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Bioaccumulation of profenofos and its impact on hematological parameters of Nile tilapia, *Oreochromis niloticus* (Linnaeus, 1758)

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Abstract: The effect of acute and chronic exposure of pesticide profenofos on its accumulation and some hematological parameters of the Nile tilapia (*Oreochromis niloticus*) were studied. The 96 hrs LC_{50} of profenofos to Nile tilapia was determined to be 0.87 mg/l. No detectable profenofos in aquaria water was observed after 14, 21 and 28 days of trial period and during recovery. Profenofos were accumulated in higher rate fish tissues during the acute exposure. However, during chronic period a gradual increase was observed through 1 to 7 days followed by gradual decrease until it was not detectable at the end of 28 days. Regarding hematology, highly significant increase in WBCs counts during both the acute and chronic exposure to was observed. Highly significant decrease in RBCs counts, Hb content and Hct % was noticed during experimental periods. For most of the recorded data, it showed marked improvement during the recovery period. There was a significant increase in MCHC during both acute and chronic periods which were restored to its normal values by the end of chronic period. During the acute intoxication, MCH and MCV values showed highly significant decrease. Thereafter, MCH and MCV restored its normal level, except MCV on day 7th during the chronic exposure. In conclusion, profenofos exert an alteration of fish hematological parameters and profenofos deposited in their flesh.

Key words: Nile tilapia, Acute, chronic exposure, pesticide, profenofos, hematological parameters, bioaccumulation

Introduction

Egypt which is mainly an agricultural country has relied heavily on pesticides to control pests harmful mainly to cotton, maize, rice, wheat, bean and other cultivations (Kotb, 2007). The monitoring of pesticides residue in River Nile has been studied by several investigators; (Osfor *et al.*, 1998; Zidan *et al.*, 2002; Malhat and Nasr, 2011). Profenofos (selecron) as organophosphorus insecticide, acaricide, is highly toxic (Class II) recorded by

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PMWC (1997). Profenofos has low persistence and are readily decomposed. It was used extensively during last years for selective control of mites on cotton, maize and other vegetables in Egypt. Therefore, profenofos like other pesticides may find its way to water system and adversely affecting aquatic life particularly fish (Ayas *et al*,. 1997; Mahboob *et al.*, 2015). Consequently, several major fish kills have occurred in recent years which have been traced to pesticides use. Nile tilapia, *Oreochromis niloticus,* is the most economic fish, highly consumable and is one of the major sources of protein for human beings in Egypt, and it can also be a source of threating to human health through transporting the toxic material directly to consumers (Zaghloul, 2000; Dahshan *et al.,* 2013). The objectives of this study are to show the short and long term effects of pesticide profenofos on some hematological parameters of *O. niloticus* and its bioaccumulation.

Materials and Methods

Pesticide

The used pesticide has a common name Profenofos and commercial name is Selecron which introduced by Syngenta Agro-Pazel Switzerland. It has a formula, $C_{11}H_{15}$ $BrClO_3PS$ and Chemical name, O-(4-bromo-2-chlorophenyl) O-ethyl S-propylphosphorothi-oate. Profenofos has a molecular weight 373.6, density 1.455 (20ºC), solubility 28 mg/l (25ºC), and hydrolysis 14.6 days (pH7).

Fish

Nile tilapia, *Oreochromis niloticus* used in present study have an average total length 16-18.3 cm and average total weight 80-100g. The fish were collected from Islamic Company for Animal Productions, Alkanater, Egypt and transported to the laboratory in plastic ice-box containing dechlorinated-oxygenated water. Fish were acclimatized for three weeks in aerated fiber glass tanks before use. The physico-chemical characteristics of the test water were measured and recorded as pH 7.9, temperature 25ºC and hardness 194.03±1.83 mg/l as $CaCO_3$. Fish were fed on a commercial diet (30% protein). Water of aquaria was changed once daily to remove any feces and uneaten food from the previous day. Fish were transferred randomly into experimental glass aquaria and were acclimatized for one week before starting the exposure to tested pesticide.

