Effects of Certain Pesticides on the Histopathology of Liver of an Air Breathing Fish Channa Gachua (Bloch)

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Abstract : The present work related to effects of certain pesticides on histopathology of Liver of an air breathing teleost, Channa gachua (Bloch). exposed to lethal and sublethal concentration of electroplating industry effluent for 24, 48, 72 and 96 hrs (LC_{50} at 20%, 19.5%, 19%, 18.5% respectively). Similarly the test fish were also exposed to acute concentrations of heavy metals like Copper, Nickel and Zinc. For Copper, the LC_{50} values were found to be 180 ppm, 88 ppm, 40 ppm, and 20 ppm respectively. The LC_{50} values for Nickel were found to be 1200 ppm, 600 ppm, 300 ppm and 150 ppm respectively. The LC_{50} values for the heavy metal Zinc was found to be 630 ppm, 528 ppm, 429 ppm and 330 ppm respectively. At the end of exposure period, the fishes which survived were sacrificed liver are removed and fixed in bouins' fixative for 24 hrs. Simultaneously a control aquarium was also maintained. The results are significant from environmental pollution and human health point of view.

IndexTerms **- Channa gachua, Histopathology, Liver, Heavy metal, Environmental pollution.**

I. INTRODUCTION

The costs which society and mankind have to pay for the above damage include diseases, failing health, hunger, starvation, death and adverse effects on property caused by pollution. Pollution exerts direct effects on human health and property as well as some indirect effects on the ecological systems that make life impossible on this planet. In the former case, any insult to the environment is immediately reflected in some adverse effects without there being any major intermediate involvement of the ecological systems. In case of the indirect effects, the ecological systems constitute an essential link between the insult and the cost aspect. For instance, when people drink polluted water and develop hepatitis, it is a direct effect of water pollution. But water pollution can also produce adverse effects on aquatic and terrestrial ecosystems and such effects result in subsequent costs to mankind, hence exemplifying the indirect effects.

Most of the Indian rivers and freshwater streams are seriously polluted by industrial wastes or effluents which come along with waste waters of different industries such as petro-chemical complexes, fertilizer factories, oil refineries, pulp and paper, electroplating, textile, sugar and steel mills, tanneries, distileries, coal washries, synthetic material, plants for drugs, fibers, rubber, plastics, etc. The wastes of these industries and mills include heavy metals like Chromium, Copper, Zinc, Nickel, Cadmium, Mercury, Lead, etc., detergents, petroleum, acids, alkalies, phenols, carbamates, alcohol, cynide, arsenic, chlorine, and many other organic and inorganic toxicants. All these toxicants of industrial wastes are harmful and may cause death or sublethal pathology of the liver, kidney, reproductive system, respiratory system and nervous system in both vertebrate and invertebrate aquatic animals. The severity of damage depends upon the toxic potentiality of a particular compound or particle accumulated in the tissues.

Fishes are one of the main victims of aquatic pollution. Fishes exposed to industrial effluents show loss of equilibrium, increase in opercular movements, irregular movements, disclouration of body and finally death. This may be due to significant damage to the internal organs. These pollutants, especially heavy metals, find their way in the body of aquatic animals by means of gills, digestive tract & general body surface and accumulate in the different tissues of the body. Therefore it is necessary to study in details, the histopathological alterations or changes in structure produced by industrial effluent in different organs of fishes and investigate them thoroughly in order to access the extent of damage.

Channa gachua, the **dwarf snakehead**, is a species of snakehead. It is one of the dwarf snakeheads, and has a length of up to 20 cm (8 in). It gave its name to the aquarists' term dwarf snakeheads to denote the smaller *Channa* species.

