EFFECT OF BASAGRAN HERBICIDE TOXICITY ON GROWTH PERFORMANCE, ACETYLCHOLINE CONTENT, AND ACETYLCHOLINESTERASE ACTIVITY IN NILE TILAPIA (*OREOCHROMIS NILOTICUS*)

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Received 5/ 1/ 2014 *Accepted 20/ 2/ 2014*

Abstract

The present study was conducted to evaluate the toxic effect of basagran herbicide on growth performance, acetylcholine (ACh) content, and acetylcholinesterase (AChE) activity in Nile tilapia (*Oreochromis niloticus*). Fish (32.6± 1.22) were stocked at a rate of 15 fish/120-L aquarium and exposed to 0.0 (control), 20 (T1), 40 (T2), and 80 µL basagran/L (T3) for 3 months. Serum glucose and serum total protein as well as ACh content and AChE activity in different fish tissues were determined after 1, 15, 30, 60, and 90 days. The obtained results showed that fish growth decreased significantly with increasing the basagran concentrations. Also, significant increases in blood glucose and total protein levels were observed with increasing basagran concentrations. Furthermore, significant increases in ACh contents in the selected fish tissues were observed and their pattern was in the order: brain > gill > muscle > liver. On the other hand, AChE activities in fish gills, muscles, brains, and livers decreased significantly by increasing basagran concentrations suggesting the inhibitory effect of basagran herbicide on the AChE system. Inconsonance with the decrease in the AChE activities, there are corresponding increases in Ach contents of the different fish tissues suggesting a decrease in the cholinergic transmission and consequent accumulation of ACh in the same tissues; however this response is basagran dose-dependant.

Key word: Basagran herbicide, toxicity, acetylcholine, acetylcholinesterase, growth, Nile tilapia.

INTRODUCTION

Herbicide contamination of surface water derived from agricultural practices is a problem of worldwide importance. In fish, previous studies on herbicides and pesticides have focused on the effects of contaminant exposure on acetylcholinesterase (AChE) activity (Chuiko, 2000; Bretaud *et al*., 2000; Dutta and Arends, 2003). The measurement of this enzyme, present in the cholinergic synapses and motor end plates, has been used by different authors to monitor carbamate and organophosphate effects in insects and vertebrates including fish (Chuiko, 2000; De La Torre *et al*., 2002; Fernández-Vega *et al*., 2002). Dutta and Arends (2003) showed reduced AChE activity in tissue of fish that were exposed to the organochlorine endosulfan. Assays of AChE as a biomarker in different tissues provide sensible methods for detecting water contamination by many pesticides or herbicides (Sancho *et al.*, 2000). Disturbances in AChE activity can also affect locomotion and equilibrium in exposed organisms and may impair feeding, escape, and reproductive behavior (Saglio and Trijasse, 1998; Bretaud *et al*., 2000).

Rice is one of the most important crops in Egypt and basagran herbicide is extensively used in paddy rice fields for weed control. Aquatic contamination by basagran may occur in and around agricultural areas and may adversely affect aquatic fauna including fish. Additionally, the fish culture in rice fields is commonly practiced by Egyptian farmers. Since basagran herbicide is used for weeds control in the rice fields, side effects on fish are to be expected (Svobodova *et al*., 1993). However, little attention has been given to the possible occurrence of sublethal toxicity of herbicides to fish (De La Torre *et al*., 2002).

Nile tilapia (*Oreochromis niloticus*) is a native freshwater fish of Egypt and it is generally cultured in fishponds and rice fields, which may be treated by basagran herbicide for weed control. It is also known that

pesticides and certain chemical compounds which inhibit AChE activity are known to disrupt the normal behavioral patterns in toxicity animals (Bignami *et al*., 1975). The behavioral changes observed in the intoxicated animals like repeated opening and closing of opercula covering, hyper-extension of all fins, cock-screw swimming, S-jerks, coughing, burst-swimming can be directly related to the inhibition of peripheral and/or central nervous system due to inhibition of cholinesterase activity (Kurtz, 1977).

Manildo *et al*. (2007) found correlation between brain AChE sensitivity and brain AChE levels for some fish species was indicated that by selecting a sentinel species with low AChE activity levels one might risk biomarker's, efficiency by selecting a species also with less sensitivity to the toxic substances. The amount of enzyme activity is the common parameter when brain AChE is used as biomarker to pesticide exposure. Sara *et al*. (2011) reported that inhibition of cholinesterases has been widely used as an environmental biomarker of exposure to organophosphates and carbamate pesticides. Also, Vanessa *et al*. (2012) given that the activity of the enzyme AChE is one of the most recurrently used biomarkers of exposure to pesticides and there are controversial results concerning the effects of endosulfan exposure and AChE activity in fish.

