Bangladesh J. Zool. ***52****(2): 237-251, 2024* *Article*
ISSN: 0304-9027
eISSN: 2408-8455

CADMIUM CHLORIDE INDUCED HISTOPATHOLOGICAL ALTERATIONS IN THE SELECTED ORGANS OF NILE TILAPIA *OREOCHROMIS NILOTICUS* (L.)

Shawkat Ara Begum, Mahabuba Hasnath* and Kazi Abdul Aziz

Department of Zoology, University of Chittagong, Chittagong 4331, Bangladesh

ABSTRACT: Histopathological alterations in the selected organs of a freshwater fish *Oreochromis niloticus* (L.), exposed to sub lethal concentration (17.5 ppm) of cadmium chloride ($CdCl_2$), were studied at laboratory conditions. Static bioassay test was carried out to determine the sub lethal concentration. The changes in gill, liver, kidney, stomach and intestine of the fish were observed after 7 and 30 days exposure. The degenerative changes exhibited in fish organs were more severe and pronounced at long term exposure than that of at short term. The histopathological changes observed in the gills were fusion of secondary lamellae due to hyperplasia and hypertrophy, lifting and rupture of lamellar epithelium, oedema of primary lamellar tip, necrosis, damage of pillar cell system, congestion in blood vessels and haemorrhagic gill rays. Changes in the liver included disruption of cords, highly chromatic and irregular shaped shrunken hepatocytes, pyknotic nuclei, rupture and loss of cell boundaries, congestion in blood vessels and sinusoid, and presence of yellow brown bodies. Degeneration of tubular epithelium, tubular lumen with cellular debris, necrotic and swollen glomeruli, and haemopoietic tissue degeneration were marked in the kidney of exposed fish. Stomach tissues revealed rupture and separation of mucosal epithelium, swollen epithelial cells, degeneration of gastric gland cells, scattered blood cells all over the tissue and necrosis in the mucosal epithelium and lamina propria. In intestine, disintegration and rupture in mucosal epithelium, sloughing off epithelial and mucous cells, blood congestion in lamina propria, increased goblet cells, etc., were observed during the present study.

Key words: Histopathology, cadmium chloride, *Oreochromis niloticus,* gill, liver, kidney, stomach, intestine.

INTRODUCTION

Environmental pollution, especially aquatic environment, is now a global concern. Among the pollutants, heavy metals, recognized as the strong biological poisons because of their persistent nature, toxicity, tendency to get

**Author for correspondence:* <mahabuba@cu.ac.bd>
 DOI: https://doi.org/10.3329/bjz.v52i2.77285

accumulate in organisms and bio magnification (Dinodia *et al.* 2002), are the major contaminants of the aquatic environment. The levels of these natural trace components are increasing, at alarming rates, due to effluent of both industrial and household wastes, geo-chemical structure, agricultural and mining activities (Ali *et al.* 2019). Some heavy metals, at low levels, are essential for living organisms (Akter *et al.* 2021, Rohani *et al.* 2022), others, even at a trace level, are toxic and detrimental (Sardar *et al.* 2013, Sarkar *et al.* 2016). Among the aquatic organisms, fish accumulate heavy metals and affected adversely (Baki *et al.* 2019, Mohanta *et al.* 2019).

Cadmium is one of the major contaminant of aquatic ecosystem, having acute and chronic effects on the environment even at very low concentration (Islam *et al.* 2017). Cadmium related contamination of the aquatic ecosystem has greatly increased in the last few decades and have constantly being increasing (Giles 1988). Cadmium is released into the aquatic environment from wide range of natural and anthropogenic sources (Ali *et al.* 2019, Shukla *et al.* 2023). Waterborne cadmium can be accumulated in different organs of fish from polluted water bodies (Rajeshkumar and Li 2018, Parvin *et al.* 2019, Pinkey *et al.* 2024), and also from commercial fish feeds contaminated with heavy metals (Saha *et al.* 2021, Sarkar *et al.* 2022, Bhowmik *et al.* 2023) resulting in varying degree of structural and functional alterations (Zeitoun and Mehana 2014, Shahjahan *et al.* 2022).

Oreochromis niloticus (L.), commonly called tilapia is a part of the daily diet of more than 50% of the population (Islam *et al.* 2021). It is widely cultured in Bangladesh to meet the increased protein demand and due to its profitability, rapid growth rate, high market demand and increased consumer acceptance (ADB 2005).

Histological studies, along with physiological and biochemical data, provide the more complete and accurate description of the activity of a chemical agent (Mehrle and Mayer 1985). It is the basic effort for understanding the real impact of pollutants on the ecosystem (Gardner and Laroche 1973) and provides the first detectable indicator of injury in cells, tissues and organs (Baki *et al.* 2019). Information on metal and organ specific histopathological changes in fish is very scarce in Bangladesh. So, the present study was aimed to investigate the histopathological alterations in different organs of *Oreochromis niloticus* (L.) exposed to sub lethal concentration of cadmium chloride ($CdCl_2$) under laboratory conditions.

MATERIAL AND METHODS

Collection of fish and their acclimatization: Live specimens of *Oreochromis niloticus* (L.), collected from a local hatchery, were brought immediately to the

laboratory of the Department of Zoology, University of Chittagong for acclimatization to laboratory conditions. The experiment was carried out with only live and healthy acclimatized specimens. Feeding was stopped for 24 hours before starting the bioassay experiment.

Dose and duration: The fish were exposed to sub lethal concentration (17.5 ppm) of $CdCl_2$ salt for short (7days) and long term (30 days).

Collection and fixation of the organs: After the stipulated time, tissues from gill, liver, kidney, stomach and intestine were collected from both control and $CdCl_2$ treated specimens.

Preparation of histological slides: Histological slides were prepared following Humason (1972). Transverse and cross sections of tissues were taken by a rotary microtome (OSK, 8225, OGAWA SEIKI CO. LTD) at 3-5µm thickness. The photomicrographs of the histological slides were taken by using a digital camera (Cannon Power Shot A 640). Normal tissue structure was studied following Bevelander (1965), Reith and Ross (1966), Khanna (1988) and Junqueira *et al.* (1995).

