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EVALUATION OF ABAMECTIN TOXICITY ON SOME BIOCHEMICAL CONSTITUENTS AND OSMOREGULATION IN FRESHWATER FISH *Oreochromis niloticus* (Tilapia niloticus)

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ABSTRACT

Indiscriment use of pesticides have been elevated the risk of contamination in environment and aquatic organisms. Considering to the previous fact, the present study was done to investigate the alterations of some blood constituents and ionic regulation; as well as the histopathological alterations in tissues of gills and kidneys of freshwater fish, *Oreochromis niloticus*, following prolonged exposure to sub-lethal concentrations of abamectin insecticide at 14 days. The obtained results showed that abamectin treatment caused hypotriglyceridemia and hyperglycemia in fish exposed to the high concentration (103.68µgL^{-1}). In addition, hypercholesterolemia was detected in abamectin-exposed fish (by 50.48 and 103. 68µgL^{-}) after 14 days of exposure. Also, a marked increase in the creatinine level was observed in abamectin-exposed fish by 103.68µgL^{-1} on day 14, with a significant decline in the level of sodium ions (Na^{+}) and a significant elevation in the level of chloride ions (Cl^{-}) in fish exposed to low concentration (50.48µgL^{-1}) of abamectin. In contrast, a significant decrease in the level of chloride ions (Cl^{-}) was detected in the high concentration of abamectin. Marked decrease in the level of plasma bound calcium (B.Ca) with hypoproteinemia and hypophosphatemia were recorded in fish exposed to low concentration (50.48µgL^{-1}) of abamectin. On the contrary, a marked enhancement in the triiodothyronine level (T3) was noticed in fish following exposure to 50.48µgL^{-1} of abamectin. The histopathological studies on the gills and kidneys revealed necrosis of lamella and infiltration of acidophils leukocyte in gills with degenerative changes in kidney tubules were observed at both concentrations of abamectin.

Key Words: Abamectin, *Oreochromis niloticus,* Blood constituents, Ionic regulation, Histopathological alterations

INTRODUCTION

Abamectin (ABM) also, known as avermectin B_{1a} used in both as a pesticide and as an anti-helminthic drug in animals. This is the product of *Streptomyces avermitilis* obtained through naturally occurring fermentation. ABM is a mixture of homologues B_{1a} and B_{1b} and is defined as containing a minimum of 80% B_{1a} and a maximum of 20% B_{1b} (Fisher and Mrozik, 1989). The avermectins are highly lipophilic and poorly soluble in water but readily soluble in most organic solvents (Roth *et al*., 1993). In fish, the avermectins can also pass the blood / brain barrier and could cause toxic effects (Hφy, *et al*., 1990). However, avermectins are rapidly photodegraded in water to less bioactive compounds by oxidative and photo-oxidative mechanisms [$t_{1/2}$= 4 – 21 h] (Halley *et al*., 1993). Nevertheless, they could still pose a risk to the aquatic environment, especially when used frequently in large numbers of animals. Jenčic *et al.* (2006) studied the toxic effect of abamectin (ABM) on rainbow trout (*Oncorhynchus mykiss*). They found that the histological changes in organs showed a direct toxicity of ABM for rainbow trout, since degene

rative changes in brain and kidney and to a minor extent in liver were established. The biochemical processes represent the most sensitive and relatively early events of pollutant damage. Thus, it is important that pollutant effects be determined and interpreted in biochemical terms, to delineate mechanisms of pollutants action and possibly ways to mitigate adverse effects. In view of this paucity reported on the various adverse effects of abamectin on biological systems of freshwater fish, *Oreochromis*

niloticus, **therefore, the aim of this study was to determine the acute toxicity of abamectin under static conditions and evaluate the toxic effects of abamectin on lipid profile and plasma glucose level, as well as on osmoregulation and calcium metabolism, in addition to thyroid hormones status. Furthermore, the histopathological alterations in gills and kidneys of abamectin exposed fish will be detected.**

MATERIAL AND METHODS

Pesticide used:

Abamectin (Vertimec 1.8% EC) was supplied by Syngenta Co. The chemical name, 5-O-demethyl avermectin A_1a (i) mixture with 5-O- demethyl- 25-de (1-methyl propyl-25- (1-methyl ethyl) avermectin A_1a (ii).

