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Assessment of acetamiprid toxicity in *Oreochromis mossambicus* through histological and ultrastructural analysis

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Abstract

This study investigates how *Oreochromis mossambicus's* liver, brain, and gill tissues are affected by the pesticide acetamiprid following a 28-day exposure period. Commonly employed in agriculture, acetamiprid can infiltrate aquatic environments and have an impact on organisms that are not its intended target. Scanning electron microscopy was used to examine tissue samples, and Probit analysis was used to calculate the Lethal concentration (LC_{50}). Hepatocyte enlargement and degeneration in the liver, brain cell atrophy and tissue collapse, and gill damage, including hemorrhages and epithelial rupture, were among the toxic consequences that were noted. These structural alterations suggest that acetamiprid presents a serious health danger to aquatic life and possibly to people who eat contaminated fish.

Key words : *Oreochromis mossambicus,* Hepatocytes, Toxicity, Porosity, Bioindicators, Neonicotinoids, Acetamiprid.

Even though pesticides are essential for managing agricultural pests, they are naturally poisonous and can seriously harm aquatic organisms that are not their intended target when they leak into water bodies through runoff, leaching, or atmospheric deposition. Neonicotinoids such as acetamiprid (ACE), which have been used extensively since the 1990s, have been found in surface waters all over the world and are known to bioaccumulate in aquatic animals, especially fish, through skin absorption and gill uptake[4,6]. In fish, this bioaccumulation causes oxidative damage, tissue deterioration, physiological stress, and behavioral changes[2,11]. Chronic and sublethal exposures alter osmoregulation, enzyme activity, and endocrine function, which may result in death or decreased chances of reproduction[12,15]. It is crucial to use biomarkers to track pesticide-induced toxicity because of the ecological and nutritional significance of fish like *Oreochromis mossambicus,* which

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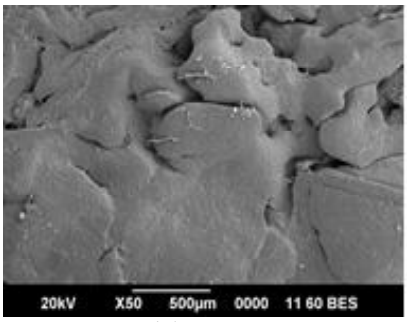
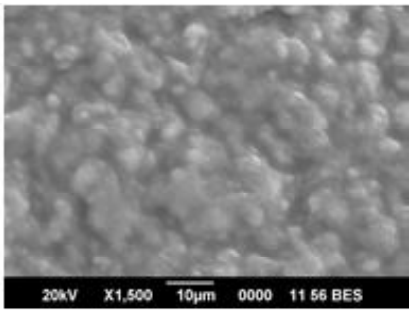
are widely cultivated and eaten[13]. Cellular damage and environmental stress responses can be effectively revealed by histopathological and ultrastructural examinations of target organs, including the liver, gills, and brain[1,4]. Furthermore, further targeted toxicological research is required because recent studies have shown that acetamiprid has genotoxic and cytotoxic effects in a variety of freshwater fish species[7]. Thus, the current work uses histological and ultrastructural evaluations of liver, brain, and gill tissues to examine the toxic effects of acetamiprid on *Oreochromis mossambicus*.

Samples of *Oreochromis mossambicus* weighing 24 ± 2.5g were taken from a freshwater pond close to Parassala and put in a glass tank that had been cleaned. The fish were fed commercial fish meal every day for 20 days until they adapted. Based on initial LC_{50} data, a sublethal concentration of acetamiprid (21.5 ppm) was chosen and given for 28 days. Every experiment was carried out in triplicate, with control groups kept alongside test groups. To maintain stability, the exposure medium was changed every day. Fish were sacrificed after the exposure period, and the tissues from their liver, brain, and gills were gathered. After being dehydrated using a series of graded ethanols, the tissues were air-dried, fixed in 4% glutaraldehyde, gold-coated, and inspected under a scanning electron microscope for ultrastructural investigation.

