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HISTOPATHOLOGICAL AND BIOCHEMICAL STUDIES ON THE ACUTE TOXICITY OF DIMETHOATE ON NILE TILAPIA FISH

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ABSTRACT

Dimethoate (DM) is one type of organophosphorus insecticide, that kills insects and mites both systemically and immediately upon contact and harms a range of non-target animals, including fish when it enters the aquatic habitat. This study aimed to determine the potentially harmful histopathological and biochemical impacts of Dimethoate on Nile tilapia fish. Sixty *Oreochromis Niloticus* freshwater fish, each weighing an average of 130 ± 10 g, were divided into two groups in triplicates. There were 6 groups, 3 control groups, and 3 experiment groups that received Dimethoate at a dose of 8 mg/l in water for 15 days. Blood samples were collected and used for biochemical indexes. For histological analysis, samples of the brain, gills, liver, and kidney were collected. The findings of the study demonstrated that fish treated with Dimethoate exhibited pale gills, anxious symptoms, as well as congestion and hemorrhages in many internal organs, comprising the brain, kidney, and liver. There was a significant increase in the blood levels of nitric oxide, lipid peroxide, and glutathione peroxidase and a significant fall in catalase levels in the Dimethoate group when compared to the control group. Pesticides, especially DM, should not be used carelessly in agriculture and allowed to accumulate in streams because of deleterious effects on fish.

Keywords: Tilapia nilotica, Dimethoate, histopathological changes, behavioral nervous manifestations, antioxidants.

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INTRODUCTION

Pesticide exposure is considered the primary occupational hazard among agriculturalists in developing countries, and the primary origin of pollutant and health hazards in surface waters and wastewaters (Evgenidou *et al.,* 2007). Pesticides are classified depending on their chemical nature into organochlorines, organophosphates, carbamates, halogenates, hydrocarbons, heterocyclic compounds, chlorinated phenoxy substances, amines, and urea, phenolic compounds, and pyrethroids (Lawson *et al.,* 2011 and Ogamba *et al.,* 2015). Organophosphates (Ops) are one of the many kinds of pesticides that are often utilized, and various populations are exposed to a variety of their metabolites (Ghorab & Khalil, 2015). Organophosphates (Ops) are more widely used by different pesticide classes due to their high insecticide properties, weak mammalian poisoning, less stability, and quick ecological biodegradability (Srivastava *et al.,* 2010).

Dimethoate is very selective as an insecticide because the comparative proportion of esterases and amidases, the degrading enzymes, is low in insects related to those of mammals (Rose and Hodgson, 2004). Via the major portal ways, it enters the aquatic environment as drainage from agricultural lands into surface waters, leaching into groundwater, washing from ambient precipitation, spray drift, spraying, and direct application (Ihsan *et al.,* 2018). It influences fish commonly through dermal uptake, or immediate uptake via the gills during breathing (Bhat *et al.,* 2010). Dimethoate works primarily as a nerve toxin as the toxicity of DM like other organophosphorus pesticides is built up via inhibition of acetyl-cholinesterase (AChE) that exists in mammals, birds, fish and insects (Pandey *et al.,* 2009). Dimethoate is one of the OPI effects on the brain by its suppressing activity of AChE which is crucial in neurotransmission at cholinergic synapses, so brain histopathology is essential to detect the deleterious result of Dimethoate in the brain of fish (Akter *et al.,* 2020).

The usage of fish as reliable sensors of chemical pollution is widespread because of how they respond to tiny quantities of dangerous substances and as their ability to measure the biological impacts of poisons and environmental quality, they are often utilized as sentinel animals (Ayas *et al.,* 2007). The Nile tilapia (*Oreochromis niloticus*), which has a high sensitivity to potential chemical side effects, was chosen as the model organism (Uner *et al.,* 2006). *Oreochromis niloticus*, which refers to one of the most significant fish classes, is considered a suitable biological model due to how easy it is to manipulate, cultivate, and care for in the lab (Garcia-Santos *et al.,* 2006).

The present study was performed to investigate DM toxicity in Nile tilapia fish by histopathological examination of different tissues, and biochemical estimation of oxidative markers.

MATERIALS AND METHODS

I-Materials

Chemicals used:

- Dimethoate trade name (Caminova 40% EC) OP insecticide solution purchased from Iso- Vit Company for veterinary medicine, Assiut.
- Antioxidant enzyme kits (lipid peroxides kits, nitric oxide kits, glutathione peroxide, and catalase kits) are purchased from the bio-diagnostic company in Cairo.

Experimental Fish

Oreochromis niloticus of both sexes with approximate body weight (130 ± 10 g) gathered from the River Nile at Assiut Governorate, Egypt. Using electric air pumping compressors, continuous aeration was maintained in each aquarium as fish were adapted to lab settings for 10 days in

maintenance glass aquariums (70-liter capacity) at room temperature (25 ± 1.5), ph. (7.3 ± 0.03) and dissolved oxygen (7.2 ± 0.2). Fish were provided with commercial pellet food from the Department of Aquatic Animal Medicine, Faculty of Veterinary Medicine, Assiut University, two times daily at a daily feeding rate of 3% of body weight. The diet consisted of 20% crude protein, 4% crude fat, 5% crude fiber, 12% crude ash, and 10% crude moisture.