No information is available on changes in ACh content and AChE activity in response to basagran exposure in Nile tilapia. Therefore, the present work was undertaken to find out to what extent the changes in Ach content and AchE activities in different tissues as a possible early biomarker in Nile tilapia exposed to basagran herbicide. Also, fish growth, blood glucose, and total protein were evaluated as well.

MATERIALS AND METHODS

The experimental design.

Apparently healthy Nile tilapia (O. *niloticus*) were collected from Abbassa Fish Farm and transferred to the wet Lab., Central Laboratory for Aquaculture Research (CLAR), Abbassa, Abou-Hammad, Sharkia, Egypt. Fish was transferred to the wet laboratory and acclimated for two weeks during which fish were fed a commercial diet (25% crude protein) for satiation twice a day. After that, fish (32.6± 1.22 g) were stocked in glass aquaria at a rate of 15 fish per 120-liter aquarium. The aquaria were supplied with dechlorinated tap water and constant aeration. The dissolved oxygen was 6.2±0.3 mg/L, the temperature was 27 ± 2 C° and pH was 7.3 ± 0.2.

Basagran herbicide (3-isopropyl-1 H-2, 1, 3-benzothiazin-4 (3H)-one 2,2-dioxide) is aqueous solution containing 48% pentazon as active material produced by Passive Co., Germany. The 96- hour half lethal concentration (96-hr LC_{50}) was found to be 400 µL/L for Nile tilapia **(**Mousa *et al*., 2005). Four fish groups were exposed to four sub-lethal doses of basagran; 0.0 (control), 20 (T1), 40 (T2), and 80 µL/L (T3) in quadriplicates for 3 months. Fish were fed a 25% crude protein diet up to satiation twice daily. A half of aquarium's water was siphoned daily and replaced by new aerated tap-water containing the same basagran concentrations. At the end of the experiment, fish from each aquarium were group-weighed to evaluate the weight gain and specific growth rate as follows:

Weight gain (g/fish) = $W_2 - W_1$

Where W2= final weight, W1= initial weight, and T= experimental period (days)

Survival (%) = {(Total number – dead number) / Total number} x 100

Biochemical analysis:

At the end of the exposure periods, fish were picked up randomly at the same time from each aquarium, anesthetized in tricaine methane sulfonate (MS-222; 30 ml/L) before they were killed by transaction of the spinal cord. Blood was drawn from the caudal peduncle region (v. caudalis). The blood samples were taken from all fish groups after 1, 15, 30, 60 and 90 days of the experiment to determine changes in glucose according to Trinder (1969) and total serum protein according to Henry (1964).

Estimation of acetylcholine (ACh) content:

Fish were dissected and gills, dorsal muscles, brains, and livers were obtained and preserved at – 20 ^{o}C for further assays. The ACh contents in the different fish tissues were estimated as described by Augustinson (1957). One gram of each tissue was transferred to test tubes and kept in boiling water bath for 10 min to inactivate AChE enzyme to release bound ACh. The tubes were cooled and the contents were homogenized in 2.0 ml of distilled water and 2.0 ml of alkaline hydroxylamine hydrochloride and 1.0 ml of 1:1 diluted HCl with H_2O was added. The contents were centrifuged and 1.0 ml of ferric chloride was added to the supernatant. The optical density of the sample was measured at 540 nm in spectrophotometer (Systronic, 169 model) against a blank. The blank consists of 2.0 ml of distilled water, 2.0 ml of alkaline hydroxylamine hydrochloride, 1.0 ml of diluted HCl and 1.0 ml of ferric chloride solution. The values were expressed as µM of ACh/g wet weight of tissue.

Estimation of acetycholinesterase (AChE) activity:

Acetylcholinesterase activity was estimated by the method of Metcalf (1951). Three percentage of homogenate of brain, muscle, gill and liver tissues were prepared in cold 0.25M sucrose solution and

homogenated. Supernatant was used for the AChE enzyme assay. The reaction mixture of 3.0 ml contained: 12µm of acetylcholine chloride, 100 µm of sodium phosphate buffer (pH 7.4) and 1.0 ml of homogenate. After incubating at 37 ◦C for 30 min the reaction was stopped by adding 2.0 ml of alkaline hydroxylamine hydrochloride solution followed by 1.0 ml of HCl (1:1 HCl:H2O). The contents were thoroughly mixed and filtered. To clear the filtrate, 1.0 ml of 0.37 M ferric chloride solution was added and the colour was read at 540 nm in a spectrophotometer (Systronic, 169 model) using blank. The blank preparation is same as homogenate and the values were expressed as µM of ACh hydrolyzed/mg protein/hour.

