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Impact of a Short – Term Malathion Exposure of Nile Tilapia, (*Oreochromis niloticus*): The Protective Role of Selenium

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Abstract: Malathion is an organophosphate pesticide widely used to control a variety of insects in agriculture. It can reach the aquatic ecosystems affecting non target organisms like fish. The purpose of this study was to determine LC_{50} of malathion and to investigate the possible protective effects of selenium on malathion-induced toxicity in Nile tilapia. The fish were exposed to sub lethal concentrations of malathion (1/2 and 1/4 LC_{50}) for 15 days, and selenium (5.54 mg/kg of fish weight) was simultaneously administered. Blood and liver samples were collected at the end of the experiment. Biochemical parameters [serum glucose, cortisol, acetylcholinesterase (AchE)], haematological profiles [white blood cells (WBCs), red blood cells (RBCs) counts, haemoglobin (Hb) concentration, haematocrit (Ht) level], and oxidant/antioxidant statuses [lipid peroxidation (LPO) level, superoxide dismutase (SOD), glutathione-S-transferase (GST) and catalase (CAT) activities] were analysed. The findings of the present study revealed that short-term exposure to malathion at sub lethal concentrations induced biochemical and haematological alterations in *Oreochromis niloticus* and led to oxidative damage. Moreover, the administration of selenium considered as an effective way to counter the toxicity of malathion in tilapia fish.

Keywords: Malathion, *Oreochromis niloticus*, Fish Physiology, Oxidative Stress, Selenium

1. Introduction

For centuries, pesticides have been used in agriculture to improve food production by eliminating unwanted pests and controlling disease vectors [1]. Among common pesticides, organophosphorus (OP) compounds are broadly used in medicine, industry and cultivation [2]. Malathion (O,O-dimethyl-S-1,2-bisethoxycarbonylethylphosphorodithioate) is a non systemic, wide spectrum pesticide in the organophosphate chemical family and is extensively used throughout the world [3], which is mostly preferred for its short half-life and high selectivity to target pests [4,5]. Malathion is used excessively to protect crops which eventually affects the aquatic ecosystem including fishes. Malathion gets oxidized to malaoxon by cytochrome P450 enzymes and it is considered to be more toxic than the parent compound [6].

The primary site of action of malathion is the central and peripheral nervous systems because they inhibit Acetylcholinesterase (AChE), the enzyme that hydrolyses the neurotransmitter acetylcholine (ACh). In the presence of an inhibitor of AChE, synaptic ACh may increase to abnormally high concentrations, which is assumed to precipitate a "cholinergic crisis" that can be debilitating and fatal [7]. So that, the measurement of AChE activity is a proven indicator of OP exposure in a number of non-target aquatic organisms including fish [8, 9]. The exposure of fish to several chemical agents may induce changes in haematological parameters, which are frequently used to estimate fish health. Therefore, haematological techniques are the most commonly used method to determine the sublethal effects of contaminants [10,11].

Bioaccumulation of toxic substances prompts redox reactions generating free radicals, especially free oxygen radicals, but also other reactive oxygen species (ROS) are produced, that induce biochemical changes in fish tissues [12,13]. Oxidative stress happens when an imbalance occurs between production and removal of ROS. The ROS can be detoxified by an enzyme defense system, comprising superoxide dismutase (SOD), catalase (CAT), while organic peroxides can be detoxified by the activity of glutathione-S-transferase (GST) [14]. Several studies proved that changes in the levels of antioxidant enzyme activities

can be used as possible biomarkers in different aquatic organisms [15]. Selenium (Se) is a vital trace element in animal nutrition, including fish [16] which has been reported to counteract ROS and protect the structure and function of proteins, DNA and chromosomes against oxidation injury [17,18]. The synergistic effect of Se has proven powerful in reducing storage and toxicity of ROS [19,20]. Se is required in the diet for normal growth and physiological function of fish [21] which assumes a critical cell reinforcement part since it is a GPx cofactor. GPx scavenges H_2O_2 and lipid hydroperoxides, utilizing diminishing counterparts from glutathione and protecting membrane lipids and macromolecules from oxidative damage [22]. Also, Se presents in the active site of GPx contributes both to its catalytic activity and spatial conformation [23]. The aim of the present study was to: a) determine LC_{50} of malathion in Nile tilapia, *Oreochromis niloticus*, b) study the changes of biochemical, haematological parameters and oxidative metabolic in *O. niloticus* as a result of malathion exposure. Lastly, clarifying the possible ameliorating effects of dietary (Se) in struggling the previous parameters.

