EFFECTS ON PATHOLOGICAL AND TOXICOLOGICAL ASPECTS OF NILE TILAPIA Oreochromis niloticus OF USING MALATHION TO ERADICATE Lethocerus niloticum INSECTS

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ABSTRACT

Male tilapia fish (Oreochromis niloticus) and water bugs (Lethocerus niloticum) were studied in a presence of malathion (diethyldimethoxy thiophosphoryl thiosuccinate) of commercial grade 57%. Fish were fed during the experimental period once daily on a commercial dry pellet ration, while water bugs were fed on fish offales. They all were acclimatized to the same laboratory conditions for 2 weeks before the beginning of the experiment. Different malathion concentrations were experimented. The present study revealed that the minimum malathion concentration level that induced mortality of water bugs without any lethality on Oreochromis niloticus fish was 0.26 mg/L for 96 hrs exposure period. However, some toxic effects and histopathological alterations were observed at that condition. Gonadosomatic index, serum Testosterone, T3 and T4 concentration levels were determined. Brain AChE and LDH activities were also analyzed. Fish liver, spleen, gills, testes, brain and thyroid gland samples were histopathological studied.

Keywords: tilapia, water bugs, malathion, histopathological

INTRODUCTION

Water bugs (Lethocerus species) are commonly found in fish ponds. They normally feed on other pond life such as insect tadpoles, salamanders, small fish and snails (Huntly, 1998). Water bugs also act as a host for fish parasites (De et al., 1995).

Malathion is an organophosphate insecticide that has been widely used in agricultural activities to control harmful insects (NIOSH, 1976). It was used to control invertebrate in temporary ponds in the African Sahel (Lahr et al., 2008). Unfortunately, malathion seems to be toxic not only to insects but also to fish (Krueger et al., 1960). Several studies were carried on fish to determine the half lethal concentration (LC50) of malathion. Shim and self (2004) reported 96 hrs (LC50) values for the herbivorous fish to be from 0.14 to 8.7mg/L for different organophosphorous compounds including malathion. Pathiratne and George (2007) reported that tilapia were very sensitive to malathion concentration of 2 ppm. Malathion seems to reduce the thyroid gland activity of catfish (Yadlav and Singh, 1986). It acts as a neurotoxin due to its ability to block neurotransmission by inhibiting the enzyme acetylcholinesterase (O'Brien, 1960). Hematological changes (Mishra and Srivastava, 1983) as well as histopathological changes (Walsh and Ribelin, 1975; Reddy, 1988) have also been reported. The presence of water bugs Lethocerus niloticum in Lake Mariut in Egypt was recorded by Sorour (2008). Therefore, this study was carried out to determine the minimum malathion concentration level that induced mortality of water bugs without any lethality
on *Oreochromis niloticus* fish. The effect of such level on gonadosomatic index, level of testosterone and the thyroid hormones, as well as the changes in the activities of brain tissue acetylcholinesterase (AChE) and lactate dehydrogenase (LDH) of tilapia has also been studied. Furthermore the histopathological alterations in the affected fish organs (liver, spleen, gills, testes, brain and thyroid gland) were also recorded at that concentration level.

**MATERIALS AND METHODS**

Male tilapia fish (*Oreochromis niloticus*) of 85 ± 2 g and 15± 1 cm length were purchased from a commercial producer (Fouky farm in Kalyobia governorate). Fish were randomly distributed in glass aquaria of 50 liter each at Abassaa research center. Dechlorinated tap water at temperature of 27 ± 2 °C with continuous oxygen supply through air pumps was used. Fish were fed during the experimental period once daily on a commercial dry pellet ration (25% protein). A 12 hrs photo period was artificially maintained. They were acclimatized to the laboratory conditions for 2 weeks before the beginning of the experiment. Water bugs (*Lethocerus niloticum*) were purchased from a local commercial specific experimental animal house producer. They were distributed and kept in glass aquaria of a same volume and under a condition similar to that of fish. Water bugs were acclimatized to the laboratory condition for 2 weeks before the beginning of the experiment and fed on fish offales during this period. Malathion (diethyldimethoxy thiophosphoryl thiosuccinate) of commercial grade 57% was used in the present study.

