



Hematological and Histological Effects of Heavy Metals on Fish (*Channa punctatus*) of Gomati River, Lucknow

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ABSTRACT

Background: Sediment deposition and accumulation expose sediment-dwelling aquatic animals to lead. Heavy metals regulation is urgent and nanotoxicology data is essential for risk assessment. Heavy metal products will likely impact the food and feed industry. Classifying essential, non-essential and toxic elements.

Methods: *Channa punctatus* fish from Lucknow, India, were tested for toxicity. Water samples were taken after acclimation, feeding and fasting. Spectrophotometers measured heavy metals.

Result: Our experiment shows lead toxicity in *Channa punctatus* fish, causing haematological changes, organ abnormalities, oxidative stress, antioxidant suppression and increased lipid peroxidation.

Key words: Fish, Hematological, Heavy metals, Histological.

INTRODUCTION

Due to its toxicity and non-degradability, heavy metal contamination threatens world health (Razak *et al.*, 2021). Heavy metals-essential, non-essential and toxic-pollute aquatic habitats at high concentrations (Uddin *et al.*, 2020). Lead, cadmium and chromium are poisonous but do not affect metabolism (Rubio-Iglesias *et al.*, 2020). Wastewater with excessive heavy metal levels can damage fish critical tissues histopathologically (Drishya *et al.*, 2016). Haematological analysis measures haemoglobin, red and white blood cell counts, haematocrit, corpuscular volume and erythrocyte sedimentation rate (Fazio, 2019).

As fish health declines, environmental toxicants affect initial health markers like micro-hematocrit by correlating contaminants and chosen haematological parameters (Gallaughier, 1994). Haematological measures like haemoglobin, haematocrit and erythrocyte count can indicate heavy metal-induced stress in fish blood (Barcellos *et al.*, 2004). Since fish are sensitive to environmental changes and pollution, their health is an important indication of water contamination in aquatic environments (Mahboob *et al.*, 2015). Lead, copper and zinc contamination of Lucknow's Gomati River caused fish blood characteristics to change, suggesting a health risk (Gupta *et al.*, 2023). The study explores how heavy metals affect Lucknow's Gomati River fish's haematology and histology.

MATERIALS AND METHODS

Model organism

Toxicity studies utilised 8-10 cm long *Channa punctatus* fish weighing 28±0.6 g. These came from Lucknow's fish market. Fish acclimated to lab conditions for a week. Before the study, the fish were fed powdered feed and fasted 24 hours. According to U.S. EPA (1986), temperature, dissolved oxygen and pH were monitored. This metal toxicant was analytical lead acetate.

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Water sample collection

Sampling sites

River Gomati flows through Lucknow from west to east, spanning 5 km. Five sites were chosen for regular water sampling. These are the Vaikunth Dham (site I), Riverfront (site II), Hanuman Setu (site III), Shaheed Smarak (site IV) and Dali Ganj (site V) (Fig 1, Table 1). The Gomati River, a major river in India, is a major source of sewage and effluents, with the southern riverfront being heavily polluted by municipal drains. The river also contains heavy metals from various sources, including electric crematoriums, waste drainage and effluents from temples and fruit markets. The river's water quality is significantly affected by these sources.

Sampling procedure

Gomti River water samples were collected in June and July and their dissolved oxygen, temperature and

transparency were measured. Bacterial analysis involved measuring Pb, Cu and Ni levels using an Aurora A-11200 Atomic Absorption Spectrophotometer.

Hematological analysis

This experiment's haematological parameters were calculated using conventional methods for accuracy.

Histopathological analysis

Study histopathological changes, Microtome (Histo-line MR 2258) cut tissues to 3mm. Paraffin wax with tissue slice ribbons was applied to albumen and glycerine slides after heating in hot water (Murmu *et al.*, 2020; Paul *et al.*, 2021). Overnight oven-dried tissue slides at 37°C. Each slide was xylene-cleared for 3 minutes. Lower the slide in ethanol alcohol for 1-2 min. Slides were hematoxylin-stained 3-5 min. Canada balsam-mounted slides were photographed under a light microscope to compare histological changes to control slides (Jiraungkorskul *et al.*, 2016; Kumar *et al.*, 2024).

Statistical analysis

One-way ANOVA was used to analyse the results after Leven's test for normality and homogeneity and conversion if needed.