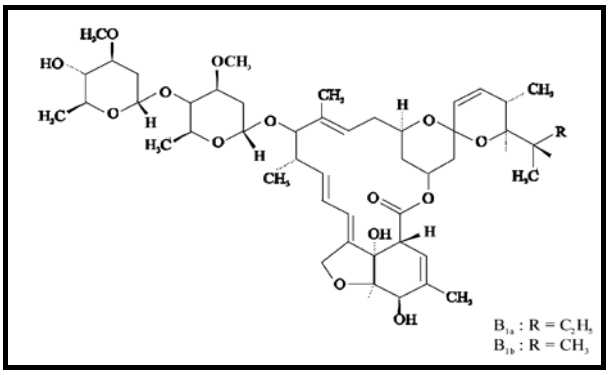

Fig. (1): Chemical structure of abamectin (ABM).

Fish and maintenance regimen:

Nile tilapia (*Oreochromis niloticus*) were purchased from commercial fish supplier (Foky Farm, El-Kaluobia, Egypt) with average weight of 85.8 ±2.83 g and average length of 15.65 ± 0.116 cm were used. The fish were held in glass aquaria (50 × 50 × 50 cm) supplied with aerated and de-chlorinated tap water. The water had the following characteristic: Temperature: 25.00 ± 0.26°C, pH: 7.28 ± 0.12, Total hardness: 229.5 ± 3.39 mg/L as $CaCO_3$, Alkalinity 3.01 ± 0.40 meq/L. A 12h light / 12h dark photoperiod was maintained. Fish were fed commercial fish feed daily at a rate of 1% of the average body weight as recommended by Sprague (1969). Also, fish were acclimated for 2 weeks under the laboratory conditions before starting of the experiments.

Experimental treatments and sampling:

Fish, acute toxicity test:

Four-days static acute toxicity test (OECD, 1984, No. 203) was performed to determine the LC50 of abamectin to *Oreochromis niloticus* in laboratory conditions.

Fish, prolonged toxicity test, -14 days study:

After determining the 96-h LC_{50} value of abamectin, which was 207.360 μgL^{-1}, the experiment was done according to the procedure described by OECD (1992) No. 204. The fish *Oreochromis niloticus* were exposed to 50.48 μgL^{-1} which represent the safe threshold concentration that computed by using the equation of Doudorff *et al.* (1951), and to 103.68 μgL^{-1} which represent the half of 96-h LC_{50} of abamectin, in addition to fish group served as control. After 7 and 14 days of exposure, the fish were collected individually and blood samples were collected from cadual vein with hepranized syringes and then the blood centrifuged and the plasma was separated, aliquoted and frozen at -20°C for biochemical measurements. Fish were killed at the end of the experiment. Gills and kidneys were collected in 10% formal saline for 24 hours. Washing, dehydration, cleaning and embedding in paraffin bees wax were done on the fixed tissues specimens.

Paraffin tissue sections were obtained and stained by haematoxylin and eosin (Banchroft *el al.,* 1996), for histopathological examination by light microscope.

Biochemical estimations:

Determination of triglycerides (TG_S), total cholesterol (T. Chol.) and low-density lipoprotein-cholesterol (LDL-Chol.) were performed according to the methods of Buccalo and Davais (1975), Trinder (1969) and Wieland and Seidel (1983), respectively.

Also, plasma glucose level was measured by using the method of Darham and Trinder (1972). Moreover, the sodium (Na^+) Potassium (K^+) and Chloride (Cl^-) ions were measured according to the methods of Maruna (1958), Sunderman and Sunderman (1958) and Skeggs and Hochstrassat (1964), respectively.

