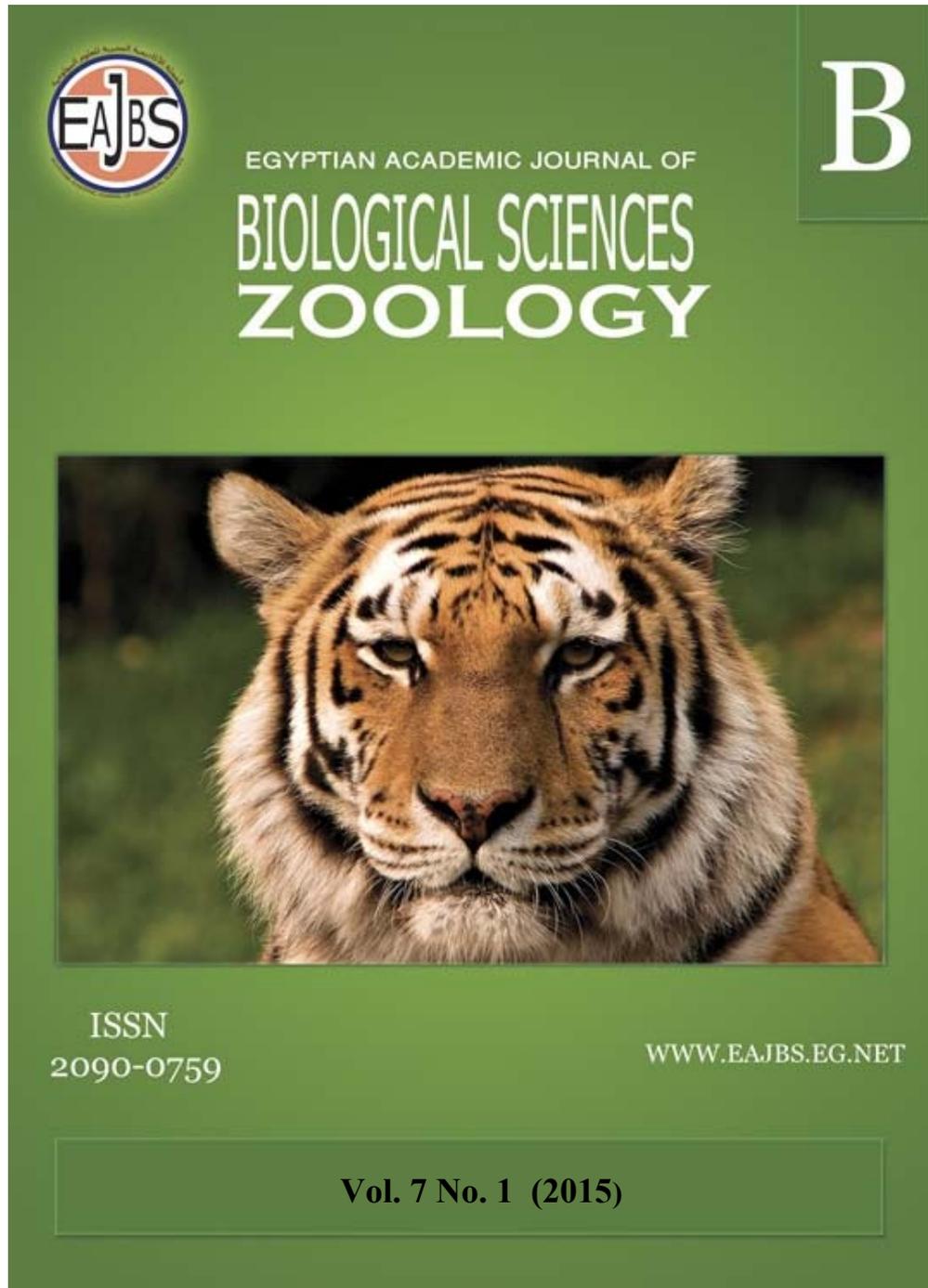


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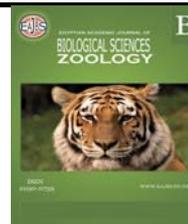