Statistical Analysis:

The obtained data were subjected to two-way analysis of variance (ANOVA) to test the effect of different doses of Basagran and the exposure period. Duncan's Multiple Range test was used as a post-hoc test to compare between means at $P \leq 0.05$. The software SPSS, version 10 (SPSS, Richmond, VA, USA) was used as described by Dytham (1999).

RESULTS

At the end of the experiment, the fish growth performance showed significant reduction in growth due to basagran exposure and the effect was dose-dependent (Table 1). Meanwhile, the control fish group showed the highest growth. The fish survival decreased significantly in fish groups treated with basagran as compared to that of the control fish group and the lowest fish survival was observed in T3 (59.0±1.08%).

Table 1. Growth performance of Nile tilapia exposed to different basagran concentrations for 90 days.

Treatments	Basagran concentrations (ug/L)			
Parameters	Control (0.00)	T1 (20)	T2 (40)	T3 (80)
Initial weight (g)	32.1±1.21[a]	32.6± 1.22[a]	32.5±1.34[a]	32.2± 0.88[a]
Final weight (g)	63.4±2.14[a]	55.3± 0.08[b]	51.5±1.33[c]	47.2± 2.01[c]
Weight gain (g)	31.3±1.37[a]	22.7± 1.91[b]	19.0± 1.08[c]	15.0± 0.54[c]
Fish survival (%)	96.9±1.05[a]	80.7± 1.35[b]	67.5±2.04[c]	59.0± 1.08[d]

Means with the same letter in the same row are not significantly different (P = 0.05).

In the present study, the blood glucose level increased significantly in basagran-treated fish groups on the 1st and 15th day after exposure (Fig 1). This hyperglycemia continued till the 90th day.

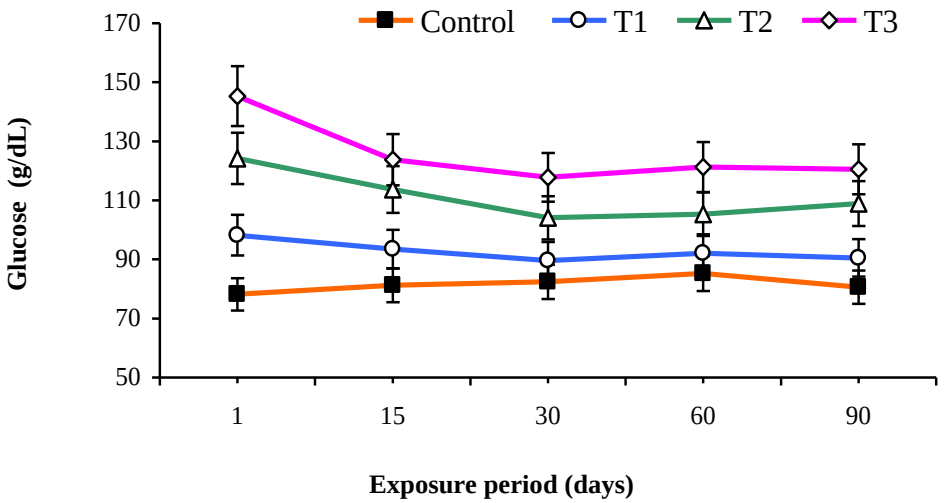

Figure 1. Glucose levels (mg/dL) of Nile tilapia exposed to different sub-lethal basagran doses for different periods.

Contrarily, the total serum protein decreased significantly in fish groups treated with the different basagran concentrations from the 1st day

to the end of the experiment, recording severe decrease on the 15th day (Fig 2).

