after low dose exposure ($P < 0.05$) as well as high dose exposure ($P < 0.01$) in kidney as compared to control after 45 & 90 days exposure. [Figure 4].

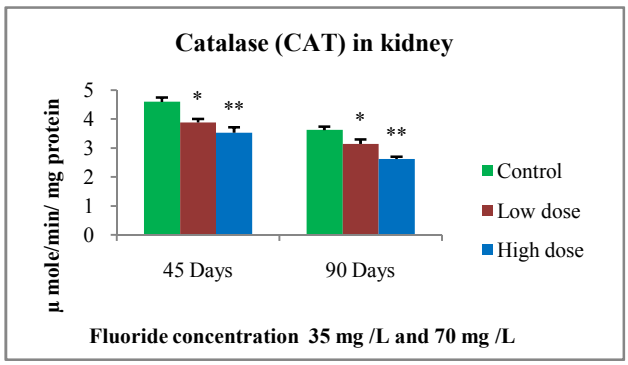

Figure 4 Effects of fluoride exposure on Catalase after 45 & 90 days. Values as Mean ± SE. Comparison with control and treated group are decreases ($P < 0.05$) and high significantly ($P <0.01$) different from each other.

Gpx activity was decreased significantly after low and high dose exposure (P<0.01) in kidney tissue as compared to control after exposure of 45 & 90 days **[Figure 5].**

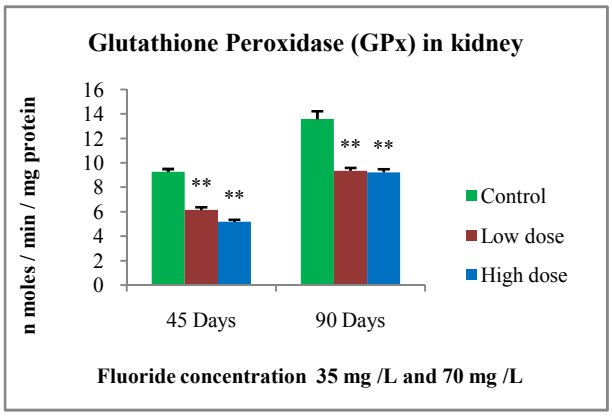

Figure 5 Effects of fluoride exposure on GPx after 45 & 90 days. Values as Mean ± SE. Comparison with control group are decreases significantly ($P < 0.01$) different from each other.

GST activity was increased significantly ($P < 0.01$) in kidney tissue after treated with low and high dose exposure at 45 & 90 days **[Figure 6].**

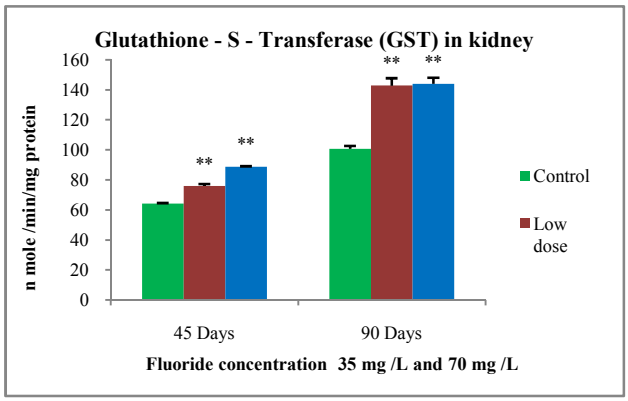

Figure 6 Effects of fluoride exposure on GST after 45 & 90 days. Values as Mean ± SE. Comparison with control group are increase significantly ($P < 0.01$) different from each other.

