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REVIEW ARTICLE

CYPERMETHRIN (A SYNTHETIC PYRETHROID) INDUCED HISTOPATHOLOGICAL ALTERATIONS IN GILL, LIVER AND KIDNEY OF A NON-TARGET ORGANISM, FISH

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Abstract: Histopathological changes due to various pesticides have been extensively studied in different fish species. Histopathological studies have already been tested and proposed as efficient and sensitive tools for the monitoring of fish health and environmental pollution in natural water bodies. Organs like gill, liver, intestine and kidney are the best suited organs for histopathological observations. The toxic potential of cypermethrin, a synthetic pyrethroid was reviewed with emphasis on histopathological effects in various fish species. Data provided in this review could be useful in environmental management of pesticide pollution.

Key Words: Cypermethrin; Histopathological Alterations; Gill; Liver; Kidney.

INTRODUCTION

Bioassay experiments are used to determine the toxicity of chemicals and to indicate which organisms are the most sensitive to such chemicals. Histological study is a rapid method for detection of pollutants effects on various tissues of fish and it has been extensively used to determine the deleterious effects of various toxic chemicals. Histopathological alterations have been widely used as bio-monitoring tools of health status of fish exposed to chemical compounds both in laboratory experiments (Boran *et al*., 2012) and field studies (Stentiford *et al*., 2003). Histopathological changes in fish organs have been increasingly studied as biomarkers for assessing aquatic contamination in environmental monitoring studies (Ameur *et al*., 2012; Fricke *et al*., 2012). Histopathology may therefore prove to be a cost effective tool to determine the health status of fish populations and hence reflect the health of the entire aquatic ecosystem in the bio-monitoring process (Nikalje *et al*., 2012). Gills, liver, kidney, intestine, ovary and skin are the most suitable organs for histological examination in order to determine the effect of a toxicant. Tissue changes in test organisms exposed to lethal and sub lethal concentration of a toxicant are functional responses of organisms which provide information on the nature of the toxicant. Histological changes associated with pesticides in fish have been studied by many authors.

Gill histopathology

Since gills remain in close contact with the external environment, gill is considered the primary target organ for the contaminants (Camargo and Martinez, 2006). They absorb even minute concentrations of pyrethroid pesticides and are an important way of uptake and the first site where pyrethroid induced lesions may occur. Cypermethrin exposure induce marked pathological alterations in fish gill architecture viz. epithelial lifting, bulging of tips of primary gill filaments, degenerated secondary gill lamellae, curling of secondary gill filaments, atrophy of secondary lamellae and fusion of secondary gill filaments. Lethal concentrations of cypermethrin will cause well marked severe alterations such as shortened and clubbing of ends of secondary gill lamellae, fusion of adjacent secondary gill lamellae and necrosis in the primary lamellae. Hyperplasia, hypertrophy of nuclei, pyknotic nuclei, vacuolization and degeneration of epithelial and pillar cells and lifting of the epithelial layer from the secondary gill lamellae were also significant at higher concentrations.

Histopathological changes due to cypermethrin were indicative of less oxygen supply to the test fish, resulting in hypoxic respiratory responses. Gill damage caused by the toxicant is important from the aspect of morbidity as it retards growth and affects reproduction (Das and Mukherjee, 2000). Damage of the gills may be due to impairment in gaseous exchange efficiency of the gills oedematous of the lamellae. The cellular damage in terms of epithelium proliferation and necrosis can adversely affect the gaseous exchange and ionic regulation and observed oedematous changes in gill filaments are probably due to increased capillary permeability (Olojo *et al*., 2005). Alterations like fusion of some secondary gill lamellae are examples of defense mechanism, since; in general, these result in the increase of the distance between the external environment and the blood and thus serve as a barrier to the entrance of contaminants (Camargo and Martinez, 2007). As a consequence of the increased distance between water and blood due to epithelial lifting, impaired oxygen uptake is evident. The damaged pillar cells can result in an increased blood flow inside the lamellae, causing dilation of the marginal channel, blood congestion or even an

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aneurysm (Rodriguez *et al.,* 2002). The formation of aneurysm is related to the rupture of the pillar cells (Martinez *et al.,* 2004) due to a heavier flow of blood or even because of the direct effects of contaminants on these cells.

Liver histopathology

Liver plays a fundamental role in uptake, biotransformation and detoxification of foreign compounds (Gernhöfer *et al*., 2011) in body and is thus a target organ of xenobiotics. It is also one of the most affected organs by contaminants in water (Camargo and Martinez, 2007) and as a consequence it undergoes different levels of damage. Tissue changes in liver are linked with histological abnormalities of kidney and gill. Once cypermethrin absorbed, it is transported by blood circulation to liver for transformation and/or storage, and if transformed in the liver it may be excreted trough the bile or pass back into blood for possible excretion by kidney or gill. Discrete pathological alterations include degenerated hepatopancreatic tissue, blood cells among hepatocytes, appearance of blood streaks among hepatocytes, formation of vacuoles along with atrophy, necrosis, disappearance of hepatocytic cell wall and disposition of hepatic cords are the changes observed during cypermethrin exposures which are further intensified at lethal exposures.