Five aquarium with five different malathion concentrations of 0.08, 0.17, 0.35, 0.7 and 1.4 mg/L, respectively, each was stocked with a group of ten fish. No control group (table 1, 2) was included as it is expected to have no effects on the fish or the bugs mortality. However the control group was included in studies of the hormonal and enzymes changes (table 3). Dead fish were collected and mortality percent for each group was calculated after 96 hrs (Table 1). Other five aquarium, with the same previous concentrations of malathion, each was stocked with a group of ten water bugs. Dead bugs were collected daily and mortality percent for each group was calculated after 96 hrs (Table 2).

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The data of that experiment suggested that average concentration of malathion that cause death to 100% of water bugs and least mortality percent of fish was 0.26 mg/L. Therefore, another experiment was conducted to examine the effect of such concentration on gonadosomatic index, hormonal changes and enzymatic activities in brain as well as pathological changes of fish exposed to that concentration. Two groups of ten fish were stocked in two different aquariums. The first one was filled with clear water and served as control group while the other contained 0.26 mg/L malathion and considered as the test group. Five live fish were collected from each group after 24 hrs and 96 hrs, respectively, for the examination of the pathological and toxicological effects.
Gonadosomatic index was determined according to Munkittrick and Dixon (1988). Testosterone concentration in serum was determined by radioimmunoassay (RIA) according to the method of Tremblay et al., (1972). On the other hand, T₃ and T₄ were determined according to the method of Brown and Eales, (1977). AChE activity in brain was determined according to Szasa (1968), while the LDH activity was determined according to the method of Annon (1971). Live fish were subjected to post-mortem examination and any lesion seen was recorded. For the histopathological study, samples from liver, spleen, gills, testes, brain and thyroid gland were fixed in 10% neutral buffered formalin, dehydrated in alcohol, cleared in xylol and embedded in paraffin. About 4μ-thick sections were prepared and stained with Hematoxlene and Eosine (Carleton et al., 1967).

Student’s t-test was used to compare the significance of differences between means of control and treated groups according to Snedecor (1971).

RESULTS

The data presented in Table 2 showed that the concentration level of malathion of 0.7 mg/L caused death to 100% of the exposed water bugs. However, this concentration level caused also death to 50% of the exposed fish (Table 1). The concentration level of 0.35 mg/L caused death to 80% and 10% of water bugs and fish, respectively. On the other hand, at the 0.17 mg/L malathion neither water bugs nor fish was affected. Therefore, the present data also showed that the concentration level of malathion which caused mortality to the greatest number of water bugs and might had no lethal effect on tilapia lies between the concentration levels of 0.17 and 0.35 mg/L. Therefore, an average concentration of malathion of 0.26 mg/L may be recommended in the present work to be effectively used in eradicating water bugs in tilapia ponds.

The effect of the average concentration level on gonadosomatic index was presented in Table (3). No significant (P<0.05) decrease in the gonadosomatic index after 24 hrs (0.013 ± 0.0004) was observed compared to the control group (0.014 ± 0.0002). On contrary, the same index showed significant (P<0.05) decrease (0.012 ± 0.001) after 96 hrs compared with control group.

The effect of the average concentration level on the testosterone and thyroid hormones is presented in Table (3). There was a significant (P<0.05) decrease in testosterone level (79.2 ± 1.68) after 96 hrs exposure period compared with the control group (81.4 ± 1.97). Similar trend was recorded for the thyroid hormones levels (T₃ & T₄) i.e. significant (P<0.05) decrease (62.76 ± 1.08 and 1.82 ± 0.025, respectively) after the same period of exposure, compared with the control group (64.62 ± 0.49 and 2.30 ± 0.02, respectively). Moreover, the levels of hormones showed no significant decrease during 24 hrs exposure to malathion.