II. REVIEW OF LITERATURE

It is generally reported that the histopatholoical biomarkers are useful as indicators of the general health of the fish and are considered as a mirror that reflects of the exposure to a variety of anthropogenic pollutants (Van der Oost *et al*.,

2003). Fish are especially susceptible to environmental variations and respond more sensitively to pollutants than numerous mammals. Their liver is a very interesting model for the study of interactions between environmental factors and hepatic structures and functions (Brusle and Anadon, 1996). Thus research on fish liver is important especially in the field of problems induced by aquaculture conditions and aquatic pollution (Gochfeld, 2003; Mela *et al*., 2007). In teleosts the liver is the primary organ for biotransformation of organic xenobiotics, and probably also for the excretion of harmful trace metals, food digestion and storage, and metabolism of sex hormones (Heath, 1995; Hinton *et al*., 2001). Liver of fish is sensitive to environmental contaminants because many contaminants tend to accumulate in the liver and exposing it to a much higher levels than in the environment or in other organs (Heath, 1995) The organ most associated with the detoxification and accumulation process is the liver and due to its function, position and blood supply, it is also one of the organs most affected by contaminants in the water (Camargo and Martinez, 2007). It also plays a prominent role in fish physiology, both in anabolism (protein, lipid, carbohydrate) and catabolism (glycogenolysis, detoxification) and it acts as storage center for many substances, mainly glycogen (Devi and Mishra, 2013 a).

III. EXPERIMENTAL

In the set of experiment, the test fish *Channa gachua* were exposed to two sub lethal concentrations of electroplating industry effluent for 30 days (chronic) prepared as mentioned in the earlier chapter of chronic toxicity. i.e., $1/5^{th}$ of 96 hrs (3.70%) and $1/10^{th}$ of 96 hrs (1.85%). Similarly for the heavy metal Chromium, the fishes were exposed for 30 days at 12 ppm ($1/5^{th}$ of 96 hrs) and at 6 ppm ($1/10^{th}$ of 96 hrs). For the heavy metal Copper, the two sublethal concentrations were found to be 4 ppm ($1/5^{th}$ of 96 hrs) and 2 ppm ($1/10^{th}$ of 96 hrs) for a period of 30 days. The fishes were also exposed to two sublethal concentrations of heavy metal Nickel at 30 ppm ($1/5^{th}$ of 96 hrs) and 15 ppm ($1/10^{th}$ of 96 hrs) for 30 days. For The heavy metal Zinc – as ZnSO4 the two sublethal concentrations were found to be 66 ppm ($1/5^{th}$ of 96 hrs) and 33 ppm ($1/10^{th}$ of 96 hrs) respectively. Simultaneously a control aquarium was also maintained. At the end of the experiment the fishes were killed and the target organs were utilized for histopathological study. All tissues were fixed in the bouins' fixative for 24 hrs and processed according to the standard procedures of routine microtechnique. Blocks were prepared in paraffin wax and sections were cut on rotary microtome to a thickness of 4-6 µ. For staining, the double staining method was followed by using hematoxylin and eosin (H & E) and mounting was done in DPX. The photographs were taken by a digital camera.

IV. RESULTS

Histological changes produced by electroplating industry effluent and the 3 heavy metals –Copper, Nickel and Zinc on the target organs like liver of the fish, *Channa gachua* have been examined in the present study.

Liver is a digestive gland composed of hepatic cells or hepatocytes and lattice fibers support these cells. The hepatocytes have a distinct central nucleus with densely stained chromatin margin and a prominent nucleolus. Normally these polygonal cells of liver are tightly packed with distinct shape of nucleus. The islets of langerhans lie scattered in the hepatocytes. Blood capillaries known as sinusoids are irregularly distributed between the hepatocytes. The sinusoids are lined by endothelial cells with prominent nuclei. (Fig 1)

The fishes exposed to the lethal concentration for a period of 24 hrs at 20% dilution of electroplating industry effluent showed marked pathological changes in liver as degeneration of sinusoids and disintegration of islets of langerhans, shrinkage of central vein, accumulation of blood cells in central vein with necrosis and degenenration in the hepatic tissue were observed. Intercellular space became wider due to connective tissue damage. (Fig 2)

The fishes exposed to lethal concentration of electroplating industry effluent for a period of 96 hrs at 18.5% dilution show vacuolation in hepatic tissue, vacuolation in islets of langerhans, shrinkage of central vein, ruptured sinusoids with hemorrhages at several places, intercellular space became wider due to degeneration and necrosis in the connective tissue. (Fig 3)