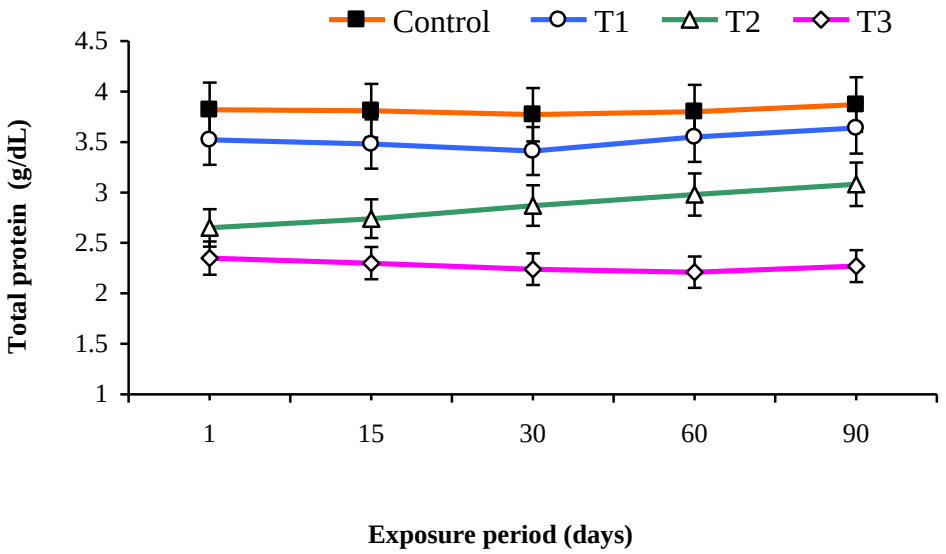

Figure 2. Total protein levels (mg/dL) of Nile tilapia exposed to different sub-lethal basagran doses for different periods.

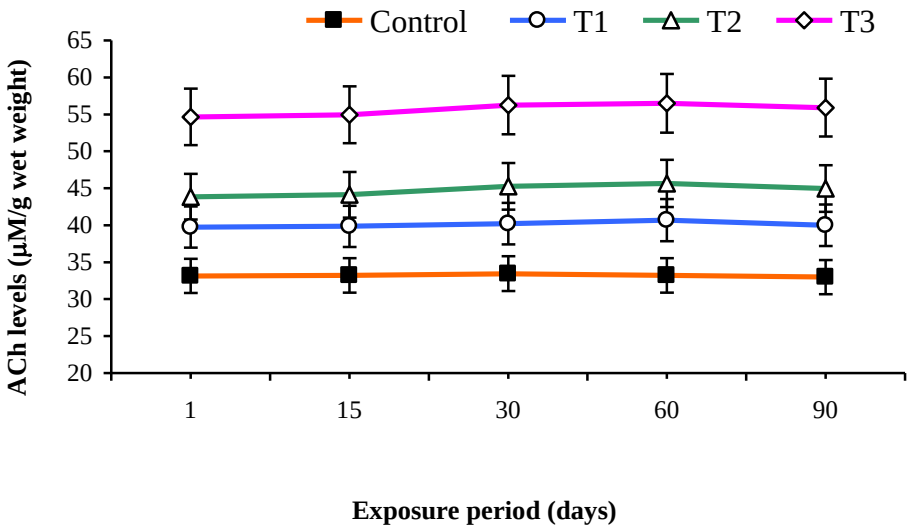

Figure 3. ACh levels (µM/g wet weight) in gills of Nile tilapia exposed to different sub-lethal basagran doses for different periods.

ACh content in the gills of Nile tilapia exposed to sub-lethal basagran concentrations for different exposure periods increased significantly in all treated fish groups on the 1st and 15 days of exposure (Fig 3). Higher ACh content continued till the 90th day in the fish group treated with different concentrations of basagran compared with those of the control group.

In Fig 4, ACh content in fish muscles at different basagran concentrations increased significantly in on the 1st and 15 days of the experiment. Higher ACh content was continued till the 90th day in T1, T2 and T3 (41, 48.65 and 57.87 µM/g wet weight, respectively) compared with those of the control fish group (39.89 µM/g wet weight).

