Progressive Agriculture 28 (3): 240-248, 2017 ISSN: 1017 - 8139

Progressive Agriculture
Journal homepage:http://www.banglajol.info/index.php/PA

Effects of profenofos induced histopathology and recovery patterns in silver barb (*Barbonymus gonionotus*)

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Abstract

Histopathology is promising field for research in aquatic toxicology as it provides the real picture of the toxic effects of xenobiotics in vital functions of a living organism. The present study aims to evaluate the toxic effect of pesticide namely profenofos on silver barb. Liver and kidney of silver barb were examined histologically after exposure to sublethal concentrations (0.01 ppm, 10% of LC_{50} and 0.05 ppm, 50% of LC_{50}) of profenofos for 0, 7, 15 and 30 days. Histological recovery was also studied by maintaining the pesticide-exposed fish in a freshwater system for an additional 7, 15 and 30 day. Kidney and liver of exposed individuals exhibited some remarkable changes in their histology in comparison to control and recovery group. Hepatic lesions in the liver tissues of fish were characterized by cloudy swelling of hepatocytes, lipoid vacuoles, pycnotic nuclei and focal necrosis. Epithelial hypertrophy, narrowing of the tubular lumen, atrophy of the glomerulus, broader Bowman's capsule, necrosis in the epithelial cells and pycnosis in the hematopoietic tissue were observed in kidney tissues of experimental fish. These lesions grew with increasing concentration. Although some of the changes were reversible, the rest were less pronounced after a recovery period.

Key words: Histopathology, profenofos, *Barbonymus gonionotus,* organophosphate pesticide

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Introduction

Pesticides have been one of the most effective weapons discovered by man to protect agricultural products from the attack of pests. But the extensive use of pesticides pose a constant threat to the aquatic life by altering the habitat behavior pattern, growth and reproductive potential (Jarvinen, 1977). Although there are considerable research activities in the field of pesticides, there is wide variation in the amount of information available concerning the effect of particular pesticides on selected non-target organisms. Among the organisms studied, fishes have drawn more attention due to their economic importance (Ganesan et al. 1989). The extent severity of tissue damage of a particular compound as toxicant depends on the toxic potentiality of it in the tissues of organisms (Tilak et al. 2001). Susceptibility to chemical injury varies greatly in the tissues and cells of the same animal. It is even greater in different animal groups. However, the location of the major damage may be determined by the mode of action of the chemical. The mode of action of each poison and the pattern of tissue vulnerability has been well defined and the toxic level of each agent at which a fairly standard distinctive pattern of tissue damage has been studied.

Organophosphorus (OP) pesticides are finding increasing use in recent years since they are biodegradable and therefore persist in the environment only for a short time. Because of their low persistence, repeated applications of these pesticides are being practiced for the control of pests in agricultural fields and thereby large quantities find their way into water bodies (Jyothi and Narayan, 1999).

Profenofos is one of the organophosphate insecticides extensively used in agriculture in our area. The Environment Centre of National Toxicology declared that, there are a dozen highly dangerous chemicals including profenofos. Profenofos is a hard insecticide, which has become a matter of concern because of its potentiality and hazardous effect. The present study was performed to evaluate the sub-lethal effects of profenofos on histopathological alterations in the vital organs like liver and kidney of silver barb as a laboratory animal model.

Histopathological changes have been widely used as biomarkers in the evaluation of the health of fish exposed to contaminants, both in the laboratory (Wester and Canton, 1991; Thophon et al., 2003) and field studies (Hinton et al., 1993; Schwaiger et al., 1997; Teh et al., 1997). One of the great advantages of using histopathological biomarkers is that category of biomarkers allows examining specific target organs, including gills and liver that are responsible for vital functions, such as respiration, excretion and accumulation and biotransformation of xenobiotics in the fish (Gemhofer et al., 2001), and serve as warning signs of damage to animal health (Hinton et al., 1993).
The present study aims at evaluating the toxic effect of profenofos on histopathological alterations in the vital organs like kidney, liver and its recovery patterns of toxicity in siver barb.

Materials and Methods

***Test species*:** Freshwater silver barb, length 8.7±0.47cm and weight 10.62±1.46g were procured from the local fish farm at Mymensingh, Bangladesh. The collected fishes were acclimated to laboratory conditions in dechlorinated tap water for 15 days. The fishes were fed with natural feed including *Bosmina coregoni, Ceriodaphnia reticulate, Cyclops bicolor, Cypriois occidentalis, Diaotomus pygmaeus, Diaphanosoma tropicum, Diaptomus gracilis, Moina brachiata, Moina macrocopa* etc. and commercial fish feed during acclimation.