2. Materials and Methods

2.1. Chemicals

2.1.1. Malathion

The insecticide that was used in the present study was an emulsifiable concentrate of malathion 57% (El- Nasr Pharmaceutical chemical Co., Egypt) with molecular weight of 330.35 and chemical name: (O,O dimethyl S-1,2 dicarbo-ethyl-thiophosphate).

2.1.2. Selenium and Biochemical kits

Selenium as sodium selenite pentahydrate was obtained from Merck (Darmstadt, Germany). Biochemical kits (glucose, cortisol and AchE) were purchased from Gamma Trade Co., (Cairo, Egypt). All other chemicals used in the study were of analytical grade.

2.2. Fish

A total number of 320 *O. niloticus* with average body weight 150±10 g. were obtained from Abassa fish farm, El-Sharkya governorate, Egypt. Fish were transferred to the laboratory in 100 liters well aerated fiberglass tanks, followed by clinical examination to assure the absence of any abnormalities or external lesions on fishes. The fish were kept in identical glass aquaria measuring (80 x 40 x 40 cm) supplied with continuous aerated dechlorinated water 26±2◦C and pH was 7.5±0.5. Fish were fed with commercial diet containing 32% protein twice daily and kept two weeks for acclimation.

2.3. Determination 96 h LC_{50} of Malathion

The half lethal concentration (LC_{50}) of malathion was determined with definitive test by the static renewal bioassay method of Litchfield and Wilcoxon [24]. Briefly, five groups of eight fish each were exposed to various concentrations of malathion (0.55, 1.1, 2.2, 4.4 and 8.8 ppm), plus the control group.

2.4. Sub Lethal Studies

Two hundred forty (240) fish were divided into six groups (40 fish each), distributed in glass aquaria at a rate of 10 fish /aquarium. Each group was exposed for 15 days to one of the following treatments:

Group I: Control group, fish were reared in dechlorinated tap water and fed on a commercial diet (25% protein).

Group II: Control group, fish were reared in dechlorinated tap water and fed on a commercial diet (25% protein) supplemented with 5.54 mg Se/kg diet according to Tawwab and Wafee [25].

Group III: Fish were exposed to1/2 LC_{50} (1.35ppm) of malathion and fed on a commercial diet (25% protein).

Group IV: Fish were exposed to1/4 LC_{50} (0.68ppm) of malathion and fed on a commercial diet (25% protein).

Group V: Fish were exposed to1/2 LC_{50} (1.35ppm) of malathion and fed on a commercial diet (25% protein) supplemented with 5.54 mg Se/kg diet.

Group VI: Fish were exposed to1/4 LC_{50} (0.68ppm) of malathion and fed on a commercial diet (25% protein) supplemented with 5.54 mg Se/kg diet.

2.5. Collection of Blood Samples

At the end of the experiment, blood was collected from the caudal vein of the individual fish after anesthetization with benzocaine. Blood samples which were collected from the fish were divided into two parts: part (I): blood was allowed to clot at room temperature and centrifuged at 3000 r.p.m. for 15 minutes and sera was separated for the biochemical parameters: glucose, cortisol and (AchE). Part (II): blood was performed on the same day using sodium citrate as an anticoagulant [26] for the haematological parameters: WBCs and RBCs count, Hb content and Ht values.

2.5.1. Biochemical Analysis

Serum glucose, cortisol and AchE were estimated by the method of Trinder [27], Foster and Dunn [28] and Kendel and Böttger [29], respectively.

2.5.2. Hematological Analysis

WBCs and RBCs were counted by haemocytometer method of Kanaeu [30]. Hb concentrations were estimated by Cyanmethaemoglobin method of Van Kampen and Zijlstra [31] and Ht was determined by the microhematocrit method of Jain [32].

2.6. Sample Preparation

Samples of liver tissue were homogenized in a Potter–Elvejhem glass/Teflon homogenizer. The homogenates were centrifuged at 11,000xg for 30 min. and the supernatant was used for enzyme and lipid peroxides determination. All procedures were done at 4 ◦C.