RESULTS AND DISCUSSION

Hematological observations

Table 2 shows the haematological parameters for each heavy metal group after fish exposure at varied doses. After both exposure intervals, Heavy metal groups had reduced RBCs, haemoglobin and Hct (p0.05). Significantly reduced exposure period's primary influence for these components (p0.05). Increased heavy metal dose lowered MCV (p0.05). Mean corpuscular haemoglobin (MCH) was similar in all groups. Heavy metal exposure increased mean corpuscular haemoglobin (MCHC).

Red blood cell (RBC)

Table shows similar exposure lowered RBC effects. After 5 days of exposure to 10 mg/L, 12 mg/L, 24 mg/L and 26 mg/L dosages, RBC levels declined to 6.1 ± 0.5 , 5.0 ± 0.0 , 5.23 ± 1.05 and 4.85 ± 0.07 (orderly). For 10 days of exposure to 10mg/L, 12 mg/L, 24 mg/L and 26 mg/L dosages, RBC levels were 5.1 ± 0.28 , 4.10 ± 0.0 , 5.05 ± 0.07 and 4.78 ± 0.02 , respectively. The 5th and 10th day RBC control values were 6.25 ± 1.20 and 6 ± 1.55 (Fig 2).

Hemoglobin (Hb)

During 5 days of treatment to 10 mg/L, 12 mg/L, 24 mg/L and 26 mg/L dosages, Hb levels fell by 7.2 ± 0.42 , 6.15 ± 0.07 ,

Table 1: Location of sampling sites.

Site name	Latitude	Longitude
Vaikunth dham	26.8579°N	80.9671°E
River front	26.8546°N	80.9706°E
Hanuman setu	26.8606°N	80.9374°E
Shaheed smarak	26.8633°N	80.9284°E
Dali ganj	26.8728°N	80.9276°E

Table 2: Summarization of heavy metal different dose impact on hematological parameters.

Concentration	10 mg/ml			12 mg/ml			24 mg/ml			26 mg/ml			Control		
	5 D	10 D	5 D	10 D	5 D	10 D	5 D	10 D	5 D	10 D	5 D	10 D	5 D	10 D	5 D
Blood parameter															
Hb	7.2±0.42	6.95±0.07	6.15±0.07	5±0.14	4.95±0.07	4.3±0.42	4.95±0.07	4.3±0.42	4±0	3.6±0.39	9.5±0.70	8.7±0.28	9.5±0.70	8.7±0.28	6±1.55
RBC	6.1±0	5.1±0.28	5.0±0.0	4.10±0.0	5.23±1.05	5.05±0.07	5.23±1.05	5.05±0.07	4.85±0.07	4.78±0.02	6.25±1.20	6±1.55	6.25±1.20	6±1.55	6±1.55
MCV	20.45±0.62	20.54±0.74	21.80±0.84	19.6±0.59	24.04±1.20	21.55±0.72	24.04±1.20	21.55±0.72	21.05±0.07	20.43±0	23.0±9.87	20.34±5.29	23.0±9.87	20.34±5.29	20.34±5.29
MCH	13.35±0.36	11.77±0.67	11.35±0.07	10.40±0.41	13.39±0.70	11.67±0.15	13.39±0.70	11.67±0.15	12.1±0.94	10.32±0.45	11.60±0.67	11.85±0.04	11.60±0.67	11.85±0.04	11.85±0.04
MCHC	24.13±0.05	208.62±1.35	212.0±1.56	218.36±0.53	215±0.05	205.77±6.92	215±0.05	205.77±6.92	210.27±0.41	214.26±0.40	184.35±6.33	182.56±8.20	184.35±6.33	182.56±8.20	182.56±8.20
WBC	23.0±1.23	1952.2±24.04	1302.5±60.10	1293.5±7.77	1914±1.41	1872.5±7.77	1914±1.41	1872.5±7.77	1805±9.89	126.5±0.70	4133±9.19	4277.8±69.29	4133±9.19	4277.8±69.29	4277.8±69.29
Haematocrit	15.77±0.84	15.47±1.13	12.1±0.28	10.4±0.28	15.70±0.71	15.28±0.54	15.70±0.71	15.28±0.54	15.11±0	14.48±0.22	20.84±0.67	21.26±0.41	20.84±0.67	21.26±0.41	21.26±0.41



Fig 1: Sampling area map. (Source: Google map).