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Effect of Ammonia Stress on Blood Constitutes in Nile Tilapia

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ABSTRACT

This experiment was done to evaluate the effects of ammonium nitrate on *Nile tilapia*. The fish were exposed to sub lethal concentrations of ammonium 2, 4 and 8 mg L⁻¹ of ammonium nitrate respectively for a period of 7 days, 14 days and 21 days respectively to study the changes in hematological parameters and the serum constitutes. General decline in the hematological parameters and the serum constitutes were observed beside the rate of serum constitutes decreased progressively with the increase of toxicant concentration and duration of the exposure. It can be concluded from this study that disturbance in the RBCs and WBCs, hematocrit value, hemoglobin content, serum total protein, serum glucose level, serum activities of (AST, ALT) and (creatinine and uric acid) concentrations as a result of stress of ammonium nitrate on Nile tilapia *O. niloticus* reflect the disturbance in all metabolic function and can be used as marker of pollution. Also, the changes in the hematological parameters indicate that they can be used as indicators of ammonium nitrate related stress in fish on exposure to elevated ammonium nitrate levels in the water.

INTRODUCTION

Nitrogen compounds have been identified as major metabolic products in fish culture. Nitrite may reach toxic concentrations in high density aquaculture systems and in flowing waters due to industrial contamination and fertilizer use. It is an intermediate product in the bacterial oxidation of ammonia to nitrate in conditioned aquaculture systems (Collins, 1975). This nitrogen compound is highly toxic to aquatic organisms and poses a potential threat to cultured fish. When the methemoglobin content of the blood exceeds 70 to 80 % of the total hemoglobin, fish become torpid, unresponsive and disoriented (Klinger, 1957). In aquatic body toxicants present above the normal level i.e., at lethal concentrations bring about mortality of fish and also increase the rate of oxygen consumption in survived fish. The acute toxicity of ammonium sulphate to juvenile rainbow trout *Oncorhynchus mykiss*. Ammonium sulphate was the most toxic compound to rainbow trout compared to composite fertilizers (Erol Capkin, *et al.*, 2010). Respiratory blood pigment hemoglobin manifests the transport of oxygen. Nitrite an intermediate product of ammonia nitrification, may reach toxic concentration in aquaculture

systems when imbalances occur among species of nitrifying bacteria. Nitrite is present at unusually high concentrations in lakes (McCoy, 1972). One physiological response to nitrite is an increase in methemoglobin. The hemoglobin becomes oxidized i.e., the ferrous ion (Fe^{++}) is oxidized to ferric ion (Fe^{+++}) and unable to bind and carry molecules of oxygen. Hence, the toxicity of nitrite to fish received much attention (Russo and Thurston, 1977). Fish with elevated levels of methemoglobin may suffer from anoxia. (Huey *et al.*, 1980; Tomasso, 1981).

In conclusion, the changes observed indicate that hematological parameters can be used as an indicator of ammonium nitrate related stress in fish on exposed to elevated ammonium nitrate levels and glucose level, total protein, the activities of AST and ALT and creatinine and uric acid levels in the *Nile tilapia* that exposed to ammonium nitrate.

