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Effect of Selenite and Selenate Pollution on Water Quality and Meat Pollution of Nile Tilapia

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ABSTRACT

The current experiment was conducted compare the effect of two concentration levels of selenium (30 and 60 micrograms/l) in water on Nile tilapia and plankton community using selenite and selenate as sources of pollution. Water quality in rearing tanks and meat pollution of Nile tilapia were assessed. Two replicate tanks per treatment were maintained. The experiment included five treatments: the control treatment without selenium pollution, two selenite pollution treatments and two selenate pollution treatments. All tanks were fed and fertilized at the same rate for a duration of 75 days. Juvenile Nile tilapia grew at reduced rate in the selenite treatments compared to those of the selenate treatments. Selenium concentration in water were 30 and 60 micrograms per litter at the start of the experiment, which declined to a concentration of 0.25to 0.36µg/l by the end of the experiment due to its bioaccumulation in the food chain (i.e. plankton) and detrital pool. An average of 343 to 411-folds of selenium bioaccumulation from water to aquatic food organisms was observed. Pollution in Nile tilapia flesh averaged 24.14-44.11µg/g which is considered above the safe level recommended for fish consumption.

Keywords: selenium, selenite, selenate, community respiration, toxicity, meat pollution, Nile tilapia.

INTRODUCTION

Introduction Selenium (Se), as an essential micronutrient for animals and humans (Eisler, 2000), plays important roles in antioxidant defense systems, regulation of thyroid hormone metabolism, and cell growth (Patching and Gardiner, 1999). However, levels of accumulated selenium in the body that exceed nutritional requirements result in large harm to organisms (Li *et al*., 2008).

Industrial and agricultural activity has hastened the release of selenium from geologic sources and made them available to fish and wildlife in aquatic and terrestrial ecosystems around the globe (Hamilton, 2004). Agricultural drain water, sewage sludge, fly ash from coal-fired power plants, oil refineries, and mining of phosphates and metal ores are all sources of selenium contamination of the aquatic environment (Eisler, 2000 and Lemly, 2002a). Once in the aquatic environment, it can rapidly attain levels that are toxic to fish and wildlife because of its bioaccumulation in the food chains and resultant dietary exposure (Lemly, 2004).

Several field studies proved that over a period of several years, low water-borne

concentrations of selenium in both freshwater and brackish systems can cause adverse impacts in fish and wildlife (Hyne *et al.,* 2002). These studies highlighted the importance of food chain uptake and bioaccumulation of selenium, particularly when the source water is selenite-dominated rather than when it is selenate-dominated (Hyne *et al.,* 2002). Selenium contamination has occurred world-wide in association with common and economically important activities such as fossil fuel processing, mining and irrigation (Van Kirk and Hill, 2006).

High concentrations of selenium are toxic, causing the generation of reactive oxygen species (ROS), which can induce DNA oxidation, DNA double-strand breaks and cell death (Umysová *et al.,* 2009). Selenium occurs in several different oxidation states which include oxidized selenates and selenites. Each form is known to differ in bioavailability and toxicity to aquatic organisms (Sappington, 2002).

Selenium release to the environment has devastated wildlife populations in several large scale incidents, like at Belews Lake in North Carolina, or at Kesterson reservoir and San Joaquin Valley in California, leading to deformities and massive reproductive failure in fish and birds (Hamilton, 2004: cited in Morlon *et al.,* 2005).The widespread, expanding use of coal for electric power production makes the associated risk of selenium contamination a global issue (Lemly, 2004).

The objectives of the current study were to explore the effect of two sources of selenium at concentration levels (30 and 60 micrograms l^{-1}) in water on aquatic community respiration, selenium bioaccumulation in the food chain, growth performance and meat pollution of Nile tilapia.

MATERIALS AND METHODS

The experiment was conducted in the Fish Experimental Unit, Faculty of Agriculture, Cairo University, Egypt, during summer 2010(8^{th} August – 21^{th} October). The study lasted 75 days.