Fishes exposed to the sublethal concentration of electroplating industry effluent at $1/5^{th}$ of 96 hrs (3.70% dilution) for a chronic period of 30 days revealed shrinkage of central vein, hemorrhage and degeneration of sinusoids, vacuole formation in islets of langerhans, disintegration of bile canaliculi and vacuole formation between hepatocytes with swelling of hepatocytes. (Fig 4)

Fishes exposed to sub lethal concentration of electroplating industry effluent at $1/10^{th}$ of 96 hrs (1.85% dilution) for a chronic period of 30 days recorded vacuolation between hepatocytes, degeneration of islets of langerhans,

vacuolation in bile canaliculi, ruptured sinusoids, shrinkage of central vein, with necrosis and degenenration of hepatic tissue. (Fig 5)

The fishes exposed to the heavy metal Copper (Cu) as $CuSO_4$ for a period of 24 hrs at 180 ppm recorded marked pathological changes in liver as degeneration and rupture of sinusoids, vacuole formation between hepatocytes with necrosis and degenenration in the hepatic tissue were observed. Vacuole formation in bile canaliculi with degeneration of islets of langerhans was also observed. Intercellular space became wider due to connective tissue damage. (Fig 6)

The fishes exposed to the heavy metal Copper as (Cu) $CuSO_4$ for a period of 96 hrs at 20 ppm show marked changes in the liver like shrinkage of central vein, accumulation of blood cells in the central vein, loosening of hepatic tissue due to degeneration and necrosis of the connective tissue with vacuolation, necrosis and hemorrhage in hepatic tissue. (Fig 7)

Fishes exposed to the sublethal concentration of heavy metal Copper (Cu) as $CuSO_4$ at $1/5^{th}$ of 96 hrs (4 ppm) for a chronic period of 30 days revealed pathological changes in the liver like rupture of sinusoids, vacuole formaton in hepatic tissue with swelling of hepatocytes, necrosis and degeneration in hepatic tissue and shrinkage of central vein. (Fig 8)

Fishes exposed to the sublethal concentration of heavy metal Copper (Cu) as $CuSO_4$ at $1/10^{th}$ of 96 hrs (2 ppm) for a chronic period of 30 days recorded pathological changes in the liver like ruptured sinusoids, degeneration of connective tissue with vacuolation and necrosis in hepatocytes. (Fig 9)

The fishes exposed to the heavy metal Nickel (Ni) as $NiSO_4$ for a period of 24 hrs at 1200 ppm recorded marked pathological changes in liver as degeneration and rupture of sinusoids, vacuole formation between hepatocytes with necrosis and degeneration in connective tissue were observed. Destruction of bile canaliculi with degeneration of islets of langerhans was also seen. Intercellular space became wider due to connective tissue damage. (Fig 10)

The fishes exposed to the heavy metal Nickel (Ni) as $NiSO_4$ for a period of 96 hrs at 150 ppm show marked pathological changes in the liver like shrinkage of central vein, accumulation of blood cells in the central vein, rupture of sinusoids, loosening of hepatic tissue due to degeneration and necrosis in the connective tissue. Degeneration of islets of langerhans with hemorrhage in hepatic tissue was seen. (Fig 11)

Fishes exposed to the sublethal concentration of heavy metal Nickel (Ni) as $NiSO_4$ at 1/5th of 96 hrs (30 ppm) for a chronic period of 30 days revealed pathological changes in the liver like rupture of sinusoids, vacuole formaton in hepatic tissue with swelling of hepatocytes, necrosis and degeneration in hepatic tissue, degeneration of bile canalicule and shrinkage of central vein. (Fig 12)

Fishes exposed to the sublethal concentration of heavy metal Nickel (Ni) as $NiSO_4$ at $1/10^{th}$ of 96 hrs (15 ppm) for a chronic period of 30 days recorded pathological changes in the liver like ruptured sinusoids, degeneration of connective tissue, necrosis in hepatic tissue, destruction of bile canaliculi with disintegration of islets of langerhans. (Fig 13)

The fishes exposed to the heavy metal Zinc (Zn) as $ZnSO_4$ for a period of 24 hrs at 630 ppm recorded marked pathological changes in liver as degeneration and rupture of sinusoids, vacuole formation between hepatocytes with necrosis and degeneration in connective tissue were observed. Degeneration of islets of langerhans was also seen. Intercellular space became wider due to connective tissue damage. (Fig 14)