Experimental design:
According to (Sprague, 1973), the lethal concentration of DM 40%EC on Nile tilapia was determined using a static renewal bioassay method, which is 25 mg/L. A pilot experiment was conducted to determine the appropriate environmentally relevant concentration (8 mg/L) for the study, using glass tanks filled with different concentrations of DM solutions (3 mg/l, 5 mg/l, and 8 mg/l) for 5 days.

In this study, two groups of *Oreochromis niloticus* in triplicates were used:
Group 1 (control group): 30 fish (10 fish in each tank) did not receive any treatment
Group 2 (control group): 30 fish (10 fish in each tank) received (8 mg/l) DM which is equal to (1/3) LC50 DM**.**

II- Methods

Biochemical evaluation:
Three fish from each group were randomly picked after 15 days, gently caught, and sent to the lab for testing. They were anesthetized, and killed in clove oil solution, and blood and tissue samples were collected. Blood was clotted, centrifuged, and stored at -20℃ for biochemical analyses using a UV/VIS Spectrophotometer. The homogenate of the liver and muscles was centrifuged at 18000 g at 4ºC for 30 min before the determination of catalase (CAT), and glutathione peroxidase (GPx) activity.

- Malondialdehyde (MDA) was determined in serum to examine Lipid peroxidation (LPO) using a colorimetric assay kit according to (Utley *et al.,* 1967).

- CAT activity was detected in collected serum using the method defined by (Aebi, 1984).

- Homogenized liver tissue was used to track the GPX enzyme activity, according to Paglia and Valentine, (1967). Additionally, liver homogenate was used to quantify the endogenous nitrite content, which Montgomery and Dymock, (1961) identified as an indication of NO production.

Gross examination:
Careful P.M. examination was carried out on all experimental fishes and gross lesions were recorded in the affected organs.

Histopathological examination:
After the fish were sacrificed after the study after 15 days, the brain, kidney, liver, and gills were taken away and subsequently stored in a 10% neutral buffered formalin solution for 24 hours. Following fixation, all tissue samples were regularly fixed and periodically handled as follows for traditional histopathological analysis:

Histopathological analysis using light microscopy, five-micron sections were prepared and stained with hematoxylin and eosin (Bancroft & Stevens, 1982).

Histopathological scoring:
Histopathological scoring of tissue lesions is an effective way to evaluate research tissues and validate morphological findings. By rating the severity of the histological damage by techniques that have already been available (Rotta *et al.,* 1999; Sahin *et al.,* 2006), a semi-quantitative evaluation of the damage was achieved. No changes (0), minor (1+), mild (2+), moderate (3+), severe (4+), and extremely severe (5+) are the levels of alterations for each section.

Statistical analysis

All of the data's uniformity of distribution and homogeneity of variance were evaluated utilizing the Kolmogorov-Smirnov test and Bartlett's test as well as using the Statistical Analysis System's one-way ANOVA for statistical analysis (Hoshmand, 2006). The tested groups were compared to discover if there were any metrics where there was a (P0.05) significant difference.

RESULTS

A- Clinical sings (Behavioral sings):

Throughout the experiment, The Control Group exhibited normal opercula movement and skin color. They frequently displayed rapid, coordinated movements and were highly lively. They were acutely sensitive to even the slightest disturbance or outside stimulus. Fish in the control group had shiny colors and behaved normally.

Two days after the study started, fish exposed to 8 mg/l Dimethoate displayed the aberrant behavioral changes listed in Table (1). Fish in this group were acting differently from fish in the control group. More severe behavioral changes included losing appetite and stability, swimming erratically and hysterically, circling, convulsions, and remaining immobile on the tank floor. Fast swimming, surfacing activity frequency, and gulping of surface water were recorded for 5-10 days. Dark- Color discoloration of the body surface, overproduction of mucus, as well as accelerated operculum movement were all noted clinically within the first 10-15 days. Severe convulsive reflexes upon stimulation were abundant. The mucous secretion increases considerably in the exposed fishes and turbidity of the water of the test troughs increases gradually during continuous DM exposure. Finally, the fish affected by the toxin exhibited a lack of stability and a spiral swimming pattern, as well as being extremely weak and swimming to the bottom.

Table 1: Behavioral alterations of *Oreochromis niloticus* subjected to a sub-lethal dose of Dimethoate.

	Behavioral signs					
Duration	**loss of hunger and equilibrium**	**erratic and hysteric swimming**	**staying motionless on the aquarium bottom**	**surfacing activity frequency and gulping of surface water**	**excessive mucus secretion**	**Colour darkening of the body surface**
5 days	+	++	++	+	++	+
10 days	++	+++	++	+++	++	++
15 days	+++	++	+++	+++	+++	+++

- No significant, +low severity, ++moderate severity, +++high severity

B- Biochemical results

Table 3: Values of different biochemical indices in fish of different groups.