RESULTS AND DISCUSSION

Histopathological alterations in the gill, liver, kidney, stomach and intestine of fish *Oreochromis niloticus* (L.) due to short (7 days) and long term (30 days) exposure to sub lethal concentration (17.5 ppm) of cadmium chloride ($CdCl_2$) were observed. The degenerative changes in fish organs were more severe and pronounced at long term exposure than that of at short term.

Effects on gill: The gill of *O. niloticus* (control) show the typical pattern of normal gill histology consisting of primary gill lamellae borne by gill arches and secondary gill lamellae lined by a single layer of squamous epithelium. Pillar cells system and blood capillaries present between the two layers of epithelia of the secondary lamella. Between the secondary gill lamellae, the primary gill lamellae are lined by a thick stratified epithelium, constituted by chloride, mucous and pavement cells (Fig. 1. A). $CdCl_2$ induced extensive changes in the gill structure of *O. niloticus* during 7 days exposure at sub lethal concentration included cellular proliferation (hyperplasia) and swelling of the epithelial cells of the lamellar epithelium causing fusion of secondary lamellae, resulting in disappearance of inter lamellar spaces between them, hypertrophied gill rays and excessive blood congestion and necrotic areas in the gill lamellar epithelium, detachment and rupture of lamellar epithelium at the base of the primary lamellae with mucous cell congestions, damaged blood vessels and oedema at tips of the primary lamellae (Fig. 1. B and C). The degenerative changes in fish gill at long term (30 days) exposure to $CdCl_2$ included complete fusion of secondary lamellae due to hyperplasia and hypertrophy of the

epithelium, degenerated pillar cell systems having congestion of blood vessels and hemorrhagic gill ray duo to rupture of blood capillaries, necrosis and hypertrophied cells with pyknotic nuclei, and lifting of the lamellar epithelium at the base (Fig. 1. D).

The fish gills, participate in respiration, osmoregulation and excretion, remain in close contact with the external environment and particularly sensitive to changes, even minor chemical or physical, in the surrounding water, are the primary target of the contaminants (Movahedinia *et al.* 2009). $CdCl_2$ exposure can alter the gill morphology and damaged the gill shape (Adam *et al.* 2019). The severity of the alterations increased with dose and time (Otlodil *et al.* 2017).

Like the present study, hypertrophy and hyperplasia of the lamellar epithelium and fusion of the secondary lamellae were also reported in tilapia (Otlodil *et al.* 2017, Gulzar *et al.* 2023) and many other fish species (Ahmed *et al.* 2014, Jayakumar and Subburaj 2017) exposed to cadmium. Initially, such changes represent an adaptation to new conditions and is a protecting mechanism against excessive penetration of toxins from water to the blood vessels in the gills and thus to the blood (Hinton and Lauren 1990, Fernandes and Mazon 2003).

Lifting of lamellar epithelium, was a common symptom exhibited by the present cadmium stressed fish gills. The oedematous separation of gill epithelium from the basement membrane, due to the increased capillary permeability or lowered efficiency of the epithelial cells in maintaining normal water balance (Roberts 2001) is often associated with cell infiltrates and is a symptom of disorders of osmoregulation (Movahedinia *et al.* 2012). In the present investigation, degenerative changes observed in gills, specially damaged pillar cell system, which were more prominent in long term exposure, might affect the gill by reducing the supply of blood and can cause respiratory impairment (Gupta and Rajbanshi 1988).

Effects on liver: The normal hepatocyte has a homologous cytoplasm and a large central or sub-central spherical nucleus. The parenchymatous cells separated by blood sinusoids, and portal veins, hepatic arteries, hepatic veins and bile ducts were distributed throughout the liver (Fig. 2. A and B).

The marked histological changes in the liver of *O. niloticus* at 7 days exposure included degeneration of cell membrane, cytoplasm and nucleus resulting in loss of its normal polygonal shape. The cytoplasm was highly chromatic and vacuolated. Nuclei pyknotic, atrophied, displaced and reduced in size and shape. Blood vessels and sinusoids were congested, and hemolysis in blood vessels and hepatoportal vessels observed. Brown bodies were found all over the liver tissue (Fig. 2. C).

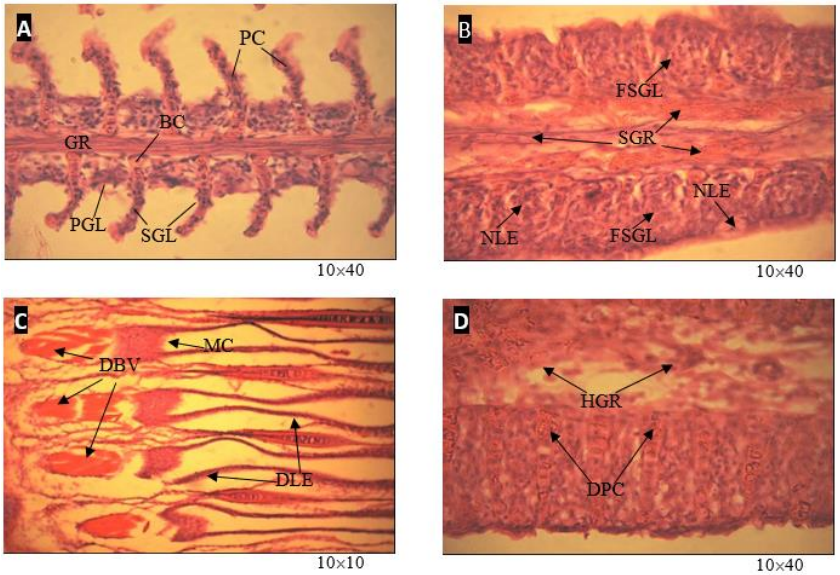

Fig. 1. Photomicrographs of gill of *Oreochromis niloticus*: A. Control; B and C. At 7 days exposure to 17.5 ppm $CdCl_2$; D. At 30 days exposure to 17.5 ppm $CdCl_2$. Abbreviations: BC -Blood capillary; DBV - Damaged blood vessel; DLE - Detachment of lamellar epithelium; DPC -Degenerated pillar cell system with blood congestion; FSGL - Fused secondary gill lamellae; GR - Gill ray; HGR - Haemorrhagic gill ray; MC - Mucous cell congestion; NLE - Necrotic areas in the lamellar epithelium; PC - Pillar cells; PGL - Primary gill lamellae; RBV - Ruptured blood vessel; SGL- Secondary gill lamellae; SGR - Swollen gill ray.