Also, inorganic phosphate (Pi) was determined with the method described by by Kilchling and Freiburg (1951). The plasma urea and creatinine concentration were determined by using the methods of Coulomb and Farreau (1963) and Husdan and Rapoport (1968). The total calcium was measured according to the method of Stern and Lewis (1957) and free ionized calcium (Ca^{2+}) level was calculated according to the formula of Puerro and Alexandre (1995). While, the bound calcium was calculated by subtracting the free-ionized calcium (Ca^{2+}) from the total calcium values, and also, the plasma total protein was measured according to the method of Weichselbaum (1946).

Hormonal analysis:

Plasma thyroxine (T4) and triiodothyronine (T3) were assayed with Commercial Radio Immunoassay

kit (RIA) according to the method of Britton *et al.* (1975).

Statistical analysis:

Data are expressed as mean ± Standard error. Significant of the values obtained were tested using the student "t" test (Gad and Weil, 1989).

RESULTS

The results presented in Table (1) demonstrated that triglycerides (TGs) level was decreased markedly in fish exposed to the high concentration (103.68 µgL^{-1}) of abamectin after 7 and 14 days of exposure in comparison with the control group. Meanwhile, a significant elevation in the levels of total cholesterol (T. Chol.) and low-density lipoprotein cholesterol (LDL-Chol.) were recorded in fish following treatment with both concentrations of abamectin for 14 days of treatment.

Also, there was a significant increase in the level of plasma glucose in fish exposed to the high concentration (103.68 µgL^{-1}) of abamectin over the experimental period.

Fish exposed to both concentrations of abamectin for 7 days, had a marked increase in the level of sodium ions (Na^{+}), whereas a significant decrease in the level of this ion was detected in fish exposed to the low concentration (50.48 µgL^{-1}) of abamectin for 14 days. No-significant alterations in the level of potassium (K^{+}) were observed in fish treated with abamectin through abamectin exposure (Table 2).

In contrast, there was a significant increase in the level of chloride ions (Cl^{-}) in fish exposed to 103.68 and 50.48 µgL^{-1} of abamectin on day 7 and 14, respectively.

The urea level was only decreased markedly in fish after treatment with 50.48 µgL^{-1} of abamectin for 7 days of treatment. With respect of creatinine level, there was a significant decrease in the level of creatinine following exposure to both concentrations of abamectin for 7 days. Also, this trend was observed in fish treated with 50.48 µgL^{-1} on day 14 of exposure, whereas, a marked elevation in the creatinine level was recorded in fish exposed for 14 days to 103.68 µgL^{-1} of abamectin tested (Table 2).

Fish exposed to abamectin had insignificant differences in the levels of total calcium (TCa) and free-ionized calcium (Ca^{+2}), whereas a significant reduction in the levels of plasma total protein and bound calcium (B.Ca) were observed over the experimental period (Table 3). The same trend was observed in inorganic phosphate level in abamectin-exposed fish (by 103.68 µgL^{-1}). Daily exposure for 14 days to 50.48 µgL^{-1} of abamectin led to a marked enhancement in the level of plasma triiodothyronine (T3) in abamectin-exposed fish (Table 4).

The results of the histopathological examination showed that fish which were exposed to the low concentration of abamectin exhibited necrosis of gills lamellae (Fig. 2). While, other which had high concentration of abamectin showed granular of acidophils leukocytic cells infiltration in the base of filaments (Fig. 3). There was focal haemorrhages in between the vacuolar degenerated renal tubules (Fig. 5).