The liver, brain, and gill tissues of fish exposed to acetamiprid showed notable histological and ultrastructural changes in comparison to the control group. Fish tissues frequently accumulate more pesticides than their aquatic surroundings, which can cause problems with eating, immunity, and growth[5,13]. Histological biomarkers provide trustworthy information about these harmful exposures[6]. The liver tissues of control fish had normal polygonal hepatocytes with intact lobular shape and central nuclei (Figure 1A–1B). However, fish treated with acetamiprid showed signs of hepatotoxicity, including necrosis, granular cytoplasm, loss of lobular pattern, and hepatocyte swelling (Figure 1C–1D)[4,11]. Numerous pesticide exposures, such as sinusoidal dilatation and vacuolation, have been linked to comparable histological damage in the past[1,15]. Control fish brain tissue showed an intact neuronal network and healthy cerebellum and cerebrum structures (Figure 1E–1F), while treated specimens displayed necrotic lesions, neuronal atrophy, glial infiltration, and porosity (Figure 1G–1H), which suggested neurodegeneration and disruption of the central nervous system[7,14]. These results are consistent with past research on vacuolar alterations brought on by pesticides in fish brain tissues[3]. Due to their close exposure to the aquatic environment, the gills displayed noticeable structural and functional alterations. While treated fish showed lamellar fusion, epithelial rupture, bleeding, and mucosal loss (Figure 1K–1L), indicating poor respiration and osmoregulation, control fish displayed normal lamellar architecture with ordered epithelial layers and vascular components (Figure 1I–1J)[2,12]. Lamellar disorganization and cell necrosis were verified by SEM analysis, which was in line with previous gill toxicopathology studies[8]. These results demonstrate that acetamiprid exposure, even at sublethal levels, can cause substantial tissue damage to essential organs. The detected histological lesions highlight the



1A



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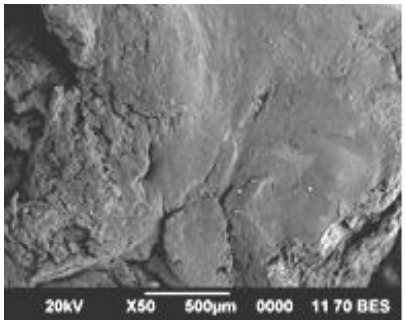

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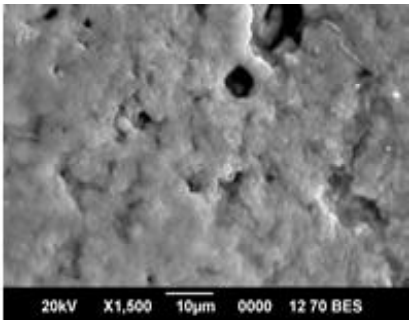
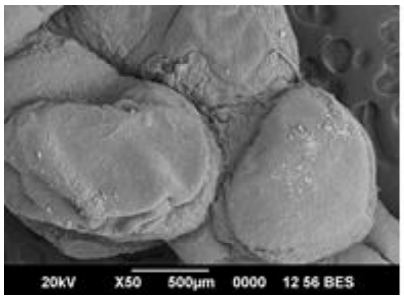
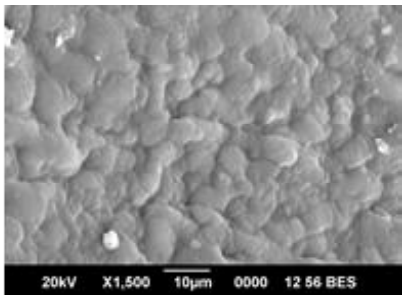