MATERIAL AND METHODS

Experimental Animals

Nile tilapia 200 - 250 gm body weight was obtained from Central Lab of aquaculture research, Abbasa. The fish were held in large tanks contained water from their farm, where the physical properties of this water are the following (pH was approximately 7.8, dissolved oxygen was approximately 6.2 ml / L, and salinity was approximately 7.1 %) for a period of two months. The fish were fed with 30 % protein during this time. The fish were divided into four groups, control group and three groups exposed to 2, 4 and 8 mg L⁻¹ of ammonium nitrate respectively for a period 7 days, 14 days and 21 days respectively. Each group consists of two-glass aquaria 30 x 60 x 40 cm³. Each glass aquarium contains 10 fish. The blood samples were taken from six fish after 7 days, 14 days and 21 days of exposure to ammonium nitrate from arterial caudalis with heparinized syringes from control group and 2, 4 and 8 mg/l of ammonium nitrate exposure groups respectively according to the methods of Soivio *et al.* (1972). The blood was divided into two parts; the first part was analyzed for RBCs and white blood cell counts, differential white blood cell counts, hematocrite value and hemoglobin content. The derived hematological indices of mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) were calculated. The second parts of the blood were centrifuged at 4000 rpm for 15 mints to separate the serum for measuring glucose level, total protein, and the activities of AST and ALT and creatinine and uric acid levels.

Analytical Techniques

Blood samples were drawn from arterial caudalis with heparinized syringes. Red blood cells and white blood cells were determined by hemocytometres improved neubaure, hawksley, Germany. Hematocrite value was determined by using a microcapillary tubes where, these tubes filled with blood immediately after blood collection and centrifuged in a microcapillary centrifuge. The amount of red blood cells was determined as a percentage of total blood (hematocrit) by the use of microcapillary reader. Hemoglobin content measured according to Dacie and Lewis (1975). The remaining blood samples were centrifuged for determination of concentration of serum glucose, total protein, and the activities of AST, ALT activities, creatinine and uric acid. The glucose was determined by enzymatic colorimetric method according to Trinder (1969). The total protein was determined by rapid colorimetric method according to Peter (1968). The activities of AST and ALT were determined by colorimetric method according to Reitman and Frankel (1957).

The creatinine and uric acid levels were determined by colorimetric methods according to Henry (1974).

Statistical Analysis

Data were reported as means \pm S. E. (n). Significant differences ($p < 0.05$) within each group were tested with Student's two-tailed t – test and one-way ANOVA by SPSS for windows XP2 2002. Comparisons between groups were tested by Student's two-tailed t – test unpaired design, ($p < 0.05$)

RESULTS

The total content of hemoglobin estimated in the blood of *Nile tilapia* was highly significant decreased when the *Nile tilapia* was exposed to different concentrations of ammonium nitrate and a decrease in total hemoglobin and decrease in derived hematological indices of mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) content was observed as shown in Tables from 1 to 12. Whereas under ammonium nitrate exposure, the hemoglobin content decreased and blood content RBCs, WBCs, MCV, MCH, MCHC also decreased as shown in Tables (1-12).

Table 1: Red blood cells (RBCs) $\times 10^6$ changes in *Nile tilapia* that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	RBCs x 106 after 7days	RBCs x 106 after 14days	RBCs x 106 after 21days
Control	2.6800 \pm 0.19235	2.6800 \pm 0.19235	2.6800 \pm 0.19235
2 mg / l of ammonium nitrate	2.4200 \pm 0.19235	2.4200 \pm 0.19235	2.4200 \pm 0.19235
4 mg/l of ammonium nitrate	2.0400 \pm 0.05477*	1.4000 \pm 0.54772*	1.4000 \pm 0.54772*
8 mg/l of ammonium nitrate	1.7000 \pm 0.15811*	1.4000 \pm 0.54772*	1.2000 \pm 0.44721*

Table 2: White blood cells (WBCs) $\times 10^6$ changes in *Nile tilapia* that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	WBCs x 103 after 7 days	WBCs x 103 after 14 days	WBCs x 103 after 21 days
Control	8.6600 \pm 0.16733	8.6600 \pm 0.16733	8.6600 \pm 0.16733
2 mg / l of ammonium nitrate	7.9200 \pm 0.27749	7.9200 \pm 0.27749	7.9200 \pm 0.27749
4 mg/l of ammonium nitrate	7.0400 \pm 0.11402*	6.2000 \pm 0.83666*	4.6000 \pm 0.89443**
8 mg/l of ammonium nitrate	6.4800 \pm 0.31145*	5.2000 \pm 0.44721*	3.0000 \pm 0.00**