The fishes exposed to the heavy metal Zinc (Zn) as $ZnSO_4$ for a period of 96 hrs at 330 ppm showed marked pathological changes in the liver like shrinkage of central vein, accumulation of blood cells in the central vein, rupture of sinusoids with destruction of bile canaliculi. Vacuolation in the hepatic tissue, loosening of hepatic tissue due to degeneration and necrosis in the connective tissue was also seen. (Fig 15)

Fishes exposed to the sublethal concentration of heavy metal Zinc (Zn) as $ZnSO_4$ at $1/5^{th}$ of 96 hrs (66 ppm) for a chronic period of 30 days revealed pathological changes in the liver like vacuole formation with necrosis and degeneration in hepatic tissue, shrinkage of central vein. Destruction of bile canaliculi with vacuolation in islets of langerhans was also recorded. (Fig 16)

Fishes exposed to the sublethal concentration of heavy metal Zinc (Zn) as $ZnSO_4$ at $1/10^{th}$ of 96 hrs (33 ppm) for a chronic period of 30 days recorded pathological changes in the liver like degeneration of connective tissue, shrinkage of central vein, accumulation of blood cells in central vein, ruptured sinusoids, with vacuolation between hepatocytes. (Fig 16)

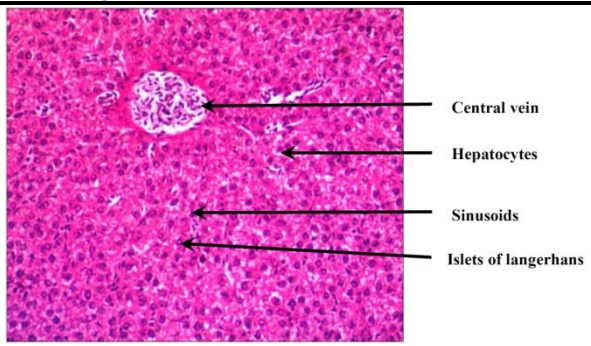

Fig 1: T. S. of Liver of *Channa gachua* (Normal) (H/E) (400X)

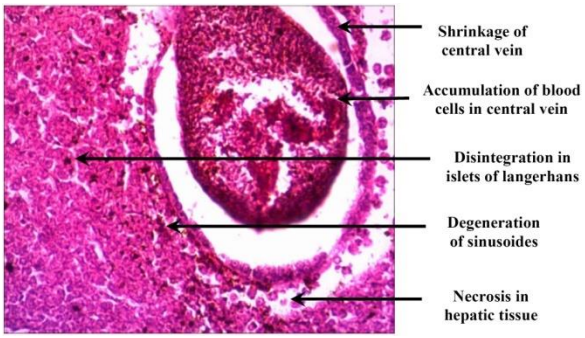

Fig 2: T. S. of Liver of *Channa gachua* after exposure to electroplating industry effluent at 20% concentration for a period of 24 hrs.(H/E) (400X)

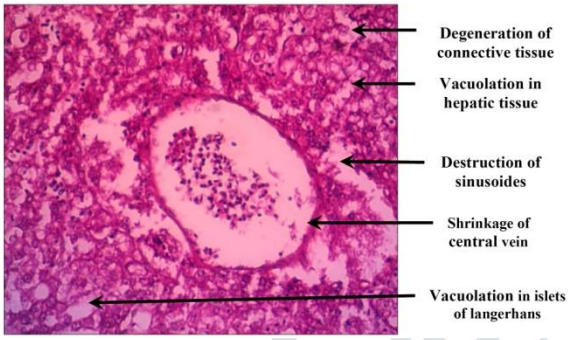

Fig 3: T. S. of Liver of *Channa gachua* after exposure to electroplating industry effluent at 18.5% concentration for a period of 96 hrs. (H/E) (400X)