Determination of profenofos LC_{50}

Ten fishes were transferred to each individual profenofos concentration (0.7, 0.8, 0.85, 0.87, 0.9, 1, 3, 4 and 5 mg/l) containing aquaria. The experiment was continued for 96 hrs of each concentration. Water was changed daily keeping profenofos concentration constant during 96 hrs. The fish died were counted and removed every day. By the end of the fourth day, the cumulative mortality percentages were

then calculated as 96h LC_{50} by probit analysis method (Finney, 1971). This experiment was repeated twice and the average of mortality was taken. The 96h LC_{50} value for profenofos was recorded as 0.87 mg/l.

Experimental design

Fish were divided into four main groups. All fish groups were fed on commercial diet (30% protein).

Group I (100 fish):

Fish of control group for both treatments were reared in dechlorinated tap water.

Group II (40 fish):

Acute group, fish were exposed to 0.435 mg/l profenofos (1/2 LC_{50}) for 96hrs. Ten fish were sacrificed every 24 hrs.

Group III (80 fish):

Chronic group, fish were exposed to 0.087 mg/l profenofos (1/10 LC_{50}), ten fish were sacrificed after 1, 2, 3, 4, 7, 14, 21 and 28 days.

Group IV (10 fish):

Recovery group, fish were exposed to 0.087 mg profenofos/l for 28 days then transferred to clean, dechlorinated water for another 28 days then sacrificed.

Determination of profenofos residues

In the acute experiment muscle, gill and liver tissue samples were taken from control and profenofos-exposed fish. Tissue and water samples were taken after 1 hr, 24 hrs, 48 hrs, 72 hrs and 96 hrs of exposure. Also samples of fish tissues and water were taken after 1 hr, 1 day, 2, 3, 4, 7, 14, 21 and 28 days and also after 28 days as recovery of profenofos chronic exposure. Profenofos in water and fish tissue samples were extracted and analyzed according to EL-Sheamy *et al.* (1991) by using pre-coated silica gel GF254 plates (20 x20 cm) with layer thickness of 0.25 mm of silica gel. Five ml of methanol was taken and added to 20 µl of each sample to be injected for determining the residues by using gas liquid chromatography [GLC-Hewlett Packard, BC model 6890], adjusted at flame photometric detector (FPD), column (1.5x4mm) Pyrex glass column, oven temperature 250ºC, carrier gas N_2, flow hydrogen 75 ml/min, air flow rate 100ml/min, retention time 2.6 min.

Sampling

Blood was obtained directly from puncture of the heart of each fish. The blood samples were collected in heparinized tube and some in non-heparinized tube to obtain sera. Each fish was dissected to collect pieces of muscle, liver and gill tissues.

Blood parameters and indices

Total number of leucocytes (WBCs) and erythrocytes (RBCs) were counted using improved Hemocytometer using Shaw's solutions (Shaw, 1930; Hesser, 1960). Hemoglobin content (Hb) was estimated using the cyanomethemoglobin method described by

(Van and Zijlstra, 1961). The hematocrit percent (Hct%) value was determined using small hematocrit pipette, where it was centrifuged at 3000 r.p.m for 15 minutes until the blood corpuscles were separated from plasma. Mean corpuscular hemoglobin concentration (MCHC) was calculated as MCHC = Hb (g/100 ml blood) / Hct% X 100. Mean corpuscular hemoglobin (MCH) as MCH = Hb (g/100 ml blood) / RBCs (10^6/mm^3) X 10. Mean corpuscular volume (MCV) as MCV = Hct%/RBCs (10^6/mm^3) X 10. All mentioned blood indices were calculated according to Gupta (1977).

Statistical analysis

The statistical analysis was estimated according to the method of Ridgman (1990). The test of least significant difference (LSD) was used to separate the means when a significant value for F was obtained in the ANOVA test.