Treated groups Groups	Catalase measurement	Glutathione peroxidase	Nitric oxide measurement	Malonaldehyde level
Group I (control)	$(0.397\pm.034)1^{a}$	$(0.006\pm.002)^{1a}$	$(13.233\pm4.963)^{1a}$	$(13.439\pm 5.749)^{1a}$
Group II (Dimethioate intoxicated group)	$(0.341\pm.047)^{1a}$	$(0.029\pm.007)^{1ab}$	$(30.295\pm12.453)^{1a}$	$(28.857\pm 5.534)^{1a}$

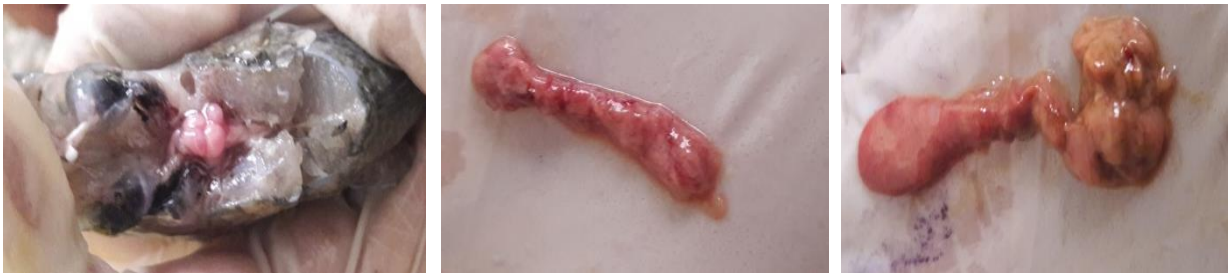
The data are displayed as mean SD. The means at the identical raw (time relation) with distinct superscript characters differ considerably (P 0.05). Means in the same column that have various superscript letters (Dose relation) vary considerably (P 0.05).

As in Table 3, after 15 days of treatment, a serum biochemical analysis of the Dimethoate-intoxicated group revealed a non-significant rise in glutathione peroxidase values in Group II (the Dimethoate group) and a non-significant decline in catalase values in Group II (the Dimethoate group) in contrast with the control group. However, there was a significant rise in serum nitric oxide and MDA levels when contrasted with the control group.

C- Histopathological results:

I- Gross findings:

NO gross findings appeared in the fish of the control group. After 15 days of exposure to 8 mg/l, Dimethoate revealed congestion of all internal organs (Brain, Gills, Kidney, and Liver) (Fig.1).



(Fig.1): After 15 days of Dimethoate of exposure to 8 mg/l, Nile tilapia (*Oreochromis niloticus*) showing congestion of the brain, kidney and liver.

II-Histopathological findings:

Control group (Group I):

The tissues from the control group did not exhibit any histological alterations when they were inspected under a light microscope. Additionally, the analyzed sections from this group showed normal hepato-pancreas and a normal liver with maintained morpho-histological components. Gills, kidneys, liver, and brain were among the other organs that were normal and still had their original morpho-histological structures.

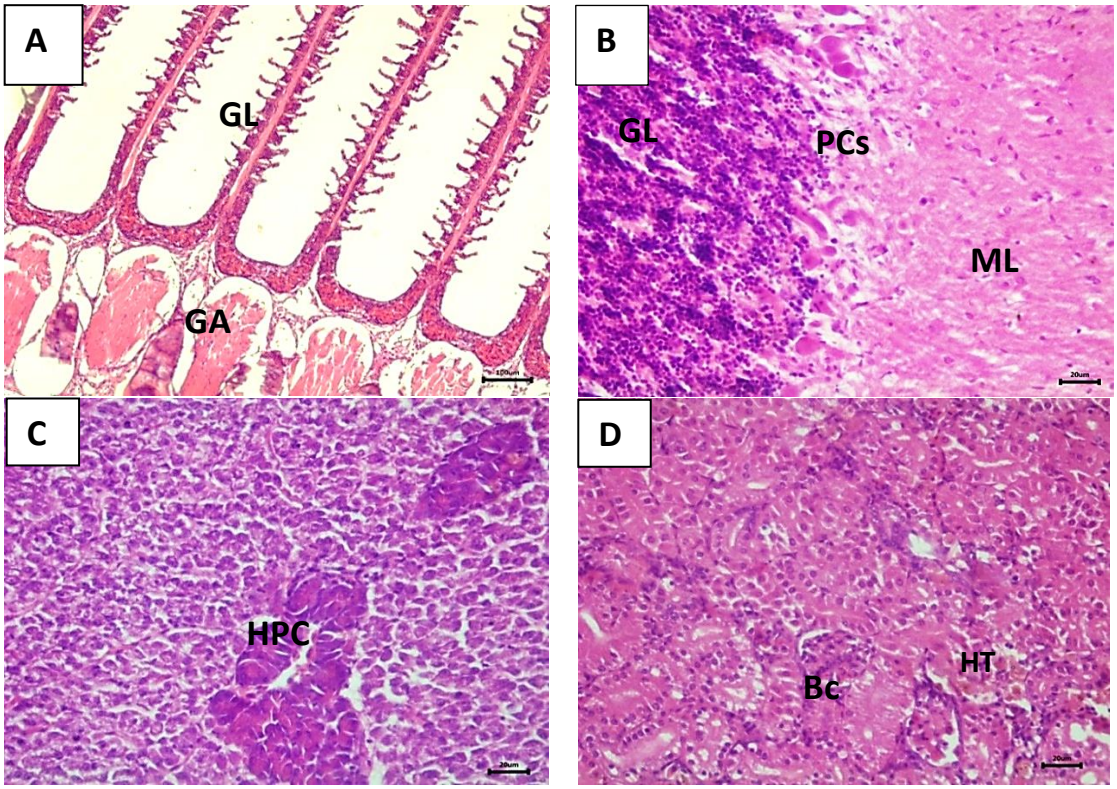
The control group shows the typical architecture of the gill including gill lamellae, with identical interlamellar space, primary gill lamellae, secondary gill lamellae, and gill arch that could all be seen in (Fig. 2 A).