***Water quality*:** During the period of acclimatization and experimentation the water used was clear, dechlorinated ground water pumped from a deep well. Water temperature, dissolved oxygen and pH were monitored according to APHA (1995) in all test tanks every day. The temperature was measured by a thermometer; dissolved oxygen and pH were measured by DO meter (Model 2020, UK) and pH meter (Model YSI 58, USA), respectively.

***Experimental toxicant*:** Profenofos**:** Profenofos is an organophosphate insecticide pesticide. It is a wide-spectrum insecticide with easy biodegradation and a high bioactivity for antitoxic pests. Profenofos can be used to control pests in fruit trees and vegetables with an excellent effect on bollworms aphids and many other sucking insects.

***Experimental design*:** Acute toxicity test: Profenofos was purchased from the authorized dealer of Mymensingh. Ten fishes were randomly selected from the stock and exposed to different concentrations of profenofos (0.4, 0.3, 0.2, 0.1, 0.05 ppm) for 96 hrs to determine the median lethal concentration (LC_{50}). Water was replaced daily with fresh profenofos mixed water to maintain constant level of profenofos during exposure period. Percent mortality was calculated and the values were transformed into probit scale. Probit analysis was carried out as per Finney (1947). The LC_{50} value for profenofos was 0.1 ppm. Based on the result of the 96 hrs LC_{50} of profenofos, 120 fishes of *B. gonionotus* were exposed for 7, 15 and 30 days to the sub-lethal concentrations of 10% and 50% value of the LC_{50} of the profenofos.

***Experimental study*:** Sublethal exposure: The experimental design and calculations for the acute toxicity were based on the procedure given. The acclimated test fishes were divided into three groups each containing 15 fishes with three replications. Group I was used as control reared in toxic free water. The test fish were belonging to group II, exposed to 0.01ppm and the group III, exposed to 0.05 ppm.The water was changed along with waste food and fecal materials periodically by slowly siphoning the water from each aquarium and at that time aquarium were refilled and redosed with toxicant.

***Recovery studies*:** For recovery, after 7, 15 and 30 days of pesticide exposure, the treated fish were transferred to clean tap water and kept for another 7, 15 and 30 days for recovery.

***Tissue sample collection*:** Tissue samples from the profenofos and control fish were collected after 7, 15 and 30 days of exposure and another 7, 15 and 30 days of depuration respectively. For each sampling 3 fish samples were sacrificed from control and treated groups.

***Histological pursuit*:** After exposure to sub lethal concentrations the fish samples were dissected out and different tissues like liver and kidney were fixed in buffered formalin for 24 hours and subjected to standard histological techniques.

Results

Control liver: Liver is an important organ of various metabolic processes. The normal liver of a teleost fish is characterized by large polyhedral cells which are arranged as a minute network of canalculi; between the liver cells. The nuclei are vesicular with large nucleolus and irregular distribution of bite ducts (Plate 4 A).

***Treated liver*:** Cloudy degeneration, pycnotic nuclei and vacuolization, were observed in *B. gonionotus* exposed to 0.01 ppm of profenofos {(Plate 1(B), 2(F), 3(J)}. At 0.05 ppm profenofos several histological alternation were observed in liver such as cloudy degeneration (CD), hepatocyte (HC), necrosis (N), pyknosis (P), hemorrhage (H); hepato-pancreas (HP) large vacuoles (LV) and portal blood vessel (PBV), focal necrosis (FN), vacuolization (V), and binucleated hepatocyte (BH) {(Plate 4(C), 5(G), 6(K)}.

***Recovery of liver*:** A similar fashion of histological alternations dilation of sinusoid (DS), haemorrage (H), vacuolization (V) were also observed in the 0.01 profenofos treated fish in the recovery grops of 7, 15 and 30 days {(Plate 1(D), 2(H), 3(L)}. Haemorrage (H), necrosis (N), binucleated hepatocyte (BH), cloudy degeneration (CD), vacuolization (V), binucleated hepatocyte, karyolysis (K) were recorded in 10% profenofos treated fish {(Plate 1(E), 2(I), 3(M))}.