2.6.1. Lipid Peroxidation (LPO)

Lipid peroxides was detected according to the thiobarbituric acid reactive substances (TBARS) method of Buege and Aust [33]. Briefly, 200 µL of 11,000xg supernatant were mixed with thiobarbituric acid (TBA)solution and incubated in a boiling bath for 30min. After cooling, the reaction mixture was centrifuged and the absorbance of supernatant was measured at 535nm.TBARS concentration was estimated as malondialdehyde (MDA) equivalents, using the extinction coefficient of MDA–thiobarbituric acid complex(156mM−1 cm−1). Results were defined as pmol TBARS per mg protein.

2.6.2. Antioxidant Enzymes

SOD activity was estimated according to the method of Paya et al. [34], with monitor modifications. The nitrotetrazolium blue chloride (NBT, Sigma N-6876) was used as revelation molecule instead of cytochrome c. Estimations were conducted in the presence of 100 nM potassium phosphate buffer (pH 7.8), EDTA 10 mM, NBT 10 mM, hypoxanthine 10 mM (Sigma H- 9377), and xanthine oxidase 0.023 U mol^{-1} (Sigma X- 4500). The decrease in NBT was measured at 560 nm and constant temperature (25◦C). The rate of NBT reduction in the absence of tissue was used as reference rate. One unit of SOD was expressed as the amount of protein needed to decrease the reference rate to 50% of maximum inhibition.

GST activity was determined according to Habig et al. [35], using1-chloro-2,4-dinitrobenzene (CDNB) as substrate. The reaction mixture contained enzyme sample, 100 Mm phosphate buffer (pH6.5) and 10mMCDNB. The absorbance of GS-DNB complex was observed at 340nm (extinction coefficient GST CDNB:9.6mM-1 cm-1). One unit of GST was expressed as the amount of enzyme required to catalyze the formation of 1nmol of GS-DNB per min.

CAT activity was detected by the decomposition of hydrogen peroxide spectrophotometrically at 240nm, using an extinction coefficient of 40M−1 cm−1. The reaction mixture contained enzyme sample, 50 mM potassium phosphate buffer (pH7.4) and 10mM hydrogen peroxide using the method described by Aebi [36]. Results were expressed as units of CAT per mg protein. One unit of CAT was defined as the enzyme amount decomposing 1 µmol of H_2O_2 per min.

2.7. Statistical Analysis

Data were statistically analyzed using analysis of variance, one way "ANOVA", and Duncan's multiple range test to evaluate comparison between means at P< 0.05 using SPSS (14.0) software [37].

3. Results

3.1. LC_{50}

Nile tilapia were exposed to different concentrations of malathion for 96 h. the results revealed that the 96 h-LC_{50} was 2.71 ppm (Table 1) and therefore 1/2 and 1/4 of the median lethal concentrations (1.35 and 0.68 ppm) were used for acute toxicity valuation.

Table 1. *Half lethal concentration (LC_{50})/96h of malathion in O. niloticus.*

Malathion Conc. (ppm)	No. of alive fish	No. of dead fish	a	b	a x b
0.00	8	0	0.00	0	0
0.55	7	1	0.55	0.5	0.275
1.1	5	3	0.55	2	1.1
2.2	3	5	1.1	4	4.4
4.4	2	6	2.2	5.5	12.1
8.8	0	8	4.4	7	30.8
					∑a x b=48.675

Half lethal concentration of malathion = Highest conc. – ∑a x b /n
LC_{50} = 8.8 - 48.675/8 = 2.71 ppm
Where:
a: Constant factor of difference between groups.
b: Mean value of dead fish between each two successive groups.
n: Number of fish in each group.

3.2. Biochemical Parameters

3.2.1. Glucose

Blood glucose level was significantly increased by malathion exposure (Table 2) in groups (III and IV) compared to the control. Co-treatment with Se resulted in decrease glucose level in groups (V and VI) to reach the normal control groups.

Table 2. *Effect of malathion on biochemical parameters of O. niloticus after 15 days of exposure.*

Parameters / Groups	Glucose (mg/dl)	Cortisol (µg/dl)	AchE (µ/l)
Group I	53.01±1.08 ^{d}	19.09±0.52^{c}	506.84±3.70^{a}
Group II	51.69±1.31^{d}	18.78±0.31^{c}	503.37±2.35^{ab}
Group III	68.27±2.79^{a}	40.72±1.72^{a}	330.48±5.17^{d}
Group IV	60.58±0.75^{b}	30.93±2.15^{b}	396.49±7.66 ^{c}
Group V	57.81±2.54^{c}	24.49±1.71^{bc}	422.64±7.36^{c}
Group VI	53.77±0.99^{d}	21.09±2.46^{c}	460.61±5.05^{b}

Data are represented as means ± SE (n = 10). Values with different superscript letter in the same column for each group are significantly different ($P < 0.05$).