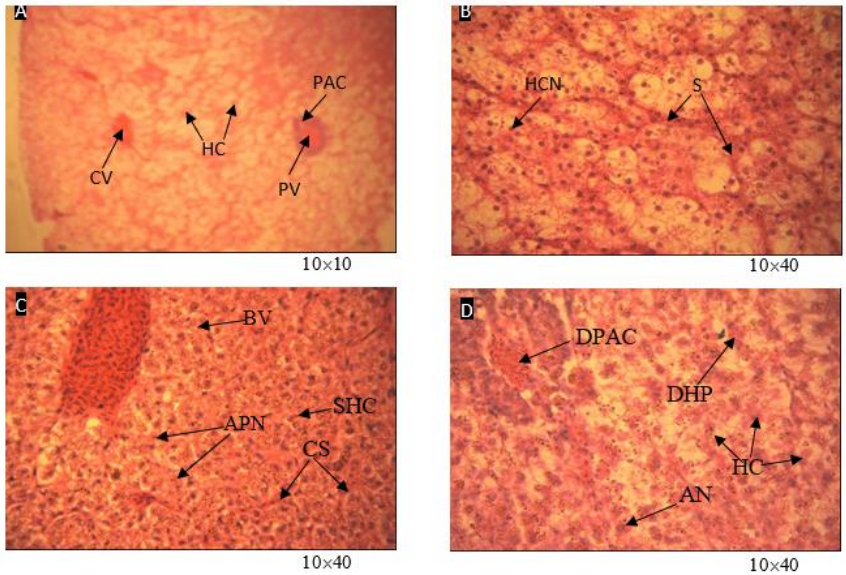

Fig. 2. Photomicrographs of liver of *Oreochromis niloticus*: A. and B. Control; C. At 7 days exposure to 17.5 ppm $CdCl_2$; D. At 30 days exposure to 17.5 ppm $CdCl_2$. Abbreviations: AN - Aggregation of nuclei; APN - Atrophied pyknotic nuclei; BV - Blood vessel; CS - Congested sinusoids; CV - Central vein; DHP - Disruption of the arrangement of hepatic parenchyma; DPAC - Disintegrated pancreatic acinar cell; HC - Hepatic cells; HCN - Hepatic cell with central or sub central nucleus; PAC - Pancreatic acinar cell; PV - Portal vein; S - Sinusoids; SHC - Shrinkage of hepatocyte.

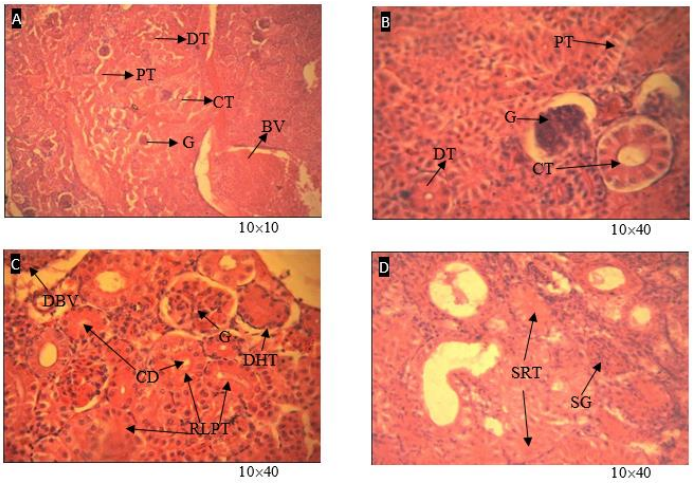

Fig. 3. Photomicrographs of kidney of *Oreochromis niloticus*: A. and B. Control; C. At 7 days exposure to 17.5 ppm $CdCl_2$; D. At 30 days exposure to 17.5 ppm $CdCl_2$. Abbreviations: BV - Blood vessel; CD - Cellular debris; CT - Collecting duct; DBV - degenerated blood vessel; DHT - Degenerated haemopoietic tissue; DT - Distal tubule; G - Glomerulus; PT - Proximal tubule; RLPT - Reduced lumen of the proximal tubules; SG - Swollen glomerulus; SRT - Swollen epithelial cells of the renal tubule.