Table 3: Hemoglobin (gd L-1) changes in *Nile tilapia* that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	Hemoglobin (gd L-1) 7 days	Hemoglobin (gd L-1) 14 days	Hemoglobin (gdL-1)after21 days
Control	13.4800 \pm 0.25884	13.4800 \pm 0.25884	13.4800 \pm 0.25884
2 mg / l of ammonium nitrate	12.4800 \pm 0.30332	12.4800 \pm 0.30332	12.4800 \pm 0.3033**
4 mg/l of ammonium nitrate	11.1400 \pm 0.16733**	11.2000 \pm 0.836**	9.2000 \pm 0.923**
8 mg/l of ammonium nitrate	10.5200 \pm 0.37014**	8.4000 \pm 0.19**	4.6000 \pm 0.140**

Table 4: Haematocrit (%) changes in *Nile tilapia* that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	Haematocrit (%) after 7 days	Haematocrit (%) after 14 days	Haematocrit (%) after 21 days
Control	35.2000 \pm 0.18708	35.2000 \pm 0.18708	35.2000 \pm 0.18708
2 mg / l of ammonium nitrate	34.3600 \pm 0.40373	34.3600 \pm 0.40373	34.3600 \pm 0.403*
4 mg/l of ammonium nitrate	33.6800 \pm 0.4324**	31.4000 \pm 0.341**	21.6000 \pm 0.209**
8 mg/l of ammonium nitrate	33.1400 \pm 0.167**	24.4000 \pm 0.335**	14.8000 \pm 0.949**

Table 5: MCV (μ g) changes in *Nile tilapia* that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	MCV (μ g)after 7 days	MCV (μ g)after 14 days	MCV (μ g) after 21 days
Control	133.6000 \pm 0.04959	133.6000 \pm 0.0459	133.6000 \pm 0.04959
2mg / l of ammonium nitrate	127.8000 \pm 0.483**	127.8000 \pm 1.48324	127.8000 \pm 0.483**
4 mg/l of ammonium nitrate	127.2000 \pm 0.836**	116.0000 \pm 0.418**	100.0000 \pm 0.15.81**
8 mg/l of ammonium nitrate	122.2000 \pm 0.2280**	95.0000 \pm 0.414**	54.0000 \pm 0.11.40**

Table 6: MCH (μg) changes in *Nile tilapia* that exposed to different concentrations and different periods of ammonium nitrate

parameters /groups	MCH (μg) after 7 days		MCH (μg)after 14 days		MCH (μg)after 21 days	
Control	46.5400	\pm 0.23022	46.5400	\pm 0.23022	46.5400	\pm 0.23022
2 mg / l of ammonium nitrate	45.3200	\pm 0.31145	45.3200	\pm 0.31145	45.3200	\pm 0.31145
4 mg/l of ammonium nitrate	43.2000	\pm 0.1788**	35.0000	\pm 0.0500**	30.0000	\pm 0.0707**
8 mg/l of ammonium nitrate	40.2000	\pm 0.447**	24.0000	\pm 0.0418**	14.0000	\pm 0.05477**

Table 7: MCHC (%) changes in *Nile tilapia* that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	MCHC (%) after 7 days		MCHC (%) after 14 days		MCHC (%) after 21 days	
Control	39.1800	\pm 0.086610	39.1800	\pm 0.086610	39.1800	\pm 0.086610
2 mg / l of ammonium nitrate	33.8600	\pm 0.24**	33.8600	\pm 0.240**	33.8600	\pm 0.24**
4 mg/l of ammonium nitrate	33.1600	\pm 0.20**	30.2000	\pm 0.0311**	25.0000	\pm 0.0500**
8 mg/l of ammonium nitrate	32.1000	\pm 0.7**	22.0000	\pm 0.02738**	12.0000	\pm 0.0273**