There were no morphological alterations to the brain. The typical architecture of the

control fish brain tissue reveals the hippocampus (HI) and normal cerebellum, both of which are composed of neural cells with distinctive nuclei (Fig. 2B).

The liver structure of the control fish was not significantly altered. Hepatocytes were arranged in the Nile tilapia liver samples from the control group, forming a group of cells near the sinusoidal capillaries and the hepato-pancreas (HPC). (Fig. 2 C).

The kidneys of the control fish had normal features without any histopathological alterations. The control group's kidneys showed typical renal corpuscles and renal glomeruli that were surrounded by hematopoietic tissue (Fig. 2 D).



(Fig. 2): The control group showing (A) Gills with the typical organization include gill lamellae (GL), with identical inter lamellar space (ILS), epithelial cell (EC), endothelial cell (ENC), primary gill lamellae (PGL), secondary gill lamellae (SGL) (H&E), gill arch (GA); (B) A brain with a typical histological structure in which the cerebellum is composed of Purkinje cells (PCs), granular layer (GL), and molecular layer (ML); (C) Liver with normal pattern of the hepato-pancreatic tissue (HPT); (D) Kidney with typical architecture of renal tubule (RT), Bowman's capsule (Bc) and hematopoietic tissue (HT); bar=20, H&E.

Dimethoate intoxicated group (Group II):

Gills

The gills of fishes after 15 days of DM (8 mg/l) revealed extensive epithelial lifting and hyperplasia of the epithelium with fusion of adjacent lamellae in many areas owing to filamentary epithelium proliferation accompanied by severe curling of secondary lamellae (Fig 3 A). Shortening of secondary lamellae in other areas with severe fusion of adjacent secondary gill lamellae infiltrated with inflammatory cells was also observed (Fig 3 B). The angiopathic changes were also noticed in gills characterized by dilation of the central venous with blood congestion with respiratory epithelial edema (Fig. 3 C). Telangiectasis was also observed in the gill's secondary lamellae with extensive bulging or clubbing of tips\ ends of secondary gill filaments (club-shaped filaments) (Fig 3D).

Brain:

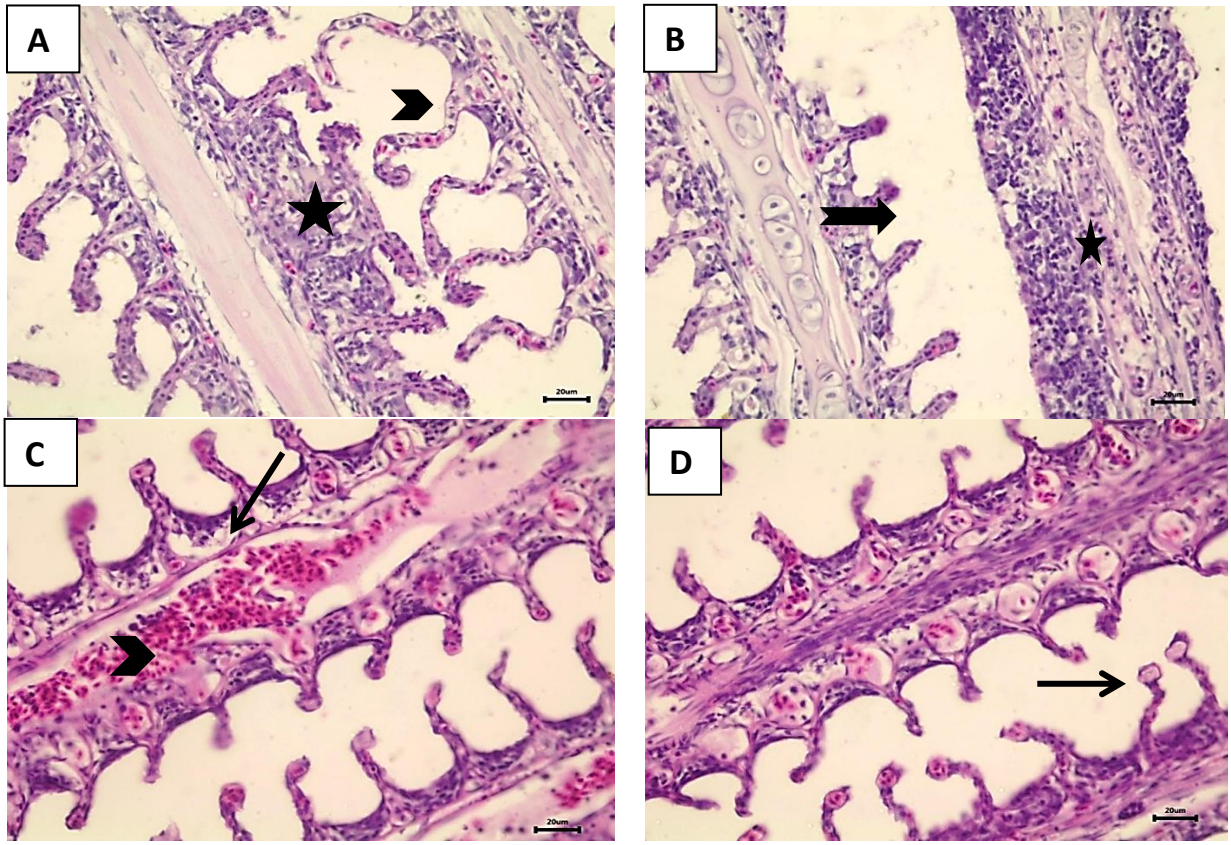
After 15 days of being subjected to DM (8 mg/l), the brain of fish displayed numerous histopathological defects, such as Separation or lifting and thickening of the meninges accompanied by submeningial edema (Fig. 4 A). Angiopathic changes also were noticed characterized by congestion of cerebral blood vessels accompanied with perivascular edema (Fig 4B). In the cerebellum, extensive neuronal degeneration was also found characterized by disorganization of Purkinje cells with pyknosis of the nucleus and necrotic areas in the inner granular layer of the cerebellum (Fig 4 C). There was necrosis of neurons and cytoplasmic vacuolization and disorganized neuronal tissue with blurry nuclei were detected in the degenerated neurons (Fig.4D).