Table (1): Effects of different treatments of abamectin on lipid profile and glucose level in *O. niloticus.*

Time	7 days			14 days		
Treatment / Parameter	Control	50.48 µg/l	103.68 µg/l	Control	50.48 µg/l	103.68 µg/l
TG (mg/dl)	63.568 ± 7.62	44.600 ± 3.78	34.542 ± 3.52*	57.481 ± 7.47	59.179 ± 7.87	34.199 ± 2.85*
TC (mg/dl)	89.032 ± 2.47	94.677 ± 13.45	77.580 ± 9.65	84.995 ± 13.23	122.89 ± 8.32*	136.45 ± 4.54*
LDL – C (mg/dl)	87.705 ± 10.43	133.243 ± 23.98	90.658 ± 9.04	93.093 ± 1.50	114.831 ± 7.19*	133.24 ± 7.44***
Glucose (mg/dl)	47.582 ± 4.26	78.727 ± 11.97	76.386 ± 8.78*	52.117 ± 2.45	54.557 ± 6.42	73.272± 2.33**

Mean ± S.E. * P< 0.05, ** P<0.01, *** P<0.001,
TG = Triglyceride, TC = Total Cholesterol,
LDL-C = Low Density Lipoprotein –Cholesterol

Table (2): Effects of different treatments of abamectin on electrolytes level and renal function in *O. niloticus.*

Time	7 days			14 days		
Treatment / Parameter	Control	50.48 µg/l	103.68 µg/l	Control	50.48 µg/l	103.68 µg/l
Na^+ (mmol/L)	176.79 ± 8.073	283.289 ± 29.642*	366.040 ± 9.549***	173.73 ± 3.471	155.96 ± 3.587*	174.000 ± 3.315
K^+ (mmol/L)	4.456 ± 0.291	3.765 ± 0.223	3.848 ± 0.194	4.567± 0.333	4.090 ± 0.736	5.211 ± 0.620
Cl^- (mmol/L)	116.95 ± 1. 27	114.425± 4.101	150.508 ± 13.717*	116.419 ± 1.208	164.932 ± 10.270**	101.990 ± 0.571
Urea (mg/dl)	3.127 ± 0.138	2.999 ± 0.241	2.010 ± 0.349*	2.097± 0.337	2.449 ± 0.369	2.714 ± 0.115
Creatinine (mg/dl)	0.316 ± 0.032	0.163 ± 0.024**	0.162 ± 0.036**	0.316 ± 0.024	0.183 ± 0.024**	1.100± 0.273*

Mean ± S.E., * P< 0.05, ** P<0.01, *** P<0.001, Na^+ = Sodium ion, K^+ = Potassium ion and Cl^- = Chloride ion

Table (3): Effects of different treatments of abamectin on calcium profile in *O. niloticus*

Time	7 days			14 days		
Treatment / Parameter	Control	50.48 µg/l	103.68 µg/l	Control	50.48 µg/l	103.68 µg/l
T.Ca mg/dl	10.148 ± 0.333	10.248 ± 0.844	9.475 ± 1.077	9.462 ± 1.100	8.516 ± 0.323	8.854± 0.719
Ca^{2+} mg/dl	6.363 ± 0.226	7.224 ± 0.475	7.259 ± 0.474	5.896 ± 0.529	6.816 ± 0.150	6.368 ± 0.099
B.Ca mg/dl	3.785 ± 0.117	2.515 ± 0.305**	1.714 ± 0.129***	3.566 ± 0.482	1.699 ± 0.194*	1.958 ± 0.344*
Pi mg/dl	5.956 ± 0.389	6.478 ± 0.917	4.836 ± 0.711	6.340 ± 0.536	5.105 ± 0.769	4.666 ± 0.330*
P.T.P. g/dl	2.996 ± 0.114	2.137 ± 0.208**	0.998 ± 0.085***	2.742 ± 0.272	1.803 ± 0.073*	1.596 ± 0.218*

Mean ± S.E., * P< 0.05, ** P<0.01, *** P<0.001, T.Ca = Total calcium, Ca^{2+} = Free-ionized calcium, B.Ca = bound calcium, P.T.P. = Plasma total protein Pi = Inorganic phosphate

Table (4): Effect of different treatments of abamectin on thyroid hormones (T4 and T3) in *O. niloticus.*