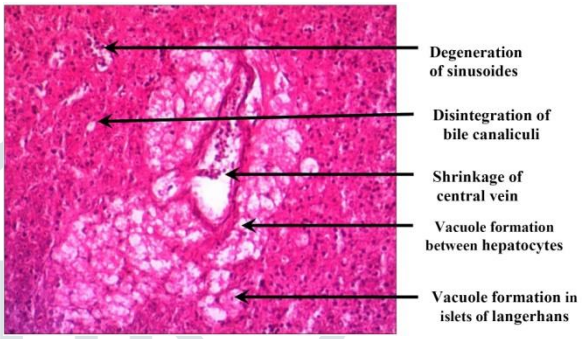

Fig 4: T. S.of Liver of *Channa gachua* after exposure to electroplating industry effluent at 1/5$_{th}$ of 96 hrs (3.70% concentration) for a chronic period of 30 days. (H/E) (400X)

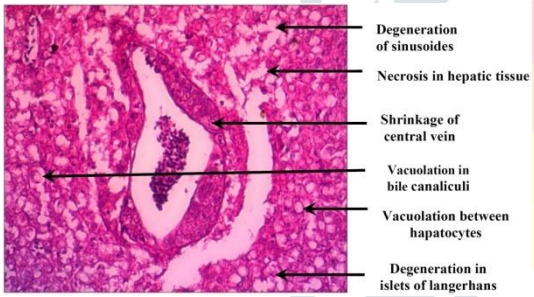

Fig 5: T. S.of Liver of *Channa gachua* after exposure to electroplating industry effluent at 1/10$_{th}$ of 96 hrs (1.85% concentration) for a chronic period of 30 days. (H/E) (400X)

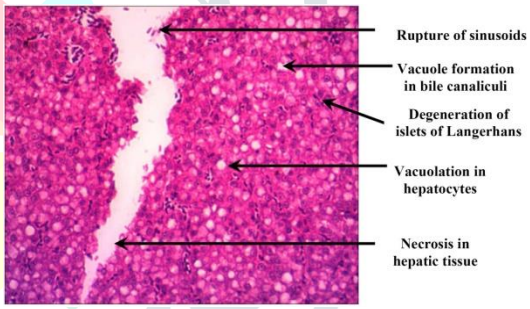

Fig 6: T.S of Liver of *Channa gachua* after exposure to heavy metal Copper (Cu) as $CuSO_4$ at 180 ppm for a period of 24 hrs. (H/E) (400X)

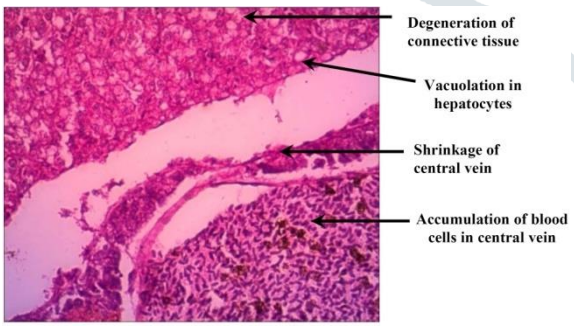

Fig 7: T.S. of Liver of *Channa gachua* after exposure to heavy metal Copper (Cu) as $CuSO_4$ at 20 ppm for a period of 96 hrs. (H/E) (400X)

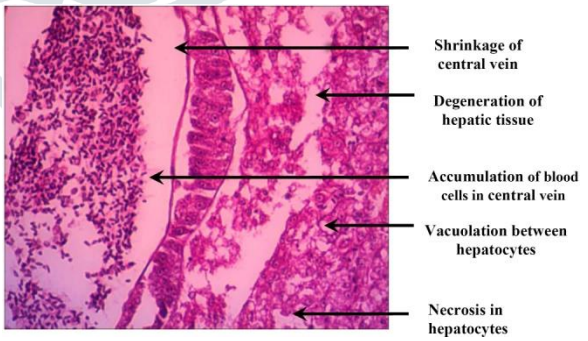

Fig 8: T.S of Liver of *Channa gachua* after exposure to heavy metal Copper (Cu) as $CuSO_4$ at 1/5$_{th}$ of 96 hrs (4 ppm) for a chronic period of 30 days. (H/E) (400X)