Liver:

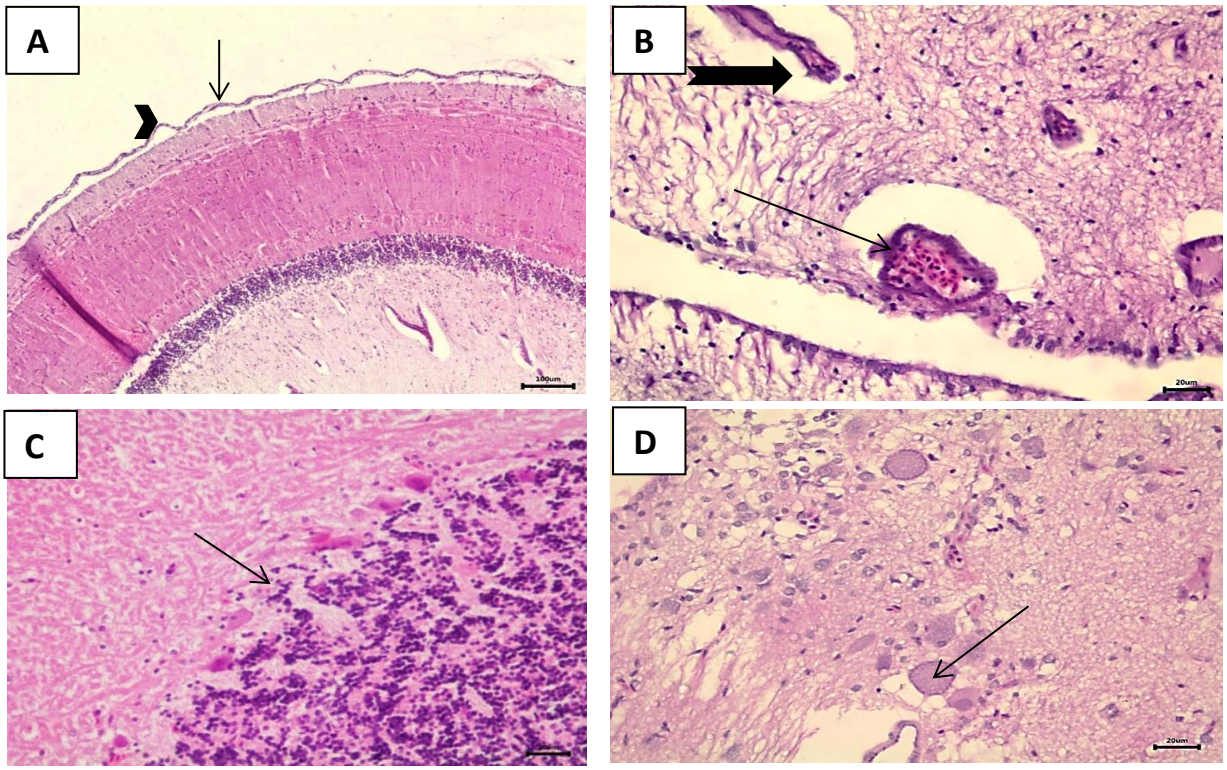
Severe vacuolar degeneration in all hepatic tissue with indistinct cellular boundaries and pyknotic nuclei was observed in the DM intoxicated group in addition to vascular changes characterized by congestion of central vein and hepatic sinusoids (Fig.5A). Inflammatory cellular reaction characterized by perivascular infiltration with inflammatory cells was also detected (Fig. 5B). In the same group hepatic tissue exhibited severe degeneration and necrosis; the necrotic hepato- pancreatic tissue infiltrated with mononuclear inflammatory cells (Fig. 5C).