A static outdoor rearing system was used to carry out the experiment. Rectangular concrete tanks (2.2*1.2*1.0m) were filled with fresh water obtained from a well and were used as rearing units. Ten concrete tanks were used to evaluate the effect of two sources of selenium pollution (selenite and selenate) applied at two concentration levels in water (30 and 60 micrograms l^{-1}) on water quality, growth performance and meat pollution of Nile tilapia.

All tanks were filled with water to 75 cm depth at stocking. All treatments were randomly assigned to rearing tanks.

Experimental design

Nile Tilapia juveniles (initial weight=54.7-61.6 grams/fish) were randomly distributed into 10 experimental tanks and stocked at a rate of 10 juveniles each. Fish were weighed and lengthed before stocking in each tank. The different doses of selenium were applied once in each tank at the start of the experiment and stopped there after till the end of the experiment. The experiment consisted of five treatments, with two replicate tanks per treatment as follows:

High dose selenite –selenium treatment (SeO_3 -Se= 30 micrograms l^{-1})

The high dose selenite treatment was applied once upon a time (at the start of the experiment) at the rate of 0.03 gram selenium per cubic meter of water in each concrete tank and stopped thereafter. This selenite-selenium concentration was equivalent to a concentration of 30 micrograms $l^{-1}$of water.

Hyper dose selenite –selenium treatment (SeO_3 -Se, 60 micrograms l^{-1})

The hyper dose selenite treatment was applied once (at the start of the experiment) at

the rate of 0.06 gram selenium per cubic meter of water in each concrete tank and stopped thereafter. This selenite-selenium concentration equivalent to a concentration of 60 micrograms l^{-1} of water.

High dose selenate–selenium treatment (SeO_4 -Se, 30 micrograms l^{-1})

The high dose selenate treatmentwas applied only once (at the start of the experiment) at the rate of 0.03 gram selenium per cubic meter of water in each concrete tankand stopped thereafter. This selenate-selenium concentration equivalent to a concentration of 30 micrograms l^{-1} of water.

Hyper dose selenate–selenium treatment (SeO_4 -Se, 60 micrograms 1^{-l})

The hyper dose selenate treatment included one application of selenate-selenium (at the beginning of the experiment) at a rate of 0.06 gram selenium per cubic meter of water in the concrete tank and stopped thereafter. This selenate-selenium concentration was equivalent to a concentration of 60 micrograms l^{-1} of water.

Control treatment

The control treatment did not receive any selenium pollution. This treatment was fed and fertilized at the same rate as other treatments.

Feed and fertilizer

Fish were feed low protein supplemental diet (17.0 % crude protein as fed). The diet consisted of a combination four dietary ingredients (soybean meal, corn, fish meal and vitamin-mineral mix). The diet was processed into pellet form at the Fish Research Unit. These dietary ingredients were incorporated at 22.0%, 71.0%, 5.0%, and 2.0% of total diet composition (Table 1). Tank water was fertilized weekly at the dose of 1.0 gram nitrogen and 0.25 gram phosphorus as active fertilizer material in order to increase algal and plankton abundance in tank water. Supplementary low-protein diet was adopted in the current experiment in order to encourage fish to feed on the plankton. Fish were fed at 2.0% of body-weight, six days a week.

Growth performance

Growth performance of cultured fish was measured in terms of final individual fish weight (g), total length (mm), specific growth rate (SGR % day), weight gain (g/fish) and condition factor (CF). The growth performance parameters were calculated as follows:

Table1. ***Proximate analysis of diet (as fed) and chemical composition of selenite and selenate.***

Component	Diet %	Chemical composition	Selenium %
Protein	16.90	----------	----------
Lipid	2.80	----------	----------
Ash	3.39	----------	----------
Crude fiber	2.00	----------	----------
NFE	63.51	----------	----------
Selenite	------	Na_2SeO_3	41.8 %
Selenate	-------	Na_2SeO_4	45.6 %

Individual fish weight

Individual fish weights were measured at the start and end of the experiment, using a digital balance to the nearest 0.1 g.

