EFFECT OF COPPER ON SOME BLOOD PARAMETERS AND IT'S BIOACCUMULATIONS IN NILE TILAPIA, *OREOCHROMIS NILOTICUS.*

WAFEEK, M.[1] AND A. M. AKAR[2]

1- *Physiology and Biochemistry Department.*
2- *Central Laboratory for Aquaculture Research, Abbassa, Sharkia, Egypt.*

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Abstract

Nile tilapia (*Oreochromis niloticus*) were fed to satiation on a Cu-loaded diet (000 and 2000 mg Cu kg−1 dry weight (dw) feed), or a control diet (3 mg Cu kg -1 dw feed), for 45 days. Then all fish were fed the control diet for a further 21 days to assess recovery. Biochemistry, haematology, and tissue ion content (Cu, Na+, and K+) were measured. No mortalities occurred during the experiment. Dietary copper exposure was confirmed by elevated serum cortisol and glucose of Cu-exposed fish compared to controls after 45 days ($P < 0.05$), liver enzymes (ALT and AST) were take the same trend. Copper exposure was confirmed by elevated Cu concentrations in the intestine, liver and gills of Cu-exposed fish compared to controls after 45 days ($P < 0.05$). All fish showed normal tissue Na+ and K+, and hematology throughout the experiment, fatty change was observed in the liver of Cu-fed fish during the exposure period. The recovery phase was characterized by a reduction in intestinal and branchial Cu levels back to control values. However, the liver of the Cu-fed fish showed a further rise in Cu content and marked hepatic lipidosis (increased intracellular fat stores) post-exposure, suggesting redistribution of Cu to the liver and delayed hepatotoxicity.

Keywords: Copper exposure, glucose, cortisol, AST, ALT, Liver, HSI gills, intestine, moisture, recovery, Nile tilapia, *Oreochromis niloticus*

INTRODUCTION

Oreochromis niloticus is a widely used species in aquaculture for food supply and has been suggested as a bio indicator of water contamination (Almeida *et al.*, 2002). Copper is one of the most abundant transition metals in nature and an essential constituent of all living tissues. When it presents at high concentrations, it becomes toxic to living organisms, including fish (Pelgrom *et al.*, 1995). Also, Copper (Cu) is an essential micronutrient for vertebrate animals and has numerous functions in cellular biochemistry including vital roles in cellular respiration, and as a co-factor for over 30 different enzymes (Linder, 1991). Toxic copper concentrations occur in nature mainly through mining and smelting, leaching from bedrock due to acid mine drainage and precipitation, and from industrial and agricultural activities (IPCS, 1998).

Gills are the first target of waterborne pollutants (Perry and Laurent, 1993) and the main place for waterborne copper uptake (Campbell *et al.*, 1999).

Cu toxicity in freshwater fish is between 16 and 730 mg Cu kg−1 dry weight feed (Clearwater *et al.*, 2002). Nile tilapia is one of the most important freshwater finfish in world aquaculture (Likongwe *et al.*, 1996, Barriga-Sosa *et al.*, 2004). Among the numerous regions now inhabited by Nile tilapia, many are under threat from metal pollutants including copper (Khallaf *et al.*, 1998, 2003 and Shakweer, 1998). Wild Nile tilapia is known to ingest contaminated lake water during feeding with consequent deleterious effects on gut function (Getachew, 1988). Recovery from dietary Cu exposure has rarely been investigated in fish (trout) Handy (1992), and the recovery phase of this experiment therefore adds to the sparse literature on the reversibility of dietary Cu toxicity to fish.

Teleost fishes have a nutritional requirement of about 3–10 mg Cu kg−1 dry weight (dw) feed, depending on the species, feeding regime and life stage (Clearwater *et al.*, 2002). Although the precise Cu requirements of Nile tilapia, *Oreochromis niloticus*, are uncertain, values for tilapia species in aquaculture are suggested (e.g. 4mg Cu kg−1 feed for *O. niloticus* × *O. aureus* hybrids, (Shiau and Ning 2003).