Protein activity was decreased significantly ($P < 0.01$) at low dose exposure for 45 days and after 90 days exposure it was also decreases significantly ($P < 0.05$) and ($P < 0.01$) respectively**[Figure 7].**

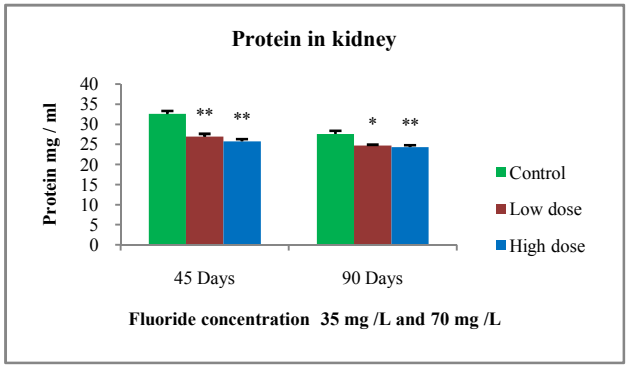

Figure 7 Effects of fluoride exposure on Protein after 45 & 90 days. Values as Mean ± SE. Comparison with control group are decreases significantly ($P < 0.05$) and very high significantly ($P <0.01$) different from each other.

Histology of Kidney

Histologically kidney of *H. fossilis* is made up of large number of nephrons. Each nephron can be distinguished into malphigian tubules i.e. Bowman's capsule and glomerulus, a short neck segment, proximal tubule, distal tubule and collecting duct. Blood capillaries and haemopoietic tissues are present in the inter tubular spaces.

Low concentration

After 45 days of exposure, hypertrophy and degenerative changes were observed in the nephrons, degenerative changes were more prominent. The brush border was found deformed in renal tubules with gradual atrophy. Focal necrosis in renal tubular cells was more prominent. After 90 days of exposure, the capillaries of glomeruli showed more degenerative changes and they were found collapsed and shrunken. Vacuolisation was seen in between renal corpuscle. Nuclear decline i.e. karyolysis and karyorrhexis were seen in tubular epithelium

High concentration

After 45 days of exposure, nuclear decline, severe damage of renal tubules was manifested with increased vacuolization. The capillaries of glomeruli showed pronounced degenerative changes and they were found collapsed and shrunken. Heavy vacuolization and haemosiderin between renal tubules was seen. After 90 days of exposure, deformed brush border of proximal tubules, the parietal layer of bowman's capsule was found ruptured with nuclear deterioration. Prominent focal necrosis was observed at this stage.

A severe necrotic change was seen. Almost complete loss of nephron architecture was observed. Bowman's capsule showed degenerative changes such as necrosis in epithelial cells and accumulated cellular debris in the capsule (**Figure 8**).

Fig.1: Control group showing normal appearance of Bowman's capsule, glomerulus, renal tubules and haemopoietic cells (arrow).

Fig.2: Low concentration exposed group showing swelling, necrosis, vacuolization, cytoplasmic degeneration and nuclear pyknosis and reduction in haemopoietic cells (arrow).

Fig. 3: High concentration exposed group showing disorganized nephrons, karyolysis, hypertrophy, necrosis, vacuolization, swelling, disruption in renal tubules and degradation of renal cells (arrow).

DISCUSSION

In present study, elevated level of LPO was observed in kidney of *H. fossilis* exposed to fluoride. The rise in LPO may be due to the increase in the generation of the free radicals. These free radicals attack cell structures within the body, causing damage to cell membrane and enzyme systems. Similar observation has been reported by various workers (Vasylkiv *et al.,* 2010; Velma and Tchounwou, 2010) after chromium induced oxidative stress in fish. In this study increased LPO levels were observed after fluoride exposure. Such increased LPO levels were also found by previous investigators in brain (Shivarajashankara *et al.,* 2002 Yadav *et al.,* 2015 a), myocardium (Basha and Sujitha, 2011), liver (Chinoy, 2004) and kidney (Birkner *et al.,* 2006).