These results showed no significant (P<0.05) changes in both AChE and LDH activities of brain tissue at 24 hrs exposure period. While induced significant inhibition in the activities of fish AChE and lactate LDH in the brain.
tissue at 96 hrs where the values being 64.0 ± 1.41 and 59.6 ± 0.89 u/L, respectively, in comparison to the control values, 68.0 ± 0.31 and 61.2 ± 1.24 u/L, respectively. (Table, 3).

Histopathological examination of fish treated with 0.26 mg/L malathion for 24 hrs revealed no alterations in examined organs especially testes and thyroid gland (Fig. 1, 2).

The gross examination of fish exposed to the average concentration level (0.26 mg malathion/ L) for 96 hrs revealed abnormal black discoloration of the body, severely congested gills and rough scales. The histopathological examination of such group showed the apparent changes. In the liver, vacuolar degeneration of the hepatocytes and severe congestion of hepatoporal blood vessels were observed (Fig. 3). Mononuclear cell infiltration was noticed in portal area and around the hepatoporal blood vessels. In such cases, mild degenerative changes were seen in hepatopancreatic region. In other cases, histopathological examination showed mild pathological alterations including congestion of the hepatic blood vessels and granular degeneration of some hepatocytes. The examination of spleen revealed depletion of lymphocytes with decreased melanin intensity of melanomacrophage centers (Fig. 4). In other cases, splenic ellepsoids showed marked thickening of their capillary wall, swelling of the endothelial lining with activation of the haematopoietic cells around the blood vessels of the ellepsoids (Fig. 5). The examined gills revealed severe congestion of the lamellar capillaries (Fig. 6), while in other fish, gill edema and aggregation of eosinophilic granular cells (EGC) was the common picture (Fig. 7). In other cases, hyperplasia of the cells constituting gill lamellae together with fusion of secondary lamellae was a common lesion observed (Fig. 8). The examined testes of this group showed degeneration and atrophy of the germinal epithelium of seminiferous tubules and a few spermatocytes in their lumen (Fig. 9). The brain of the fish in this group showed focal gliosis with oedema (Fig. 10). Thyroid gland examination revealed devoience of some follicles from colloid (Fig. 11), but without evidence of cellular hyperplasia.

DISCUSSION

The present results revealed that the 96 hrs LC$_{50}$ of malathion was 0.70 mg/L. Shim and Self (2004) reported 96 hrs LC$_{50}$ values for the herbivorous fish to be from 0.14 to 9.7 mg/L for different organophosphorous compounds including malathion. Shukla et al., (2002) determined the LC$_{50}$ of malathion for the fingerlings of Channa punctatus and found it was in the range of 10.95 to 3.22 mg/L for 24 to 96 hrs exposure periods. Jagan et al., (1989) recorded the LC$_{50}$ of malathion for carp fish (Cyprinus carpio) to be 0.138 ppm. Tsuda et al. (1997) determined the 48 hrs LC$_{50}$ for malathion in Killfish to be 1.8 mg/L. Pathiratne and George (2007) reported that tilapia were very sensitive to malathion (96 hrs LC$_{50}$ = 2 ppm). Variation between the present results concerning the estimation of LC$_{50}$ of malathion and those of other investigators may be attributed to several factors such as species, age,
body weight, time of exposure, the method used and the sensitivity of fish as well as stress factors. Concerning the reduction of gonadosomatic index of fish exposed to malathion the present results were in agreement with the findings reported by many investigators. Haider and Upadhyaya (2005) found that the commercial formulation of four organophosphorous insecticides birlane, jardona, phosdrin and malathion caused loss of stage II and III oocytes.