Results

Profenofos residues

***Acute exposure*:** The amounts of profenofos residues in experimental ponds water showed a significant ($P<0.05$) gradual degradation. The residues recorded decomposition percent of 9.2% to 24.4% after one hour to 96 hrs, respectively. Concerning profenofos residues in fish muscles, data showed accumulated residues during the acute period. It revealed a sharp increase ($P<0.05$) in profenofos muscle concentrations. Residue levels revealed the same pattern in gills and liver where it reached its peak by the end of experimental period (Table 1).

Tab. 1: Residues of profenofos in aquaria and tissues of Nile tilapia after acute exposure to 0.435 mg profenofos/l.

Time	Aquaria (mg/l)	Muscles (mg/g)	Gills (mg/g)	Liver (mg/g)
Control	N.D	N.D	N.D	N.D
1 hr	$0.40^{a} \pm 0.001$	$12.3^{e} \pm 0.2$	$12.0^{d} \pm 0.2$	$0.8^{d} \pm 0.001$
24 hrs.	$0.40^{ab} \pm 0.004$	$18.4^{d} \pm 0.7$	$12.7^{cd} \pm 0.01$	$1.7^{c} \pm 0.003$
48 hrs.	$0.36^{ab} \pm 0.002$	$27.9^{c} \pm 0.04$	$13.4^{c} \pm 0.01$	$1.9^{bc} \pm 0.004$
72 hrs.	$0.33^{b} \pm 0.001$	$34.4^{b} \pm 0.08$	$33.9^{b} \pm 0.01$	$2.1^{b} \pm 0.01$
96 hrs.	$0.30^{b} \pm 0.001$	$58.4^{a} \pm 0.3$	$41.4^{a} \pm 0.3$	$6.5^{a} \pm 0.1$

- All data are mean of 10 individuals. Data are expressed as mean ± SE.
- Variation between similar single letters in each component is not significant. N.D; no detection for Profenofos.

Chronic exposure: Profenofos exposure of 0.087 mg/l was detected as residue in water. Rapid and higher degradation of analyzed organophosphorus insecticide in contaminated water was observed. The residues reached 0.074 mg/l after one hour of water contamination, while data showed no detectable residues after 21 and 28 days of trail periods or during the recovery period. Data of profenofos residues in fish muscle, gill and liver tissues showed no detectable residues in control group. In profenofos-exposed groups, residues were increased gradually through 1 to 7 days followed by gradual depression at the remained periods (14, 21 and 28 days). No detectable profenofos residues in muscles at 28 days and after recovery periods in all tested tissues (Table 2).

Tab. 2: Residues of profenofos in aquaria and tissues of Nile tilapia after chronic exposure to 0.087 mg profenofos/l.

Time	Aquaria (mg/l)	Muscles (mg/g)	Gills (mg/g)	Liver (mg/g)
Control	N.D	N.D	N.D	N.D
1 hr	$0.074^{a} \pm 0.003$	N.D	$6.706^{f} \pm 0.04$	N.D
1 day	$0.062^{b} \pm 0.001$	$1.67^{f} \pm 0.03$	$7.5^{ef} \pm 0.14$	$6.7^{g} \pm 0.04$
2 days	$0.052^{c} \pm 0.002$	$2.55^{db} \pm 0.05$	$8.8^{d} \pm 0.05$	$7.7^{f} \pm 0.04$
3 days	$0.031^{d} \pm 0.001$	$3.73^{c} \pm 0.01$	$11.84^{c} \pm 0.2$	$8.7^{e} \pm 0.025$
4 days	$0.012^{e} \pm 0.001$	$4.73^{b} \pm 0.06$	$17.9^{b} \pm 0.3$	$12.6^{c} \pm 0.06$
7 days	$0.01^{f} \pm 0.003$	$6.59^{a} \pm 0.06$	$26.8^{a} \pm 0.06$	$29.9^{a} \pm 0.12$
14 days	$0.007^{g} \pm 0.001$	$2.13^{e} \pm 0.01$	$12.5^{c} \pm 0.2$	$14.7^{b} \pm 0.07$
21 days	N.D	$1.11^{g} \pm 0.04$	$7.9^{ab} \pm 0.2$	$9.4^{d} \pm 0.08$
28 days	N.D	N.D	$3.7^{de} \pm 0.02$	$5.0^{h} \pm 0.03$
Recovery for 28 days	**N.D**	**N.D**	**N.D**	**N.D**

- All data are mean of 10 individuals. Data are expressed as mean ± SE.
- Variation between similar single letters in each component is not significant. N.D; no detection forProfenofos.