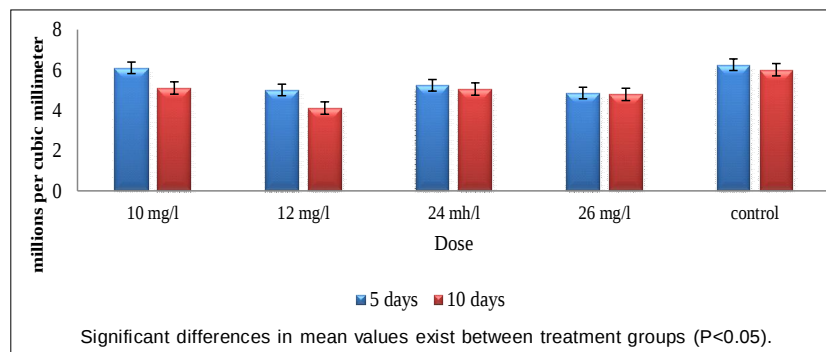


Fig 2: *Channa punctatus*' RBC levels were measured after 5 and 10 days of heavy metal exposure.

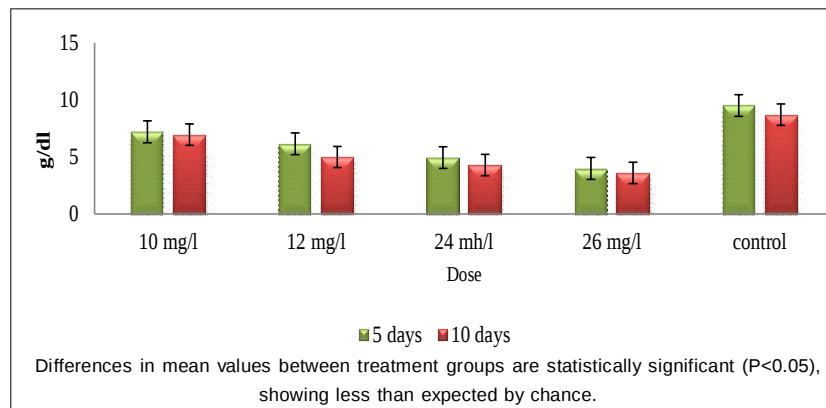


Fig 3: *Channa punctatus* Hemoglobin (Hb) levels were measured after 5 and 10 days of heavy metal treatment.

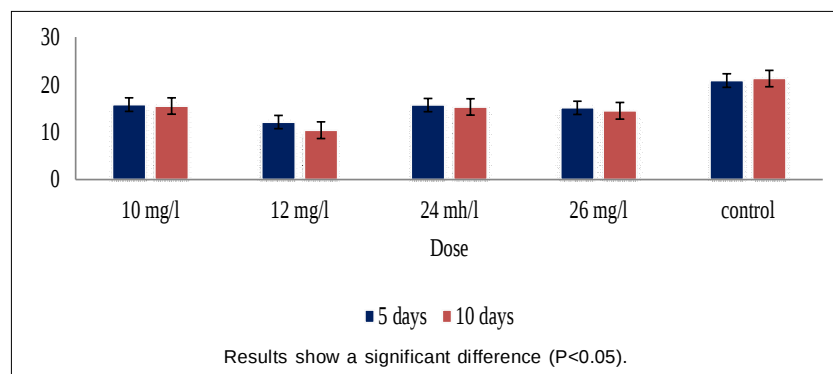


Fig 4: Hematocrit (Hct) levels (mean ± SD) in *Channa punctatus* were measured after 5 and 10 days of heavy metal treatment.

4.95±0.07 and 4±0, respectively. Exposure to 10 mg/L, 12 mg/L, 24 mg/L and 26 mg/L dosages resulted in Hb levels of 6.95±0.07, 5±0.14, 4.3±0.42 and 3.6±0.39 after 10 days. The 5th and 10th day Hb control values were 9.5±0.70 and 8.7±0.28, respectively. Exposed haemoglobin levels decrease with concentration compared to control (Fig 3).

Hematocrit (Hct)

Finally, the study examined haematocrit levels using exact doses. Haematocrit levels fell by 15.77±0.84, 12.1±0.28, 15.70±0.71 and 15.11±0 after 5 days of exposure with 10 mg/L, 12 mg/L, 24 mg/L and 26 mg/L dosages. After a 10-day exposure to 26 mg/L, 24 mg/L, 12 mg/L and 10 mg/L dosages, the MCH level was 15.47±1.13, 10.4±0.28, 15.28±0.54 and 14.48±0.22. The control values for HCT were 20.84±0.67 and 21.26±0.41 on days 5 and 10 (Fig 4).