Table (1) Mortality data of Oreochromis niloticus fish exposed to different concentration levels of malathion

<table>
<thead>
<tr>
<th>Fish Group</th>
<th>Fish/group</th>
<th>Dose mg/L</th>
<th>Dead fish</th>
<th>Mortality %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st</td>
<td>10</td>
<td>0.08</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2nd</td>
<td>10</td>
<td>0.17</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3rd</td>
<td>10</td>
<td>0.35</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>4th</td>
<td>10</td>
<td>0.70</td>
<td>5</td>
<td>50</td>
</tr>
<tr>
<td>5th</td>
<td>10</td>
<td>1.40</td>
<td>6</td>
<td>60</td>
</tr>
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</table>

Table (2): Mortality data of water bugs exposed to different concentration levels of malathion

<table>
<thead>
<tr>
<th>Water Group</th>
<th>Water bugs /group</th>
<th>Dose mg/L</th>
<th>Dead water bugs</th>
<th>Mortality %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st</td>
<td>10</td>
<td>0.08</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2nd</td>
<td>10</td>
<td>0.17</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3rd</td>
<td>10</td>
<td>0.35</td>
<td>8</td>
<td>80</td>
</tr>
<tr>
<td>4th</td>
<td>10</td>
<td>0.70</td>
<td>10</td>
<td>100</td>
</tr>
<tr>
<td>5th</td>
<td>10</td>
<td>1.40</td>
<td>10</td>
<td>100</td>
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</table>

Table (3) Effect of average concentration level of malathion on gonadosomatic index, some hormonal and enzymatic activities of Oreochromis niloticus

<table>
<thead>
<tr>
<th>Control Zero Malathion</th>
<th>Treatment 0.26 mg/L. Malathion</th>
</tr>
</thead>
<tbody>
<tr>
<td>24 h.</td>
<td>24 h.</td>
</tr>
<tr>
<td>96 h.</td>
<td>96 h.</td>
</tr>
<tr>
<td>Ganado somatic index %</td>
<td>0.014 ± 0.002</td>
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<td></td>
<td>± 0.0002</td>
</tr>
<tr>
<td>Testosterone ng/dL</td>
<td>81.4 ± 1.97</td>
</tr>
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<td></td>
<td>± 0.97</td>
</tr>
<tr>
<td>Triiodothyronine T3 ng/dL</td>
<td>65.5 ± 0.15</td>
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<td></td>
<td>± 0.15</td>
</tr>
<tr>
<td>Thyroxin T4 ng/dL</td>
<td>2.3 ± 0.01</td>
</tr>
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<td></td>
<td>± 0.01</td>
</tr>
<tr>
<td>Acetylcholin esterase ACHE u/L</td>
<td>68.0 ± 0.31</td>
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<td></td>
<td>± 0.31</td>
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<tr>
<td>Lactate dehydro genase LDH u/L</td>
<td>61.2 ± 1.24</td>
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<td>± 1.24</td>
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</tbody>
</table>

*Significant at p< 0.05
Fig. (1): Testis of Oreochromis niloticus fish exposed to 0.26 mg/L malathion for 24-h, showing normal seminiferous tubules impacted with spermatozoa. [H&E stain, X40]

Fig. (2): Thyroid gland of Oreochromis niloticus fish exposed to 0.26 mg/L malathion for 24-h, showing normal follicles. [H&E stain, X40]
Fig. (3): Liver of *Oreochromis niloticus* fish exposed to 0.26 mg/L malathion for 96-h, showing vacuolar degeneration of the hepatocytes and severe congestion of hepatoportal blood vessels. [H&E stain, X40]

Fig. (4): Spleen of *Oreochromis niloticus* fish exposed to 0.26 mg/L malathion for 96-h, showing lymphocytic depletion and melanomacrophage center devoid of melanin. [H&E stain, X40]
Fig. (5): Spleen of *Oreochromis niloticus* fish exposed to 0.26 mg/L malathion for 96-h, showing swelling of endothelial cells of ellepsoidal capillaries (arrow), thickening of capillary wall and activation of hemopoietic element. [H&E stain, X40]