Time	Abamectin treatment for 14 days		
Treatment / Parameter	Control	50.48 µg/l	103.68 µg/l
T4 mmol/L	7.237 ± 0.201	8.490 ± 0.0.554	7.075 ± 0.641
T3 mmol/L	1.857 ± 0.208	3.976 ± 0.346***	2.251 ± 0.084

Mean ± S.E. *** P<0.001,
T4 = Thyroxine, T3 = Triiodothyronine

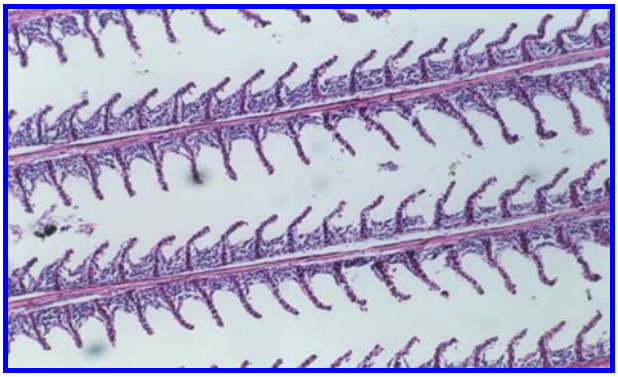

Fig (1): Gills of control fish showing the normal histological structure (HXE X40).

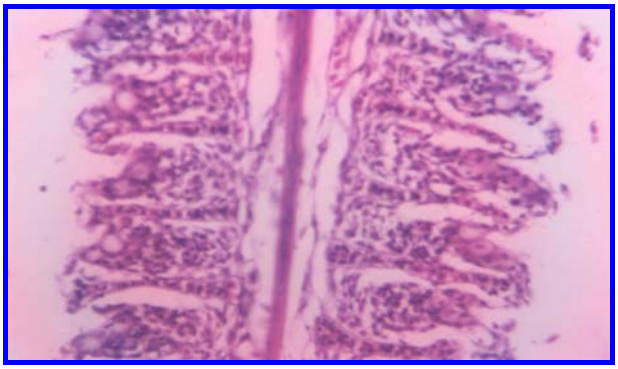

Fig (2): Gills of fish exposed to 50.48 µL-1 of abamectin for 14 days showing necrosis of lamella (HXE X 160).

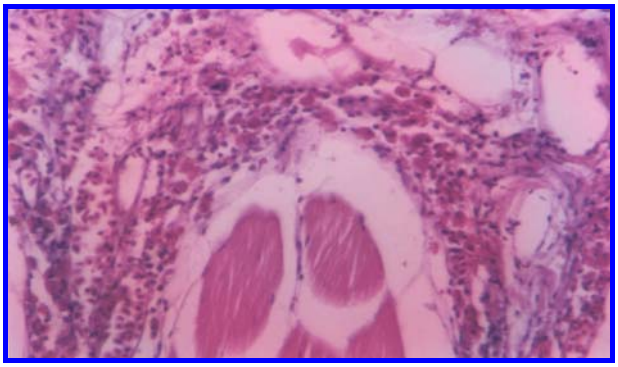

Fig (3): Gills of fish exposed to 103.68 μL^{-1} of abamectin for 14 days showing infiltration of granular acidophilic cells in the base of fillament (HXE X160).

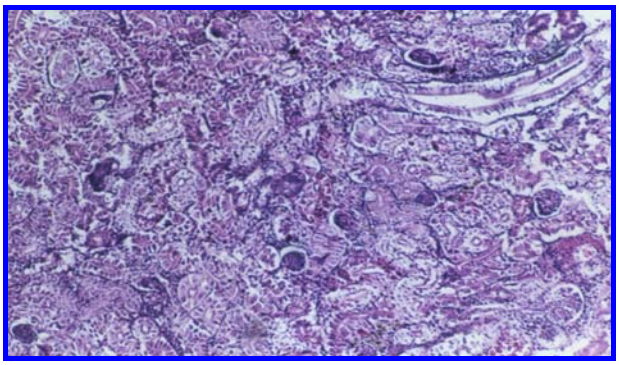

Fig (4): Kidney of control fish showing the normal histological structure (HXE X40).