Generally, the maximum residue content of profenofos in investigated vital organ tissues of tilapia were 29.9 in liver, 26.8 in gills and 6.59 mg profenofos/g in muscles. Thereafter by the end of chronic exposure data cleared 5.0 mg/g in liver, 3.7 mg/g in gills and no detectable profenofos in the muscle tissue (Table 2).

Hematological parameters

Acute exposure: Nile tilapia, *Oreochromis*

niloticus exposed to 0.435 mg/l (half the 96h LC_{50}) showed highly significant increase of WBC_s count ($P<0.05$) during the acute exposure. The maximal mean value of white blood cell count was recorded in fish after 72 hrs of exposure which represented 48.4% increase of control value. While RBC_s count, hemoglobin content and hematocrit percent were decreased after 96 hrs by about the half value of that recorded in case of control fish (Table 3). The depression of blood indices as MCHC, MCH and MCV was significant ($P<0.05$) by the end of acute exposure (Table 4).

Tab. 3: Blood parameters of Nile tilapia after acute exposure to 0.435 mg profenofos/l.

Time	WBCs ($\times 10^3/mm^3$)	RBCs ($\times 10^6/mm^3$)	Hb (g/dl)	Hct %
Control	$682.1^{d} \pm 5.1$	$1.97^{a} \pm 0.3$	$14.3^{a} \pm 0.1$	$31.4^{a} \pm 0.4$
24 hrs.	$810.6^{c} \pm 3.4$	$1.05^{b} \pm 0.2$	$7.7^{b} \pm 0.9$	$12.6^{b} \pm 0.2$
48 hrs.	$998.9^{a} \pm 3.1$	$0.93^{c} \pm 0.5$	$6.5^{c} \pm 0.08$	$11.8^{b} \pm 0.2$
72 hrs.	$1012.3^{a} \pm 2.2$	$0.93^{c} \pm 0.1$	$5.5^{d} \pm 0.07$	$11.6^{bc} \pm 0.2$
96 hrs.	$937.4^{b} \pm 3.7$	$0.81^{d} \pm 0.7$	$5.4^{d} \pm 0.08$	$10.3^{c} \pm 0.1$

- All data are mean of 10 individuals. Data are expressed as mean ± SE.
- Variation between similar single letters in each component is not significant.

Tab. 4: Blood indices in Nile tilapia after acute exposure to 0.435 mg profenofos/l.

Time	MCHC (g/100 ml)	MCH (pg/cell)	MCV (μ^3m/cell)
Control	$45.64^{d} \pm 0.3$	$72.83^{a} \pm 0.2$	$159.65^{a} \pm 0.1$
24 hrs.	$60.902^{a} \pm 0.3$	$73.549^{a} \pm 0.3$	$120.99^{b} \pm 0.3$
48 hrs.	$54.942^{b} \pm 0.3$	$69.351^{ab} \pm 0.2$	$128.09^{b} \pm 0.2$
72 hrs.	$47.941^{d} \pm 0.3$	$58.979^{c} \pm 0.2$	$124.04^{b} \pm 0.1$
96 hrs.	$52.194^{c} \pm 0.3$	$66.358^{b} \pm 0.3$	$127.08^{b} \pm 0.3$

- All data are mean of 10 individuals. Data are expressed as mean ± SE.
- Variation between similar single letters in each component is not significant.