The main objective of this study is to determine the effects of copper exposure on copper accumulation, Na+/ K+-ATPase activity in gill tissue, plasma ion levels, AST and ALT and in stress response indicators such as plasma, glucose concentration and cortisol concentration, in Nile tilapia and to investigate all parameters after the recovery period.

MATERIALS AND METHODS

Eighty Nile tilapia fish with an average weight 75.2±2.6g were obtained from the Central Laboratory for Aquaculture Research, Abbassa, Sharkia, Egypt and placed into a recirculation system consisting of four 150 L experimental tanks (50 fish per tank) with flowing, filtered, aerated, and dechlorinated tap water at 26±1 °C. All fish were fed a control diet (3 mg Cu kg−1 dw feed), to satiation for 14 days in order to acclimate to experimental conditions. Thereafter fish were placed in 9 aquaria (20 fish per aquarium) presented three treatments, one (control group) (3 mg Cu kg−1 dry weight (dw) feed): treatment 1, second diet (1000 mg Cu kg−1 dry weight (dw) feed) treatment 2, and third (2000 mg Cu kg−1 dry weight (dw) feed) treatment 3, fish were fed for 45 days. With then all aquaria fed the control diet for 21-day recovery period. Throughout the experiment fish were fed to satiation once a day, at 11:00 am. Care was taken to ensure that no uneaten food remained in the aquaria during feeding, and Cu did not leach from the feed. Copper concentrations in water samples collected 10 min. before and after feeding remained low throughout the experiment. Fish were randomly sampled from each aquarium after 45 days of exposure and 21 days of recovery for hematology, biochemistry and tissue ion analysis. Fish were not

fed the day before sampling times in order to empty the gut and to facilitate dissection. Hepatosomatic index for each fish (HSI (%) = liver weight (g)/body weight (g) ×100) were determined.

Haematology

Three to five fish per aquarium, sampled randomly, after 45-days from the start of the experiment and at day 21 after recovery period for hematology. Fish were anaesthetized with diluted MS222 and whole blood was collected via the caudal vein into heparinized syringes, then fish were weighed and recorded. Haematocrit value (HCT) and haemoglobin (Hb) concentration were determined immediately. In addition, blood was collected and serum separated (3000 rpm for 10 min) for blood glucose determination. The glucose concentration in plasma samples was measured in triplicate using spectrophotometer at 420 nm. Cortisol levels in plasma were measured by immunological method (Sibar, Perugia, Italy) (Arakawa *et al.*, 1979).

Tissue ion analysis

After blood sampling fish and terminal anesthesia in accordance with ethical approval, tissues were carefully dissected for trace metal analysis according to Handy *et al.* (1999). Briefly, the second gill arches from opercula cavities, posterior intestine, muscles and then liver were harvested. The tissues used for ion analysis (Handy et al., 1999). Tissues for Cu^{++}, Na^{+} and K^{+} analyses were oven dried to a constant weight, digested in 5ml of concentrated nitric acid, then diluted to 20 ml with deionizer water and analyzed by inductively coupled plasma were measured by atomic absorption of Thermo.400 with graffiti furnace (England) Thermo Company model 2005 for Cu, K+ and Na+. Percentage tissue moisture content was calculated from wet and dry tissue weights.

Water quality

Water samples were collected twice weekly in each period throughout the experiment, 10 min prior to and 10 min post-feeding and analyzed according to APHA, (2000).

Statistical analysis

Statistical analysis was performed using the analysis of variance (ANOVA). Duncan's Multiple Range Test was used to determine the significant differences between means at $P<0.05$. Standard division of treatment means was also estimated. All statistics were carried out by using Statistical Analysis Systems (SAS) program (SAS, 2000).

RESULTS

The difference in blood glucose (in mg/dl) and cortisol hormone concentrations (ng/ml) were not significant between treatment 2 with higher copper dose compared with treatment 1 or control one, after the exposure period or after the recovery

period, as showed in (Table 1). Serum transaminases enzymes (AST and ALT) activity recorded significant differences among the three groups after the exposure period, whereas after the recovery period the ALT activity and hepatosomatic index was still highly significant in treatment 2. The HIS recorded the highest percentage in fish treated with the highest copper dose after the first periods and second.