Table 8: Lymphocyte changes in *Nile tilapia* that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	Lymphocyte after 7 days		Lymphocyte after 14 days		Lymphocyte after 21 days	
Control	20.0000	\pm 0.31623	20.0000	\pm 0.31623	20.0000	\pm 0.31623
2 mg / l of ammonium nitrate	18.7000	\pm 0.452*	18.7000	\pm 0.45*	18.7000	\pm 0.452**
4 mg/l of ammonium nitrate	16.0000	\pm 0.15**	15.6000	\pm 0.0114**	12.6000	\pm 0.0207**
8 mg/l of ammonium nitrate	12.4000	\pm 0.167**	10.0000	\pm 0.01414**	7.2000	\pm 0.0164**

Table 9: Monocyte changes in *Nile tilapia* that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	Monocyte after 7 days		Monocyte after 14 days		Monocyte after 21 days	
Control	2.0000	\pm 0.79057	2.0000	\pm 0.79057	2.0000	\pm 0.79057
2 mg / l of ammonium nitrate	0.7000	\pm 0.273**	0.7000	\pm 0.273**	0.7000	\pm 0.273**
4 mg/l of ammonium nitrate	0.5400	\pm 0.240**	0.4200	\pm 0.130**	0.2400	\pm 0.151**
8 mg/l of ammonium nitrate	0.1600	\pm 0.089**	0.1200	\pm 0.044**	0.1000	\pm 0.00**

Table 10: Neutrophile changes in *Nile tilapia* that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	Neutrophile after 7 days		Neutrophile after 14 days		Neutrophile after 21 days	
Control	16.1000	\pm 0.54314	16.1000	\pm 0.54314	16.1000	\pm 0.54314
2 mg / l of ammonium nitrate	14.3800	\pm 0.396*	14.3800	\pm 0.396*	14.3800	\pm 0.39*
4 mg/l of ammonium nitrate	13.0000	\pm 0.1581**	12.0000	\pm 0.0122**	10.4000	\pm 0.0114**
8 mg/l of ammonium nitrate	10.8000	\pm 0.836**	8.2000	\pm 0.0204**	4.8000	\pm 0.0130**

Table 11: Eosinophile changes in *Nile tilapia* that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	Eosinophile after 7 days		Eosinophile after 14 days		Eosinophile after 21 days	
Control	15.2800	\pm 0.31145	15.2800	\pm 0.31145	15.2800	\pm 0.31145
2 mg / l of ammonium nitrate	14.2400	\pm 0.28810	14.2400	\pm 0.28810	14.2400	\pm 0.28810
4 mg/l of ammonium nitrate	11.8000	\pm 0.83666**	9.6000	\pm 0.0167332**	7.0000	\pm 0.0282843**
8 mg/l of ammonium nitrate	9.2000	\pm 0.83666**	6.2000	\pm 0.83666**	4.6000	\pm 0.0230217**

Table 12: Basophile changes in *Nile tilapia* that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	Basophile after 7 days		Basophile after 14 days		Basophile after 21 days.	
Control	12.3000	\pm 0.34641	12.3000	\pm 0.34641	12.3000	\pm 0.34641
2 mg / l of ammonium nitrate	11.3800	\pm 0.35637	11.3800	\pm 0.35637	11.3800	\pm 0.35*
4 mg/l of ammonium nitrate	10.0000	\pm 0.707**	8.8000	\pm 0.0164**	6.0000	\pm 0.0187**
8 mg/l of ammonium nitrate	7.8000	\pm 0.83**	5.0000	\pm 0.0100**	2.6000	\pm 0.089**

Also, there were fluctuations of the activities of AST and ALT and creatinine and uric acid levels in the Nile tilapia that exposed to ammonium nitrate due to damages of protein and hyperglycemia and glycogenolysis by liver and excreted by kidney as shown in Tables (14 – 17). There were decrease in the serum glucose and serum total protein levels in the Nile tilapia that exposed to ammonium nitrate due to

damages of protein and hyperglycemia and glycogenolysis by liver and excreted by kidney as shown in Tables (13 & 18).