Kidney

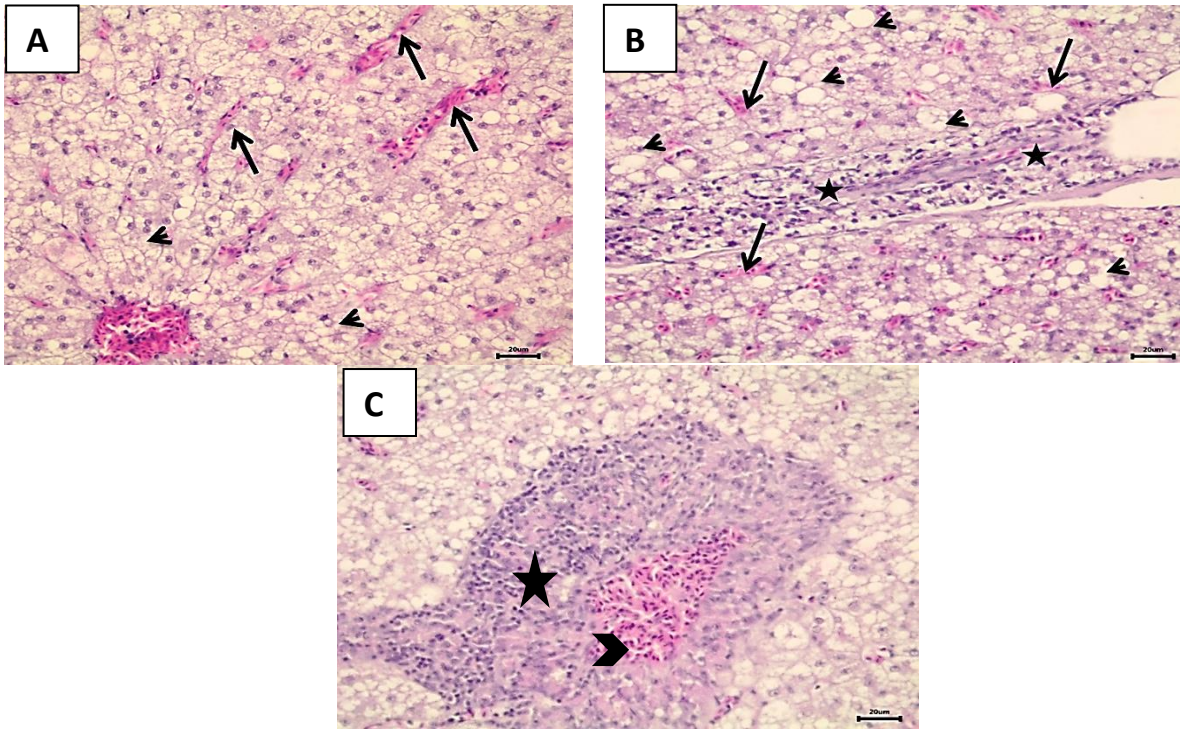
Dimethoate (8 mg/l) exposure caused broad glomerulus shrinkage, bowman's space expansion, and renal tubules with deteriorated epithelial and dilated lumens in certain regions, as well as complete cytolysis of the epithelium of renal tubules (Fig. 6A). Focal regions of interstitial tissue infiltration with inflammatory cells were also observed (Fig 6B).



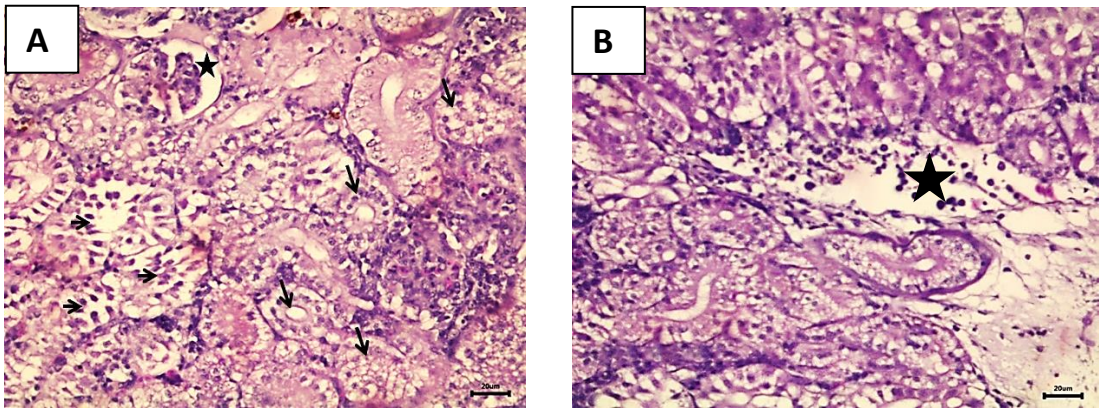
(Fig. 3) Gills of 15 days Dimethoate exposed fishes showing (A) Hyperplasia in the epithelium of primary lamellae with fusion of secondary gill lamellae (star), Curling of secondary gill lamellae (arrows head); (B) Shortening and fusion of secondary gill lamellae (notched arrows), infiltration of the fused secondary gill lamellae with inflammatory cells (star); (C) Hemorrhage in primary lamellae (arrows head) with epithelial edema (arrows); (D) Club-like elongated secondary lamellae (telangiectasis) (arrows) (H&E), bar=20.



(Fig. 4) Brain of 15 days Dimethoate exposed fishes showing (A) Separation and thickening of the meninges (arrow), submeningial edema (arrowhead); (B) Congestion of cerebral blood vessels (arrow) and perivascular edema (notched arrow); (C) disorganization of Purkinje cell layer (arrow); (D) Blurry nuclei were observed in the degenerated neurons (arrow) (H&E), bar=20.



(Fig. 5) Liver of 15 days Dimethoate exposed fishes showing (A) Congestion of the central vein (star), vacuolar degeneration of the hepatocytes (head arrows), dilatation and congestion in blood sinusoid (arrows); (B) Inflammatory cellular aggregation (star), dilatation and congestion in blood sinusoid (arrows), vacuolar degeneration of the hepatocytes (head arrows); (C) Mononuclear inflammatory cells in the necrotic hepato- pancreatic tissue (star), hemorrhage (head arrows),(H&E), bar=20.