There were no treatment-dependent changes in either whole blood haemoglobin levels or haematocrit values ($P > 0.05$) after 45 days exposure to excess dietary Cu. Haemoglobin levels were 5.88±0.21, 5.91±0.17, and 5.66±0.19 g/dl for control, treatment 1 and treatment 2 at day 45, respectively. Haematocrit (HCT) percentage was no significant difference in control fish at the end of the exposure period or after recovery period among the three groups of fish under the different treatments (Table 2).

Dietary copper exposure was confirmed by elevated Cu concentrations in the intestine, liver and gills of Cu-exposed fish compared to control ones after 45 days (Table 3). After the recovery phase where all fish were fed the control diet, Cu concentrations in the intestines and gills of the Cu-exposed fish decreased clearly and the differences were not significant compared with their respective control fish which fed throughout the control diet experiment. However, liver Cu concentrations continued to rise in the Cu-exposed group during the recovery phase (Table 3).

There were no significant treatment dependent differences in Na^+ levels in liver and intestine, in K^+ levels in gills and liver, tissue moisture content in liver, intestine and muscles among treatments include the control after the exposure period. There were no significant in Na^+ and K^+ levels in organs, tissues moisture content in any organ, except Na^+ in gills among treatments include control after the recovery period and K^+ in the intestine(Table 3).

DISCUSSION

This study shows that these fish accumulate excess Cu in the gill, liver, and intestine. Importantly, Nile tilapia does not recover quickly from dietary Cu exposure. The liver showed further increases in Cu content and fatty change during the recovery phase. Copper levels in the tissues of control fish in this study are broadly similar to previous reports for tilapia (Shiau and Ning, 2003, Co˘gun and Kargin, 2004). The Cu accumulation in Nile tilapia reflected the route of exposure with large increasing in the Cu content of the liver and intestine which in agreement with previous studies on rainbow trout (Kamunde *et al.*, 2001, Campbell *et al.,* 2002, Kamunde and Altwood, 2003), and marine grey mullet (Baker *et al.*, 1998). The gills showed an increase in Cu content during exposure, which cannot be explained by aqueous Cu uptake, because waterborne Cu levels remained low throughout the experiment and gill morphology,

was normal. Food regurgitation was not observed in this study. The small increase in gill Cu content therefore probably reflects systemic Cu in the gill tissue, and distribution of some dietary Cu to the gill from the gut in Nile tilapia. This phenomenon has been previously observed with high dietary Cu doses in fish (rainbow trout, Handy, 1996, Kamunde *et al.*, 2001, and grey mullet, Baker *et al.* (1998). Copper levels in the gill and intestine of Cu-exposed fish returned to control levels during the recovery phase, but hepatic Cu levels continued to increase post-exposure. This suggests that Nile tilapia can redistribute accumulated Cu for excretion via the liver. However, Handy (1992) made similar observations in rainbow trout where livers of Cu-fed fish showed a 30% increase in the proportion of whole body Cu held in the organ during a 12-day recovery phase. Several authors have also noted redistribution of newly acquired Cu to the liver of trout during aqueous exposures (Grosell *et al.*, 2001).

The absence of treatment-dependent changes in tissue moisture content, total content of Na^+ or K^+ in tissues, and HCT suggest that dietary Cu did not cause any major osmotic disturbances (as in trout, Handy *et al.*, 1999). Total sodium and potassium levels, and moisture content in gill, liver and intestine of Nile tilapia fed on a normal diet (control, 3mgCu kg−1 food) or a diet containing elevated copper (1000 and 2000 mg Cu kg−1 food) for 45 days, followed by a recovery period on normal food for a further 21 days were showed that no significant difference in moisture content was observed between control and Cu-exposed fish, or over time within treatments. These are very similar to previous reports for tilapia, Shiau and Ning (2003) who found that the values for haemoglobin and HCT range from 5.2 to 6.3 g dl−1 and 26–36%, respectively for Cu diets containing 1–5 mg Cu/kg feed. Altwood *et al.* (2003) reported that HCT ranged 23–30% depending on diet formulation and water temperature.