***Chronic exposure*:** Fish exposed to 0.087 mg profenofos/l (1/10 LC_{50}) showed a highly significant increase of total WBCs count ($P<0.05$) during the exposure (Table. 4). It reaches its beak after 14 days of exposure. After the recovery period WBCs count still recorded higher values (11.6%) than that of the control group but lower than that of fish

exposed to profenofos. On the other hand, exposure of tilapia to profenofos revealed an irregular decrease (P<0.05) of RBCs count till 28 days of chronic exposure. Concerning the recovery period, RBCs showed enhancement in its mean counts, but still lower than corresponding value of the control group. Regarding the hemoglobin content (Hb), profenofos-exposed tilapia, revealed highly significant decrease (P<0.05) during the trial period. The RBCs number almost restored to the control value after 28 days of recovery. The same trend was recorded in Hct% values, in case of profenofos chronic exposure (Table 5).

Tab. 5: Blood parameters of Nile tilapia after chronic exposure to 0.087 mg profenofos/l.

Time		WBCs (X 10^3/mm^3)	RBCs (X 10^6/mm^3)	Hb (g/dl)	Hct %
1 day	N.E	$682.1^{f} \pm 5.1$	$1.97^{a} \pm 0.3$	$14.3^{b} \pm 0.1$	$31.4^{b} \pm 0.4$
	E	$762.8^{de} \pm 3.1$	$1.434^{d} \pm 0.2$	$12.3^{d} \pm 0.2$	$27.1^{d} \pm 0.2$
2 days	N.E	$682.1^{f} \pm 5.1$	$1.972^{a} \pm 0.3$	$14.3^{b} \pm 0.1$	$31.4^{b} \pm 0.4$
	E	$806.1^{cd} \pm 4.6$	$1.23^{e} \pm 0.08$	$11.9^{de} \pm 0.1$	$21.4^{e} \pm 0.2$
3 days	N.E	$682.1^{f} \pm 5.1$	$1.97^{a} \pm 0.3$	$14.3^{b} \pm 0.1$	$31.4^{b} \pm 0.4$
	E	$780.3^{cde} \pm 4.4$	$1.07^{f} \pm 0.8$	$11.7^{e} \pm 0.07$	$20.2^{f} \pm 0.2$
4 days	N.E	$682.1^{f} \pm 5.1$	$1.97^{a} \pm 0.3$	$14.3^{b} \pm 0.1$	$31.4^{b} \pm 0.4$
	E	$818.7^{c} \pm 2.0$	$0.98^{fg} \pm 0.08$	$11.6^{e} \pm 0.1$	18.3 ± 0.2^{g}
7 days	N.E	$671.8^{f} \pm 3.2$	$1.91^{ab} \pm 0.2$	$13.3^{c} \pm 1.6$	$32.8^{a} \pm 3.2$
	E	$987.4^{a} \pm 13.6$	$1.00^{f} \pm 0.9$	$10.9^{f} \pm 1.1$	$16.8^{h} \pm 1.4$
14 days	N.E	$683.1^{f} \pm 3.3$	$1.92^{ab} \pm 0.3$	$14.1^{b} \pm 1.2$	$31.4^{b} \pm 0.2$
	E	$997.2^{a} \pm 2.2$	$0.87^{h} \pm 0.1$	$10.5^{f} \pm 0.8$	$13.9^{i} \pm 1.1$
21 days	N.E	$681.1^{e} \pm 2.8$	$1.97^{a} \pm 0.3$	$13.7^{f} \pm 1.5$	$30.2^{c} \pm 2.1$
	E	$870.9^{b} \pm 3.9$	$0.89^{gh} \pm 0.7$	$9.6^{g} \pm 1.1$	$12.0^{j} \pm 1.1$
28 days	N.E	$668.3^{f} \pm 1.7$	$1.87^{b} \pm 0.2$	$14.9^{a} \pm 2.1$	$30.9^{bc} \pm 2.5$
	E	$819.9^{c} \pm 2.2$	$0.89^{gh} \pm 0.7$	$8.9^{h} \pm 1.2$	$11.8^{j} \pm 1.2$
Recovery for 28 days	N.E	$683.1^{f} \pm 3.3$	$1.87^{b} \pm 0.2$	$13.7^{f} \pm 1.5$	$31.4^{b} \pm 0.2$
	E	$797.7^{ed} \pm 2.3$	$1.62^{c} \pm 0.1$	$13.1^{c} \pm 1.4$	$30.5^{bc} \pm 1.8$