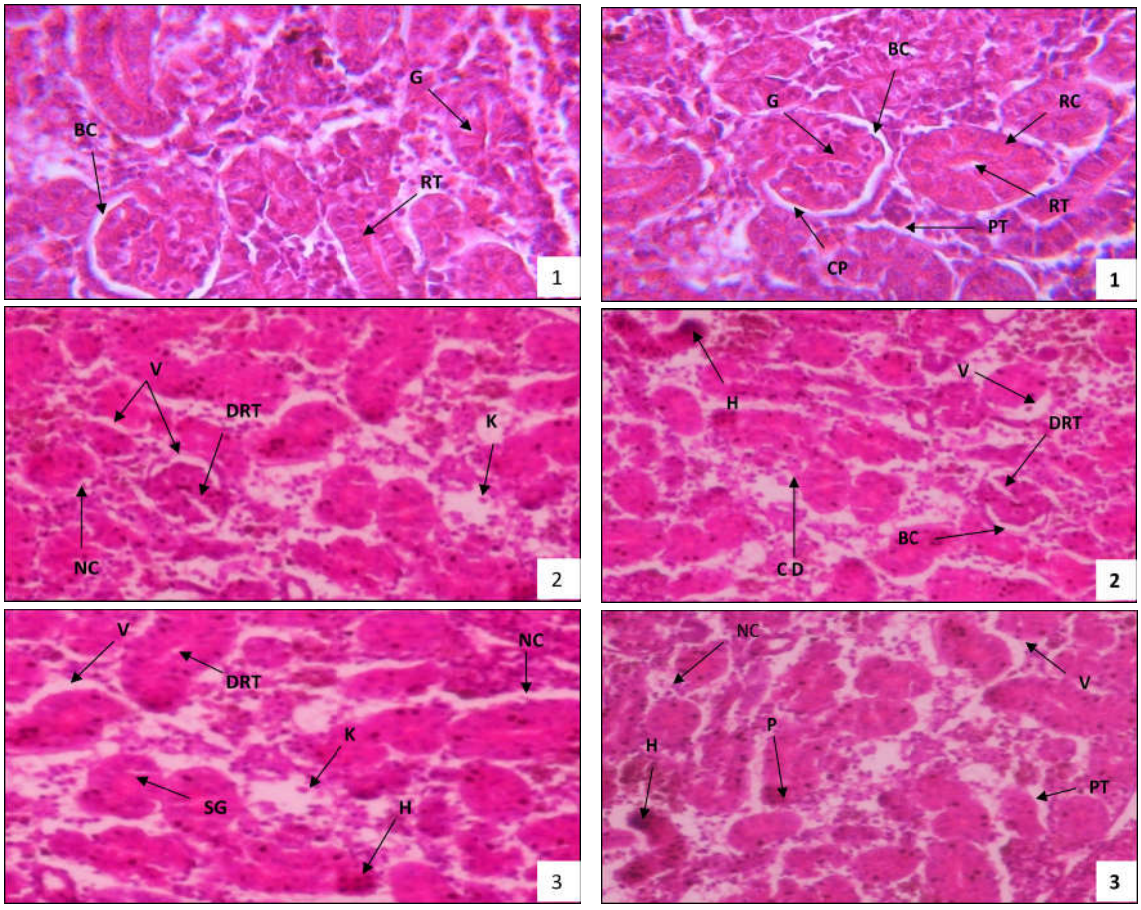

Figure 8 Photomicrograph of T.S. of Kidney of *H. fossilis* after 45 & 90 days exposure to fluoride respectively.

Lipid peroxidation end product, MDA, was significantly increased in kidney tissue of lead-treated *O. Niloticus*. In the present study lipid peroxidation was increased after fluoride exposer in kidney. Increased LPO levels in kidney caused by fluoride have reported in rats (Blaszczyk *et al.,* 2008; Rao *et al.,* 2009).

GSH is an important non-protein cellular thiol that in conjunction with GPx plays a regulatory role in cell proliferation (Bhuvaneswari *et al.,* 2001). GSH and GSH dependent enzymes are involved in scavenging the electrophilic moieties (Michiels *et al.,* 1994). It is known that GSH is one of the most powerful antioxidants in mammalian and is essential for normal cell functioning, replication and cell signalling system (Exner *et al.,* 2000). It is a scavenger of free radicals as well as a co-substrate for peroxide detoxification by glutathione peroxidases (Winterbourn, 1995).