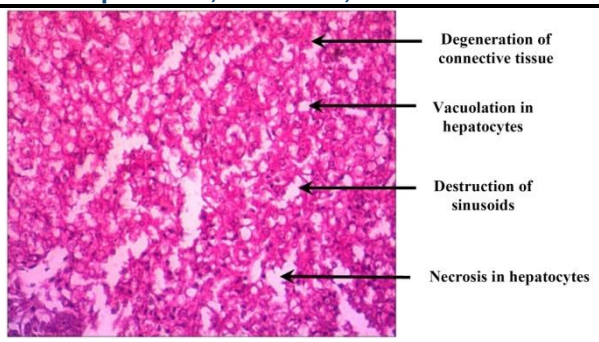

Fig 9: T.S of Liver of *Channa gachua* after exposure to heavy metal Copper (Cu) as $CuSO_4$ at $1/10_{th}$ of 96 hrs (2 ppm) for a chronic period of 30 days. (H/E) (400X)

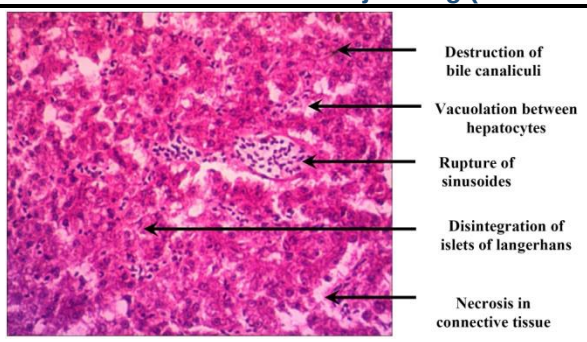

Fig 10: T.S of Liver of *Channa gachua* after exposure to heavy metal Nickel (Ni) as $NiSO_4$ at 1200 ppm for a period of 24 hrs. (H/E) (400X)

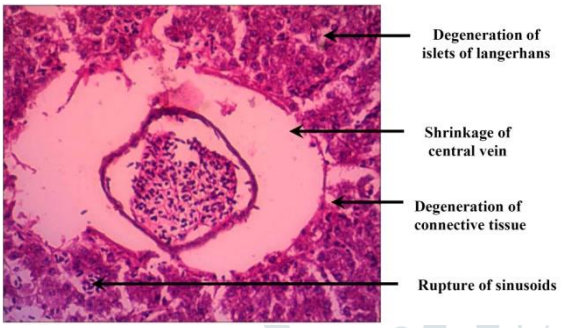

Fig 11: T.S of Liver of *Channa gachua* after exposure to heavy metal Nickel (Ni) as $NiSO_4$ at 150 ppm for a period of 96 hrs. (H/E) (400X)

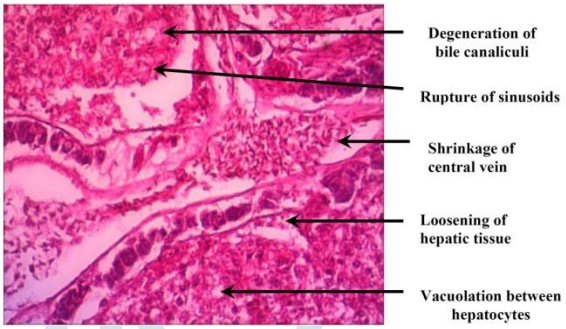

Fig 12: T.S of Liver of *Channa gachua* after exposure to heavy metal Nickel (Ni) as $NiSO_4$ $1/5_{th}$ of 96 hrs (30 ppm) for a chronic period of 30 days. (H/E) (400X)

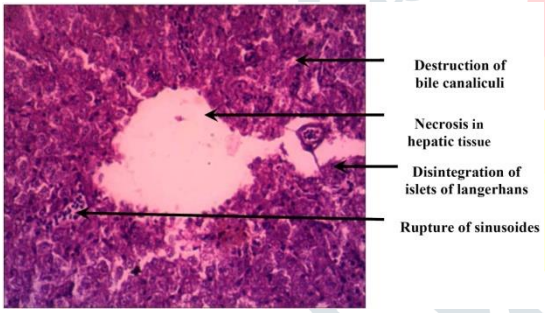

Fig 13: T.S of Liver of *Channa gachua* after exposure to heavy metal Nickel (Ni) as $NiSO_4$ at $1/10_{th}$ of 96 hrs (15 ppm) for a chronic period of 30 days. (H/E) (400X)