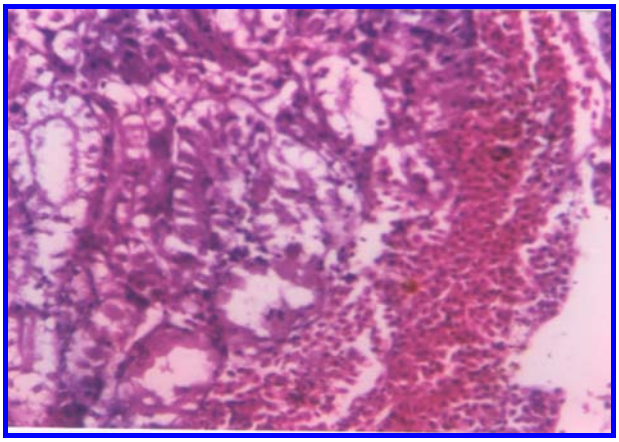

Fig (5): Kidney of fish treated by 50.48 and 103.68 μL^{-1} of abamectin for 14 days showing focal haemorrhages in between the vacuolar degenerated renal tubules (HXE X40).

DISCUSSION

The acute toxicity of abamectin to fish strongly depends on the species tested. The most sensitive fish was rainbow trout with 96h LC_{50} of 3.2 μgL^{-1}. While, the least sensitive fish species was carp with a 96h LC_{50} of 42 μgL^{-1} (Jenčič *et al.*, 2006). The molecular of avermectin B_{1a}, the major component of abamectin, is high and the water solubility is low (7.8 μgL^{-1}) and its K_{0w} is 9900 (Wislocki *et al.*, 1989 and Halley *et al.*, 1993). However, Vanden-Heuvel *et al.* (1996) found that abamectin does not bio-concentrate strongly in bluegill sunfish due to its rapid elimination during depuration period.

Short-term exposure to stressors may result in changes that whilst not being lethal, but impair the ability of the fish to function normally (Jobling, 1995). Hypotriglyceridemia was observed in fish exposed to high concentration of abamectin on day 14; this may be due to release of catecholamines (epinephrine and nor-epinephrine) which increase the formation of cAMP and activate lipolysis. However, low oxygen tension increase sympathetic activity, resulting in hyperglycemia and increase free fatty acids (FFAs) in plasma. Meanwhile, a reduction in the level of TGs may be attributed to exceed the rate of TGs accumulation within the hepatic cells rather than secretion of heptic TGs into plasma (Ruckebush, *et al.*, 1988). In the present study, the low concentration of abamectin was not effective with respect to cholesterol levels, whereas the high concentration increased the levels of T. Chol. and LDL-Chol. on day 14.

Hypercholesterolemia could be explained by an enhanced production by the liver (and other organs) or release of cholesterol damaged cell membranes and/or this may be due to inhibit the conversion of cholesterol into steroid sex hormones (Mukherjee, *et al.*, 1991; Muant, 1981 and Singh and Reddy, 1990).

Plasma glucose level was measured in *Oreochromis niloticus* exposed to abamectin to determine whether abamectin induced changes in plasma level of cortisol, T3 and T4 hormones, which are important for regulation of metabolic process. Hyperglycemia was detected only in fish exposed to the high concentration of abamectin over the course of abamectin exposure. The hypothalmo-pituitary internal axis is activated by numerous chemical and physical stressors which elevate blood levels of cortisol, which is hyperglycemic hormone, and stimulating glycolysis and gluconeogenesis from protein and lipid sources (Pickering, 1993). This endocrine response preceded by a surge of catecholamines stimulates lipolysis, glycogenolysis and gluconeogenesis (Sheridan, 1986 and Vijayan and Moon, 1992).