The absence of elevated blood cortisol and glucose in the present study support the notion that fish did not suffer overt osmoregulatory stress. Blood cortisol and glucose values were sir ıilar to Altwood *et al.*(2003) who reports thåt Nile tilapia held at 25 °C the values within the normal resting glucose range suggested for unstressed rainbow trout (Hille, 1982).

CONCLUSIONS

Copper exposure induced an early maximum inhibition of gill, liver muscle and intestine of Nile tilapia but the recovery period treats some organs. The study suggests present normal diet for fish a period of time (21 days) (recovery period) enough to remove a detectable copper dose from some investigated different organs better than exposure period.

Table 1. The effect of different doses of copper after exposure period (45 day) and recovery period (21 days) on serum glucose (mg/dl), cortisol (ng/ml) AST (U/l), ALT (U/l) and Hepatosomatic index (HIS) % of Nile tilapia.

Items / Treat	After exposure period (45 days)			After recovery period (21 days)		
	Control	Treat 1	Treat 2	Control	Treat 1	Treat 2
Glucose	78±1.5a	81±1.9a	79±3.9a	82.4±2.2a	85.3±1.7a	88.7±3.8a
Cortisol	56.2±1.1a	57.3±0.9a	59.1±1.7a	59.2±1.1a	62.3±1.5a	63.1±0.9a
AST	32.2±2.3c	39.1±2.6b	55.3±3.2a	33.5±3.6a	35.4±2.2a	36.5±2.6a
ALT	22.3±1.4c	28.4±1.9b	33.2±1.5a	23.2±1.9b	25.3±1.7b	29.6±1.6a
HIS	2.71±0.07b	2.88±0.07b	3.12±0.09a	3.01±0.11c	3.42±0.14b	4.12±0.18a

Means with the same letters in the same row for the same item within the same period are not significantly different.(P> 0.05) using ANOVA.

Table 2. The effect of different doses of copper after exposure period (45 day) and recovery period (21 days) on haemoglobin (HB g/dl) concentrations and haemotocrite ratio (Hct) % of Nile tilapia.

Items / Treat	After exposure period (45 days)			After recovery period (21 days)		
	Control	Treat 1	Treat 2	Control	Treat 1	Treat 2
HB	5.88±0.21a	5.91±0.17a	5.66±0.19a	5.92±0.22a	5.78±0.19a	6.10±0.24a
Hct	32.6±0.7a	32.3±0.9a	31.5±0.8a	33.1±1.1a	32.8±0.9a	33.5±0.8a

Means with the same letters in the same row for the same item within the same period are not significantly different.(P> 0.05) using ANOVA.

Table 3. The effect of different doses of copper after exposure period (45 days) and recovery period (21 days) on bioaccumulations of copper µg Cu g-1 dry weight of tissue, sodium, potassium in serum and dry tissue in mEqul/L and moisture in % of Nile tilapia.