- All data are mean of 10 individuals. Data are expressed as mean ± SE.
- Variation between similar single letters in each component is not significant. N.E; non-exposed to profenofos, E; exposed to profenofos

MCHC showed a significant gradual increase (P<0.05) started after the second day of chronic exposure to 0.087 mg profenofos and continued for 21 days. MCHC decreased value was observed by the end of 28 days of pesticide exposure. The recovery period almost recorded

the same value as in exposed fish after 4 weeks but representing higher value than its control fish. Regarding MCH in case of profenofos-exposed fish revealed a highly significant increase during the whole experimental period ($P<0.05$) compared to the control values. The higher values of MCH were noticed even after the recovery period. With respect to MCV values, data showed significant increase during the first four days of exposure ($P<0.05$), then gradually decreased significantly after 21 days and 28 days compared to the control values (Table 6).

Tab. 6: Blood parameters of Nile tilapia after chronic exposure to 0.087 mg profenofos/l.

Time		MCHC (g/100 ml)	MCH (pg/cell)	MCV (μ^3m/cell)
1 day	N.E	$45.75^{g} \pm 0.3$	$72.88^{fg} \pm 0.3$	$159.63^{dc} \pm 0.4$
	E	$45.08^{hi} \pm 0.4$	$85.62^{d} \pm 0.4$	$190.53^{a} \pm 0.1$
2 days	N.E	$45.75^{g} \pm 0.3$	$72.88^{fg} \pm 0.3$	$159.63^{dc} \pm 0.4$
	E	$55.38^{f} \pm 0.5$	$97.29^{c} \pm 0.5$	$175.34^{b} \pm 0.15$
3 days	N.E	$45.75^{g} \pm 0.3$	$72.88^{fg} \pm 0.3$	$159.63^{dc} \pm 0.4$
	E	$58.09^{e} \pm 0.9$	$109.63^{b} \pm 0.5$	$186.81^{a} \pm 1.2$
4 days	N.E	$45.75^{g} \pm 0.3$	$72.88^{fg} \pm 0.3$	$159.63^{dc} \pm 0.4$
	E	$63.66^{d} \pm 1.04$	$117.65^{a} \pm 0.5$	$186.25^{a} \pm 1.1$
7 days	N.E	$40.26^{j} \pm 3.2$	$69.00^{g} \pm 3.3$	$170.82^{bc} \pm 0.2$
	E	$66.13^{c} \pm 9.6$	$109.88^{b} \pm 8.9$	$166.52^{bcd} \pm 0.8$
14 days	N.E	$44.97^{hi} \pm 3.02$	$74.11^{f} \pm 2.3$	$164.86^{cd} \pm 0.3$
	E	$75.17^{b} \pm 9.9$	$119.76^{a} \pm 7.6$	$161.99^{cde} \pm 0.2$
21 days	N.E	$34.71^{k} \pm 3.4$	$53.57^{h} \pm 2.6$	$154.49^{e} \pm 1.7$
	E	$80.09^{a} \pm 6.03$	$107.11^{b} \pm 12.2$	$134.15^{f} \pm 0.8$
28 days	N.E	$48.15^{g} \pm 1.6$	$79.77^{e} \pm 2.6$	$165.76^{cd} \pm 0.3$
	E	$44.66^{b} \pm 8.6$	$98.29^{c} \pm 11.3$	$131.89^{f} \pm 0.9$
Recovery for 28 days	N.E	$33.36^{k} \pm 3.4$	$55.99^{h} \pm 3.3$	$167.61^{bcd} \pm 0.3$
	E	$42.91^{i} \pm 7.8$	$81.62^{e} \pm 5.7$	$190.53^{a} \pm 1.5$

- All data are mean of 10 individuals. Data are expressed as mean ± SE.
- Variation between similar single letters in each component is not significant. N.E; non-exposed to profenofos, E; exposed to profenofos