***Controlled kidney*:** In the control group basic unit of kidney in fish consists of a renal corpuscle, Bowman's capsule and glomerulus and various segment of the renal tubules, namely proximal tubule, intermediate segment, distal tubule and collecting duct were seen. Proximal tubules have prominent brush borders bathed in the vascular bed in the interstitial tissues. Distal tubules and collecting ducts, both devoid of brush borders, and were sparsely distributed. The intermediate segments between proximal and distal tubules were rarely seen. The renal corpuscles are located in close vicinity of renal tubules and blood vessels in the interstitial tissue. Pigments and leucocytes were very common in the interstitial tissue (Plate 4 A).

***Treated kidney*:** Fish exposed exposed to 0.01 ppm profenofos exposure showed histological alteration in intestine such as necrosis of haematopoietic tissue (NHT), vacuolization (V), necrosis (N), dilation of glomeruli (DG), necrotic changes (NC), dilation of renal tubule (DRT) narrowing of tubular leumen (NTL) {(Plate 4(B), 5(F), 6(J)}. Ruptured kidney tubules (RKT), pycnotic nuclei (PN), hypertrophy (H), ruptured renal corpuscle (RRC), tumor (T), ruptured collecting duct (RCD), necrosis (N), degenerated hepatopoietic tissue (DHT), degeneration of glomerular cells (DGC), inter cytoplasmic vacuolization (ICV),

ruptured renal corpuscle (RRC) and necrosis (N) were noticed at 0.05 ppm sublethal concentration of profenofos exposure {(Plate 4(C), 5(G), 6(K)}.

Recovery of kidney: cloudy degeneration, degeneration of renal epithelial cells, dilation of glomeruli, degeneration of renal epithelial necrotic changes were observed in silver barb exposed to 0.01 ppm profenofos exposure {(Plate 4(D), 5(H) 6(L))}. Degeneration of renal epithelial cells, necrotic changes, pycnotic nuclei and dilation of renal tubule were noticed at 0.05 sublethal concentration of profenofos {(Plate 4(E), 5(I), 6 (M)}.

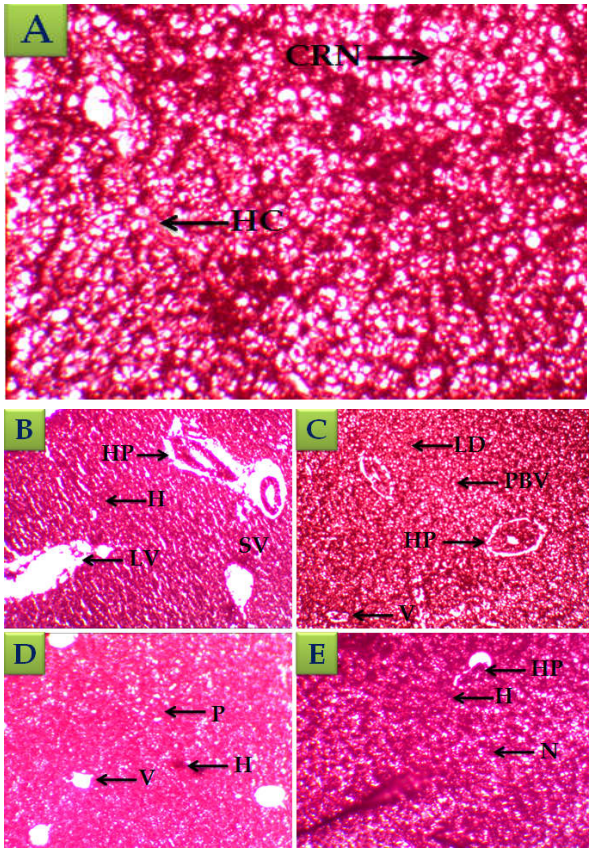


Plate 1. Histological changes of liver: 7 days after profenofos exposure and 7 days after recovery of liver (H and E stained, X100) in silver barb; (A) Control; (B) Profenofos exposure of 10% of LC_{50}; (C) Profenofos exposure of 50% of LC_{50}; (D) recovery at 10% of LC_{50}; (E) recovery at 50% of LC_{50}.