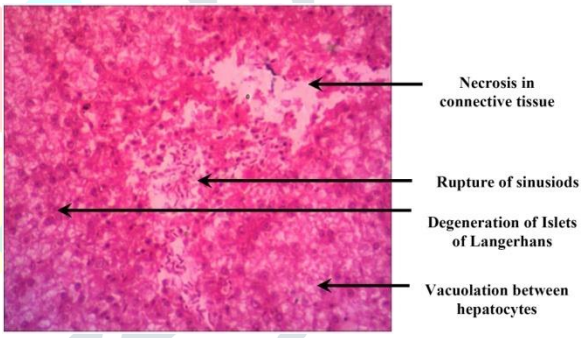

Fig 14: T.S of Liver of *Channa gachua* after exposure to heavy metal Zinc (Zn) as $ZnSO_4$ at 630 ppm for a period of 24 hrs. (H/E) (400X)

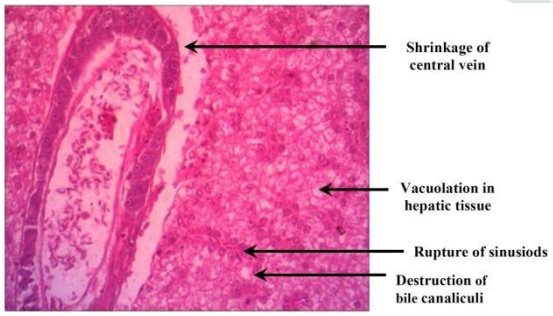

Fig 15: T.S of Liver of *Channa gachua* after exposure to heavy metal Zinc (Zn) as $ZnSO_4$ at 330 ppm for a period of 96 hrs. (H/E) (400X)

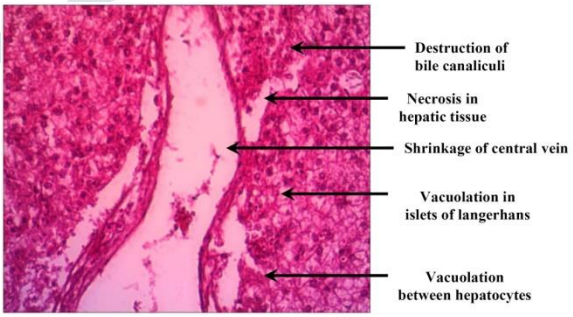

Fig 16: T.S of Liver of *Channa gachua* after exposure to heavy metal Zinc (Zn) as $ZnSO_4$ at $1/5_{th}$ of 96 hrs (66 ppm) for a chronic period of 30 days. (H/E) (400X)

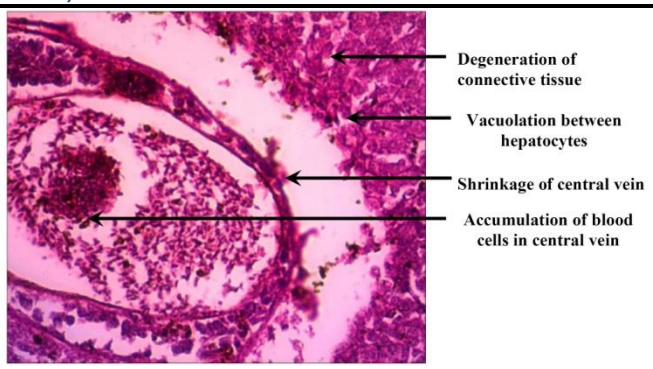

Fig 17: T.S of Liver of *Channa gachua* after exposure to heavy metal Zinc (Zn) as ZnSO4 at 1/10th of 96 hrs (33 ppm) for a chronic period of 30 days.(H/E) (400X)

V. DISCUSSION

The liver is the primary organ of detoxification of organic xenobiotics. Tissue changes in liver are linked with histological abnormalities of gills and kidney. Once absorbed, the toxicant is transported by blood circulation to liver for denaturation and is then finally passed in the blood for possible excretion by kidney or gills. Alterations in the liver are used as an important biomarker of environmental pollution. Histopathological alterations in liver of fishes on exposure to pollutants have been reported by many workers.