Fig. (6): Gills of *Oreochromis niloticus* fish exposed to 0.26 mg/L malathion for 96-h, showing severe lamellar capillary congestion. [H&E stain, X40]
Fig. (7): Gills of *Oreochromis niloticus* fish exposed to 0.26 mg/L malathion for 96-h, showing oedema and eosinophilic granular cells infiltration. [H&E stain, X40]

Fig. (8): Gills of *Oreochromis niloticus* fish exposed to 0.26 mg/L malathion for 96-h, showing hyperplasia and fusion of secondary lamellae. Notice: mucous secreting cells activation and eosinophilic granular cell aggregation. [H&E stain, X40]
Fig. (9): Testis of *Oreochromis niloticus* fish exposed to 0.26 mg/L malathion for 96-h, showing degeneration of germinal epithelium. Notice: the seminiferous tubules are devoid of spermatozoa. [H&E stain, X40]

Fig. (10): Brain of *Oreochromis niloticus* fish exposed to 0.26 mg/L malathion for 96-h, showing focal gliosis. [H&E stain, X40]
accompanied by significant decline in gonadosomatic index of fresh water teleost, *Mystus vittatus* (Bloch) exposed to malathion. Shukla *et al.* (2002) reported that the sublethal concentration of malathion had adverse effects on growth and metabolism, and exposure of fingerling *Channa punctatus* fish to 2.5 mg/L malathion induced significant reduction in growth. Khillare and Wagh (1988) showed that the fish *Puntius stigma* collected from the river Kham Aurangabad, India showed changes in survival, feeding, growth rate and oxygen consumption during chronic exposure to endosulfan, malathion and sevin (carbaryl). Ramakrishnan *et al.* (1997) exposed the *Oreochromis mossambicus* to the sublethal concentration of malathion (0.3-1.2 ppm).

The present results showed significant (P<0.05) reduction in the testosterone level of 96 hrs malathion exposed fish. This decrease is supported by the histopathological findings of the affected testes. There was marked degeneration and atrophy of the germinl epithelium of the seminiferous tubules with a few spermatogonia in their luminae. Several researches proved that some types of pesticides as organophosphates, pyrethroids, fungicides and herbicides may possess antiandrogenic effect in different livestock (Brody *et al.*, 1983; Eil and Nisula, 1990 and Wolf *et al.*, 2003). They also showed that pesticides can interact competitively with androgen receptors and sex hormones binding globulin, a mechanism by which exposure to pesticides may result in disturbances in endocrine effect related to androgen action. Singh and Singh (1987) studied the effect of
cythion (organophosphorous) and BHC (organochlorine) on the level of sex hormones (testosterone, estradiol-17 beta and estrone) in catfish *Clarias batrachus*. Sublethal concentrations of either pesticide apparently affected sex hormones production after 4 weeks of exposure, as was evident by the decrease of their levels in blood plasma. They suggested that cythion and BHC toxicity affected sex hormones production due to the impairment in the synthesis and/or release of these steroids. In the present investigation, the reduced serum T₃ & T₄ levels in 96-hours exposed fish clearly indicated altered thyroid function. These results were in agreement with those of Sinha et al., (1991) and Aktar et al. (1996). Sinha et al., (1991) concluded that malathion decreased the level of T₃ & T₄ and T₃/T₄ ratio in *Clarias batrachus* fish without altering the level of T₄ in blood circulation. Aktar et al., (1996) exposed rats to malathion and observed that malathion induced significant decrease in serum concentration of T₃ & T₄. The reduced T₄ level by malathion might be due to the sequential retardation of iodine accumulation and its conversion into hormonal form i.e. hormone biosynthesis (Singh and Singh, 1980). Leatherland and Sonstegard (1978) reported significant reduction in the levels of both T₃ & T₄ in *Oncorhynchus kaisutch* fed with an organophosphorous, mirex. They suggested that the extrathyroidal conversion of T₄ to T₃ was inhibited. The present results demonstrated clearly that malathion had an inhibitory effect on biosynthesis of T₄ and the extrathyroidal conversion of T₄ to T₃.