Discussion

The present study shows a gradual decrease of the residues of profenofos in polluted water either in acute or chronic experiment for 96 hrs or 28 days. After 21 days no residues were detected in water. This may suggest that organophosphorus compounds have low persistence and are readily decomposed as reported by Dogheim *et al.* (1996) and Mahboob *et al.* (2015). The rate of degradation is based on the binding between each chemical and surrounding matrix which characterized by the physical-chemical properties of those phases (Kennedy *et al.*, 2001). Half-life of profenofos was reported as 7 days in soil (Crossan and Kennedy, 2008). It may appear that more drought means lengthened half-life. Indeed low concentration of profenofos in water after 14 days and entire decaying after 21 days would affect its accumulation later in investigated organs. As a result of profenofos persistence, liver accumulated the greatest amount of insecticide followed by gills and finally muscles. As expected, the chronic exposure ~~to~~ of profenofos showed higher residues than acute exposure. Partial decomposition of profenofos in liver and gill tissues started after 14 days and then full decaying after 28 days of recovery. No residues were detected in muscle at the end of chronic exposure. The order of profenofos accumulation in tissues may due to that liver is considered as a blood filter of toxicants, makes it the highest reservoir. Profenofos was biotransformed in liver into two metabolites, hydroxyprofenofos and desthiopropylprofenofos (Abass *et al.,* 2007). Gills are the main exit passage of water and in direct contact with pesticide-polluted water. The high residues of profenofos in fish attributed to the rapid penetration and binding of insecticide residues in fish tissues (EL-Sheamy *et al.*, 1991; Venkateswara Rao *et al.*, 2003).

Currently tilapia showed highly significant increase of leucocytes' count as a result of acute and chronic profenofos exposure. Similar observations were mentioned after exposure of Nile tilapia to organophosphate pesticides (Ibrahim *et al.*, 2005; El-Sayed *et al.*, 2007; El-Sayed and Saad, 2008). They referred such increase in leucocyte counts to the alteration in defense mechanism. Oppositely, leucopenia was observed in common carp, *Cyprinus carpio* exposed to sublethal concentration of profenofos which was explained as the malfunction of the hematopoietic system caused by toxicant stress (Marie *et al.*, 1998). It seems that tilapia may be more resistant than carp. Significant decrease in erythrocyte counts, hemoglobin contents and hematocrit percent values were recorded in the present study during both acute and chronic periods of profenofos exposure. Such effect was also recorded in *O. niloticus* subjected to other organophosphate pesticides (Abo-Hegab *et al.*, 1993; Sweilum, 2006; El-Sayed and Saad,

2008). Also after chronic profenofos toxicity in eastern rainbow fish *Melanotaenia duboulayi* (Kumar and Chapman, 1998). They revealed that the reduction of RBCs count, Hb content and Hct value may be due to a harmful effect of pesticide on spleen, liver and anterior kidney. On the other hand, that reduction in hematology was attributed to the defect in the hematopoietic organs or destruction of erythrocytes by the pesticide which led to anemic condition (Fouda 2004). Further, the reduction in hemoglobin content can be attributed to hemodilution of blood due to bleeding in the gills and elimination of RBCs as a result of extravasation of blood (Novotony and Beeman, 1990; Heath *et al.*, 1997). The increase of mean corpuscular hemoglobin concentration (MCHC) observed herein is an indication of hemolysis. The decreasing in both mean corpuscular hemoglobin (MCH) and mean corpuscular volume (MCV) were recorded after acute and chronic exposure to profenofos in *O.niloticus*. Such alterations may be a result of the changes in RBCs count and Hb content. The disturbance of these calculated blood indices were showed also by (Marie *et al.*, 1998; Fouda, 2004) after organophosphate exposure in other fish species. They pointed out that the decreased MCV and MCH along with increased MCHC are indicative of hypochromic microcytic anemia.

In conclusion, long time of the profenofos decaying reached 14 days in water and 28 days in fish tissues. This late decomposition may cause changes in fish which is clear in hematological disturbances. Therefore, for human safety it is important to prohibit using profenofos.

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