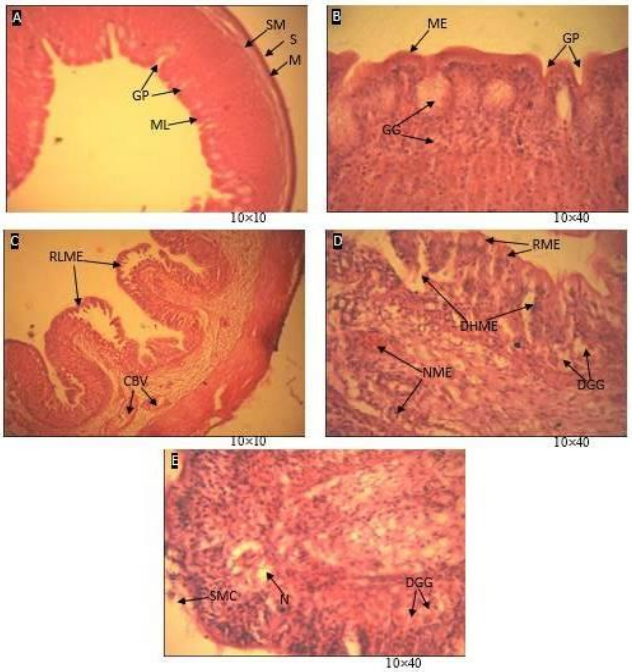

Fig. 4. Photomicrographs of stomach of *Oreochromis niloticus*: A. and B. Control; C. and D. At 7 days exposure to 17.5 ppm $CdCl_2$; E. At 30 days exposure to 17.5 ppm $CdCl_2$. Abbreviations: CBV - Congested blood vessel; DGG -Degenerated gastric glands; DHME – Degenerated, hypertrophied mucosal epithelial cell; GG - Gastric glands; GP - Gastric pits; M - Muscularis; SMC - Sloughed off mucous cells in the lumen; ME - Mucosal epithelium; ML - Mucosal layer; N -Necrosis; NME - Necrosis in mucosal epithelium and lamina propria; RLME - Rupture and lifting of mucosal epithelia; RME - Ruptured mucosal epithelium; S – Serosa; SM – Submucosa.

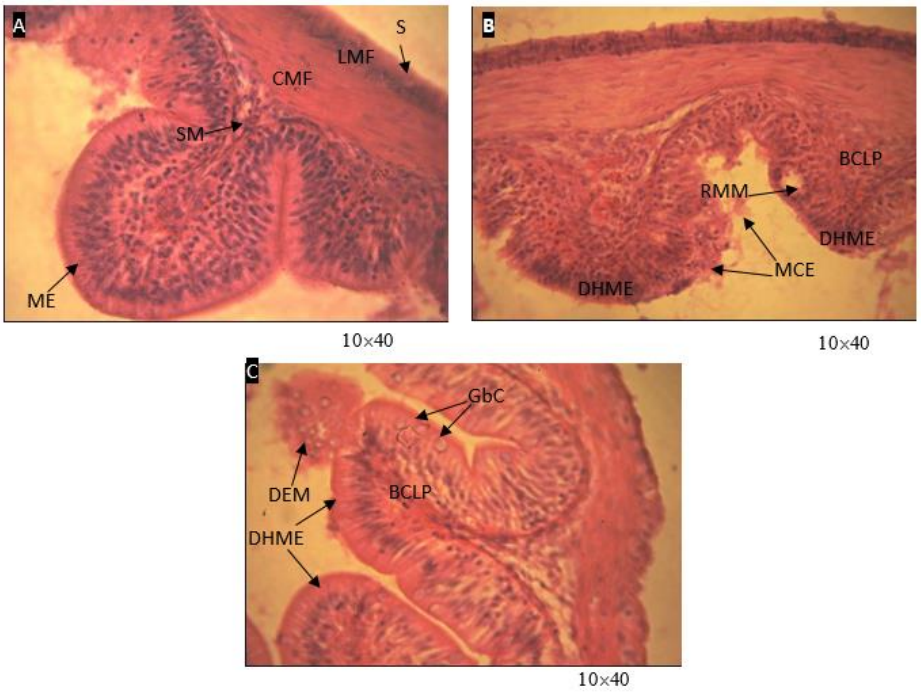

Fig. 5. Photomicrographs of intestine of *Oreochromis niloticus*: A. Control; B. At 7 days exposure to 17.5 ppm $CdCl_2$; C. At 30 days exposure to 17.5 ppm $CdCl_2$. Abbreviations: BCLP -Blood congestion in the lamina propria; CMF - Circular muscle fibre; DEM - Disintegration of epithelial and mucous cells at the tip of the villi; DHME - Degenerated, hypertrophied mucosal epithelial cells; GbC - Goblet cells; LMF - Longitudinal muscle fibre; MCE - Mucous and epithelial cells exudation; ME - Mucosal epithelium; RMM - Ruptured mucosal membrane; S - Serosa; SM - Submucosa.

During the long term exposure, the hepatic cells were extremely damaged, cytoplasm eosinophilic, atrophied with pyknotic nuclei. Normal cordal arrangement of the hepatic parenchyma was disrupted. Due to loss of the cell boundaries many nuclei were found to aggregate at one place bearing empty areas beside in different places of hepatic parenchyma and these were prominent near the cell boundaries. Sinusoids and blood vessels were congested and brown pigments were scattered all over the tissues. Pancreatic acinar cells also disintegrated (Fig. 2. D).

Liver, mostly were associated with the detoxifications and biotransformation process and due to its function, position and blood supply it is also one of the organs heavily affected by contamination of water (Camargo and Martinez 2007).

The degenerative changes like hepatocyte degeneration, pyknotic nuclei, necrosis, vacuolation, congestion in blood vessels and sinusoid, etc., reported during the present study, were observed in *O. niloticus* exposed to $CdCl_2$ (Omer

et al. 2012, Younis *et al.* 2013) and waterborne heavy metals (Nofal *et al.* 2019, Musa and Imam 2023).

The duration dependent changes, found presently, corroborates with Dar *et al.* (2011) and these changes may be attributed to direct toxic effects on hepatocytes since the liver is the only site of detoxification (Soufy *et al.* 2007). Vacuolation of hepatocytes, seen in the present investigation, might indicate an imbalance between the rate of synthesis of substances in the parenchymal cells and the rate of their release into the circulation system (Gingerich 1982).

The bile stagnation in liver, in most fish studied, in the form of brownish yellow granules in the cytoplasm of the hepatocytes, indicates that bile is not being released from the liver (Pacheco and Santos 2002). The presence of brown bodies in the liver tissue might be the cause of metabolic problem of the liver due to histopathological changes (Camargo and Martinez 2007).