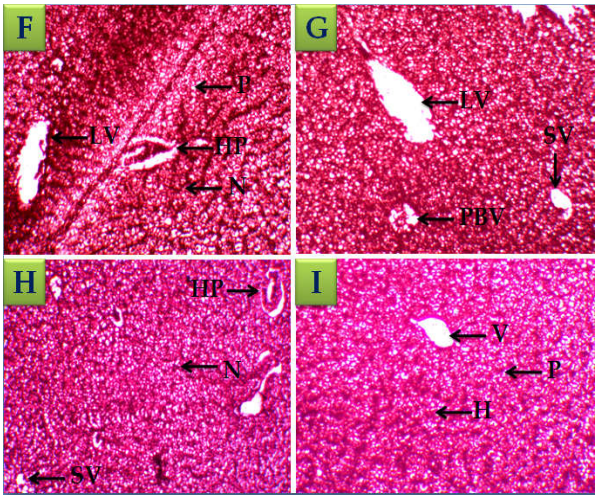
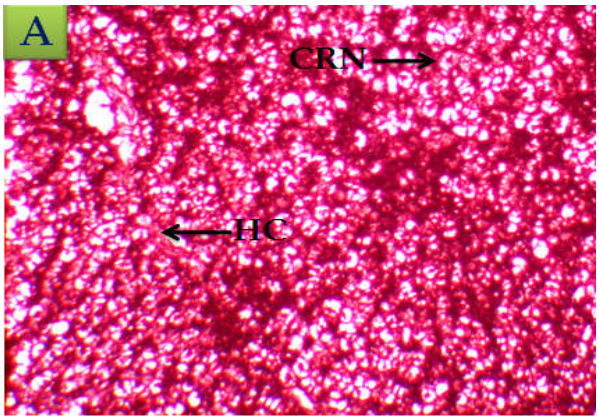


Plate 2. Histological changes of liver: 15 days after profenofos exposure and 15 days after recovery of liver (H and E stained, X100) in silver barb; (F) Profenofos exposure of 10% of LC_{50}; (G) Profenofos exposure of 50% of LC_{50}; (H) recovery at 10% of LC_{50}; (I) recovery at 50% of LC_{50}.

Arrows are indicating central and round nucleus (CRN); hepatocyte (HC); small vacuoles (SV); necrosis (N); pyknosis (P); vacuoles (V); hepato-pancreas (HP); hemorrhage (H); large vacuoles (LV) and portal blood vessel (PBV).



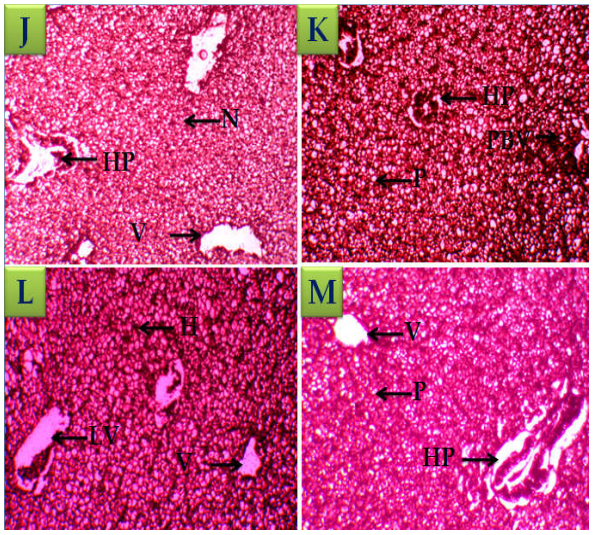


Plate 3. Histological changes of liver: 30 days after profenofos exposure and 30 days after recovery of liver (H and E stained, X100) in silver barb; (A) Control; (J) Profenofos exposure of 10% of LC_{50}; (K) Profenofos exposure of 50% of LC_{50}; (L) recovery at 10% of LC_{50}; (M) recovery at 50% of LC_{50}. Arrows are indicating central and round nucleus (CRN); hepatocyte (HC); necrosis (N); pyknosis (P); vacuoles (V); hemorrhage (H); hepato-pancreas (HP) large vacuoles (LV) and portal blood vessel (PBV).