Table 13: Glucose mg / dl changes in *Nile tilapia* that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	Glucose mg / dl after 7 days	Glucose mg / dl after 14 days	Glucose mg / dl after 21 days
Control	242.4000 ± 02.88097	242.4000 ± 02.88097	242.4000 ± 02.88097
2 mg / l of ammonium nitrate	201.6000 ± 01.81**	201.6000 ± 01.81**	201.6000 ± 01.81**
4 mg/l of ammonium nitrate	193.6000 ± 08.61**	178.0000 ± 013.0**	150.0000 ± 022.36**
8 mg/l of ammonium nitrate	174.0000 ± 013.11**	140.0000 ± 012.24**	70.0000 ± 030.00**

Table 14: GOT changes in *Nile tilapia* that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	AST U/L after 7 days	AST U/L after 14 days	AST U/L after 21 days
Control	8.3800 ± 0.39623	8.3800 ± 0.39623	8.3800 ± 0.39623
2 mg / l of ammonium nitrate	7.0800 ± 0.08845	7.0320 ± 0.0845	6.0800 ± 0.0885
4 mg/l of ammonium nitrate	8.2000 ± 0.83666	7.0000 ± 01.87083	5.0000 ± 01.00**
8 mg/l of ammonium nitrate	6.2000 ± 0.4**	4.6000 ± 0.89**	2.6000 ± 0.54**

Table 15: GPT U/L changes in *Nile tilapia* that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	ALT U/L after 7 days	ALT U/L after 14 days	ALT U/L after 21days
Control	11.7200 ± 0.47645	11.7200 ± 0.47645	11.7200 ± 0.47645
2 mg / l of ammonium nitrate	13.1200 ± 0.39623	13.1200 ± 0.39623	13.1200 ± 0.39623
4 mg/l of ammonium nitrate	11.2000 ± 0.83666	9.6000 ± 02.07*	6.0000 ± 01.58**
8 mg/l of ammonium nitrate	8.8000 ± 0.83**	5.6000 ± 01.51**	3.2000 ± 0.44**

Table 16: Serum uric acid level mg % changes in Nile tilapia that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	uric acid mg % after 7 days	uric acid mg %after 14 days	uric acid mg %after 21 days
Control	7.9800 ± 0.29496	7.9800 ± 0.29496	7.9800 ± 0.29496
2 mg / l of ammonium nitrate	9.3400 ± 0.64653	9.3400 ± 0.64653	9.3400 ± 0.64653
4 mg/l of ammonium nitrate	9.8000 ± 0.83666	6.8000 ± 01.64317	5.8000 ± 01.78**
8 mg/l of ammonium nitrate	7.2000 ± 0.83666	4.8000 ± 0.83**	2.4000 ± 0.547**

Table 17: Serum creatinine level mg % changes in Nile tilapia that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	creatinine mg % after 7 days	creatinine mg % after 14 days	creatinine mg % after 21 days
Control	1.7600 ± 0.11402	1.7600 ± 0.11402	1.7600 ± 0.11402
2 mg / l of ammonium nitrate	1.3000 ± 0.811	1.3000 ± 0.15*	1.3000 ± 0.58*
4 mg/l of ammonium nitrate	1.3000 ± 0.20000	1.2000 ± 0.4*	1.2000 ± 0.54*
8 mg/l of ammonium nitrate	1.0800 ± 0.083**	1.2000 ± 0.44*	1.0000 ± 0.00**

Table 18: Total protein g% changes in Nile tilapia that exposed to different concentrations and different periods of ammonium nitrate.

parameters /groups	Total protein g% after 7 days	Total protein g% after 14 days	Total protein g% after 21 days
Control	5.7000 ± 0.24495	5.7000 ± 0.24495	5.7000 ± 0.24495
2 mg / l of ammonium nitrate	5.3200 ± 0.19235	5.3200 ± 0.19235	5.3200 ± 0.19235
4 mg/l of ammonium nitrate	4.2000 ± 0.83*	4.2000 ± 01.095	3.2000 ± 0.836**
8 mg/l of ammonium nitrate	3.2000 ± 0.4**	2.2000 ± 0.44**	1.4000 ± 0.54**