3.2.2. Cortisol

Blood cortisol level was significantly increased by malathion exposure (Table 2) in comparison with the control groups. Administration with Se in groups (V and VI) enhanced cortisol level to reach normal control levels

3.2.3. AchE

AchE activity showed a marked reduction in groups (III and IV) compared with the control ones (Table 2). Co-treatment with Se in groups (V and VI) resulted in AchE activity that was still reduced, but closer to the values of the control group.

3.3. Haematological Parameters

3.3.1. WBCs Count

WBCs showed significant increment in the groups exposed to both concentrations of malathion alone (Table 3).

Administration with Se in groups (V and VI) decreased WBCs to nearly normal values.

Table 3. *Effect of malathion on blood parameters of O. niloticus after 15 days of exposure.*

Parameters / Groups	WBC_S ($10^3/mm^3$)	RBC_S ($10^6/mm^3$)	Hb (g/dl)	Ht (%)
Group I	0.86±0.02 [c]	2.78±0.05[a]	7.42±0.77 [a]	33.67±1.59[a]
Group II	0.89±0.03[c]	2.73±0.12[a]	7.23±0.53[ab]	34.52±1.12[a]
Group III	2.63±0.23[a]	1.31±0.09 [d]	4.19±0.18 [d]	21.84±1.26 [d]
Group IV	1.54±0.23[b]	1.74±0.08 [c]	5.32±0.39 [c]	27.23±1.13[c]
Group V	1.03±0.27[bc]	2.32±0.32[b]	6.53±0.32[b]	30.20±1.60 [b]
Group VI	0.97±0.09 [c]	2.22±0.23[b]	6.72±0.58[b]	31.36±1.45[b]

Data are represented as means ± SE (n = 10). Values with different superscript letter in the same column for each group are significantly different ($P < 0.05$).

3.3.2. RBCs Count

RBCs revealed significant decrease in groups (III and IV) compared to the control (Table 3). Co-treatment with Se provided a marked normalization of RBCs when compared to malathion groups.

3.3.3. Hb

Hb values indicated marked decrease in the groups exposed to malathion alone (Table 3). Administration with Se in groups (V and VI) resulted in marked increase in Hb values when compared to the malathion group.

3.3.4. Ht

Ht levels showed statistically significant reduction in groups (III and IV) exposed to malathion only (Table 3). While, Co-treatment with Se improved Ht to reach normal levels.

3.4. LPO

Significant increment in LPO level was recorded in the liver tissue of fish in response to malathion exposure alone (Table 4). Co-treatment with Se in groups (V and VI) resulted in marked decrease in tissue LPO levels when compared with malathion exposed group.

Table 4. *Effect of malathion on LPO and antioxidant enzymes of O. niloticus after 15 days of exposure.*

Parameters / Groups	(LPO) (nmol / g tissue)	(SOD) (U/g tissue)	(GST) (U/g tissue)	(CAT) (U/g tissue)
Group I	38.18±1.42[c]	60.93±2.37[a]	128.77±2.99 [a]	43.93±4.15[a]
Group II	39.61±0.69[c]	60.33±0.91[a]	127.03±3.63 [a]	43.80±2.15[a]
Group III	77.97±5.21[a]	40.90±1.16[c]	84.22±1.89 [c]	26.52±2.19[c]
Group IV	61.30±2.33[a]	48.21±1.51[b]	103.91±4.43[b]	34.88±1.65[b]
Group V	49.41±4.07[b]	49.26±0.67 [b]	116.51±2.87 [b]	31.83±0.91[b]
Group VI	41.95±1.96[c]	58.32±1.65[a]	124.59±1.56 [a]	39.99±0.40[a]

Data are represented as means ± SE (n = 10). Values with different superscript letter in the same column for each group are significantly different ($P < 0.05$).

3.5. Antioxidant Enzymes

3.5.1. SOD

SOD activity was markedly decreased in the liver tissue of the groups exposed to both concentrations of malathion alone (Table 4). While, groups (V and VI) which co-treated with Se showed higher SOD activities than the malathion groups, but lower activities than the control group.