Items / Treat	Organs	After exposure period (45 day)			After recovery period (21 day)		
		Control	Treat 1	Treat 2	Control	Treat 1	Treat 2
Copper	Gills	0.06±0.01c	0.09±0.01b	0.19±0.02a	0.05±0.01a	0.06±0.01a	0.05±0.01a
	Liver	3.74±0.54c	7.42±0.72b	12.63±1.23a	4.11±0.65c	8.88±0.89b	20.23±1.71a
	Intestine	0.33±0.06c	8.25±0.19b	11.23±1.02a	0.26±0.03a	0.24±0.02a	0.35±0.09a
	Muscles	4.92±0.21b	5.21±0.22b	6.42±0.32a	4.88±0.23b	5.28±0.26b	5.89±0.26a
Sodium	Gills	264±18b	300±17a	346±29a	249±15a	250±16a	278±17a
	Liver	70±5a	72±6a	76±4a	75±6a	77±5a	81±6a
	Intestine	281±36a	278±22a	289±27a	240±15a	239±16a	251±14a
	Serum	139±2.52a	135±2.17a	129±2.31b	137±2.21a	136±2.17a	136±2.33a
Potassium	Gills	162±5a	160±4a	172±6a	145±6a	144±6a	147±7a
	Liver	219±16a	230±14a	245±18a	238±15a	235±11a	260±16a
	Intestine	236±15b	270±16a	310±23a	240±19a	251±20a	275±22a
	Serum	3.17±0.11c	3.31±0.08b	3.62±0.13a	3.25±0.08a	3.30±0.11a	3.33±0.12a
Moisture	Gills	74.2±3.6b	78.2±2.5a	82.5±2.6a	74.8±3.1a	76.2±2.3a	80.1±3.9a
	Liver	71.2±1.2a	71.4±1.3a	71.8±1.5a	72.8±1.3a	72.2±1.4a	73.2±1.5a
	Intestine	80.2±1.4a	79.3±1.2a	73.2±1.6a	79.6±0.7a	80.1±1.1a	81.31.2a
	Muscles	70.9±1.1a	71.2±1.1a	72.4±1.2a	72.1±1.2a	72.2±0.9a	73.1±1.1a

Means with the same letters in the same row for the same item within the same period are not significantly different.(P> 0.05) using ANOVA.

Table 4. The effect of different doses of copper after exposure period (45 day) and recovery period (21 days) on some water physioc-chemicals.

Items / Treat	After exposure period (45 day)			After recovery period (21 day)		
	Control	Treat 1	Treat 2	Control	Treat 1	Treat 2
Temperature °C	26.8±0.21a	26.9±0.25a	26.8±0.19a	27.1±0.22a	27.0±0.25a	27.3±0.22a
pH	7.7±0.15a	7.6±0.14a	7.5±0.14a	7.6±0.15a	7.7±0.21a	7.8±0.17a
Salinity g/l	0.22±0.02a	0.23±0.01a	0.22±0.01a	0.23±0.03a	0.24±0.02a	0.22±0.03a
T. alkalinity mg/l	270±21a	275±19a	285±21a	279±18a	281±23a	290±24a
D.O. mg/l	6.82±0.22a	6.92±0.21a	6.72±0.19a	7.91±0.23a	7.72±0.17a	7.82±0.15a

Means with the same letters in the same row for the same item within the same period are not significantly different.(P> 0.05) using ANOVA.

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تأثير النحاس على بعض عوامل الدم وتراكماته فى البلطى النيلى

محمد وفيــق[١] ، عادل محمد عكر[٢]

١ -*قسم الفسيولوجى و الكيمياء الحيوية – المعمل المركزي لبحوث الثروة السمكية بالعباسة*

٢ -*قسم التفريخ – المعمل المركزي لبحوث الثروة السمكية بالعباسة*

فى هذه الدراسة على البلطى النيلى والذى يتغذى على علائق محملة بتركيزات مــن النحــاس بجرعات ١٠٠٠و ٢٠٠٠مجم لكل كجم من العلف الجاف ووجبة التحكم بها ٣ مجم لكل كجــم مــن العلف الجاف لمدة ٤٥ يوم. وتتغذى كل الأسماك المتبقية على وجبة التحكم لمــدة ٢١ يــوم لعمــل الأستشفاء. وتم قياس العوامل البيوكيميائية والهيموتولوجية وعنصر النحاس والصوديوم والبوتاســيوم فى الانسجة والمصل. ولوحظ زيادة السكر فى الدم وكذا هرمون الكورتيزول وانزيمات الكبــد عنــد نهاية ٤٥ يوم وكذا زيادة تراكم النحاس فى الخياشيم والأمعاء والكبد ولم يلاحظ زيادة فى الصوديوم والبوتاسيوم فى تلك الأعضاء. لوحظ انخفاض تلك الزيادات فى كل الأعضاء عدا انزيمــات الكبــد وتراكم النحاس فى الكبد خلال فترة الأستشفاء او التغذية على علائق بها ٠,٣ مجم من النحاس لكــل كجم عليقة جافة.