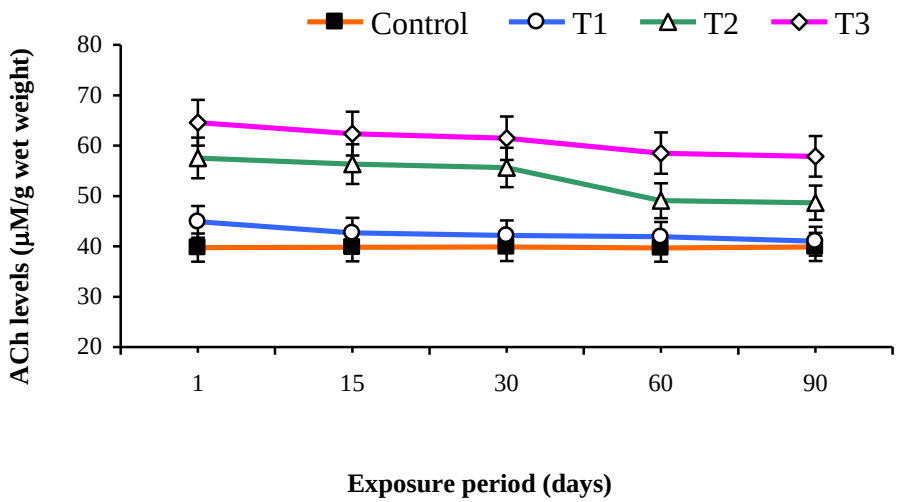

Figure 4. ACh levels (µM/g wet weight) in muscles of Nile tilapia exposed to different sub-lethal basagran doses for different periods.

Moreover, ACh content in the brain of Nile tilapia in all basagran-treated fish groups increased significantly especially in T3 on the 1st (116.46 µM/g wet weight) and 15 (95.34 µM/g wet weight) days of the experiment (Fig 5). Higher ACh content continued till the 90th day in T1,

T2 and T3 (68.24, 82.37 and 87.37 µM/g wet weight, respectively) compared with those of the control fish group (64.66 µM/g wet weight).

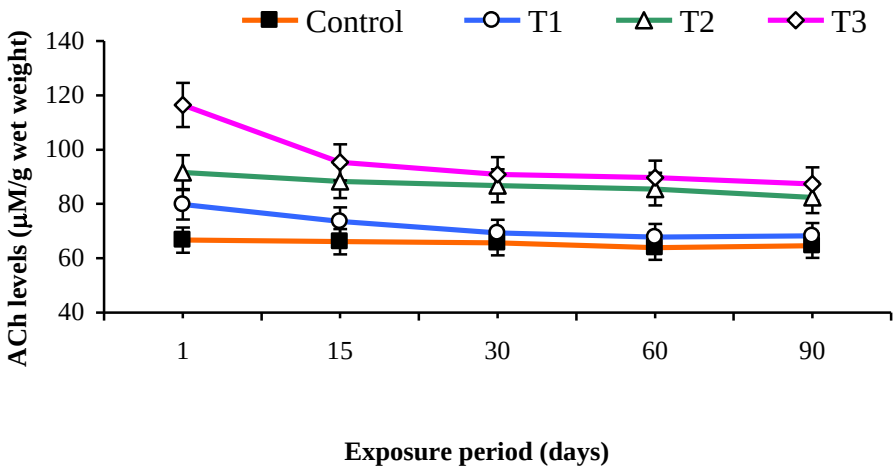

Figure 5. ACh levels (µM/g wet weight) in brains of Nile tilapia exposed to different sub-lethal basagran doses for different periods.

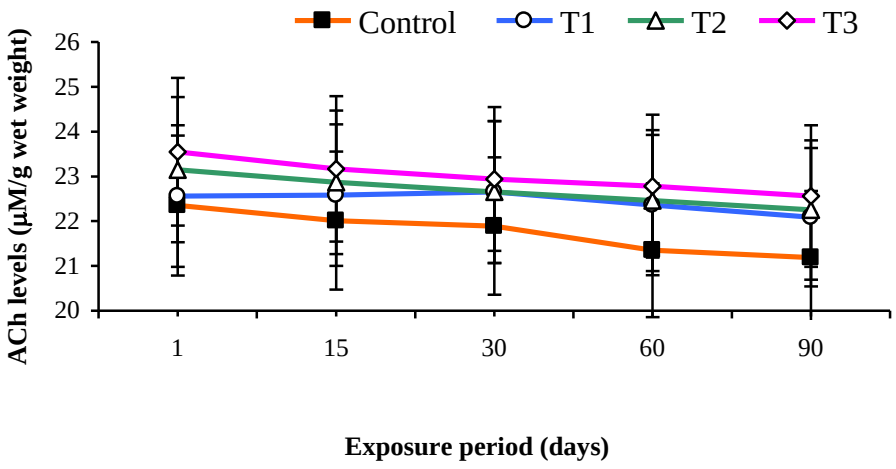

Figure 6. ACh levels (µM/g wet weight) in liver of Nile tilapia exposed to different sub-lethal basagran doses for different periods.

In Fig 6, ACh content in the liver of Nile tilapia at T3 increased significantly on the 1st and the 15th (23.55 and 23.17 µM/g wet weight, respectively) day of the experiment. Higher ACh content continued till the 90th day in T1, T2 and T3 (22.09, 22.25, and 22.56 µM/g wet weight, respectively) compared with those of the control group (21.19 µM/g wet weight).