Specific growth rate (SGR)

Specific growth rates (SGRW) were determined as:

$SGR\ (W) = (Ln\ Wt - Ln\ W_0) \times 100/t$,

Where:

Wt = Average final weight per fish

W_0 = Average initial weight per fish

t = the experimental period in days

Weight gain (WG)

Weight gains (WG) were calculated as:

WG = Final body weight - Initial body weight

Condition factor (K)

Condition factor (K) was calculated as:

$K = (100\ Wt / Lt^3)$

where:

K= Condition factor of each fish

Wt = Body weight of fish in grams

Lt = Total length of fish (cm)

Water quality analysis

Water from each tank was tested once a week for temperature (^OC), dissolved oxygen (DO), secchi disk visibility (SD) and pH.

All determinations were measured according to the Standard Methods, American Public Health Association (APHA, 1993) and Boyd and Tacker (1992). Water quality data were both replicated among tanks and over time (during the experiment) to perform statistical comparison.

Calculations that predict nighttime decline in DO were based on Boyd *et al.* (1978) and Romaire and Boyd (1979). The projection method was based on assuming that the DO decline during nighttime is essentially linear with respect to time. Boyd (1998) confirmed the high accuracy of the projection method in predicting DO concentration at dawn compared with measured values. Romaire and Boyd (1979) indicated that the nighttime dissolved oxygen model gave highly reliable prediction of early morning DO concentration.

Determination of daytime net primary production and nighttime community respiration required the measurements of the following parameters:

1. Nighttime community respiration per hour ($nCRh^{-1}$) = (dusk oxygen concentration – nighttime oxygen concentration) /nighttime period (hours).
2. Optimal nighttime community respiration (nCR) = hourly nighttime community respiration * dark period (hours).
3. Daytime net primary production (dNPP) = dusk oxygen concentration – dawn oxygen concentration.
4. Dawn oxygen surplus or deficit = dNPP – nCR.

Selenium pollution

Selenium pollution was determined in fillet of cultivated fish, plankton and water as follows: Selenium pollution in fillet of fish was determined according to the method outlined by (Black *et al*., 1965 and Cottenie *et al.*, 1982). Fillet tissue was digested using conc.H_2SO_4 and per chloric acid. The solution was analyzed using Atomic absorption (using International Lab AA/AE Spectrophotometer 157). Selenium pollution was also measured in filtered water where tank water was filtered through 0.45µm Millipore filters. Selenium concentration in filtered water was determined using Atomic absorption according to the method outlined by (Black *et al*., 1965 and Cottenie *et al*., 1982). Selenium concentration in plankton (algae and zooplankton) was determined after digestion of water using conc. $H_2\ SO_4$ and per chloric acid. Selenium concentration in digested solution was determined using Atomic absorption according to Black *et al* (1965) and Cottenie *et al* (1982). Selenium pollution in plankton per liter of tank water was determined as follows:

Selenium concentration in plankton = (digested water Se – filtered water Se). Where:

Digested water Se = Selenium concentration in digested water (micrograms l^{-1}).

Filtered water Se = Selenium concentration in filtered water (micrograms l^{-1}).

Statistical analysis

Selenium pollution, water quality and growth performance data were subjected to one-way analysis of variance to determine significant statistical differences among treatments. One-way analysis of variance was used to test the effect of different selenium pollution levels among treatments. Separation of means was performed using Duncan new multiple range test (using SPSS software, Version 1997). Differences among treatment means were considered significant at 0.05 level for each comparison.

RESULTS AND DISCUSSION

Water quality parameters

Water temperature

The averages of water temperature in rearing tanks ranged from 29.5 to 30.6 ^{O}C among treatments during the experimental period (Table 2), with no significant differences among treatments (P>0.05). There was a gradual decrease in water temperature in rearing units over time during the experimental period, reaching 28.2-29.6^{O}C at the end of the experiment.

Table 2. Water quality parameters in fertilized concrete tanks under different selenite/selenite concentrations.