Mean cell volume (MCV)

Additionally, the same exposure was used to assess MCV levels, which reduced compared to the 5 and 10 day controls. After 5 days of exposure to 10 mg/L, 12 mg/L, 24 mg/L and 26 mg/L dosages, MCV levels reduced from 20.45±0.62, 21.80±0.84, 24.04±1.20 and 21.05±0.07, respectively. After 10 days of exposure to 10 mg/L, 12 mg/L, 24 mg/L and 26 mg/L dosages, MCV levels were 20.54±0.74, 19.6±0.59, 21.55±0.72 and 20.43±0. The control values for MCV were 23.0±9.87 and 20.34±5.29 for days 5 and 10 (Fig 5).

Mean cell haemoglobin (MCH)

MCH levels were also tested using the same exposure. The MCH level reduced by 13.35±0.36, 11.35±0.07, 13.39±0.70 and 12.1±94 after 5 days of exposure to 10 mg/L, 12 mg/L, 24 mg/L and 26 mg/L dosages, respectively. Over 10 days, MCH levels ranged from 11.77±0.67 to 10.40±0.4, 11.67±0.15 and 10.32±0.45 at doses of 26 ml/l, 24 ml/l, 12 ml/l and 10 ml/l. The control values for MCH were 11.60±0.67 and 11.85±0.04 on days 5 and 10 (Fig 6).

Mean cell hemoglobin concentration (MCHC)

MCHC levels were assessed using the same treatment. MCHC levels increased by 24.13±0.05, 212.0±1.56, 215±0.05 and 210.27±0.41 after 5 days of exposure at dosages of 10, 12, 24 and 26 mg/L. Ten days of exposure resulted in MCHC values of 208.62 ±1.35, 218.36 ±0.53, 205.77±6.92 and 214.26±0.40, respectively. The 5th and 10th day control values for MCHC were 184.35±6.33 and 182.56±8.20, respectively (Fig 7).

White blood cells (WBC)

To determine WBC effects, the same dosage was used. During the 5-day treatment, WBC levels increased by 23.0±1.23, 1302.5±60.10, 1914±1.41 and 1805±9.89 with successive dosages of 26 mg/L, 24 mg/L, 12 mg/L and 10 mg/L. Over 10 days of exposure, WBC counts ranged from 1952.2±24.04 to 1293.5±7.77, 1872.5±7.77 and 126.5±0.70. WBC control levels were 4133±9.19 and 4277.8±69.29 for days 5 and 10 (Fig 8).

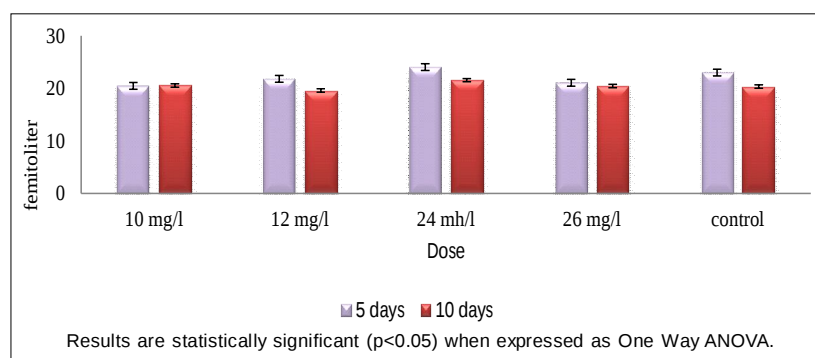


Fig 5: The MCV level (mean±SD) in *Channa punctatus* was measured after exposure to various heavy metal doses for 5 and 10 days.

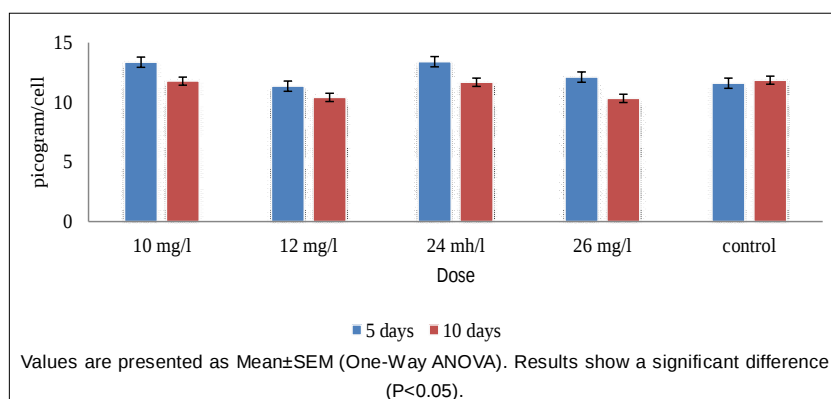


Fig 6: *Channa punctatus* was treated with different heavy metal doses for 5 and 10 days to measure its MCH level (means).