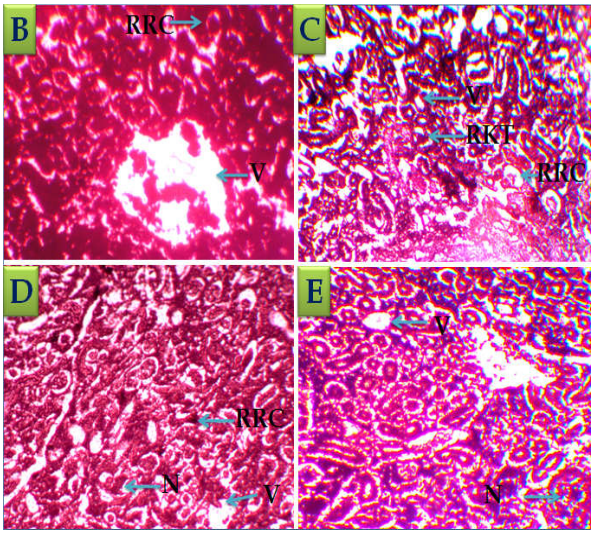
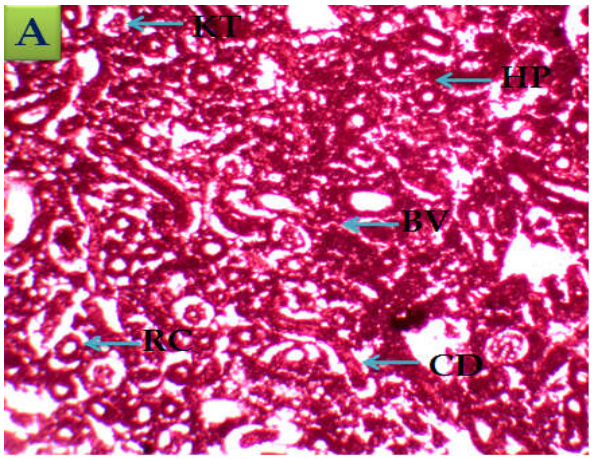
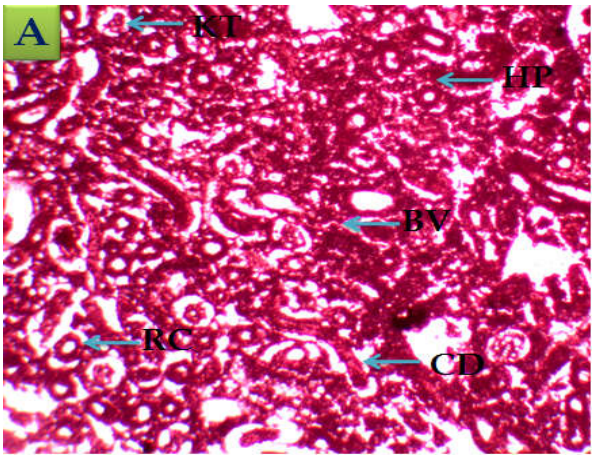


Plate 4. Histological changes of kidney: 7 days after profenofos exposure and 7 days after recovery of kidney (H and E stained, X100) in silver barb; (A) Control; (B) Profenofos exposure of 10% of LC_{50}; (C) Profenofos exposure of 50% of LC_{50}; (D) recovery at 10% of LC_{50}; (E) recovery at 50% of LC_{50}. Arrows are indicating blood vessel (BV); kidney tubules (KT); hepatopoietic tissue (HP); renal corpuscle (RC); ruptured renal corpuscle (RRC); vacuolation (V); necrosis (N); ruptured kidney tubules (RKT) and collecting duct (CD).





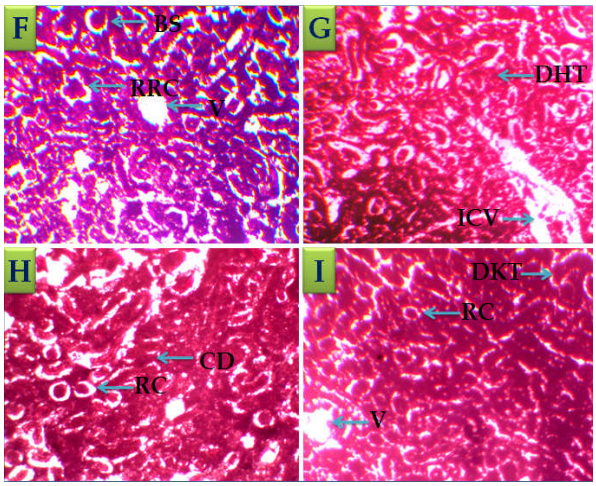


Plate 5. Histological changes of kidney: 15 days after profenofos exposure and 15 days after recovery of kidney (H and E stained, X100) in silver barb; (A) Control; (F) Profenofos exposure of 10% of LC_{50}; (G) Profenofos exposure of 50% of LC_{50}; (H) recovery at 10% of LC_{50}; (I) recovery at 50% of LC_{50}.