Effects on kidney: The kidney of *O. niloticus* is made up of numerous nephrons, each consisting of a renal corpuscle, renal tubule and collecting duct. The kidney tissue of the control fish exhibited an ordinary pattern with no abnormal changes in the cells (Fig. 3. A and B). At short term exposure, kidney tissue revealed inflammation of the epithelial cells with pyknotic nuclei of the renal tubules. Lumens of proximal tubules reduced and have cellular debris. Swollen glomeruli resulting in the reduction of Bowman's capsule space, many were atrophied also. Haemopoietic tissue and blood vessels were degenerated (Fig. 3. C). Degenerative changes recorded at 30 days exposure were heavily hypertrophied proximal and distal renal tubular cells, separating off from the basement membrane, obliterating their luminal space but the lumen of the collecting ducts were spacious with cellular debris. Cystic proximal tubules due to complete sloughing of the lining cells. Swollen and necrosed glomeruli. Haemopoietic tissue degenerated and fragmented. The renal tubular cells were acidophilic (eosinophilous) in contrast to their normal basophilic condition (Fig. 3. D).

The kidney, organ to maintain homeostasis, is severely affected by contaminants of the water (Thophon *et al.* 2003). Like the present study, tubular degeneration and dilation of capillaries in the glomerulus and reduction of Bowman's space were reported in the kidney of fishes exposed to acute and chronic exposer to cadmium (Kaoud *et al.* 2011, Salman *et al.* 2015) and combination of heavy metals (Vinodhini and Narayanan 2009). The reduced renal tubular lumen, specially the proximal tubules, and spacious collecting duct lumen with cellular debris, etc., were much prominent in long term exposure (30 days). Similar pathological symptoms observed in different fish species exposed to Cd (Gupta and Rajbanshi 1988, Kaoud *et al.* 2011) were

regarded to be due to an alteration in the metabolic activity, resulting in a disorder of the useful divalent ions transport (Oronsaye 1989).

Highly degenerated epithelial cells of the kidney tubules, even ruptured and detached from the basement membrane and became cystic which are in consistent with that of Vinodhini and Narayanan (2009).

Swollen glomeruli, blood vessels congestion and haematopoietic tissue degeneration, were the other alterations in the kidney tissue of the presently studied fish that were also in agreement with the findings of Begum *et al.* (2001) and Mohamed (2009), working with different fish species exposed to different toxicants.

Effects on stomach: The stomach tissue of the fish *O. niloticus* in control, showed the normal arrangement of the mucosal, submucosal, muscularis and serosa layer. The inner most mucosal layer raised into primary and secondary folds, composed of columnar cells, gastric glands and lamina propria. The epithelial cells of the mucosal membrane and gastric glands exhibited normal size and shape (Fig. 4. A and B).

Degenerative changes in the stomach tissue of *O. niloticus*, at 7 days exposure included rupture and lifting of the mucosal epithelial layer, necrosis in the lamina propria and mucosal membrane, hypertrophied and degenerated epithelial cells with pyknotic nuclei, damaged gland cells and congested blood vessels. No change was observed in the submucosa and muscularis layer except excess blood cells all over the tissue (Fig.4. C and D). After 30 days exposure, almost same but more prominent, degenerative changes reported were hypertrophy of the epithelial cells of the mucosal membrane with pyknotic nuclei, rupture and damage of the mucosal epithelium, necrosis in the epithelium and lamina propria. Sloughed off mucous found in the lumen. Epithelial cells of the gastric glands hypertrophied, degenerated and aggregated in the middle, separating off from the basement membrane. Scattered blood cells were found all over the tissue (Fig. 4. E).

The stomach is a target organ for accumulation of cadmium on acute and sub-acute exposures (Tanhan *et al.* 2005). The visible alterations in stomach histology, found presently, were also observed in the stomach tissue of *O. niloticus* exposed to $CdCl_2$ (Kaoud *et al.* 2011) and other fishes exposed to different heavy metals (Begum *et al.* 2001 and 2010).

The most notable alterations in the stomach tissue of the present experimental fish, were rupture and lifting of mucosal epithelium and rupture within the mucosal layer. The hypertrophy of the epithelial cells of the mucosal membrane and digestive glands is known to be a protective mechanism that usually shown in response to chemical or toxic stresses (Roberts 2001).

Effects on intestine: The typical intestinal wall is composed of the mucosa, sub mucosa, muscularis and serosa that form villi. In the present study intestinal tissue of the fish *O. niloticus* in control, was normal in their structure and showed no alterations in the histology (Fig. 5. A). In the intestinal tissue of the 7 days exposed *O. niloticus* degenerative alterations included rupture of mucosal membrane, mucous and sloughed off epithelial cells, hypertrophied epithelial cells with pyknotic nuclei of the mucosal epithelium, disintegration of epithelial cells at the tip of the villi, congestion of blood in the mucosal epithelium and lamina propria (Fig. 5. B).

The epithelial cells of the mucosal layer were extremely hypertrophied and degenerated, specially tips of the villi, increased goblet cells with wide apical tips and mucous secretion along with sloughed off cells in the lumen of the intestine was prominent (Fig. 5.C) after 30 days of exposure.

Metals uptake mainly occur via gills, but may enter the digestive tract of fish via consumption of food and water, causing a deterioration of structures and functions in the gut (Bano and Hasan 1990, Banerjee and Bhattacharya 1995). The presently found severe degenerative and necrotic changes, desquamation and congestion of submucosal blood vessels in the intestinal mucosa and infiltration of inflammatory cells in intestine were associated with cadmium toxicity (Omer *et al.* 2012) and the oedema between the submucosa and mucosa may be due to absorption of toxic heavy metals (Hanna *et al.* 2005). The pathological alterations, found in the intestine of the present studied fish, were agreement with the observations of Younis *et al.* (2013) and Kaoud *et al.* (2011) and were also common in different fish species exposed to different toxicants (Mohamed 2009, Salman *et al.* 2015). The increased number of goblet cells, blood congestions, degeneration in the connective tissue of the lamina propria and scattered blood cells all over tissue, exhibited in the present study, were regarded as response to the irritation of internal mucosa by the metals (Crespo *et al.* 1986).

CONCLUSION

Exposure to sub lethal concentration (17.5 ppm) of cadmium chloride ($CdCl_2$) caused remarkable histopathological alterations in the gill, liver, kidney, stomach and intestine of *Oreochromis niloticus* (L.), which became more severe and pronounced at long term exposure than that of at short term. The degenerative changes in these vital organs might have resulted in physiologic and metabolic dysfunctions, which further led to behavioral alterations and growth impairment. In the long-run, therefore, cadmium chloride exposures to, even, sub lethal concentrations may pose serious threat to fish health and their population, eventually to human.