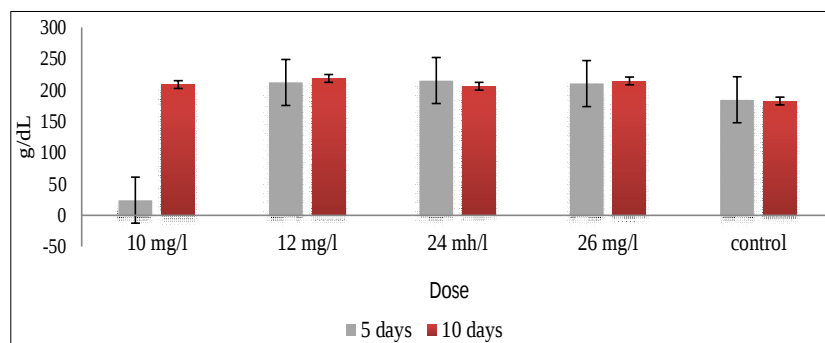


Fig 7: The MCHC level (mean $\pm$ SD) in *Channa punctatus* were observed on exposing to different dose of heavy metal for 5 days and 10 days the outcome is statistically significant i.e., ($p < 0.05$).

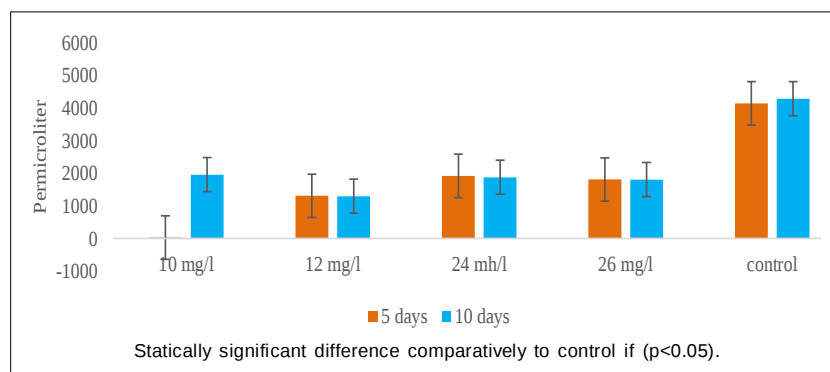


Fig 8: The WBC level (mean $\pm$ SD) in *Channa punctatus* was observed on exposure to different doses of heavy metal for 5 days and 10 days.

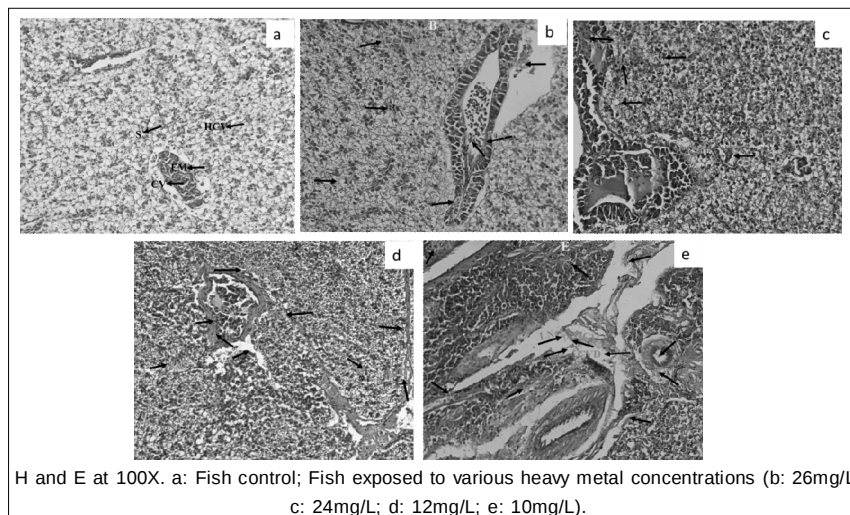


Fig 9: Five-day *Channa punctatus* liver section.