DISCUSSION

In aquatic body toxicants present above the normal level i.e., at lethal concentrations bring about mortality of fish and also increase the rate of oxygen consumption in survived fish. The acute toxicity of ammonium sulphate to juvenile rainbow trout *Oncorhynchus mykiss*. Ammonium sulphate was the most toxic compound to rainbow trout compared to composite fertilizers (Erol Capkin, *et al.*, 2010). Decrement in hemoglobin has affected the oxygen carrying capacity of the blood hemoglobin levels decreased gradually and correspondingly the oxygen consumption levels have fallen due to the absorption of more toxicant through the

gills. During experiment the control fish demanded more oxygen in the early hours later got settled and maintained a constant uptake of oxygen, while the exposed fish demanded more oxygen in the early hours and later showed a decrease in the oxygen demand. The increase in the intake of oxygen by the fish may be due to stress caused by the toxicants ammonium nitrate decrement in the oxygen consumption was caused not only due to the damage of the gills but also due to the decrease in the hemoglobin content. The exposed fish consumed more oxygen. The values obtained in the whole animal respiration i.e., the intake of oxygen can be correlated to the hematological changes. When the hemoglobin content decreases its capacity to combine with oxygen also decreases. Hence it leads to a decrement in the rate of oxygen consumption. Decrease in oxygen consumption has been reported for many pesticides (Veeraiah and Durga Prasad, 1998 and Erol Capkin, *et al.*, 2010). A gradual decrease in the intake of oxygen from initial stages to final stages was observed in the test fish, under the exposure of ammonium nitrate. The slight decrement in the intake of oxygen in control fish in the final stages of exposure can be attributed to the decrease in the oxygen levels of water in the container (this decrement is due to oxygen consumed by the test fish). The decrement is higher concentrations of ammonium nitrate exposure than the lower concentrations and so higher concentrations of ammonium nitrate are more toxic than the lower concentrations. This is in agreement with the hematological changes by the effects of ammonium nitrate on hemoglobin of *Cyprinus carpio* (Perrone, 1977) and Ammonium sulphate was the most toxic compound to rainbow trout (Erol Capkin, *et al.*, 2010). Toxic effects of ammonium nitrate include oxidation of hemoglobin to methemoglobin, a form incapable of binding molecular oxygen (Brown and McLey, 1998). Fish with methemoglobin can be detected by the color of the blood and also by brown color of the gills. As ammonium nitrate rises, the fraction of methemoglobin in the blood reduces the oxygen carrying capacity of the blood (Cameron, 1975). Fish with elevated levels of methemoglobin may suffer from anoxia (Tomasso, 1981). Hotchkiss *et al.*, (1992) have observed that the base substitutions are impaired during DNA replication exerting mutagenic effect. The potential role of ammonium nitrate in the toxicity of aquatic animals which reach definitely high concentrations in fish farms due to fertilization and artificial feeding. Also, there is decrease in the glucose level in the Nile tilapia that exposed to different concentrations of ammonium nitrate due to high stress of ammonium nitrate on Nile tilapia. This is in agreement with Shokr, *et al.*, (2002); Shokr and Azza (2004&2005); Shokr (2007) who reported that *Tilapia zilli* was affected by polluted aquatic environment. This is in agreement with the findings of Shokr (2007) when they exposed the *Tilapia ziulli* and Nile tilapia *O. niloticus* to a toxic environment. This they attributed to stimulation of the immune mechanism of the fish to eliminate the effects of the pollutants. Total protein in the Nile tilapia was decreased under the effect of different concentration of ammonium nitrate due to high stress which fish exposed to ammonium nitrate to Nile tilapia. This is in agreement with Shokr (2007) when they exposed the *Tilapia ziulli* and Nile tilapia *O. niloticus* to a toxic environment. This they attributed to stimulation of the immune mechanism of the fish to eliminate the effects of the pollutants. These results are in agreement with Shokr, *et al.*, (2002); Shokr and Azza (2004&2005); Shokr (2007) who reported that *Tilapia zilli* was affected by polluted aquatic environment. This is in agreement with the findings of Shokr (2007) when they exposed the *Tilapia ziulli* and Nile tilapia *O. niloticus* to a toxic environment. This they attributed to stimulation of the immune mechanism of the fish to eliminate the effects of the pollutants. These results are