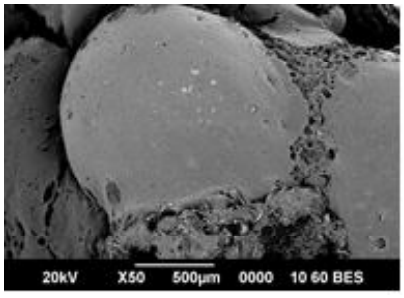
1D



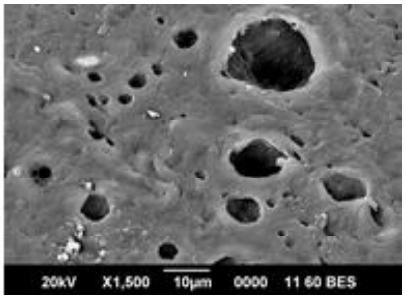
1E



1F



1G



1H

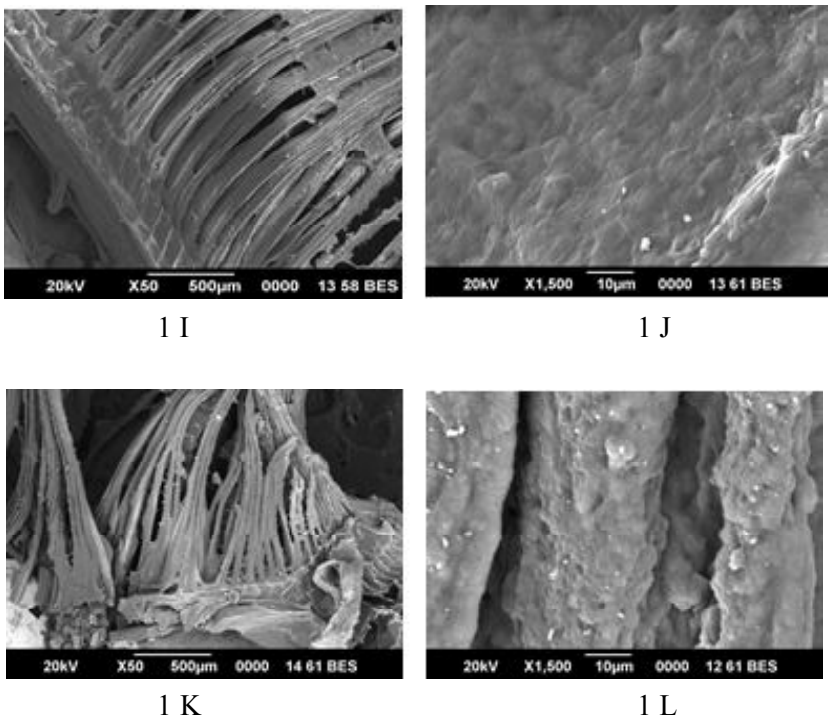


Figure 1 Scanning Electron Microscopic images of Liver, Brain and Gill at 28th day of exposure: *A (50 X) & B (1500 X) Control fish Liver, C (50 X) & D (1500X) Acetamiprid treated fish liver, E (50 X) & F (1500 X) Control fish Brain, G (50 X) & H (1500X) Acetamiprid treated fish Brain, I (50 X) & J (1500 X) Control fish Gill, K (50 X) & L (1500X) Acetamiprid treated Gill,*

ecological risk caused by pesticide runoff in freshwater systems and justify the use of ultrastructural biomarkers in aquatic ecotoxicological studies, especially in the liver, brain, and gills[9,10].

Fish are particularly at risk whenever chemicals are present in the water. As a result, when these chemical pollutants get inside fish bodies, they can create significant abnormalities in the organ systems, substantially impair certain physiological and biochemical processes, and eventually cause the species to go extinct. Histopathological alterations in tissue can be used to rapidly evaluate the detrimental effects of chemicals in different tissues and organs. The results of this investigation clearly demonstrate that the insecticide acetamiprid poses a risk to the vital organs of *Oreochromis mossambicus*. Long-term exposure of organisms to pesticides puts the fish population at constant danger for health issues. Therefore, these histopathological and ultrastructural responses could be considered bioindicators to evaluate fish health in contaminated waterways.

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