Inhibition of AChE activity in fish brain after exposure to organophosphorous pesticides is very specific effect, which can be utilized for diagnostic purposes. This inhibitory effect has been taken as an index of organophosphorous insecticide toxicity (Chakraborty et al., 1978). The inhibition of AChE was maximal in the brain, muscles and gill (Nagat Ali, 1995). The inhibition of AChE activity reported in the present study was in agreement with Ansari and Kumar (1984); Johnson and Wallace (1987); Sulaiman et al., (1989); Nemcsok et al., (1990).

Ansari and Kumar (1984) reported that a 7-day exposure to 0.5-1.1 mg/L malathion significantly inhibited the brain AChE activity in the zebrafish (*Brachydanio rerio*). Johnson and Wallace (1987) recorded that malathion caused inhibition of brain AChE activity in fat head minnows and rainbow trout. Sulaiman et al. (1989) mentioned that AChE activity of the brain was depressed significantly in *Tilapia nilotica* and *Tilapia mossambica* exposed to malathion. Nemcsok et al. (1990) studied the In-vivo effect of 2 ppm of malathion on the carp (*Cyprinus carpio*) brain and liver AChE. They found that the enzyme activity decreased significantly in the examined tissues. The inhibition of AChE of brain was a dose-dependent Das and Sengupta (2003).

The present results showed that the brain LDH activity was significantly reduced in the treated fish than control group. These findings was confirmed by the histopathological examinations in the present study since the brain showed focal gliosis and oedema. These results were in agreement with those of Rani et al., (1990). They also exposed *Clarias batrachus* fish to
malathion at 5 ppm for 96 hrs. The results indicated reduction in the activity of LDH in liver, gills and brain. LDH has a role for interconversion of lactate and pyruvate in the presence of NADH and NADH$_2$. Reduction of LDH activity in brain tissues or other tissues indicated tissue damage to the affected organs with elevation in serum LDH activity (Murray et al., 1988).

Our findings revealed that *Oreochromis niloticus* exposed to malathion resulted in marked histopathological findings in the liver, spleen, gills, tests, brain and thyroid gland of the tested fish. The histopathological alterations in the liver of treated fish were in agreement with the findings reported by Anees (1978); Ramalingam (1988) and Al-Hamdanne (1998). Anees (1978) stated that malathion caused vacuolation and necrosis of hepatocytes in channel catfish. Ramalingam, (1988) reported that malathion caused comparatively more liver damage of fish *Sarotherodon mossambicus* than mercury and several changes such as necrosis, fatty degeneration and also red cell occlusion in portal vessels were observed. Several histopathological changes in carp fish exposed to malathion were recorded by Al-Hamdanne (1998). These changes include vacuolation of hepatic cells with sinusoidal congestion, kidney congestion, haemorrhage of intestinal submucosal layer and coagulative necrosis of muscles. Regarding the histopathological changes in the spleen of malathion-treated fish; Abu-Hadeed (1978) exposed Nile catfish (*Clarias lazera*) to the organophosphorous insecticide (Curacron) and his results revealed severe congestion and destruction of some lymphoid follicles of spleen. Similar findings were reported by Abou El-Magd et al., (1998) who observed congestion, moderate depletion of the lymphocytes of the splenic white pulp, thickening of splenic blood vessels, activation of the melanomacrophage centers and infiltration of the red pulp with lymphocytes in Nile catfish treated with glyphosate. Our results were almost similar to those recorded by Hussain (1993) who showed depletion of the lymphocytes in the white pulp of spleen in rats treated with some pesticides. Marked histopathological lesions in the gills of *Oreochromis niloticus* fish exposed to malathion were observed in the present study. Several investigators reported similar alterations in the gills of the affected fish. Walsh and Ribelin (1975) for instance mentioned that malathion caused lesions in the gills of exposed fish including necrosis, hyperplasia, hypertrophy and oedema. Reddy (1988) showed also that sublethal concentration of malathion (2 ppm) damage to the gill architecture in carp fish after 15 days from exposure. He also reported that this damage decreased rates of $O_2$ consumption rates and opercular movement of fish.