LITERATURE CITED

ADAM, M.A., MAFTUCHB, M., KILAWATIB, Y. and RISJANIB, Y. 2019. The effect of cadmium exposure on the cytoskeleton and morphology of the gill chloride cells in juvenile mosquito fish (*Gambusia affinis*). *Egyptian Journal* of *Aquatic Research*, **45**: 337–343. https://doi.org/10.1016/j.ejar.2019.11.011.

ADB (Asian Development Bank). 2005. An impact evaluation of the development of genetically improved farmed Tilapia and their dissemination in selected countries. Operations Evaluation Department, Asian Development Bank, Manila, Philippines.

AHMED, M.K., PARVIN, E., ISLAM, M.M., AKTER, M.S., KHAN, S. and ALMAMUN, M.H. 2014. Lead- and cadmium-induced histopathological changes in gill, kidney and liver tissue of freshwater climbing perch *Anabas testudineus* (Bloch, 1792). *Chemical ecology*, **30**: 532-540. DOI: 10.1080/02757540.2014.889123.

AKTER, S., JAHAN, N., ROHANI, M.F., AKTER, Y. and SHAHJAHAN, M. 2021. Chromium supplementation in diet enhances growth and feed utilization of striped catfish (*Pangasianodon hypophthalmus*). *Biological Trace Element Research*, **199**: 4811–4819.

ALI, H., KHAN, E. and ILAHI, I. 2019. Environmental chemistry and ecotoxicology of hazardous heavy metals: Environmental persistence, toxicity, and bioaccumulation. *Journal of Chemistry*, 1–14. doi: 10.1155/2019/6730305.

BAKI, M.A., SHOJIB, M.F.H., SEHRIN, S., CHAKRABORTY, S., CHOUDHURY, T.R., BRISTY, M.S., AHMED, M.K., YUSOFF, S.B. and KHAN, M.F. 2019. Health risk assessment of heavy metal accumulation in the Buriganga and Turag River systems for *Puntius ticto*, *Heteropneustes fossilis*, and *Channa punctatus*. *Environmental Geochemistry and Health*, **42**:531–543. https://doi.org/10.1007/s10653-019-00386-4.

BANERJEE, S. and BHATTACHARYA, S. 1995. Histopathological changes induced by chronic nonlethal levels of elsan, mercury and ammonia in the small intestine of *Channa punctatus* (Bloch). *Ecotoxicology and Environmental Safety*, **31**: 62–68.

BANO, Y. and HASAN, M. 1990. Histopathological lesions in the body organs of cat-fish (*Heteropneustes fossilis*) following mercury intoxication. *Journal of Environmental Science and Health*, Part B, **25(**1): 67-85. DOI: 10.1080/03601239009372677.

BEGUM, S.A., KHANAM, F. and HOQUE, B. 2001. Effects of chromium on histological structures of different organs of *Clarias gariepinus* (Burchel). *The Chittagong University Journal of Biological Science*, **25**(1): 1-10.

BEGUM, S.A., BANU, Q. and HOQUE, B. 2010. Histopathology of stomach tissue of catfish magur, *Clarias batrachus* under chromium, cadmium and mercury stress. *The Chittagong University Journal of Biological Science*, **5**(1 &2):09-17.

BEVELANDER, G.A.B. 1965. *Essentials of Histology.* 5th eds. The C.V. Mossby Company, Saint Louis and Toppan Company, LTD, Tokyo, Japan. Pp.329

BHOWMIK, S., DEWANJEE, S., ISLAME, S., SAHAA, D., BANIKA, P., HOSSAINF, M.K., RAHMANG, M., AL MAMUN, M.Z.U. and MAMUN, A. 2023. Nutritional profile and heavy metal contamination of nursery, grower, and finisher feeds of tilapia (*Oreochromis niloticus*) in Bangladesh. *Food Chemistry Advances*, **2**:100235. https://doi.org/10.1016/j.focha.2023.100235.

CAMARGO, M.M.P. and MARTINEZ, C.B.R. 2007. Biochemical and physiological biomarkers in *Prochilodus lineatus* submitted to *in situ* tests in an urban stream in southern Brazil. *Environmental Toxicology and Pharmacology*, **21**: 61-69.

CRESPO, S., NONNOTTE, G., COLIN, D.A., LERAY, C., NONNOTTE, L. and AUBREE, A. 1986. Morphological and functional alteration induced in trout intestine by dietary cadmium and lead. *Journal of Fish Biology*, **28**: 69- 80.

DAR, B.A., QURESHI, T.A. and KHALIQ, R. 2011. Effect of Cadmium chloride on the Histoarchitecture of certain organs of a freshwater catfish, *Clarias batrachus*. *Our Nature*, **9**: 83-88.

DINODIA, G.S., GUPTA, R.K. and JAIN, K.L. 2002. Effect of cadmium toxicity on liver glycogen in some fresh water fishes. *Proceedings of National Symposium on Environment*, **11**: 236-238.

FERNANDES, M. N. and MAZON, F. 2003: Environmental pollution and fish gill morphology. In: *Fish Adaptations* (Eds. Val, A.L. and Kapoor, B.G.) Enfield Science Publishers, pp. 203–231.

GARDNER, G.R. and LAROCHE, G. 1973. Copper induced lesions in estuarine teleosts. *Bangladesh Journal of Fisheries Research*, **30**:363-368.

GILES, M. A. 1988. Accumulation of cadmium by rainbow trout, *Salmo gairderi*, during extended exposure. *Canadian Journal of Fisheries and Aquatic Sciences*, **45**: 1045-1053.

GINGERICH, W.H. 1982. Hepatic toxicology in fish. In *Aquatic Toxicology* (Ed. Weber, L.J.) New York, Raven Press, pp. 55-105.