AChE activity in the gills of Nile tilapia exposed to sub-lethal basagran concentrations at different exposure periods decreased significantly in all treated fish groups on the 1st and 15th days of the experiment (Fig 7).

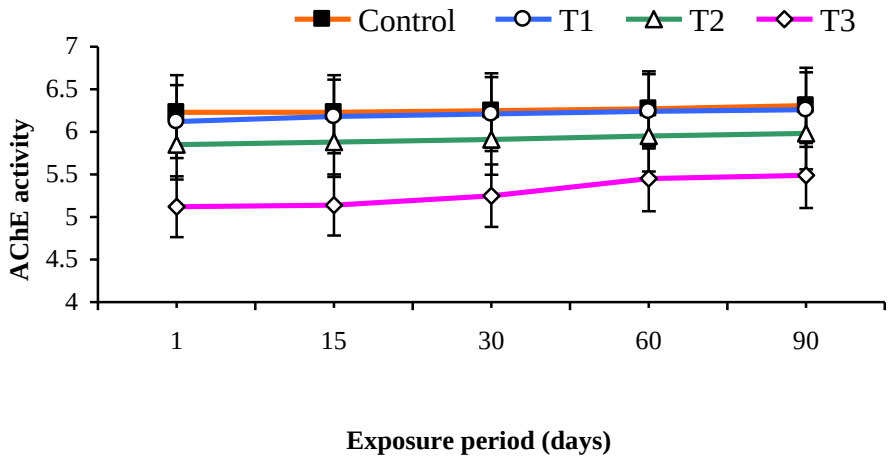

Figure 7. AChE activity (µM of ACh hydrolyzed/mg protein/hour) in gills of Nile tilapia exposed to different sub-lethal basagran doses for different periods.

The low AChE activity continued till the 90th day in all basagran groups as compared with those of the control group. In Fig 8, AChE activity in fish muscles increased significantly in all basagran-treated fish groups on the 1st and 15th days of the experiment. The low AChE activity continued till the 90th day in T1, T2 and T3 (7.04, 6.69 and 6.25 µM of acetyl choline hydrolyzed/mg protein/hour, respectively) compared with

those of the control fish group (7.28 µM of acetyl choline hydrolyzed/mg protein/hour).

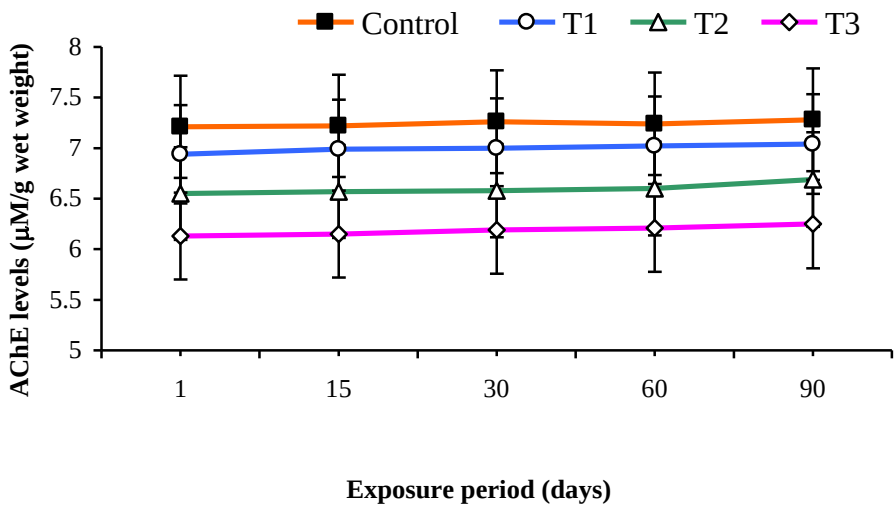

Figure 8. AChE activity (µM of ACh hydrolyzed/mg protein/hour) in muscles of Nile tilapia exposed to different sub-lethal basagran doses for different periods.

Additionally, AChE activity in the brain of Nile tilapia in T3 increased significantly on the 1st and the 15th (7.12 and 7.19 µM of acetyl choline hydrolyzed/mg protein/hour, respectively) days of the experiment (Fig 9). The low AChE activity continued till the 90th day in T1, T2 and T3 (11.35, 8.75 and 7.34 µM of acetyl choline hydrolyzed/mg protein/hour, respectively) compared with those of the control fish group (12.37 µM of acetyl choline hydrolyzed/mg protein/hour).