These changes may be attributed to the direct toxic effects of pollutants on hepatocytes. Vacuolations of hepatocytes is a common response associated with exposure of fish to a variety of toxicants which might be an indication of imbalance between the rate of synthesis of substances in the parenchyma cells and the rate of their release into the circulation. Depletion of the glycogen in the hepatocytes is usually found in stressed animals (Filho *et al*., 2001), because the glycogen acts as a reserve of glucose to supply the higher energetic demand occurring in such situations (Panepucci *et al*., 2001). Necrosis of some portions of the liver tissue is probably due to the excessive work required by the fish to get rid of the toxicant from its body during the process of cypermethrin detoxification. The inability of fish to regenerate new liver cells may also have led to necrosis.

Kidney histopathology

Highly degenerative changes may be observed in haemopoitic tissue which includes shrinkage of glomerulus, expansion of space inside Bowman's capsule, hypertrophied cells and diminished lumen tubules. Intra cytoplasmic vacuoles in epithelial cells of renal tubules, degenerating haemopoietic tissue with erythrocytes were also prominent. Besides the above changes severe necrosis, cloudy swelling in renal tubules and granular cytoplasm were also observed. Cypermethrin, while it is being eliminated through kidney may cause degenerative changes in renal tubule and glomerulus. Cypermethrin exposure induces marked abnormalities in the kidney initiated with disruption of tubular organization. Thereafter degeneration of tubular epithelial cells and lympocytic infiltration is evident. Most of these pathological changes persisted with vacuolation, clotting of blood in some sinusoids and glomerular degeneration.

Cypermethrin - Histopathological studies

Cypermethrin induced histopathological alterations were studied by Karthigayani *et al*., 2014 (*Oreochromis mossambicus*), Ojutiku *et al*., 2014, Asiwaju *et al.,* 2012 and Ayoola and Ajani, 2008 (*Clarias gariepinus*), Veni and Veeraiah (2014), Prashanth, 2013, 2011, 2006 and Prashanth and David, 2011 (*Cirrhinus mrigala*), Ullah *et al*., 2015 (*Tor putitoria*), Velisek *et al*., 2011 & 2006 (*Oncorhynchus mykiss*), Korkmaz *et al*., 2009 (*Oreochromis niloticus*), Joshi *et al*., 2007 (*Heteropneustes fossilis*), Jee *et al*., 2005 and Sarkar *et al*., 2005 (*Labeo rohita*) and Dobsikova *et al*., 2006 (*Cyprinus carpio*), Olufayo and Alade, 2012 (*Heterobranchus bidorsalis*).

Ullah *et al.,* (2015) reported severe histopathological changes in liver, gills and brain tissues of *Tor putitoria*. Glycogen vacuolation, hemorrhage, congestion, fatty infiltration and hepatic necrosis were observed in liver. Exposure to cypermethrin resulted in cellular infiltration, congestion, swollen tip of the gill filament, heterophilic infiltration and damaged gill. Discoloration, neuronal degeneration, infiltration and severe spongiosis in brain were observed. Karthigayani *et al.,* (2014) investigated the toxic effect of cypermethrin on intestine and liver tissues in *Oreochromis mossambicus* exposed to sublethal concentration of (0.008ppm) of cypermethrin for 24 to 192h. At 96h, epithelial cells and the cells of the outer wall disintegrated which would eventually result in the breakdown of the intestinal functions. Liver tissue showed damaging effect of cypermethrin on the hepatocytes that were altered with pyknotic nucleus. Ojutiku *et al.,* (2014) studied the effect of acute concentrations (0.025mg/l, 0.050mg/l, 0.075mg/l, 0.100mg/l and 0.125mg/l) of cypermethrin for 96h in *Clarias gariepinus* and reported histopathoical alterations in gills and liver. Destruction of gill lamella, epithelial hyperplasia and epithelial hypertrophy were the most common gill changes at all the doses. Hepatic lesions in the liver were characterized by degeneration of hepatocyte, vascuolization of cell cytoplasm, fatty degeneration and hypertrophy of hepatocytes. Histological comparison of tissues indicated that most damage occurred in the gill rather than in the liver. Veni and Veeraiah (2014) reported histopathological effects