In conclusion the present study revealed that the minimum calculated malathion concentration level that may induce mortality of water bugs without any lethality on *Oreochromis niloticus* fish was estimated at 0.26 mg/L for 96 hrs exposure period. However, some toxic effects and histopathological alterations were practically observed at that level. Therefore further studies are needed to verify the optimum level of malathion that could be used to eradicate *Lethocerus niloticum* without any adverse effects on the tilapia fish or human safety.
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تأثيرات سمية وباثولوجية على أسماك البلطي النيلية عند استخدام الملاطيون لإبادة حشرة بق الماء
أم مصطفى أحمد و أشرف هاشم جمعة
المؤسسة الأكاديمية للأعمال بمركز البجوث الزراعية.

يعتبر وجود بق الماء (ليثوسيرس النيلي) في أحواض الأسماك من الخطورة حيث أنه يغذي على الأسماك ويهدف هذة الحشرات إلى تحديد جرعة الفسيح الشريعي الفتليليون التي تسبب نفوق بق الأماكن دون أي تأثير خفيف على الأسماك. الهدف من تجربة جذور الأسماك البلطي النيلي إلى تحديد تركيز نصف الميت للأسماك. من ناحية أخرى تم تجريبي بق الماء (ليثوسيرس النيلي) على نفس الجرعات لمعرفة نسبة الفروق الحادة في بق الماء عند التعرض لهذه الجرعات. ومن نتائج التجربتين السابقتين يمكن تحديد مستوى الجرعة من الملاطيون الذي يسبب نفوق بق الماء دون حدوث نفوق للأسماك. هذا وقد تجريبي مجموعة أخرى من الأسماك دخول بق الماء إلى هذه الجرعة لمدة 24 ساعة و 96 ساعة للدراسة التأثير السماع وكذلك التأثير الهستوباثولوجي على أسماك الأسماك المعرضة لهذه الجرعة. وقد تم تجريبي معامل وزن الخصية إلى السماع وكذلك بعض هرمونات السيرم مثل التستوستيرون والترادي آيودو تيروتين (تي 3) والتيروكسيدين (تي 4) وأيضا النشاط الأندروبيبي لبعض خصائص النسيج الجذور مثل الكولين استيريز والثيرونين ديدروجيناز. وقد أوضح تجربة أن التركيز نصف الميت للملاثيون هو 0.70 مجم /ثرب من الماء بينما مستوي الجرعة الأممن من الملاطيون الذي يسبب نفوق بق الماء ولا يسبب نفوق للأسماك هو 0.26 مجم /ثرب. أيضا هناك نفس معيون في معامل وزن الخصية بالنسبة للسماع بعد 24 ساعة من التعرض مع نفس في مستوى الهرمون بالسيرم وكذلك تثبيط لنشاط خلايا أنسجة المع. وقد دعمت هذه النتائج بالدراسة الهستوباثولوجي التي أوضحت وجود تغيرات وباثولوجية في جميع الأسماك التي تم تجريبيها بعد هذه الفترة من التعرض لهذه الجرعة. ومن التحليلات الحيوانية لهذه المناطق وجود أن مستوي الجرعة الأدنى من الملاطيون الأسماك مفاهيم في إبادة بق الماء هو 22 مجم /ثرب ويفوق للجرعة لمدة 24 ساعة فقط حيث أنه لا يوجد أي تأثير عقسي لهذه الجرعة على الأسماك خلال هذه الفترة من التعرض.