Histopathological observation of *Channa punctatus* liver

Histopathology of liver on treatment with Heavy metal on day 5

In control fish liver tissue, the portal vein's histological structure was normal, showing sinusoids, homogenous cytoplasm and hepatocytes in the hepatic parenchyma. A histological analysis of the liver was conducted after five days of exposure to a heavy metal dose of 10 mg/liter. The

obtained results clarified the observations of blood congestion, cytoplasmic vacuolation, patchy degeneration, melanoma-macrophage centers, patchy central vein and hyperplasia with exposure to 10 milligrams per liter of pyknotic nuclei. A histological analysis of the liver was conducted after five days of exposure to a heavy metal dose of 12 milligrams per liter. The obtained results clarified the observations of cytoplasmic vacuolation, mononuclear cell infiltration, fatty alterations and hyperemia during exposure

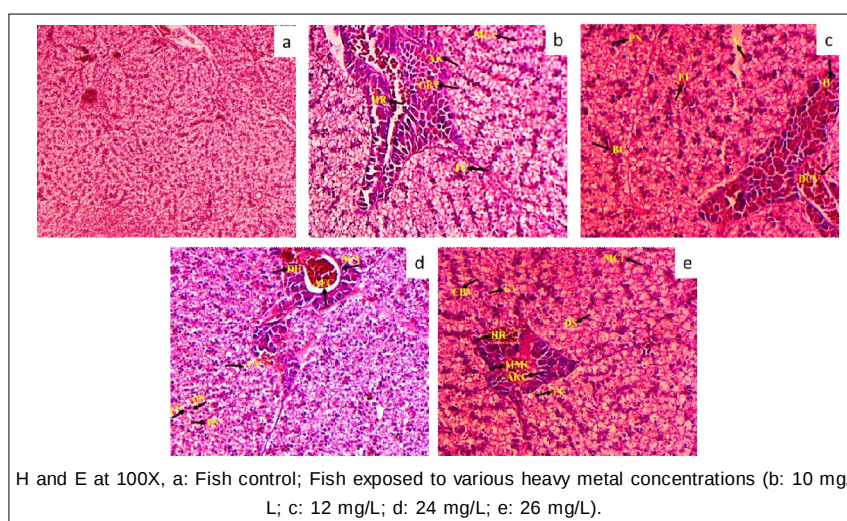


Fig 10: *Channa punctatus* liver section observations at 10 days.

to 12 milligrams per liter of melanoma-macrophage centers. After five days of exposure to a heavy metal dose of 24 mg/liter, a histological analysis of the liver was conducted. The obtained results clarified the reported fatty alterations, hyperemia, cytoplasmic vacuolation, infiltration of mononuclear cells and exposure to 24 mg/liter of macrophage centers. After five days of exposure to the 26 mg/liter doses of heavy metals, a liver histological test was conducted. The obtained data clarified that after 26 mg/liter of exposure. Hepatic vein degeneration, damaged hepatic lobule, damaged hepatocytes, fragmented kuffer cell, damaged central vein, pycnotic nuclei and cytoplasmic vacuolation were also noted (Fig 9).

Histopathology of liver on treatment with Heavy metal on day 10 day

The liver was histopathologically examined after 10 days of heavy metal exposure at 10 milligrammes per litre. On 10 mg/L exposure, the results showed. Mononuclear cell infiltration, kuffer accumulation, congested blood vessels, hyperaemia and fatty changes occurred. Histopathological liver examinations were performed after 10 days of heavy metal exposure at 12 milligrammes per litre. Results showed 12 mg/L exposure. Mononuclear cell infiltration, kuffer accumulation, congested blood vessels, hyperaemia and fatty changes occurred. Histopathological liver examinations were performed after 10 days of heavy metal exposure at 24 milligrammes per litre. Results showed 24 mg/L exposure. Hepatocyte damage, cytoplasmic vacuolation, pycnotic nuclei, dilated sinusoids, kuffer cell accumulation and central vein damage were observed. The liver was histopathologically examined after 10 days of heavy metal exposure at 26 mg/liter. Results showed 26 mg/L exposure. Cytoplasmic vacuolation, hyperaemia, melano-macrophage centres, fatty changes, congested blood vessels, dilated sinusoids, kuffer cell accumulation and mononuclear cell infiltration were observed (Fig 10).