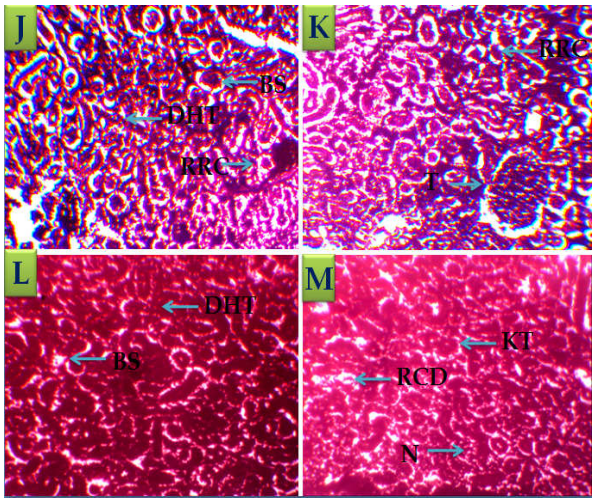


Plate 6. Histological changes of kidney: 30 days after profenofos exposure and 30 days after recovery of kidney (H and E stained, X100) in silver barb; (J) Profenofos exposure of 10% of LC_{50}; (K) Profenofos exposure of 50% of LC_{50}; (L) recovery at 10% of LC_{50}; (M) recovery at 50% of LC_{50}.

Arrows are indicating blood vessel (BV); kidney tubules (KT); hepatopoietic tissue (HP); renal corpuscle (RC); ruptured renal corpuscle (RRC); ruptured collecting duct (RCD); vacuolation (V); degenerated kidney tissue (DKT), collecting duct (CD); bowman's space (BS); degenerated hepatopoietic tissue (DHT) and intra-cytoplasmic vacuoles (ICV).

Discussion

In the present study the LC_{50} values of profenofos exposed for 96 hrs for *B. gonionotus* was found to be 0.1ppm. Percent of mortality increase with an increase in exposure time to profenofos. The acute toxicity test in the present study evidently indicates the toxicity of profenofos to the experimental fish. Dramatical changes in the physical behaviour of fish *B. gonionotus* were recorded in treated profenofos specimens. Histological changes associated with pesticides in fish have been studied by many authors (Wester and Canton, 1991; Bhuiyan et al., 2001; Gernhofer et al., 2001; Thophon et al., 2003; Velmurugan et al., 2007; Matos et al., 2007; Ogueji et al., 2013; Nannu et al., 2015; Mostakim et al., 2015).

The histological studies on fish revealed that various toxicants produce pathological changes such as necrobiotic changes in the liver, tubular damage of kidney, gill lamellar abnormalities (Vinodhini and Narayanan, 2009). Flat fish with liver and kidney lesions was found frequently in areas associated with urban pollution (Malins et al, 1980). Liver and kidney damages were correlated with development of histopathological lesions dependent on does as well as duration of exposure.

Histopathological manifestation has been observed in the tissue of gill, liver and kidney of the fish *Ctenopharyngodon idella* when exposed to technical and sublethal concentration of 20% EC of fenvalerate, a synthetic pyrethroid. The tissue damages like necrosis, vacuolar degeneration and atrophy were observed which are attributed to the effect of the pesticide (Tilak and Yacobu, 2002).

***Liver*:** In the present study liver of silver barb exposed to sublethal concentration of profenofos treatment for 7, 15 and 30 days shows cloudy degeneration, pycnotic nuclei, vacuolization, have been observed in silver barb exposed to 0.01 ppm profenofos exposure. Silver barb exposed to 0.01 ppm profenofos exposure shows histological alteration in liver tissues such as cloudy degeneration, focal necrosis, hemorrhage hepato-pancreas, large vacuoles, portal blood vessel and vacuolization.

Similarly vacuolization, binucleated hepatocyte are observed in 0.05 ppm sublethal concentration of profenofos exposure are observed dilation of sinusoid, haemorrage, vacuolization were the histological alterations observed in the 0.05 ppm profenofos treated i silver barb n 7, 15 and 30 days recovery. Besides haemorrage, binucleated hepatocyte, cloudy degeneration, vacuolization, karyolysis were recorded in 0.01 ppm profenofos treated fish after recovery. Necrosis, hemorrhage, hepato-pancreas, large vacuoles, portal blood vessel (PBV), binucleated hepatocyte, dilation of sinusoid, vacuolization, cloudy degeneration have been observed in 0.05 ppm sublethal concentration of profenofos exposure in the liver of silver barb after 30 days recovery.