3.5.2. GST

Hepatic GST tissue showed a statistically significant inhibition in groups (III and IV) exposed to malathion only (Table 4). Co-treatment with Se enhanced GST level to be normalized.

3.5.3. CAT

CAT activity was significantly reduced by malathion exposure. While, administration with Se resulted in CAT activities that were still reduced, but closer to the values of the control group (Table 4).

4. Discussion

The present study showed that the 90h-LC_{50} of malathion for *O. niloticus* was (2.71 ppm). The finding came in accordance with Pathiratne and George [38] who recorded that the 96h LC_{50} for Nile tilapia was (2.2 ppm), But it was lower than the values (5 ppm for *O. niloticus* and 8.6 ppm for minnows) documented by Kandiel et al. [39] and Durkin [40]. In contrast to the aforementioned values, Al-Ghanim [41] reported a lower 96h LC50 value (1.06 mg/l) for *O. niloticus.* Malathion was toxic to *Labeo rohita* (LC_{50} value 4.5 µg/L) [42] and *C. gariepinus* (LC_{50} 8.22 mg/L.) as recorded by Ahmad [43]. The difference in toxicity to the different fish species might be due to differences in the absorption of pesticides, their accumulation, biotransformation and excretion [44]. The magnitude of toxic effects of pesticides also depends on length and weight, corporal surface to body weight ration and breathing rate [45]. Fishes are particularly sensitive to environmental pollution of water. Hence, pollutants such as insecticides may damage certain physiological and biochemical processes when they enter into the organs of fishes [46]. Malathion, like other pesticides, can cause serious impairment to physiological and healthy status.

Hyperglycemia was evident in the blood of exposed fish which may help fish to meet critical needs of energy. Such elevation may also be due to enhanced gluconeogenesis response of stressed fish in their attempt to satisfy their new energy demands [47]. So that, increasing blood glucose levels is widely used as a secondary marker of a stress response [48]. Similar results were recorded in *Clarias gariepinus* [49] and *O. niloticus* [41] after exposure to diazinon and malathion, respectively.

Cortisol is the most important glucocorticoid hormone in teleost fish and has an important mineralocorticoid role in promoting osmoregulatory changes in fish [50]. Cortisol level was increased in malathion - exposed fish in comparison to the control ones. Cortisol also plays an important role in stress, by increasing energy availability through lipolysis, glycogenolysis, and gluconeogenesis [51], and thus it is commonly used as an indicator of stress in fish.

Induction of the stress response and the subsequent increase in cortisol level due to contaminant exposure were previously reported by [49,52] in different fishes exposed to various pesticides, suggesting that this is a generalized response to a variety of pollutants.

The main mechanism of toxic action of organophosphates is the inhibition of cholinesterase enzyme which is vital to the normal functioning of the nervous system [53]. Following OP exposure and uptake, the pesticideis metabolically converted to its oxygen analog which binds to the nucleophilic active site on the cholinesterase enzyme, mostly AChE, and causes irreversible inhibition of the enzyme's action [54]. This in turn results in an accumulation of free, unbound acetylcholine at the nerve ending and thus a continual stimulation of electrical activity and a disruption of normal nervous system functioning [55]. plasma AChE activity was inhibited in *O. niloticus* exposed to malathion alone. The obtained results are in accordance with several reports that revealed decreased plasma AChE activity under various organophosphorous pesticides toxicity conditions in different fishes such as *C. gariepinus* [49] and *Oreochromis mossambicus* [56].