Gills

Histopathological examination of gills on treatment with Heavy metal on day 5

Normal fish gills have primary and secondary lamellae. Histopathology was performed on the gills after 5 days of 10 mg/mL heavy metal exposure. The results showed that 10 mg/mL exposure caused interlamellar hyperplasia, epithelial layer detachment, aneurysm, partial fusion and almost complete secondary lamellae fusion. Histopathology was performed on the gills after 5 days of 12 mg/mL heavy metal exposure. At 12 mg/mL, secondary lamella epithelial layer detachment, mononuclear leukocyte infiltrates, telangiectasia, hyperplasia and erythrocyte infiltration occurred. After 5 days of 24 mg/mL heavy metal exposure, gill histopathology was performed. Results: 24 mg/mL caused venous sinus dilatation, coalescent interlamellar hyperplasia, partial fusion, secondary lamella hyperplasia, epithelial layer detachment, telangiectasis and hyperplasia. After 5 days of 26 mg/mL heavy metal exposure, gills were histopathologically examined. Results showed 26 mg/mL exposure caused central venous congestion, interlamellar hyperplasia, telangiectasia, cell detachment, erythrocyte infiltration and almost complete secondary lamellae fusion (Fig 11).

Histopathology of liver on treatment with titanium oxide heavy metals on day 10

A liver histopathology study was done after 10 days of 10 mg/L heavy metal exposure. Primary and secondary lamellae were thick, early aneurysm, complete fusion, parasitic cyst, respiratory epithelium lifting and clubbed together after 10 exposures. Gill histopathology was performed 10 days after 12 mg/L heavy metal exposure. After 12 exposures, primary and secondary lamellae thickened, coalescent interlamellar hyperplasia occurred and cells detached. The gills were histopathologically examined after 10 days of 24 mg/L heavy metal exposure.

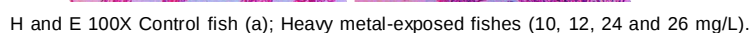
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Fig 13: 5 day *Channa punctatus* kidney section observations. H and E at 100X.

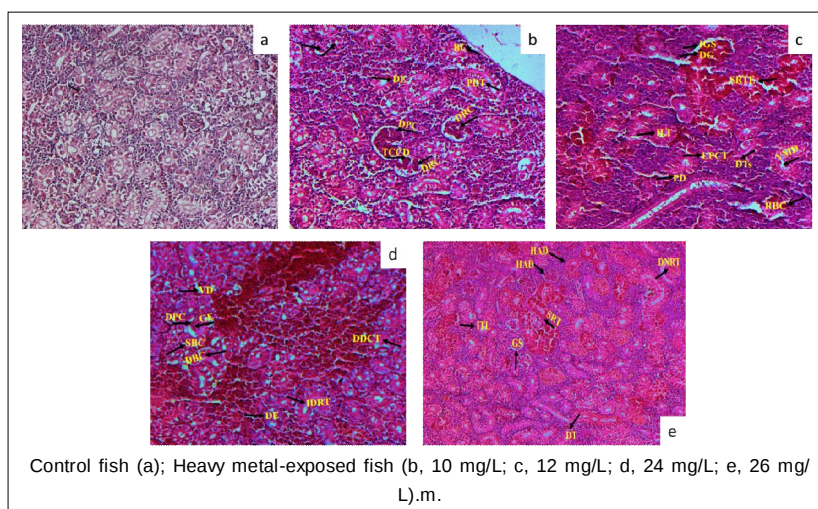


Fig 14: *Channa punctatus* kidney section observations at 10 days. H and E at 100X.

Parasite cyst, aneurysm and respiratory epithelium lifting caused lamellae to fuse at 24 mg/L. Histopathology was performed on the gills after 10 days of 26mg/L heavy metal exposure. At 26 mg/L, main and secondary lamellae thickened, coalescent interlamellar hyperplasia, tip telangiectasia and partial fusion were found (Fig 12).

Kidney

Histopathological examination of kidney on treatment with heavy metal on day 5

The kidney had normal Bowman capsules, glomerulus and Kupfer cells. Kidney histopathology was done after five days of heavy metal exposure. Epithelial cell atrophy, reduced glomerular cells, tubular cell component loss, large collecting duct, hydropic degeneration, dilated tubule and patchy degeneration occurred after 10 mg/mL exposure. A kidney histopathological examination was done after 5 days of 12 mg/L heavy metal exposure. Results showed 12 mg/mL exposure detached basal lamina epithelium, deformed Bowman space, Bowman capsule space, Erythrocyte glomeruli Degeneration of epithelium Atrophy, hydropic degeneration, renal tubule degeneration, visceral membrane damage and mesangial cell damage were caused by erythrocytes. Histopathological kidney exams were done after 5 days of 24 mg/L heavy metal exposure. After 24 exposure, glomerulus, first proximal tubule, melanophages, collecting duct, space in Bowman capsules, mononuclear cells, red blood cells, visceral membrane damage, nuclear pyknosis and patchy degeneration were observed. Histopathological kidney exams were done 5 days after 26 microgrammes per litre heavy metal exposure. Basal lamina epithelial cells detached, renal corpuscle degenerated, proximal convoluted tubule shrank, tubular cell components disappeared and podocyte cells were damaged at 26 mg/L (Fig 13).