GULZAR, R., RIAZ, Z., GILLANI, Q.U.A., MEHREEN, A., JAMEEL, F. and NAWAZ, R. 2023. Bioaccumulation and influence of cadmium chloride on histology of muscles and gills in Nile tilapia (*Oreochromis niloticus*). *Journal of Survey in Fishery Sciences*, **10**(2): 992-1000. https://doi.org/10.53555/sfs.v10i2.1509.

GUPTA, A.K. and RAJBANSHI, V.K. 1988. Acute toxicity of cadmium to *Channa punctatus* (BLOCH). *Acta Hydrochimica et Hydrobiologica*, **16**(5): 525-535. http:// dx.doi.org/10.1002/aheh.19880160513.

HANNA, M.I., SHAHEED, I.B. and ELIAS, N.S. 2005. A contribution on chromium and lead toxicity in cultured *Oreochromis niloticus*. *Egyptian Journal of Aquatic Biology and Fisheries*, **9**(4): 177-209.

HINTON, D.E. and LAUREN D.J.1990: Liver structural alterations accompanying chronic toxicity in fish: potential biomarkers of exposure. In: *Biomarkers of Environmental Contamination* (Eds. McCarthy, J.F. and Shugart, L.R.) Lewis Publishers, Boca Raton. 51–65.

HUMASON, G.L. 1972. *Animal Tissue Techniques*. Eds. 3. San Francisco: W.H. Freeman and Company.

ISLAM, M.D., RAHAMAN, A. and JANNAT, F. 2017. Assessment of micro and macro nutrients in poultry feeds available in Dhaka city, Bangladesh. *Journal of Agricultural Science*, **1**:264–268.

ISLAM, S., BHOWMIK, S., HOSSAIN, M.K., NORDIN, N., RAHMAN, M., AHMMED, M.K., PARVIN, A. and HOSSAIN, M.A. 2021. Tilapia from most of the sources in Bangladesh are safe for human

consumption: A Hazard Index (HI) based study on heavy metals. *Journal of Aquatic Food Product Technology*, **30**(8):1017-1027.

JAYAKUMAR, N. and SUBBURAJ, A. 2017. Sub-lethal cadmium toxicity induced histopathological alterations in the gill, liver and kidney of freshwater catfish (*Heteropneustes fossilis*). *Journal of Entomology and Zoology Studies,* **5**(5): 1339-1345.

JUNQUEIRA, L.C., CARNEIRO, J. and KELLY, R.O. 1995. *Basic Histology*, 8th eds. Prentice – Hall International, Inc. 269-301.

KAOUD, H.A., ZAKI, M.M., EL-DAHSHAN, A.R., SAEID, S., and EL ZORBA, H.Y. 2011. Amelioration the toxic effects of cadmium exposure in Nile tilapia (*Oreochromis niloticus*) by using *Lemna gibba* L. *Journal of Life Sciences*, **8**: 185-195.

KHANNA, S.S. 1988. *An Introduction of Fishes*. Central Book Depot. Allahabad. 61-92 pp.

MEHRLE, P.M. and MAYER, F.L. 1985. Biochemistry/physiology. In: *Fundation of Aquatic Toxicology* (Eds. Rand, G.M. and Petrocelli, S.R.) Hemisphere Publishing Co., London, pp. 264–269.

MOHAMED, F.A. 2009. Histopathological studies on *Tilapia zillii* and *Solea vulgaris* from Lake Qarun, Egypt. *World Journal of Fish and Marine Sciences*. **1**(1): 29-39.

MOHANTA, L.C., NILOY, M.N.H., CHOWDHURY, G.W., ISLAM, D. and LIPY, E.P. 2019. Heavy metals in water, sediment and three fish species of Dhaleshwari river, Savar. *Bangladesh Journal of Zoology*, **47**(2):263-272. ISSN: 0304-9027 (print) 2408-8455 (online). DOI: https://doi.org/10.3329/bjz.v47i2.44337.

MOVAHEDINIA, A., SAVARI, A., MOROVVATI, H., KOOCHANIN, P., MARAMMAZI, J. G. and NAFISI, M. 2009. The effects of changes in salinity on gill mitochondria-rich cells of juvenile yellowfish seabream, *Acanthopagrus latus*. *Journal of Biological Sciences*, **9**: 710–720.

MUSA, I.M. and IMAM, T.S. 2023. Investigation of selected heavy metals, histopathological and oxidative stress responses of Nile tilapia, *Oreochromis niloticus* in water from the Hadejia-Nguru wetlands Nigeria. *Dutse Journal of Pure and Applied Sciences*, **9**(1): https://dx.doi.org/10.4314/dujopas.v9i1a.15.

NOFAL, M.I., ZAKI, V.H., AHMED, N.A.S., ABED-AZIZ, A.F. and RASHAD, M.M. 2019. Effects of heavy metal pollution on Nile tilapia in Manzala farm: Oxidative stress biomarkers and histopathological findings. *International Journal of Fisheries and Aquatic Studies*, **7**(5): 315-328.

OMER, S.A., ELOBEID, M.A., FOUAD, D., DAGHESTANI, M.H., AL-OLAYAN, E.M., ELAMIN, M.H. and AMEERA, V.P. 2012. Cadmium bioaccumulation and toxicity in tilapia fish (*Oreochromis niloticus*). *Journal of Animal and Veterinary advances*, **11**:1601-1606.

ORONSAYE, J.A. 1989. Histological changes in the kidneys and gills of the stickleback, *Gasterosteus aculeatus* L, exposed to dissolved cadmium in hard water. *Ecotoxicology and Environmental Safety*, **17**(3):279-290. doi: 10.1016/0147-6513(89)90048-1. PMID: 2743916.

OTLUDİL, B., AKIN, H.K. and ÜNLÜ, E. 2017. Effects of sub-lethal exposure of cadmium on histopathology of gills of Nile tilapia, *Oreochromis niloticus* and the mitigating effects of *Cladophora glomerata*. *Acta Biologica Turcica*, **30**(1):24-30.