The present study showed decreased activity of GSH in kidney of *H.fossilis* after exposure to fluoride. The reduction in the GSH level may be due to direct conjugation of GSH with electrophiles species which are produced increasingly by fluoride exposure or due to inhibition of enzymes such as glutathione reductase, glutathione peroxidase, glucose-6-phosphate dehydrogenase etc. which are involved in GSH synthesis and regeneration (Bano and Bhatt, 2007). Barbiera *et al.* (2010) found that the fluoride can alter GSH levels with increased, fluoride concentration toxic reaction was induced and the GSH content was decreased. GSH level was significantly decreased in kidney after lead intoxication. Lead depletes GSH levels, several enzymes in the antioxidant defense system protect the imbalance between pro-oxidant and antioxidant. Most of the enzymes contain sulfhydryl groups at their active site and become inactive due to direct binding of lead to sulfhydryl group (Flora *et al.,* 2008; Hashish *et al.,* 2015)

Superoxide dismutase is a class of enzymes that catalyze the binding of ROS with water to dismutation of superoxide anion into molecular oxygen and hydrogen peroxide. As such, they are an important antioxidant defense in nearly all cells exposed to oxygen. In present finding, SOD and CAT level decreased in kidney with increased concentration of fluoride. The decreased activity of SOD in the kidney tissue might cause the accumulation of ROS. Fluoride exposure is considered to generate anion superoxide (O_2^-) (Garcia-Montalvo *et al.,* 2009). During the period of oxidative stress, fluoride can inhibit the activity of antioxidant enzymes such as SOD, GSH and CAT, which play an important role in the antioxidative cell defense and eliminating free radicals, owing to its interactions with enzymes. Similarly decrease in SOD in kidney was reported by (Flora *et al.,* 2008; Hashish *et al.,* 2015) as compared to control group after lead treatment of fishes. Lead showed inhibitory effects of SOD activities; this leads to impairment of cell antioxidant defence mechanisms, which would render cells exposed to oxidative attacks. Significant increase in MDA level and decreased activities of superoxide dismutase and catalase in kidney suggested that oxidative stress has mediated toxic effect in fluoride intoxicated rats (Guo and Sun, 2003).

The activity of CAT in kidney decreased due to over production of free radicals. In contrasts liver and ovary CAT activity in the exposed fish increased as compared to controls both 45 and 90 days of exposure (Yadav *et al.,* 2015 b). The alternative explanation for reduction in CAT activity by fluoride exposure may be related to the direct binding of fluoride to -SH groups of the enzyme molecule. The reduced CAT activity in kidney may also be associated with the compensatory high activity of GPx, which acts as a defense against the formation of H_2O_2 or effective antioxidant responses due to a higher renovation of kidney. Similarly, a decreased CAT activity was also observed in the kidney of *C. punctatus* after cadmium exposure (Dabas *et al.,* 2012).

Glutathione peroxidase plays a main role in minimizing oxidative damage. It is an enzyme with selenium and Glutathione s-transferase works together with glutathione in the decomposition of H_2O_2 or other organic hydroperoxides to nontoxic products at the expense of reduced glutathione (Bruce *et.al.,* 1982). In the present study, after exposure to different doses of fluoride there was decrease in the GPx activity in kidney of fish as compared to control group. It may be due to reduced activity of GSH which is used as a substrate for GPx. Enzyme activity can be decreased by negative feedback from excess of substrate or damage by oxidative modification (Tabatabaie and Floyd, 1994). Topal *et al.* (2013) have reported that the activity of antioxidant GPx and SOD in kidney after cadmium treatment was decreased significantly as compared with control. Similar finding are reported after exposure to cadmium and chromium by Talas *et al.* (2008).