Histopathological examination of kidney on treatment with Heavy metal on day 10

The kidney was histopathologically examined after 10 days of 10 mg/L heavy metal exposure. Epithelial cell atrophy, basal lamina separation, proximal convoluted tubule shrinkage, tubular cell component disappearance and renal corpuscle degeneration resulted from 10 exposure. Damaged podocytes, basophilic cluster, patchy distal tubule. Histopathology was performed on the kidney after 10 days of 12 mg/L heavy metal exposure. At 12 mg/L, glomerulus space grew. Glomerulus damage Red blood cells, patchy degeneration, renal tubular epithelium separation from basement, disorganised tubules and visceral membrane damage were observed. A kidney histopathological examination was done after 10 days of 24 mg/L heavy metal exposure. Results showed 24 mg/L caused vacuolar degeneration, podocyte cell damage; Space of Bowman capsule, detached basal lamina epithelium, distal convoluted tube disappearance, glomerular expansion, Bowman's space dilation and renal tubule diameter increase Histopathology was performed on the kidney after 10 days of 26 mg/L heavy metal treatment. After 26 mg/L treatment, basal lamina epithelial cells detached, renal capsule degenerated, proximal convoluted tubule shrank, tubular cell components disappeared and podocyte cells were damaged (Fig 14).

After five and ten days of exposure to all Lead (Pb) concentrations, fish became lethargic, parasite-infected and pale. Lead (Pb) caused the fish's pale colour and anaemia, according to haematology analysis (Gomez *et al.*, 2021). Lead (Pb) at any level caused oxidative stress, which decreased cell-protective enzyme activity, raised LPO and caused serious tissue disorders in *Channa* fish. Lead mostly affected fish livers. Pb release into aquatic ecosystems may harm aquatic life. Thus, lead (Pb) in water may harm aquatic life. After environmental release, lead (Pb)

behaviour, fate and toxicity are unpredictable. The IARC classifies lead (Pb) as a Group B carcinogen because it can cause cancer in humans (Steenland and Boffetta, 2000). Lead levels in aquatic habitats range from 0.7 to 16.8 µg/L. Lead (Pb) enters aquatic habitats worldwide at 15600 mg per year. Understanding how Lead (Pb) affects aquatic animals is crucial. Like our findings, most of these studies found no acute effects at Lead (Pb) concentrations above 30 mg/lit. Eco-toxicity research requires characterising lead (Pb) because its size and other parameters affect its properties, destiny and behaviour in biological systems (Kumar *et al.*, 2020).

Lead (Pb) intraperitoneally in rainbow trout caused kidney bioaccumulation and almost no removal ninety days later. A recent study found that rainbow trout kidneys can accumulate up to 94% lead (Pb) after intravenous injection. Histopathological kidney examinations were performed on animals exposed to the highest Lead (Pb) concentrations of 10, 12, 24 and 26 milligrammes per litre for 5 and 10 days. After 26 mg/L exposure, glomerulus space, damaged glomerulus, tubular lumen, first proximal tubule loss, patchy degeneration, renal tubular epithelium separation from basement, disorganised tubules, visceral membrane damage and red blood cells increased. Include all concentrations. Free radicals from lead (Pb) particles cause oxidative stress and cell damage, making them toxic. Aquatic species' antioxidant enzymes detoxify ROS, reducing ROS production. SOD turns O₂ into H₂O₂ (Abele *et al.*, 2011).

CONCLUSION

Channa punctatus fish show haematological changes, organ abnormalities, oxidative stress, antioxidant suppression and increased lipid peroxidation due to Pb exposure. Lead (Pb) may cause fish anaemia, according to studies. The study found no significant differences in pH or temperature between the treatment and control groups and that dissolved oxygen decreased after five and ten days of lead exposure. Lead (Pb)-exposed fish had higher nitrate and nitrite levels but lower ammonia levels. The findings suggest that Pb emissions into aquatic habitats harm aquatic life and animal welfare and that understanding Pb's toxicity is important because it is used so frequently.

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Declaration

Ethical approval

Authors declare this manuscript does not include any studies using animal and human beings.

Consent to publication

All authors read and approved the final manuscript.

Conflict of interest

The authors declare no conflict of interest.

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