Haematology has been widely used for the detection of physiopathological alterations following exposure to different stress conditions. Therefore, haematological techniques are the most common method to determine the sublethal effects of pollutants [10, 11]. In this study, the WBCs count was significantly increased in the fish exposed to each concentration of malathion. Similar results were recorded by Harabawy and Ibrahim [57] after exposed *C. gariepinus* to carbofuran pesticide. Increasing the WBCs may refer to leukocytosis [58] and may be a response of fish to pesticide exposure. Also, The results obtained showed a significant decrease in RBCs, Ht and Hb values in the malathion-exposed fish. Significant lower values of RBCs, Ht and Hb were recorded in *O. niloticus* and *Cyprinus carpio* exposed to malathion, respectively [41, 59]. The reduction in the RBCs count, Ht and Hb levels may be an indicator of anemia due to the inhibition of erythropoiesis, haemosynthesis, or osmoregulatory dysfunction or to an increased rate of erythrocyte destruction in the hematopoietic organ [60]. Oxidative stress is another mechanism for toxicity leading to cell death and disturb the physiological processes in fish [61]. Oxidative stress is related to ROS production, and it can occur when the antioxidant and detoxifying systems are deficient and not able to neutralize the active intermediates that are produced by xenobiotics and their metabolites. Lipid peroxidation is considered as a valuable indicator of oxidative damage to the cellular components [62]. The high level of LPO in liver tissue of catfish exposed to malathion can probably be attributed to excessive ROS production, which could be related to antioxidant enzyme leakage. Previous investigations have reported the induction of LPO by pesticides such as atrazine, malathion, diazinon [63,64,65]. In fish, SOD and CAT constitute the first line of defense against oxidative stress. SOD catalyzes the dismutation of superoxide into hydrogen peroxide and oxygen while CAT catalyzes the decomposition of hydrogen peroxide into water and oxygen [61]. In this study, the hepatic tissue SOD and CAT activities decreased after exposure to malathion. These data are corroborated by previous reports of different pesticides induced reduction in SOD and CAT activities in *C. carpio* and *C. gariepinus*, respectively [66, 67]. The decrease in SOD level may be probably due to dismutase $O_2^{-\bullet}$ and decompose H_2O_2. In some cases, $O_2^{-\bullet}$ by itself or after its transformation to H_2O_2 causes a strong oxidation of the cysteine in the enzyme and decreases the SOD activity [68]. Decrease in CAT activity could therefore be caused by excessive production of $O_2^{-\bullet}$ [69]. Also, the reduction in CAT activity changes the redox status of the cells; where, ROS are generated in excess or there is not enough oxygen radical scavenging activity, free radical chain reactions are stimulated and interactions with protein, lipids and nucleic acids cause cellular damage and even systemic disease in stressed fish [70].

GSTs are a group of cytosolic detoxifying enzymes found in different tissues, defend cells against the mutagenic, carcinogenic and toxic impacts of various pollutants; helping in prevention of lipid peroxidation and suppressing apoptosis [71, 61]. In this study, GST level was significantly decreased in the malathion-exposed fish. Decreased GST levels were also reported in tissues of *Gambusia yucatana* [72] and *C. gariepinus* [67] exposed to carbofuran pesteside. Where, GST can catalyze the synthetic conjugation reactions of xenobiotics or pesticides to GSH, and facilitates the excretion of chemicals by the addition of more polar groups. Therefore, GST plays an important role in homeostasis as well as in the detoxification and clearance of many xenobiotic compounds, thereby protecting tissues from more serious oxidative damage [73]. Se is an element required for normal development, growth and maintenance of homeostatic functions at trace concentrations [74]. In aquaculture, it is utilized as a supplement to live encourage, enhancing their nutritious profile to satisfy necessities of refined fish [75, 76]. In the current study, Se dietary supplementation enhanced the serum biochemical parameters and haematological profiles. Tawwab and Wafee [25] recorded a significant reduction in glucose level and other tested parameters in *O. niloticus* exposed to cadmium and treated with Se, since it reduced the lipid peroxidation in liver tissues. In addition, there were elevations of hepatic antioxidant enzymes in a concentration-dependent manner. Se is a basic part of a few catalysts including glutathione peroxidase and thioredoxine which have physiological cell reinforcement properties, and assumes a powerful part in the security of tissues against oxidative harm and can estrange the lethal impacts of some dangerous chemicals [77, 78]. Many previous literatures showed the hepato protective effects of Se against insecticides and heavy metals induced hepatic injury [74, 79, 65]. Se has powerful antioxidant activity and participates in the antioxidant defense system. It is involved in thyroid hormone metabolism, spermatogenesis [80], moderation of the immune system and prevention of cancer, acting directly as a support for the organismal health [81, 82, 83]. Thus, it is

recommended to use Se as a protective dietary supplement against malathion-induced toxicity to improve the fish health.

5. Conclusion

The results of the present study indicate that malathion at sub lethal exposure induces significant changes in the biochemical and haematological parameters and causes oxidative damage of tilapia fish. The alterations of these parameters may provide early warning signals for the determination of sub lethal toxic level of pesticides. The addition of Se in diet prohibited the toxic effects of malathion by ameliorating oxidative damage and enhancing the physiological alterations which may affect the health of fish.

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