(Fig.6): Kidney of 15 days Dimethoate exposed fishes showing (A) Shrinkage of the glomerulus, increasing of bowman`s space (star); Vacuolar degeneration in renal tubules (arrows); Cytolysis of the epithelium of renal tubules (head arrows) (B) Interstitial inflammatory cellular infiltration (star), (H&E), bar=20

Table 2: The histopathological scoring of lesions in the Dimethoate intoxicated group after 15 days of exposure

Gills	**leukocytes infiltration**	**fusion of gill lamellae**	**shortening of secondary gill lamellae**	**club-like secondary gill lamellae**	**hyperplasia of the epithelial cells of the secondary lamellae**
	+3	+5	+5	+5	+5
Brain	Meningeal oedema	Karyolysis of the neuronal nucleus	congestion of meningeal and cerebral blood vessels	Dis-organization of Purkinje cell	Purkinje cell pyknosis
	+5	+5	+5	+5	+5
Liver	Vacuolar hepatocyte degeneration	Inflammatory cell aggregation	Congestion of central vein and blood sinusoids	Hemorrhage in the hepato-pancreatic tissue	Focal necrosis with leukocyte infiltration
	+5	+3	+5	+5	+5
Kidney	Vacuolar degeneration of glomerular tuft	shrinkage of some glomeruli	increase of Bowmen's space	focal necrosis with leukocyte infiltration	Cytolysis of renal epithelium
	+5	+5	+5	+5	+3

None = 0 Mild = +1 Moderate = + 3 Sever = +5

DISCUSSION

In the current research, the objective was to assess the toxicity of DM on the brain, gills, kidneys, and liver of the Nile tilapia fish. The original group functioned as a control and the second group had an exposure of (8 mg/l) which is equal to (1/3) LC50 of Dimethoate for 15 days. Acute DM exposure was very toxic to Nile tilapia. The toxicity of DM on the Nile tilapia amplified with longer exposure times. When fishes were subjected to 8 mg/l of DM showed abnormal behavioral alterations. These abnormal behavioral alterations include loss of hunger and equilibrium, fast swimming, surfacing activity frequency, gulping of surface water, erratic and hysteric swimming, circling motion, convulsion, and remaining immobile on the aquarium bottom. Similar abnormal behavioral signs were described by, (Banik *et al.,* 2016) who stated that exposure of *Glossogobius giuris* to sub-lethal diazinon concentration, showed such as anxiety, loss of control, increased working motions, surface to-bottom motion, unexpected rapid motion and bottom resting. In the present study, we noticed that mucous secretion increases considerably in the exposed fishes, and turbidity of the water of the test troughs increases gradually during continuous DM exposure. Lokhande (2017) noticed increased mucus secretion after DM exposure to *Rasbora daniconius* as a compensatory reaction to the annoying impact of the pesticide on the body surface and mucous membrane, in addition to increased opercula movement, irregular swimming movements such as erratic motion, jerky motion, and fast swimming surface activity. Similar behavioral alterations in *Cirrhinus mrigala* subjected to diazinon and C. subjected exposed to atrazine and chlorpyrifos have both been documented (Rauf and Arain, 2013; Nwani *et al.,* 2013).

Dimethoate as an organophosphorus compound induces oxidative stress in aquatic organisms, similar to all aerobic organisms because of its ability to trigger the creation of reactive oxygen species (ROS) and trouble in antioxidant defense systems that lead to alteration in the structural and functional health of cell membrane (Sharma *et al.,* 2014). The results of the current research revealed that after 15 days of exposure, DM significantly increased the MDA values. This indicates that there is an increase in lipid peroxidation, which can be attributed to an excessive formation of ROS that may be caused by the leakage of an antioxidant enzyme. Similar lesions observed by (Ajith & and Jayaprakash, 2017) reported that Nile Tilapia (*Oreochromis niloticus*) treated with Dimethoate have significantly greater levels of LPO in their gills, livers, and kidneys. The values of nitric oxide in the Dimethoate group considerably rose over 15 days in comparison to the control group. Our findings were supported by earlier research (Moran *et al.,* 2010; Duzguner and Erdogan, 2012), which showed that a variety of environmental pollutants, including pesticides, enhance NO formation.

The catalase enzyme, which is found in peroxisomes, shields fish against oxidative stress by triggering the decomposition of hydrogen peroxide into water and oxygen (Atli & Canli, 2007; Thabet *et al.,* 2021). The current study displayed that the liver tissues of *Oreochromis niloticus* that were experimentally subjected to DM concentration had somewhat decreased

CAT activity. These results disagree with those of (Ibrahim, 2015), who discovered that the gills, kidneys, and livers of *Oreochromis niloticus* tissues treated with both DZN doses showed significantly increased CAT activity. Box *et al.*, (2007) noticed that organo-phosphate pesticides and subject to ecological contaminants triggered a significant decrease in CAT activities in various tissues of *Ictalurus nebulosus and Mytilus galloprovincialis*. Crestani *et al.* (2007) also detected a decrease in CAT activity in the liver of *R. quelen* treated with clomazone. In the present study, the elevation of GPx activities offers a potential line of defense in response to increased levels of LPO in reaction to LC50 of DM. The present results indicated that GPx activity is markedly increased after 15 days of Nile tilapia exposure to the LC50 of Dimethoate. Increased GPx activity was observed as well in treated Prochilodus lineatus as a consequence of the impacts of the herbicide (roundup) and the increased formation of oxidative stress, which would disturb the equilibrium of the ROS-antioxidant system (Modesto and Martinez, 2010). The research's findings are consistent with those in common carp (*Cyprinus carpio L.*), where it was found that ATZ interfered with various endpoints (diminished GPx and SOD activities) in addition to a rise in the MDA linked to ROS creation in the liver and gill tissues (Xing *et al.*, 2012).