Sakr et al, 2001 studied the effect of organophosphorus insecticide (hastathion) on the liver of catfish, *Clarias gariepinus*. This insecticide produced pathological changes in liver like liver cord disarray, cytoplasmic vacuolation of hepatocytes, damaged blood sinusoids, blood vessel congestion and inflammatory leukocyte infiltration. Olojo et al, 2005 reported histopathological alterations in liver like necrosis, rupture of sinusoids, and breakage in central vein of lead exposed freshwater catfish, *Clarias gariepinus*. Loganathan et al, 2006 observed degeneration of hepatocytes and necrosis in liver of freshwater fish, *Labeo rohita* (Ham.) on Zinc exposure. Mishra et al, 2006 reported alterations in liver of freshwater teleost, *Channa punctatus* (Bolch.) and observed pyknotic nuclei, degeneration of hepatic cells, dilation of blood sinusoids, vacuolation of heptocytes and necrosis on acute exposure to pesticides.

Soni and Gupta, 2006 (a) reported degenerative changes like, vacuolar degeneration with focal areas of necrosis in liver of fish *Heteropneustes fossilis* under the toxic effect of $HgCl_2$ and EDTA. Chand et al, 2006 observed shrinkage in central vein, deposition of fibrous tissue around endothelial walls of central vein, complete collapse of sinusoidal openings of central vein, widening of sinusoid with infiltration of numerous macrophages and inflammatory cells and irregular shape of hepatocytes. Nassr- Allah, 2007 reported inflammation, central vein necrosis and cell degeneration in liver tissue of *Oreochromis auries* juveniles while subjecting them to phenol. Rana, 2007 observed disintegration of hepatic cells, separation of hepatic cells from blood vessels and proliferation in pancreatic tissue. He also observed broken hepatic cells and clear atrophy in the nucleus of the hepatic cells of urea exposed fish, *Anabas testudenius*.

The results in the present investigation showed several striking changes in the liver of *Channa gachua* exposed to lethal and sublethal concentration of electroplating industry effluent and heavy metassl Chromium, Copper, Nickel and Zinc caused severe degenerative changes like necrosis of hepatic cells, disintegration of islets of Langerhans, breakage and shrinkage of central vein, disorganized hepatic cords and loss of shape of hepatocytes, vacuolation in the cytoplasm, cloudy swelling in the nucleus with large vacuoles in the hepatocytes and dilation and rupture of sinusoids, necrosis in bile canaliculi were observed. Compact arrangement of hepatocytes became wider due to connective tissue damage. The present findings are concurrent with the findings Varanka et al, 2001. Nagarajan and Arunadevi, 2006., Shindhe et al, 2006., Athikeravan et al, 2006, Fernandes et al, 2007. Cellular disintegration in the liver might be due to O2 deficiency as a result of gill disintegration (Mohammed, 2001).

VI. CONCLUSION

In the present study, histopathology of liver of air breathing teleost, Channa gachua (Bloch). Experimental fishes exposed to electroplating industry effluent and 3 heavy metals - Cu, Ni and Zn are recorded. The target organs selected for study are Liver. The control fishes showed the normal architecture in the target organs whereas the experimental fishes showed histopathological alterations in the target organs such as degenerative changes in the cells, necrosis, general inflammatory responses and neoplasia/hyperplasia in the target organs. In general, the toxicity of heavy metals to the fishes is due to chemical reactivity of these ions with cellular structural proteins, enzymes and membrane

systems. Target organs of metal toxicities are usually those organs that accumulate the highest concentration of metals in vivo. This is often dependent on the route of exposure and the chemical compound of the metal, i.e., its valance state, volatility, lipid solubility, etc. Metals cause acute as well as chronic poisoning in fishes. In the present investigation, examination of tissues from organisms served to identify the cause of death and possibly the causative agent. Suitable preparations of affected, exposed tissues are observed under light and electron microscopes, for changes in the tissues. Several specific and non-specific microscopic lesions develop in the target tissues selected that are of diagnostic significance which can be studied and analysed because of the histopathological examinations. Thus it can be concluded that, the histopathological study in the present investigation has an important application in the field of toxicology.

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