Glutathione S-transferases (GST) are a family of enzymes that catalyze the adding of the tripeptide glutathione to endogenous and xenobiotic substrates which have electrophilic efficient groups. They play an important role in the detoxification and metabolism of many xenobiotic and endobiotic compounds (Ji *et al.,* 1992). GST plays an important role in cellular detoxification of xenobiotic and toxic products of lipid peroxidation. In present study after fluoride exposure GST level increased in kidney after different duration and doses. GST activity seems to be enhanced in response to the increased free radical production as the extent of the enzyme activity corresponds to extent of LPO recorded in animals exposed to fluoride for different durations. Similar results were reported by Atif *et al.,* (2005) in the level of GST in liver and kidney after cadmium exposure on deltamethrin exposure separately as well as combinedly in *C. punctatus*. The increased GST activity was concomitant to the decreases in GSH content in all tissues analyzed. The GSH plays an important role in the detoxification of electrophiles and prevention of cellular oxidative stress (Hasspieler *et al.,* 1994; Sies, 1999).

Proteins are of the most important biological material comprising of nitrogenous constituents of the body and performing different function. In the present study protein level has been found to be decreased significantly in tissues of kidney after exposure to different concentrations of fluoride. This decreased may be due to inhibition of the metabolism of amino acid and synthesis of protein. Similarly Gupta, (2003); Kale and Mule, (2015) reported protein content was decreases in muscle, liver, gill and kidney caused by sodium fluoride. Total protein content decline in different tissues & Serum of *H. fossilis* was reported by (Bajpai and Tripathi, 2010; Yadav *et al.,* 2014). Fluoride is known to decrease protein synthesis in kidney of mice and rats (Chinoy *et al.,* 2000; Birkener *et al.,* 2006; Rao *et al.,* 2009).

In present study, exposure of fluoride induced severe cellular changes in the kidney such as swelling of convoluted tubules and shrinkage of intraglomerular capillaries. There was acute tubular necrosis which confirms irreversible injury to kidney. Epithelial cells of both proximal and distal convoluted tubules in the kidney had pyknotic nuclei and were necrosed and desquamated. The shrinkage of capillaries in the glomerulus that lead to the increase in the capsular spaces and hemorrhagic areas in between the renal tubules was well-known. Cellular observations of kidney indicated that fluoride resulted in disruption in the filtration barriers. These alterations in kidney architecture might be due to generation of reactive oxygen species by fluoride which play a deleterious role in causing nephrotoxicity. Thus in present investigation degeneration of kidney tubules and haematopoietic cells, necrosis, pyknosis and haemorrhage. That means if the process of exposure of the fluoride will continue then ultimately the whole architectural change will be noticed. These findings are similar to our observations in the same model by Deka and Mahanta, (2012) who observed degeneration of kidney tubules and haematopoietic cells, necrosis, pyknosis and haemorrhage in kidney. Mild structural changes were observed including vacuolation, necrosis, mild pyknosis and mild tubular degeneration from the kidney of fish exposed to different river water (Rakhi *et al.,* 2013). Poesina *et al.* (2014) showed the similar results in the cellular changes of the kidney, such as vacuolar dystrophy and cells necrosis of renal tubules, renal epithelial cells with interstitial haemorrhage were also observed.

CONCLUSION

Our results indicate that antioxidant enzyme assays can be used as a bio indicator for chronic exposure to fluoride in the catfish of *H.fossilis*. This could be related to the alterations in antioxidant enzyme activities and other biomarkers of oxidative stress in *H.fossilis* which may cause biochemical dysfunction in this species. In addition, the results provide evidence that enzymatic and non enzymatic biomarkers of oxidative stress can be sensitive indicators of aquatic animals. Fluoride induced histopathological alterations in kidney tissues as well as degeneration of kidney tubules and haematopoietic cell, extensive vacuolated pyknosis necrosis and haemorrhage were highly evident at points was also observed.

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