Decreased triglycerides and total cholesterol following exposure to atrazine in *O. niloticus* was reported by Hussein *et al.* (1996). Further, Khater *et al.* (1990) found that lannate treatment caused a marked decrease in the level of total lipids in *O. niloticus*.

The same observations were reported by Shalaby (1997) and Danasoury *et al.* (1997). They found that the prolonged toxicity tests (14 days) of carbosulfan and malathion on the *O. niloticus* and *C. lazera*, respectively, caused a significant elevation in the level of glucose. Also, Lotfi-Salwa (1994) demonstrated that deltamethrin injection in *Clarias lazera* caused hyperglycemia.

In contrast, Kumar *et al.* (1990) reported that blood glucose level found to be markedly decreased in fish (*Clarias lazera*) exposed to deltamethrin for 30 days.

However, recent studies have shown that exposure to pesticides alters fish physiology. Sweilum (2006) found that serum glucose and lipids level were increased significantly, whereas a marked decrease in the level of serum total protein were detected, in fish exposed to sublethal doses of dimethoate and malathion, with increasing concentration of insecticide. Also, our results consistent with results reported by John (2007), who found that the glucose and cholesterol levels were found to be significantly increased after 30 days exposure of *Mustus vilttatus* to Mitasystex (an organophosphate) and Sevin (a carbamate), while total protein was found to be decreased considerably in both cases.

Our findings revealed that fish exposed to abamectin for 7 days, had a significant increase in the level of (Na^+) ions (hypernatraemia) and chloride (Cl^-) ions (hyperchloraemia) in fish exposed to high and low concentrations on day 7 and 14, respectively, this could be attributed to stimulation of Na^+-K^+ ATPase activity, this in turn, led to increase influx of Na^+ and Cl^- ions and / or may be due to stimulation the reabsorption function in the kidney.

In contrast, hyponatraemia and hypochloraemia were recorded in fish exposed to low and high concentrations of abamectin tested on day 14, respectively. This likely due to poisoning of Na^+-K^+ ATPase or carbonic anhydrase (CA) or both of these enzymes by abamectin treatment. *In vivo* could results in inhibition of sodium (Na^+) and chloride (Cl^-) transport across the gills of *O. niloticus.*

In both freshwater and seawater teleost fishes ionic regulation is controlled by cortisol which is known to stimulate the proliferation of chloride cells, which transport sodium (Na^+) and chloride (Cl^-) ions independently of each other.

Loss of structural integrity of the gill may easily lead to a drop in the concentration of blood electrolytes, such as sodium (Na^+) and chloride (Cl^-) ions. Electrolytes losses have described, for example, after exposure of freshwater fish to pesticides (Mallatt and Stinson, 1990).

In the kidney, the clearance ratio analysis indicated that the reduction in net re-absorption efficiencies was greater for Na^+ than the Cl^- ions therefore; it seems likely that abamectin interfered with tubular Na^+ re-absorption, perhaps in a similar manner to its interference with tubular Cl^- re-absorption, such that the influence of secretary processes become greater and caused of increase urinary Na^+ loss.

Freda *et al.* (1990) reported that a reduction in plasma electrolytes levels has two important causes: First, there is an elevated passive efflux of ions across the gills, due to a more or less non selective increase in the branchial permeability to water and ions, this may lead to haemodilution by enhanced osmotic uptake of water across the gills and to passive diffusional ions losses. Second, the inhibition of active ion uptake by the chloride cells of the gills may further contributed to the negative ion balance of the blood.

Our results coincidence with the results of Hussein *et al.* (1996), they found that sodium (Na^+) and chloride (Cl^-) ions were decreased markedly in *O. niloticus* following exposure to atrazine for 14 and 28 days, whereas no significant differences in urea level was observed. Meanwhile, Mitchell *et al.* (1997) mentioned that sodium (Na^+) ions level did not alter significantly in glyphosphate-exposed fish for 10 days.