agreement with (Erol Capkin, *et al.*, 2010) who reported that ammonium sulphate was the most toxic compound to rainbow trout.

It can be concluded from this study that disturbance in the RBCs and WBCs, hematocrit value, hemoglobin content, serum total protein, serum glucose level, serum activities of (AST, ALT) and (creatinine and uric acid) concentrations as a result of stress of ammonium nitrate on Nile tilapia *O. niloticus* reflect the disturbance in all metabolic function and can be used as marker of pollution. Also, the changes in the hematological parameters indicate that they can be used as indicators of ammonium nitrate related stress in fish on exposure to elevated ammonium nitrate levels in the water.

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ARABIC SUMMERY

تأثير ضغط الامونيا على مكونات الدم في البلطي النيلي

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أجرى هذا البحث بغرض معرفة مدى تأثير الامونيوم على مكونات الدم و السيرم في اسماك المياه العذبة (البلطي النيلي). حيث تم تعريض أسماك البلطي النيلي إلى نترات الامونيوم في مياه خفيفة القاعدية (PH 7.8) قسمت الأسماك إلى أربع مجموعات (المجموعة الضابطة و ثلاث مجموعات معرضة إلى ثلاثة تركيزات و هي ٢ و ٤ و ٨ ميلجرام لكل لتر ماء لمدة ٧ و ١٤ و ٢١ يوما على التوالي).

و قد وجد نقصان واضح في عدد كرات الدم الحمراء و البيضاء و كذلك قيمة الهيماتوكريت و محتوى الهيموجلوبين في الأسماك التي عرضت إلى ٢ و ٤ و ٨ ميلجرام لكل لتر ماء لمدة ٧ و ١٤ و ٢١ يوما على التوالي مقارنة بالمجموعة الضابطة. كذلك وجد نقصان واضح في معدل السكر في المصل و البروتين الكلي و أيضا نقص واضح في نشاط الانزيمات الناقلة للمجموعة الامين (AST, ALT) و نقص في تركيز حمض اليوريك و نقص في الكرياتينين للأسماك المعرضة لتركيزات الامونيوم المختلفة.

كما أوضحت النتائج إن الأسماك المعرضة إلى ٢ و ٤ و ٨ ميلجرام لكل لتر ماء من الامونيوم لمدة ٧ و ١٤ و ٢١ يوما نقصان واضح لمكونات الدم و كذلك نقصان في سكر المصل و البروتين الكلي في المصل و نقص واضح في نشاط الانزيمات الناقلة للمجموعة الامين (AST, ALT) و نقص في تركيز حمض اليوريك و نقصان في الكرياتينين للأسماك مقارنة بالمجموعة الضابطة.

كما أكدت النتائج إن نترات الامونيوم سبب إجهادا و ضغطا على اسماك البلطي النيلي التي عرضت لها تحت تأثير جميع الجرعات و أيضا جميع الفترات الزمنية المختلفة و ظهر ذلك في نقصان مكونات الدم الانزيمات الناقلة لمجموعة الامين و بعض وظائف الكلي. مما يدل على إن نترات الامونيوم لها سمية واضحة على اسماك البلطي النيلي و لذلك يوصى البحث بمحاولة إزالة أو معالجة المياه الملوثة بهذا العنصر أو مركباته لأنها شديدة السمية على الأسماك التي يتغذى عليها الإنسان و التي تمثل معظم مصادر البروتين بالنسبة له.