Histopathological changes in the liver of freshwater fishes caused by insecticides intoxication have been recorded by Ogueji et al. (2013). Butchiram et al. (2009) reported necrosis, swelling of renal tubules, cellular hypertrophy and granular cytoplasm of kidney of *Channa punctatus* exposed to alachlor. Histopathology on *A. testudinesus*, *C. punctatus* and *B. gonionotus* in relation to 7 days of exposure to sublethal concentrations of 1.13 and 3.75 ppm; 1.13 and 2.26 ppm; and 1.13 and 2.26 ppm of diazinon 60 EC respectively resulted in hypertrophy, necrosis and pyknosis of hepatocytes, pyknosis and degenerative changes such as necrosis of tubular and haematopoietic cells of kidney were the major manifestation (Rahman *et al*., 2002).

***Kidney*:** In the present study kidney of silver barb exposed to sublethal concentration of profenofos treatment for 7, 15 and 30 days showed vacuolization, necrotic tubule, degeneration of renal epithelial cells, distortion of renal tubule, cloudy Degeneration are observed in e silver barb exposed to 0.01 ppm profenofos exposure. Silver barb exposed to 0.05 ppm profenofos exposure shows histological alteration in kidney such as necrosis of haematopoietic tissue, dilation of glomeruli, necrotic changes, dilation of renal tubule, narrowing of tubular leumen, pycnotic nuclei, hypertrophy, degeneration of renal epithelial cells, degeneration of glomerular cells, ruptured renal corpuscle, vacuolation, necrosis, ruptured kidney tubules, inter cytoplasmic vacuolization, and necrosis. Degeneration of renal epithelial cells, necrotic changes, pycnotic nuclei, dilation of renal tubule in 0.05 sublethal concentration of profenofos exposure are observed in the present study in the liver of silver barb after 7, 15 and 30 days recovery. Mandal and Kulshrestha (1980) demonstrated nephropathy in *Clarias batrachus* exposed to sumithion.

The changes observed in the kidney of the treated fish include vacuolation of epithelial cells of uriniferous tubules and degeneration of glomeruli. Elsan treatment in *Channa punctatus* resulted in a significant decrease in the dimension of Bowman's capsule and glomerulus, and the tubules lost their regular shape due to precipitation of cytoplasm and karyolysis (Banerjee and Bhattacharya, 1994). Das and Mukherjee (2000), reported dilation of tubules, necrotic changes characterized by karyorrhexis and karyolysis at the nuclei of affected cells of *Labeo rohita* exposed to hexachlorocyclohexane.

The pesticide profenofos is found to be more toxic to the fish silver barb. The acute toxicity studies in silver barb at 96 hrs LC_{50} value for profenofos is 0.1 ppm. The histological investigations in silver barb exposed to profenofos were found to be highly toxic and the histological alterations were increasing with increasing concentration and duration dependent manner. The

recovery patterns of these histological alternations showed less damage compared to profenofos treated fishes.

Conflict of interest

The authors declare that we have no conflict of interest.

References

APHA (American Public Health Association) (1995). Standard methods for the examination of water and waste water. Washington, DC.

Banerjee S, Bhattacharya S (1994). Histopathology of kidney of *Channa punctatus* exposed to chronic nonlethal level of el san, mercury and ammonia. *Ecotoxicology and Environmental Safety*, 29 (3):65–275.

Bhuiyan AS, Badrun N, Quamrun N (2001): Effects of sumithion on the histological changes of spotted murrel, *Channa punctatus* (Bloch). *Pakistan Journal of Biological Sciences,* 4(10):1288-1290.

Butchiram MS, Tilak KS, Raju PW (2009): Studies on histopathological changes in the gill, liver and kidney of *Channa punctatus* (Bloch) exposed to alachlor. *Journal of Environmental Biology,* 30(2):303-306.

Das BK, Mukherjee SC (2000): Sub-lethal effect of quinalphos on selected blood parameters of *Labeo rohita* (Hamilton) fingerlings. *Asian Fisheries Science,* 13:225–233.

Finney DJ (1947). Probit analysis: a statistical treatment of the sigmoid response curve. Cambridge: Cambridge University Press,. 256 p. [University of Oxford, Oxford, England)

Ganesan RM, Tebakumar SRD, Jayaraman J (1989). Sub lethal effects of organochlorine insecticides (endosulfan) on protein, carbohydrate and lipid contents in liver tissue of *Orechromis mossambicus*. *Proceedings of the Indian Academy of Sciences Animal Sciences*, 98(1):51-55.

Gernhofer M, Pawet M, Schramm M, Muller E, Triebskorn R (2001). Ultrastructural biomarkers as tools to characterize the health status of fish in contaminated streams. *Journal of Aquatic Ecosystem Stress and Recovery*, 8:241-260.