A significant reduction in the urea level was observed in abamectin-exposed fish (at high conc.) for 7 days; this may be due to impairment in the urea biosynthesis. Also, this trend was recorded in the creatinine level in fish exposed to the low concentration of abamectin. This may due to a

decrease in the biosynthesis of creatinine and / or may be due to reduction in the muscle mass (Finco, 1989).

Conversely, significant elevation in the creatinine level was seen in fish exposed to the high concentration of abamectin on day 14, this may be occurred as a result of reducing the glomerular filtration rate (GFR) through the kidney tubules.

On the other hand, El-Said (1997) found that fluridone treatment in *Clarias lazera* for 60 days caused a marked increase in the level of urea, whereas a significant decrease in the creatinine level was recorded.

Exposure fish to abamectin did not induce any significant change in K^+ ions, total calcium (T.Ca) and free-ionized calcium (Ca^{2+}) levels.

In contrast, hypoproteinemia was observed during the experimental period (14 days), this may be attributed to impairment in protein synthesis machinery (Bradybury *at al*., 1987) and / or probably because of renal excretion (Albumin - urea) and was due to liver disorders after the pesticides exposure. Therefore, a marked reduction in the level of B. Ca, was recorded, this likely to be occurred a consequence of reducing the level of plasma proteins, which bind with calcium in circulating blood.

The explanation for the decrease in inorganic phosphate level of blood, probably that the phosphorus released by the bones, is resulted in phospholipids, nucleoprotein, and nucleotide synthesis to counter the stress condition (John, 2007). Also, is likely to be a consequence of kidney dysfunction. However, hypophosphatemia may be related to induction of energy metabolism to meet energy demand.

Similar findings on the effect of pesticides were found in previous studies (Srivastava, *et al*., 1997a and 1997b), they reported that sublethal concentrations of deltamethrin and chlorpyrifos caused hypocalcemia and hypophosphatemia for catfish (*Heteropneustes fossilis*) following exposure for 28 days.

Patel *et al*. (2006) observed that excretion rates for Na^+ and Cl^- decreased significantly over time with exposure to waterborne lead. Excretion rates for K^+ were not significantly altered from 0 to 96 h of exposure to control conditions.

Thyroid hormones are important for the control of metabolism, growth and development and osmoregulation in fish as well as in higher vertebrates. They frequently act in synergism with growth hormone and cortisol. Another important function of thyroid hormones is the triggering of migratory behavior and the control of osmoregulation adaptation (Bonga, 1993).

Decrease hepatic T3 clearance and / or increase the conversion of T4 to T3 in hepatocytes via IDI enzyme could be account for increasing of T3 level in plasma of fish exposed to the low concentration of abamectin tested. The principle pathway of T3 generation in the peripheral, mono deiodination of T4 is primarily catabolism by enzyme type I-5-mono deiodinase (Eales, 1985). Transfer freshwater fish to seawater fish involve increase of T4 and T3 metabolism and deiodination of T4 to T3 (De-Luze, *et al*., 1987).

The histopathological changes observed in fish can be negatively affected at tissue level by the cause of abamectin exposure. Some pervious histopathological findings are similar to what we have found in this work and are conformity our histopathological results.

The light microscopic studies in the gills of *O. niloticus* developed by Capkin, *et al*. (2006) found that endosulfan toxicity in juvenile rainbow trout (*Onchorhynchus mykiss*) was irreversible when fish were exposed to minimum concentration of endosulfan. Histologically, fish gills had lamellar edema and separation of epithelium cells from lamellae. Ayas, *et al*. (2007) reported that contaminated commom carp (*Cyprius carpio*) with organochlorin compounds caused degeneration of kidney tubules.

Guimarães *et al*. (2006) revealed several histopathological alterations in gills such as edemea and blood congestion were presented and no-inflammatory processes were observed after exposure to trichlorfon (An organophosphate compound).

Conclusively, it is suggested that the abamectin treatment had modulatory effects on lipid and calcium metabolism and osmoregulation characteristics, in addition to thyroid hormones status and histology of gills and kidney of fish exposed to both concentrations of abamectin.

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