Parameters	Selenite		Selenate		control
Water temperature (C^{o})	29.7^{a}	30.4^{a}	29.5^{a}	29.8^{a}	30.6^{a}
Early morning oxygen (g O_2/m^2)	3.4^{c}	3.9^{c}	6.2^{a}	4.4^{b}	3.8^{c}
Dusk oxygen concentration (g O_2/m^2)	13.06^{a}	14.24^{a}	14.52^{a}	14.32^{a}	13.98^{a}
Nighttime Community respiration per hour (g $_2/m^2$/hour)	0.794^{a}	0.889^{a}	0.704^{b}	0.707^{b}	0.839^{a}
Daytime net primary production (dNPP) (g $_2/m^2$/daytime)	11.33^{a}	12.22^{a}	8.62^{b}	8.86^{b}	11.24^{a}
Nighttime community respiration (g O_2/m^2)	9.42^{a}	10.52^{a}	8.29^{b}	8.30^{b}	9.84^{a}
Oxygen budget at dawn (g O_2/m^2)	3.79^{a}	3.90^{a}	6.27^{a}	5.03^{a}	3.83^{a}
Community respiration per day (g O_2/m^2)	18.0 ^{a}	21.35^{a}	16.90^{b}	16.96^{b}	20.10^{a}
Secchi disk readings (cm)	16.3^{a}	18.1^{a}	16.2^{a}	18.3^{a}	17.8^{a}
Early morning pH value	7.83^{a}	8.91^{ab}	9.14^{b}	9.10^{b}	8.99^{ab}
Dusk pH value	9.40^{a}	9.58^{a}	8.87^{a}	9.67^{a}	9.53^{a}

Means in the same row with different letters are significantly different (p<0.05).

Dissolved Oxygen at dusk and daybreak

Although dusk oxygen concentrations ranged between 13.0 and 14.5 grams oxygen/m^2among treatments, the rates of community respiration were different (Table 2). Dusk oxygen concentrations were high in all treatments as phytoplankton produced enough oxygen. Gross photosynthesis exceeded community respiration in all treatments. However, oxygen production and community respiration rates were depressed in selenate treatments, compared with the selenite and control treatments, with significant differences among means ($P<0.05$).

Ghosh and Tiwari (2008) reported that a healthy balance pond provides a fluctuation in oxygen levels between day and night that leaves an adequate concentration of oxygen in the water that can support aquatic animal life during night hours.

Oxygen budget

Hypertrophic aquatic systems are characterized by a wide gap in DO concentration between dusk and daybreak times (Barcia, 1980). In the current study, the calculated DO at daybreak ranged from 3.8 to 6.2 g O_2/m^2, while DO at dusk ranged between 13.06 and 14.52 g O_2 $/m^2$ among treatments. The gap in DO concentrations between dusk and daybreak among those treatments ranged from 8.2 to 10.3 g O_2/m^2, which indicated a large gap in oxygen concentrations between dusk and dawn, and the presence of high community respiration rates.

Most culture ponds are hypereutrophic with intensive growth of algae, the daytime dissolved oxygen in euphotic zones ranges from saturated to super-saturated (Chang and Ougang, 1988). Chang and Ouyang (1988) reported that the maximum change in DO between day and night in a 24-hr period was as great as 10 mg/l.

Fish respiration, constitutes a minor (approximately 25% of total) component of whole pond respiration (Hargreaves and Steeby, 1999). Pawar *et al* (2009) reported that phytoplankton is the major oxygen consumer during nighttime.

Daytime net primary production (dNPP) and nighttime community respiration (nCR).

In the current study, daytime net areal primary production rates (g O_2/m^2/day) were calculated according to the free water sampling method (Szyper, 1996). Nighttime community respiration per hour (nCR per hour) was higher in the selenite and control treatments (0.794-0.889 g O_2/m^2/hr) compared to those of the selenate treatments (0.704-0.707 g O_2/m^2/hr), with significant differences among treatments ($P<0.05$).