Hinton DE, Baumann PC, Gardner GR, Hawkins WE, Hendricks JD, Hoque RA, M. J. A. Mirja MM, Miah MS (1993). Toxicity of Diazinon and Sumithion to *Puntius gonionotus*. *Bangladesh Journal of Tran Dev*, 6(1):19-26.

Jarvinen AW, Haffman MJ, Thorslund TW (1977). Long-term toxic effects of DDT food and water exposure on fat head minnows (*Pimephales promelas*). *Journal of Fisheries Research Broadcast*, 34:20-89.

Jyothi B, Narayan G (1999). Certain pesticideinduced carbohydrate metabolic disorders in the serum of freshwater fish *Clarias batrachus* (Linn.). *Food Chemical Toxicology,* 37:417-421.

Malins DC, McCain BB, Brown DH, Sparks DH, Hodings HO (1980). Chemical contaminants and biological abnormalities in central and soutnern Puget sound. *NOAA Technological Mem*, OMPA-2. Pp 295.

Mandal PK, Kulshrestha AK (1980). Histopathological changes induced by the sub lethal sumithion in *Clarias batrachus* (Linn.). *Indian Journal of Experimental Biology*, 18(5):547–552

Matos P, Fontainhas-fernandes A, Peixoto F, Carrola J, Rocha E (2007): Biochemical and histological hepatic changes of Nile tilapia *Oreochromis niloticus* exposed to carbaryl. *Pesticide Biochemistry and Physiology,* 89:73-80.

Mostakim GM, Zahangir MM, Mishu MM, Rahman MK, Islam MS (2015): Alteration of blood parameters and histoarchitecture of liver and kidney of silver barb after chronic exposure to quinalphos. *Journal of Toxicology,* http://dx.doi.org/10.1155/ 2015/415984

Nannu MTA, Mostakim GM, Khatun MH, Rahman MK, Sadiqul MI (2015): Hematological and histo-architectural damages in the kidney and liver of Nile tilapia on exposure to kinalux. *Progressive Agriculture,* 26:173-178.

Ogueji EO, Baba I, Usman, Auta J (2013): Histopathology of liver and gill of *C. gariepinus* (Burchell 1822) with swollen abdomen following exposure to acute and sub-lethal concentrations of chlorpyrifos-ethyl. *International Journal of Basic and Applied Sciences*, 2(3):223-229.

Rahman MZ, Hossain Z, Mollah MFA, Ahmed GU (2002). Effects of diazinon 60EC on *Anabas testudineus, Channa punctatus, and Barbodes gonionotus*. *Naga*. *The ICLARM Quarterly*, 25:8-11.

Schwaiger J, Wanke R, Adam S, Pawert M, Honnen W, Triebskorn R (1997). The use of histopatological indicators to evaluate contaminant-related stress in fish. *Journal of Aquatic Ecosystem Stress and Recovery*, 6:75-86.

Teh SJ, Adams SM, Hinton DE (1997). Histopathological biomarkers in feral freshwater fish populations exposed to different types of contaminant stress. *Aquatic Toxicology*, 37: 51-70.

Thophon S, Kruatrachue M, Upathan ES, Pokethitiyook P, Sahaphong S, Jarikhuan S (2003): Histopathological alterations of white seabass, *Lates calcarifer* in acute and sub-chronic cadmium exposure. *Environmental Pollution,* 121:307-320.

Tilak KS, Veeraiah K, Yacobu K (2001). Studies on histopathological changes in the gill, liver, and kidney of *Ctenophrayngodon idellus* exposed to technical fenvalerate and EC 20%. *Pollution Research*, 20(3):387-393.

Tilak, Yacobu (2002). Toxicity and effect of fenvaerate on fish *Ctenophargodon idella*. *Journal of Ecotoxicological Environment Mointoring*, 12(1):09-15.

Velmurugan B, Selvanayagam M, Cengiz EI, Unlu E (2007): The effects of fenvalerate on different tissues of freshwater fish *Cirrhinus mrigala*. *Journal of Environmental Science and Health*, 42(2):157-63.

Vinodhini R, Narayanan M (2009): Heavy metal Induced histopathological alterations in selected organs of the *Cyprinus carpio* Linn (Common Carp). *International Journal Environmental Research* 3(1):95-100.

Wester PW, Canton JH (1991). The usefulness of histopathology in aquatic toxicity studies. *Comparative Biochemistry and Physiology*, 100:115-117.