The present study displayed that a 15-day sub-lethal dose of DM also induced toxicity in the brain, liver, kidney, and other organs, including the gills, of Nile tilapia. Significant tissue injury and shifts in the activity of antioxidant enzymes served as proof of this. According to (Wiieyaratne and Pathiratne, 2006), histopathological biomarkers in the fish's gills can serve as pointers for the health of the fish as a whole and show the impacts of disclosure to different anthropogenic contaminants. In the current research, filamentous epithelium proliferation, broad epithelial lifting, and hyperplasia of the epithelium, edema in the filamentary epithelium, dilation of the central venous with blood congestion and hyperplasia of the epithelium with the fusion of adjacent lamellae in certain regions were noted after 15 days of DM exposure. Issa *et al.* (2011) detected numerous histopathological changes in the gills of Nile tilapia treated with lesbian, including primary lamellae hemorrhage, intraepithelial edema with lifting, sloughing of epithelial cells of the secondary lamellae, hypertrophy and hyperplasia of the epithelial cells of the secondary lamellae, lamellar aneurysm. Dimethoate's cytotoxic effects on the gill of *Oreochromis mossambicus* were studied by Parikh *et al.* (2010), who found that at high doses, secondary lamellae clubbing (telangiectasis), primary lamellae enlargement and secondary lamellae loss occurred.

In the current study, degenerative modifications in a severe number of the neurons with cytoplasmic vacuolization, and the un-distribution of Purkinje cells with mild karyolysis nucleus were observed after 15 days of exposure. Similar histopathological lesions observed by (Lakshmaiah, 2016) in common carp brain (*Cyprinus carpio*) treated with organophosphate insecticide, damage of the architecture, neuronal necrosis, intracellular edema, and pycnotic nuclei, cytoplasmic vacuolization, neural cell degeneration and swollen sinusoids and hemorrhage at considerable regions. After 15 days of exposure, there were also multifocal

areas of hepatic necrosis with pyknotic and karyorrhectic nuclei and a total breakdown of the majority of the necrosed hepatocytes. According to (Neelima *et al.,* 2015), fish exposed to cypermethrin liver exhibited hepatocyte degeneration, necrosis, inflammatory cell aggregation, dilatation, congestion in the blood sinusoid, and fibrosis. All fish treated with diazinon showed severe degenerative deviations, extensive necrosis, pyknosis of the nucleus, and vacuolation (Banik *et al.,* 2016). In the current study, the kidney of the DM toxicity group displayed vacuolation in the tubular epithelium, shrinkage of the glomerulus, expansion of space inside Bowman's capsule, and mild dilation of the lumen of some epithelial tubules. These lesions are similar to alterations in the kidney of freshwater fish (*Piaractus mesopotamicus*) exposed to an organophosphate insecticide and have been documented as vacuolar degeneration of glomerular tuft, shrinkage of some glomeruli and dilatation of others, increased capsule space of Bowman, cloudy swelling of some epithelial tubules, and dilatation of lumens and obstruction of tubules by (Mataqueiro *et al.,* 2009).

CONCLUSION

The exposure of aquatic animals especially fishes to insecticides such as Dimethioate caused multiple toxic effects causing death.

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دراسات نسيجية مرضية وكيميائية حيوية على السمية الحادة للدايمثوات على أسماك البلطي النيلي

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الديميثوات (DM) هو أحد أنواع المبيدات الحشرية الفوسفورية العضوية، التي تقتل الحشرات والعث بشكل منهجي وفوري عند ملامستها وتضر بمجموعة من الحيوانات غير المستهدفة، بما في ذلك الأسماك عندما تدخل البيئة المائية. تهدف هذه الدراسة إلى تحديد التأثيرات النسيجية والكيميائية الحيوية الضارة المحتملة للدايمثوات على أسماك البلطي النيلي. تم تقسيم ٦٠ من أسماك المياه العذبة *Oreochromis Niloticus* ، وزن كل منها في المتوسط ١٣٠ ± ١٠ جم، إلى مجموعتين في ثلاث نسخ. كانت هناك ٦ مجموعات، ٣ مجموعات مراقبة، و ٣ مجموعات تجريبية تلقت الدايميثوات بجرعة ٨ ملغم/لتر في الماء لمدة ١٥ يومًا. تم جمع عينات الدم واستخدامها للمؤشرات البيوكيميائية. للتحليل النسيجي، تم جمع عينات من الدماغ والخياشيم والكبد والكلى. أظهرت نتائج الدراسة أن الأسماك المعالجة بالدايمثوات أظهرت خياشيم شاحبة وأعراض قلق، بالإضافة إلى احتقان ونزيف في العديد من الأعضاء الداخلية، بما في ذلك الدماغ والكلى والكبد. كانت هناك زيادة كبيرة في مستويات الدم من أكسيد النيتريك، بيروكسيد الدهون، وبيروكسيديز الجلوتاثيون وانخفاض كبير في مستويات الكاتالاز في مجموعة الديميثوات بالمقارنة مع المجموعة الظابطة. ولذلك توصي الدراسة بأن لا ينبغي استخدام المبيدات الحشرية، وخاصة DM، بلا مبالاة في الزراعة والسماح لها بالتراكم في مجاري المياه لأن ذلك له آثار ضارة على الأسماك.