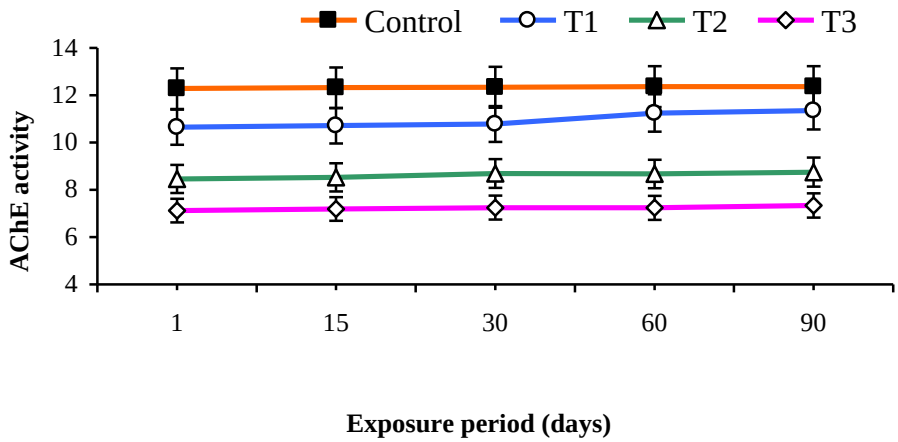

Figure 9. AChE activity (µM of ACh hydrolyzed/mg protein/hour) in brains of Nile tilapia exposed to different sub-lethal basagran doses for different periods.

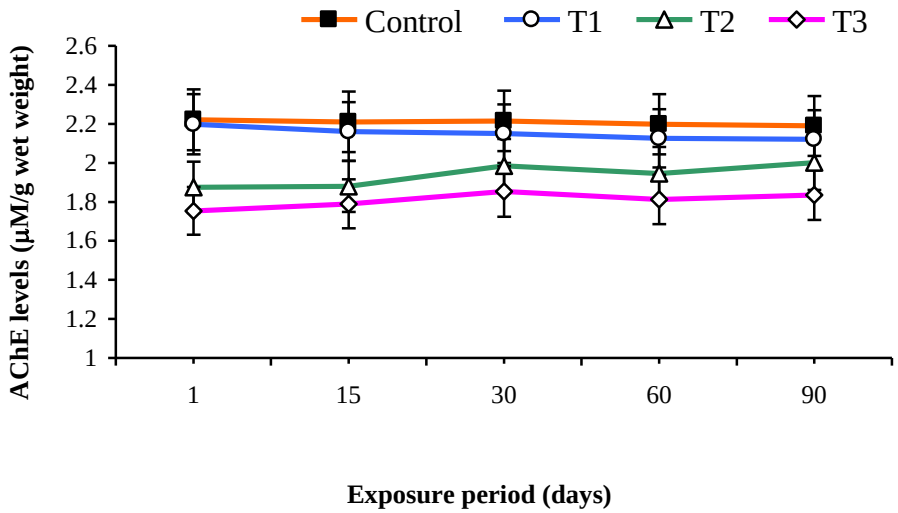

Figure 10. AChE activity (µM of ACh hydrolyzed/mg protein/hour) in livers of Nile tilapia exposed to different sub-lethal basagran doses for different periods.

In Fig 10, AChE activity in the liver of Nile tilapia in T3 increased significantly on the 1st and the 15th (1.754 and 1.79 µM of

acetyl choline hydrolyzed/mg protein/hour, respectively) days of the experiment. The low AChE activity continued till the 90th day in T1, T2 and T3 (2.121, 2.01 and 1.835 µM of acetyl choline hydrolyzed/mg protein/hour, respectively) compared with those of the control fish group (2.189 µM of acetyl choline hydrolyzed/mg protein/hour).

DISCUSSION

The erratic swimming and frequent surface movements, which was observed in basagran-exposed fish, can be attributed to hypercontraction of the muscles due to cholinesterase inhibition as previously reported by Ferguson (1989). On the other hand, Atallah *et al*. (1997) attributed such changes to the extraordinary need for the oxygen which could be attributed to coating of the gills with profuse mucus together with congestion and hyperplastic epithelium of the secondary lamellae.

Glucose levels showed significant increase with increasing the herbicide doses. These results are in agreement with those of Mousa (2004) and Mousa *et al*. (2006). Moreover, Pickering (1981) recorded that the increase in the blood glucose might have resulted from an increase in plasma catecholamines and cortecosteroid hormones and the state of hypoglycemia that occurred on the 30th day in the field treated fish group, might be due to the complete depletion in liver glycogen due to continuous exhaustion.