Nighttime community respiration was depressed in the selenate treatments compared to those of the selenite and control treatments. Consequently, the high bioaccumulation of selenate in the plankton (i.e. algae) negatively affected its nighttime community respiration. No significant differences were observed between the control and selenite treatments in terms of nighttime community respiration. Selenate negatively affected the rate of community respiration and metabolic activities of aquatic organisms (i.e., algae, plankton and bacteria), which may be due to its higher bioaccumulation in the food chain compared to those of other treatments.

Loss of oxygen from fish pond is due to fish respiration, plankton respiration, water column respiration and sediment respiration (Ghosh and Tiwari, 2008). Losordo (1980) found that water column respiration accounted on an average about 60% of the overnight DO decrease, which could be attributed to the plankton respiration rate. Water column respiration accounted for an average of 68% (the range: 53-73%) of whole pond respiration (WPR) in two tropical fish ponds (Teichert_Coddington and Green, 1993).

The daytime net primary production (dNPP) were higher in the selenite and control treatments (dNPP=11.24-12.22 g

O_2/m^2/daytime) compared to those of the selenate treatments (dNPP=8.62-8.86 g O_2/m^2/daytime). The selenate treatments had lower dNPP (67%) compared with those of the selenite and control treatments. This could be due to the negative effect of the higher selenium bioaccumulation in algal cells in the form of selenate.

Selenium can inhibit photosynthesis in both phytoplankton and higher plants (Sielicki and Burnham, 1973), and that the chloroplasts are the main site of selenosysteine formation (Pilon-smith, 2002).

The nCR: dNPP ratio

Small nCR: dNPP ratio (community respiration at night: daytime net primary production) were observed in all treatments (0.83-0.96:1), which indicated that DO was available in good concentrations during nighttime. The nCR: dNPP ratios were different among treatments. The selenite and control treatments had better oxygen concentrations during nighttime (nCR: dNPP ratio=0.83-0.87:1) compared to those of the selenate treatments (nCR: dNPP ratio=0.93-0.96:1). Consequently, selenate toxicity negatively affected the rate of D.O. production and dNPP available at dusk time, resulting in a higher nCR: dNPP ratios. Boyd (1990, 1998) reported that about 20% of dissolved oxygen in water was used by cultured species, while the remaining amount was consumed in respiration by phytoplankton, bacteria and benthic microorganisms.

The selenate toxicity negatively affected the rate of community respiration during nighttime hours, since the rates of nighttime community respiration in the selenate treatments were lower by 12-21.2% compared to those of the control and selenite treatments. This could be explained by the higher accumulation of selenium in algal cells (17.3-20.6 µg/g) in the selenate treatments, being higher than those of the selenite treatments (12.35-15.35 µg/g) (Table 3).

The selenate toxicity affected the rate of oxygen production through photosynthetic activities during daytime hours. The daytime net primary productions (dNPP) at dusk time ranged between 72.5 and 76.6% when compared to those of the control and selenite treatments. It can be concluded that selenate bioaccumulation (as Se) negatively affected algal photosynthesis during daytime hours and aquatic community respiration during nighttime hours.

The selenite toxicity did not show any effect on either algal photosynthetic activity, during daytime hours or aquatic community respiration during nighttime. Consequently, selenite toxicity could be termed as silent toxicity in terms of its non-apparent effect on algal photosynthetic ability or aquatic community respiration during nighttime. However, selenite toxicity negatively affected the growth rate of Nile tilapia than selenate.

Most of the variation in daytime net primary productivity (dNPP as g O_2/m^2/day) among treatments were explained by the effect of selenate toxicity and its bioaccumulation, on daytime oxygen production (through algal photosynthesis) and its consumption by aquatic community respiration. All treatments had excessive algal blooms (Secchi disc=16.2-18.3 cm), suggesting that the dense algal bloom did not affect DO concentration at dawn in all treatments.