in gill, liver and kidney of *Cirrhinus mrigala* and concluded that the toxicant cypermethrin (10%EC) posed a health problem in the fish vital tissues. Gill showed damaged anatomy, degradation, bulging of tips of primary gill lamellae, club-shaped secondary gill lamellae, necrosis in pillar cell nucleus and development of vacuoles in the secondary gill epithelium. Shortened and clubbing of the secondary gill lamellae, fusion of adjacent secondary gill lamellae and necrosis in the primary lamellae were also significant. Liver showed degeneration of cytoplasm in hepatocytes, atrophy, formation of vacuoles, rupture in blood vessels, necrosis and disappearance of hepatocytic cell wall and disposition of hepatic cords. Kidney exhibited severe necrosis, cloudy swelling in renal tubules, cellular hypertrophy and granular cytoplasm. All the changes according to them indicated hepatotoxic nature of cypermethrin. Renal excretion is one of the ways of eliminating the non-detoxified toxicant molecule resulting pathological changes.

Prashanth (2013) observed the effect of cypermethrin (5.13µg/l and 1.026µg/l) in liver tissue of *Cirrhinus mrigala* in which tissues were initially exhibited disarray of liver lobes, mild degree of degeneration of cytoplasm, occasional blood clots, congregation of nuclei, cloudy swelling of hepatocytes, granulisation of cytoplasm, hypertrophic, pyknotic nuclei, atrophy, hepatocytic nuclei, focal necrosis, vacuolation, shrinkage of hepatocytes, granular degeneration, rupture of blood vessels, necrosis, dissolution of laminar structure and cytoplasmic disintegration in hepatocytes were reported. In sublethal concentrations, tissues initially exhibited few changes like disarray of liver lobes, mild degree of degeneration of cytoplasm, occasional blood clots, congregation of nuclei, cloudy swelling of hepatocytes, granulization of cytoplasm, hypertrophic and pyknotic nuclei on 1st and 7th day. But on 14th and 21st day, certain degree of reorganization in the structure of liver cords was observed.

Asiwaju *et al.*, (2012) studied histopathological effects of acute toxicity of cypermethrin on *Clarias gariepinus* juveniles and observed that the gills and liver tissue showed varied morphological changes. Liver showed varying degree of deceleration of cell, hypertrophy of hepatocytes, fatty degeneration, vascular channel congestion and vacuolation of cell cytoplasms which were dose dependent. Olufayo and Alade (2012) reported that fingerlings of *Heterobranchus bidorsalis* exposed to various sublethal concentrations of cypermethrin (0.032, 0.034, 0.036, 0.038 and 0.040ml/l) for 96h showed pathological changes and alterations such as gill infiltration, inflammation in liver, vacuolation and necrosis. There was excessive necrotic degeneration in higher concentrations (0.038 and 0.040 ml/L).

Prashanth (2011) observed severe histopathological changes in kidney of *Cirrhinus mrigala* exposed to lethal (5.13µg/l) and sublethal (1.026µg/l) concentration of cypermethrin. The first sign of morphological changes in the kidney after injection of cypermethrin was found in the proximal tubule. Initial changes of these tubules included: deformation of brush border, gradual atrophy of basal cytoplasm and condensation of nuclear material. Following these initial changes, there was focal necrosis of tubular cells and pyknosis of nuclei. Degenerated cells were frequently seen extruding into the lumina of tubules, which were filled with fragments of cellular components. Focal degeneration of tubular cells was usually followed by more extensive necrosis of the whole nephron. As the focal areas of necrosis became more widespread, more and more leucocytes and macrophages surrounded the tubules. Thus the area of interstitial tissue containing leucocytes and macrophages seemed to be increased as the tubules became reduced. Prashanth and David (2011) observed effect of lethal and sub lethal concentration of cypermethrin (5.13µg/l and 1.026mg/l) for the period of 4 and 21 days in *Cirrhinus mrigala*. Histopathological alterations in intestine were found such as increase in the mucosal cell activity, degenerative changes in structure, hypertrophy of epithelial cells, swelling of lamina propria, fusion of villi due to excessive hypertrophy and oedemic lamina propria ultimately leading to rapture of villi at their tips. The damage was maximum towards the sides of villi and base but progressed with time. The damage was greater at high dose and with the time of exposure the lamina propria separated from the basement membrane inhabiting blood supply to epithelial layers. In sublethal concentration of cypermethrin, hypertophy and necrosis of epithelial cells were observed on day 1. Cellular exudates in the lumen of intestine, the circular and longitudinal muscles were desquamated at day 7 and 14. After 14 and 21 days recovery tendency was seen. Acute toxicity exposure (96h) of cypermethrin in *Oncorhynchus mykiss* caused severe teleagioectasia in the secondary lamellae of gills with the rupture of pillar cells and degeneration of hepatocytes, especially in the periportal zones in rainbow trout. Affected hepatocytes showed pyknotic nuclei and many small vacuoles or one large vacuole in the cytoplasm. The shape of vacuoles was typical for fatty degeneration of liver. Acute exposure to cypermethrin resulted in hyperaemia and perivascular lymphocyte infiltration in skin, mild hyperplasia of respiratory epithelium chloride cell activation in the gills, and vacuolization of pancreas exocrine cells (Velisek *et al*., 2011).