In the present study, AChE activity in gill, muscle, brain and liver tissues of Nile tilapia exposed to basagran decreased significantly as compared with that of control fish. These results suggest the inhibitory effect of basagran on the AChE system. Inconsonance with the decrease in AChE activity, there is a corresponding increase in ACh content of the same tissues. These results suggest the decrease in the cholinergic transmission and consequent accumulation of ACh in different fish tissues. A similar corroborative increase in Ach content consequent to a

decrease in the tissue AChE levels was reported in fish *Tilapia mossambica* exposed to malathion for 48 h (Sahib and Ramana Rao, 1980). Coppage *et al.* (1975) observed similar inhibition of AChE in the fish brain exposed to sublethal concentration of malathion for 72 h. Singh and Kumar (2000) reported decrease in AChE activity in freshwater teleost, *Catla catla* subjected to sub-chronic and acute exposure to malathion. Similar decrease in AChE activity was reported by Parma de Croux *et al.* (2002) under acute toxicity of monocrotophos in a neotropical fish, *Prochilodus lineatus*. Dos Santos Miron *et al.* (2008) found that the effects of clomazone herbicide used in rice fields on AChE activity in tissues of piava (*Leporinus obtusidens*). They found that AChE activity was reduced only in the brain and heart of fish exposed for 96 h, while AChE activity was decreased in the brain, muscle and heart tissues after 192 h of exposure. Moraes et al. (2007) evaluated the effects of commercial formulations of clomazone and propanil herbicides on AChE in teleost fish (*Leporinus obtusidens*); however, fish were exposed to field measured concentration of the herbicides clomazone and propanil (376 and 1644 μg/L, respectively) on rice paddy water for 90 days. They found decreases in specific AChE activity in fish brains and muscles. Gholami-Seyedkolaei *et al.* (2013) reported that common carp, *Cyprinus carpio* was subjected to Roundup® at 0 (control), 3.5, 7 and 14 ppm for 16 days, and the AChE activity is verified in tissues of gill, muscle, brain and liver. They found significant decreases in AChE activity of muscle, brain and liver tissues.

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تاثير سمية مبيد الحشائش بازاجران على اداء النمو ومحتوى الاستيل كولين وانزيم الاستيل كولين استراز فى اسماك البلطى النيلى

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الملخص العربى

يستخدم مبيد الحشائش بازاجران فى مقاومة الحشائش فى حقول الارز و بالتالى قد يصل هذا المبيد الى المجارى المائية بما فيها الاحواض السمكية التى تستخدم مياه الصرف الزراعى ، كما ان حقول الارز قد تستزرع باسماك البلطى النيلى مما يعرضها للضرر البيئى والفسيولوجى لهذا المبيد. لذا اجريت هذه الدراسة لتقييم تأثير مبيد بازاجران على ادائ النمو ومحتوى الاستيل كولين وانزيم الاستيل كولين استراز فى اسماك البلطى النيلى حيث عرضت اسماك البلطى النيلى الى ثلاث جرعات مختلفة من مبيد البازاجران بالاضافة الى مجموعة الضابطه وكان لكل مجموعة اربع تكرارات. لم تتعرض المجموعة الاولى لمبيد البازاجران و هى المجموعة الضابطة بينما تعرضت المجموعات الاخرى الى ٢٠ ، ٤٠ ، ٨٠ ميكرولتر بازاجران لكل لتر. لوحظ من خلال النتائج ان معدل نمو الاسماك انخفض مع زيادة تركيز مبيد البازاجران. كما لوحظ زياده الجلوكوز فى الدم فى معاملات البازاجران عنه فى المجموعة الضابطة كما زاد مع زيادة تركيز البازاجران . لوحظ ايضا ان محتوى الاستيل كولين يزداد فى انسجة السمكة حسب الترتيب الاتى المخ> الخياشيم> العضلات> الكبد المعرضة لمده ٩٠ يوم من التركيزات السابقه عنه فى المجموعه الضابطه وعلى العكس قل نشاط انزيم الاستيل كولين استراز بنفس الدرجة فى نفس الانسجة (المخ> الخياشيم> العضلات> الكبد) ونتيجة لزيادة محتوى الاستيل كولين ونقص انزيم الاستيل كولين استراز أدى ذلك الى نقل الكولينارجيك وما يترتب علية من تجمع الاستيل كولين فى نفس النسيج مما يمنع توقف الاشارة العصبية وبالتالى يحدث الشلل وربما الموت.