Averages of daytime net primary production (dNPP) in the selenite and control treatments (11.24-12.22 g O_2/m^2/daytime) exceeded their optimal nighttime community respiration (9.42-10.52 g O_2/m^2/daytime) by a factor of 1.16-1.19, suggesting net autotrophy. While the daytime net primary production in the selenate treatments (8.62-8.86 g O_2/m^2/daytime), slightly exceeded their optimal nighttime community respiration (nCR=8.29-8.30 g O_2/m^2/nighttime) by a factor of 1.06-1.03, suggesting slight autotrophy. Hargreaves and Steeby (1999) reported that average dNPP exceeded whole pond respiration (WPR) in all

ponds by a factor of about 1.3, suggesting net autotrophy.

The selenite versus selenate toxicity

Fish contamination

Although selenate toxicity negatively affected algal respiration and selenite photosynthesis, the toxicity of selenate to fish was less than that of selenite in the current experiment. Juvenile Nile tilapia grew at reduced rates (0.062 grams/ fish/ day) in the 30 µg-selenite treatment compared to that of the 30 µg-selenate treatment (0.088 grams/ fish/ day), with significant differences among treatments ($p<0.05$).The control non-polluted treatment fed supplementary diet (17% crude protein) had a daily growth rate of 0.2 grams/fish/day (Table 4). Selenite and selenate differ greatly in the ease of assimilation (Umysová *et al*., 2009). Selenate assimilation follows, in principle, that of sulfate and leads to the formation of SeCys and SeMet. Selenite is reduced to selenide and then forms selenoaminoacids (Anderson and Scarf, 1983).

Algae contamination

Selenate species in the 30 µg and 60 µg treatments bioaccumulated at high rate in the food chain (algae, plankton and fish) than those of the selenite treatments, with significant differences among treatments ($p<0.05$). This was due to the lower toxicity of selenate species and the possible tolerance of algae cells to incorporate high levels of selenate.

Selenate predominates in soils and is rapidly and more readily assimilated by plants (Spallholz and Hoffman, 2002). Most inorganic selenium is converted into the protein amino acid, L-selenomethionine (Spallholz and Hoffman, 2002). Chronic toxicity from dietary Se uptake and food chain transfer represents a far greater problem than acute toxicity associated with direct water exposure (Maier and Knight, 1994; Fan *et al*., 2002).

Selenomethionine is highly toxic to aquatic animals compared with other seleno-aminoacids. This can explain why growth rates of Nile tilapia were in the current experiment greatly reduced in the selenite treatments compared to selenate treatments, despite the effect of selenate on algal and microorganisms metabolic activities.

Selenite is highly active as redox catalysts and selenate becomes catalytic upon reduction to selenite (Andreadou *et al*., 1996; Ip *et al*., 2000: cited in Spallholz and Hoffman, 2002). The smaller total lengths and weights of fish exposed to selenium toxicity, suggested that these fish were stressed with metabolic energy shifting from growth functions to combating these stresses. This agrees with Hamilton *et al*. (2002b).

Table 3. ***Selenium content in fish flesh, plankton and water.***

Parameter	Selenite		Selenate		Control
	30 µg	60 µg	30 µg	60 µg	
Fish flesh (Se) µg/g	24.15 ± 1.74^{d}	32.65 ± 1.18^{c}	36.47 ± 0.59^{b}	44.11 ± 0.00^{a}	0.62 ± 0.00^{e}
Plankton (Se) µg/g	12.35 ± 0.85^{d}	15.35 ± 0.55^{c}	17.30 ± 0.5^{b}	20.6 ± 0.8^{a}	0.3 ± 0.00^{e}
Filtered water (Se) g/l	0.250 ± 0.02^{d}	0.365 ± 0.35^{b}	0.300 ± 0.01^{c}	0.460 ± 0.01^{a}	0.125 ± 0.005^{e}

Means in the same row with different letters are significantly different (p<0.05).

Table 4. Growth performance of Nile tilapia under different selenium concentrations.