PACHECO, M. and SANTOS, M.A. 2002. Biotransformation, genotoxic, and histopathological effects of environmental contaminants in European eel (*Anguilla anguilla*). *Ecotoxicology and Environmental Safety*, **53**: 331-347. https://doi.org/10.1016/S0147-6513 (02)00017-9.

PARVIN, A., HOSSAIN, M.K., ISLAM, S., DAS, S.S., MUNSHI, J.L., SUCHI, P.D., MONIRUZZAMAN M., SAHA B. and MUSTAFA, M.G. 2019. Bioaccumulation of heavy metals in different tissues of Nile tilapia (*Oreochromis niloticus*) in Bangladesh. *Malaysian Journal of Nutrition,* **25**(2): 237-246. https://doi.org/10.31246/mjn-2018-0153.

PINKEY, P. D., NESHA, M., BHATTACHARJEE, S., ALAMGIR, M., CHOWDHURY, Z., FARDOUS, Z., BARI, L. and KOLEY, N.J. 2024. Toxicity risks associated with heavy metals to fish species in the trans boundary river – linked ramsar conservation site of Tanguar Haor, Bangladesh. *Ecotoxicology and Environmental Safety*, **269**:115736. doi: 10.1016/j.ecoenv.2023.115736.

RAJESHKUMAR, S. and LI, X. 2018**.** Bioaccumulation of heavy metals in fish species from the Meiliang Bay, Taihu Lake, China. *Toxicological Reports*, **5:** 288–295. doi: 10.1016/j.toxrep.2018.01.007.

REITH, E. and ROSS, M.H. 1966. *Atlas of descriptive histology*. 3rd eds. Harper and Row Publishers, New York and London. 201 pp.

ROBERTS, R. J. 2001. *Fish Pathology*. 3rd Edition W.B. Saunders, London, 467pp

ROHANI, M.F., BRISTY, A.A., HASAN, J., HOSSAIN, M.K. and SHAHJAHAN, M. 2022. Dietary zinc in association with vitamin e promotes growth performance of Nile tilapia. *Biological Trace Element Research*, **200**: 4150–4159.

SAHA, B., MOTTALIB, M.A. and AL- RAZE, A.N.M. 2021. Heavy metals accumulation in different cultivated fish tissues through commercial fish feeds and health risk estimation in consumers in Bangladesh. *Chemical Reviews and Letters*, **4**:10-20.

SALMAN, N.M., AL-RUDAINY, A.J. ANAAM and FALEH, B. 2015. Histopathological effects of cadmium chloride on *Barbus sharpeyi*. *Iraqi Journal of Sciences*, **56** (1):713-722.

SARDAR, K., ALI, S., HAMEED, S., AFZAL, S., FATIMA, S., SHAKOOR, M.B., BHARWANA, S.A. and TAUQEER, H.M. 2013.Heavy metals contamination and what are the impacts on living organisms. *Greener Journal of Environmental Management and Public Safety*, **2** (4):172-179.

SARKAR, M.M., ROHANI, M.F., HOSSAIN, M.A.R. and SHAHJAHAN, M. 2022. Evaluation of heavy metal contamination in some selected commercial fish feeds used in Bangladesh. *Biological Trace Element Research*, **200**(2):844-854. doi: 10.1007/s12011-021-02692-4.

SARKAR, T., ALAM, M.M., PARVIN, N., FARDOUS, Z., CHOWDHURY, A.Z., HOSSAIN, S., HAQUE, M.E. and BISWAS, N. 2016. Assessment of heavy metals contamination and human health risk in shrimp collected from different farms and rivers at Khulna-Satkhira Region, Bangladesh. *Toxicological Reports*, **3**:346–350.

SHAHJAHAN, M., TASLIMA, K., RAHMAN, M.S., AL-EMRAN, M., ALAM, S.I. and FAGGIO, C. 2022. Effects of heavy metals on fish physiology—A Review. *Chemosphere*, **300**:134519. https://doi.org/10.1016/j.chemosphere.2022.134519.

SHUKLA, P., AHMED, A. and LODHI, S. 2023. Cadmium toxicity on aquatic ecosystem: A Review. *International Journal* of *Creative Research Thoughts,* **11**(8): 2320-2382.

SOUFY, H.M., SOLIMAN, E., EL-MANAKHLY, H. and GAAFA, A. 2007. Some biochemical and pathological investigations on monosex tilapia following chronic exposure to carbofuran pesticides. *Global Veterinaria*, **1**:45–52.

TANHAN, P., SRETARUGSA, P., POKETHITIYOOK, P., KRUATRACHUE, M. and Upatham, E.S. 2005. Histopathological alterations in the edible snail, *Babylonia areolata* (spotted babylon), in acute and subchronic cadmium poisoning. *Environmental Toxicology*, **20**(2):142-9.

THOPHON, S., KRUATRACHUE, M., UPATHAN, E.S, POKETHITIYOOK, P., SAHAPHONG, S. and JARIKHUAN, S. 2003. Histopathological alterations of white seabass, *Lates calcarifer* in acute and subchronic cadmium exposure. *Environmental Pollution*, **121**: 307-320. https://doi.org/10.1016/S0269-7491 (02)00270-1.

VINODHINI, R. and NARAYANA, M. 2009. Heavy metal induced histopathological alterations in selected organs of the *Cyprinus carpio* L. (Common carp). *International Journal of Environmental Research*, **3**(1):95-100.

YOUNIS, E.M., ABDEL-WARITH, A.A., AL-ASGAH, N.A., EBAID, H. and MUBARAK, M. 2013. Histological changes in the liver and intestine of Nile tilapia, *Oreochromis niloticus*, exposed to sub lethal concentrations of cadmium. *Pakistan Journal of Zoology*, **45**(3): 833-841.

ZEITOUN, M.M. and MEHANA, E.E. 2014. Impact of water pollution with heavy metals on fish health: overview and updates. *Global Veterinaria*, **12**(2): 219-231. DOI: 10.5829/idosi.gv.2014.12.02.82219.

(Manuscript received on 2 July 2024 revised on 25 August 2024)