Korkmaz *et al.,* (2009) observed severe histopathological lesions (lifting of epithelia, edema and hypertrophy of epithelial cells) in different organs of Nile tilapia *(Oreochromis niloticus*) exposed to cypermethrin for 10 days. Ayoola and Ajani (2008) reported cypermethrin (1.9, 4.1, 9, 21 and 45mg/l) toxicity to juvenile African catfish, *Clarias gariepinus* and observed histopathological changes in gills, liver, kidney and brain. Cellular infiltration, swollen tip of the gill filament, congestion, severe gill damage and heterophilic infiltration were observed in gill at different concentrations. There were glycogen vacuolation, fatty infiltration, hemosiderosis and congested central vein at the concentration of 1.9 to 9mg/l, severe infiltration of leukocytes, pyknotic and hepatic necrosis at 21 and 45mg/l concentration, severe necrotic, hemorrhage and vacuolation were observed in liver. Kidney tissue showed necrosis, degenerated kidney tubules pyknosis, exfoliated and swollen with pyknotic nuclei. Joshi *et al.,* (2007) studied histopathological changes in liver of *Heteropneustes fossilis* exposed to cypermethrin (¼th of LC50 i.e., 0.012ppm) for 20, 30, 40 and 60 days. After 20 days the hepatocytes became irregular and lost their polygonal shape. Some cells exhibited cloudy swelling. There were many regions in the liver where cells were highly vacuolated. Many cells had exhibited pyknosis. At the end of 30 days, focal necrosis, pyknosis and darkly stained specks of necrotic nuclei in hepatocytes were observed. Intensive vacuoation in cytoplasm and pyknotic nuclei were observed after 40 days. Extensive vacuolated pyknosis and necrosis were highly evident at some points and likely initiation of fibrosis was also observed after 60 days. *Cyprinus carpio* exposed to alimetrin 10EM in the concentration of 29.1µg/l corresponding to 29.1µg/l of cypermethrin for 96h showed severe histopathological alterations (Dobsikova *et al*., 2006). Random hyperaemia and perivascular lymphocyte infiltration in skin, respiratory epithelium hyperplasia and chloride cell activation in gills, and pancreas exocrine cell vacuolization were recorded.

Prashanth (2006) studied histopathological changes in the kidney of *Cirrhinus mrigala* exposed to lethal (5.13µg/l) and sublethal (1.026µg/l) concentration of cypermethrin and observed severe histopathological changes in the kidney. The first sign of morphological changes was found in the proximal tubule which included deformation of brush border, gradual atrophy of basal cytoplasm and condensation of nuclear material. Focal necrosis of tubular cells and pyknosis of nuclei were also observed. Degenerated cells were frequently seen extruding into the lumina of tubules, which were filled with fragments of cellular components. Focal degeneration of tubular cells was usually followed by more extensive necrosis of the whole nephron. As the focal areas of necrosis became more widespread, more and more leucocytes and macrophages surrounded the tubules. Thus the area of interstitial tissue containing leucocytes and macrophages seemed to be increased as the tubules became reduced. Velisek *et al.,* (2006) in *Onchorhynchus mykiss* reported severe teleagioectasiae in the secondary lamellae of gills with the rupture of pillar cells in the 60 % individuals at the concentration of 31.4µg/l. Degeneration of hepatocytes, especially in the periportal zones, was observed in the 40% of individuals. Affected hepatocytes showed pyknotic nuclei and many small or one big vacuole in the cytoplasm. The shape of vacuoles was typical for fatty degeneration of liver. No changes were seen in other examined organs. Significant changes such as hyperplasia, disintegration of hepatic mass and focal coagulative necrosis were found in *Labeo rohita* exposed to cypermethrin (Jee *et al*., 2005). Hyperplasia, vacuolation, disintegrated blood vessels, disrupted hepatocytes, focal coagulative necrosis, disorganized hepatic canaliculi were observed by Sarkar *et al.,* (2005) in *Labeo rohita* exposed to cypermethrin.

CONCLUSION

Histopathological changes observed in the present review clearly indicate that cypermethrin caused damage at both cellular and sub cellular level in the test organs. As a conclusion, the review of the histological investigations of cypermethrin to various fish species demonstrated direct correlation between exposure and histopathological disorders observed in various tissues.

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