Parameter	Selenite		Selenate		Control
	30 µg	60 µg	30 µg	60 µg	
Initial weight (gm)	54.7^{b}	57.3^{b}	61.67^{a}	57.0^{b}	59.35^{b}
Final weight (gm)	59.35^{cd}	56.85^{d}	68.30^{b}	60.31^{cd}	73.9^{a}
SGR (%)	0.108^{bc}	-0.014^{c}	0.136^{b}	0.008^{bc}	0.29^{a}
Weight gain (gm)	4.62^{bc}	-0.45^{c}	6.63^{b}	3.31^{bc}	14.55^{a}
CF initial	1.83 ^{b}	1.76 ^{b}	1.69^{b}	1.76^{b}	1.91^{a}
CF final	1.59^{a}	1.54^{a}	1.59^{a}	1.57^{a}	N.D.
Survival (%)	90	65	90	80	100

Means in the same row with different letters are significantly different (p<0.05).

Selenium dose

The design of the current experiment was based on the application of a selenium dose once at the beginning of the experiment at high (30 µg/l) and hyper (60 µg/l) levels and stopped thereafter. The selection of these doses was based on literature data. Dobbs *et al*. (1996) reported that 100 µg/l waterborne selenate resulted in about 50µg/g in algae and 45µg/g in rotifers using a flow through chemostat system (cited in Hamilton *et al.*, 2005b). Selenium concentrations in Belews Lake (USA) were approximately 10-20 micrograms l^{-1} in the main lake where adverse effects were measured in fish (Hamilton, 2004). The biological function of selenium depends on its dose. High concentration of selenium inhibited the mitochondria metabolism (Li *et al*., 2000), which inhibited the activity of enzymes and other functional proteins.

Waterborne selenium

The USEPA criterion of 5µg/l Se was set for the protection of aquatic life (USEPA, 1987). This criterion was based on bioaccumulation of selenium through the aquatic food chain, and the resulting dietary selenium toxicity to fish (Hamilton *et al*., 2002a). The general consensus is that dietary exposure to selenium is more toxic to fish than waterborne exposure (Hamilton *et al*., 2002a).

At the start of the current experiment, initial selenium concentrations in tank water were 30 micrograms and 60 micrograms per liter in both selenite and selenate treatments. These selenium concentrations were applied once at the start of the experiment and stopped thereafter, to simulate fish farming activities in potentially polluted water with selenium, where earthen ponds are filled once with water at the start of the growth season. By the end of the

experiment, selenium concentrations in tank water declined to nearly negligible values. These Se concentrations ranged from 0.25 to 0.36 microgram per liter in the selenite treatments, while those of the selenate treatments ranged from 0.3 to 0.46 microgram per liter, in a dose dependent manner. The control treatment had a waterborne selenium concentration of 1.5 µg/l at the start of the experiment, declining to a concentration of 0.12 micrograms per liter by the end of the experiment (Table 3). The final waterborne concentrations in all selenite and selenate treatments approached near zero Se concentrations by the end of the experiment.

Selenium in water is rapidly taken up by algae (Besser *et al*., 1993). Typically, algae took up maximal concentrations in 3–24 hours (Hamilton, 2005). Graham *et al*. (1992) reported that selenium rapidly disappeared from the water column in a pond study. Correspondingly, increased in sediments (including bacteria and biota) (cited in Hamilton *et al*., 2005a). Uptake of selenium by bacteria, algae, and aquatic consumers rapidly removes selenium from the water column and concomitantly reduces water concentrations, which can give a false impression of low waterborne selenium concentrations precluding adverse effects (Hamilton and Lemly, 1999).

Selenium bioaccumulation in food chain

Holland (1979) reported that selenium concentrations were biomagnified substantially up the food chain in the arm of Belews Lake. He found about 2 µg/L in water, 23-25 µg/g in plankton, 26-31 µg/g in benthic invertebrates, and 18-47 µg/g in three fish species (cited in Hamilton and Lemly, 1999).

In chlorella treated with selenate and selenite, the content of selenomethionine comprised 39% and 24% of the accumulated Se when treated with selenite and selenate, respectively (Neumann *et al*., 2003). This explains why selenite is more toxic than selenate to aquatic organisms, due to its high rate of transformation into selenomethionine, a highly toxic substance.

Selenium concentrations were biomagnified substantially in the food chain (natural food organisms) in the 30 µg and 60 µg selenate treatments (17.3 and 20.6 µg/g in plankton, respectively) compared to those of selenite treatments (12.35 and 15.35 µg/l, respectively), with significant differences among means ($p<0.05$).

Selenium bioaccumulated in aquatic food chain (i.e. plankton) in the current experiment. Concentrations of selenium in tank water were 30 µg and 60 µg/l at the start of the experiment in both the selenite and selenate treatments, while that of the uncontaminated control treatment had a selenium concentration of 1.5 µg/l. By the end of the experiment, selenium concentrations were biomagnified up the food chain to about 12.35-20.6 µg/g in the plankton. Bioconcentration factor of selenium in the current experiment ranged from 343 to 411 in the food chain organisms (i.e. algae and plankton) and 735-803 in fish flesh (Table 3).

In the current study, the highest concentrations of selenium were found in fish, followed by plankton, followed by filtered water (Table 3). Selenium bioaccumulated in algae at the rate of 343-411 times and in fish muscles at 735-803 times, compared to the concentration in water at the start of the experiment.

Lemly (2002b) reported that the highest concentrations of selenium were found in fish, followed by plankton, and periphyton. The Planktonic food pathways exposed fish to potential dietary concentrations of selenium that was 770 times the waterborne exposure (Lemly, 2002b). This is in agreement with the results of the current study.

The concentrations of selenium in the Belews lake water averaged 10 µg/l, selenium concentrations accumulated from 519 times (periphyton) to 3975 times (visceral tissue of fish) in the biota (Lemly, 2002a). Elevated concentrations of selenium in the sediment of Lake Macquarie up to 17 µg Se/g have been

reported (Batley *et al*., 1991; Peters *et al.*, 1999: cited in Hyne *et al*., 2002).

In terms of total selenium in aquatic food chain organisms in the present study, an average 343 to 411-folds of Se biomagnifications from water to aquatic food organisms was observed, while subsequent biomagnification from aquatic food organisms (i.e. algae) to fish flesh produced an average of 2.14 to 1.95-folds.

Fan *et al.* (2002) reported that in terms of total biomass Se, an average 1400-fold of Se biomagnification from water to microphytes was observed. Subsequent transfer from microphytes to macroinvertebrates exhibited an average of only 1.9-fold. Once accumulated in microphytes, Se can be transferred into the top predators including aquatic birds and piscivorous fish (Fan *et al.,* 2002). This agreed with the present study.

Selenium uptake in fish

Uptake of selenium by biota can be from water or diet. Some investigators have gone so far as to exclude water borne studies from consideration in discussing potential selenium toxicity thresholds in fish (DeForest *et al*., 1999).

In the current study, food-chain Se accumulation in the plankton ranged between 12.35 to 20.6 µg/g among treatments at the end of experiment, with significant differences among means ($p<0.05$). Waterborne content of selenium had values approaching zero levels (0.25 to 0.46µg/l). Consequently, selenium uptake in fish must have come from dietary uptake of contaminated algae and algal detritus since water borne selenium concentrations were very low by the end of the experiment. In the current study, fish flesh selenium residues of 24.15 to 44.11 µg/g were observed in Juvenile tilapia, feeding on contaminated plankton (12.35-20.6 µg/g as Se).

Uptake of selenate and selenite by algae, aquatic plants and bacteria, and subsequent transformation to selenoamino acids (e.g., selenocysteine and seleno-methionine) are the key pathways for biotransformation of dissolved inorganic Se into organic selenium, which are subsequently incorporated into proteins (Orr *et al*., 2006). Fish in some lentic areas downstream the mines were found to contain whole-body Se concentrations of up to 42 mg/kg dry weight (Orr *et al*., 2006).

Nile tilapia raised in the non-polluted waters of the control treatment had selenium concentration in fish flesh at 0.62 µg / g. This is in agreement with those reported by Hamilton *et al*. (2000) who reported that natural fish from non-polluted waters in field studies had selenium concentrations in tissue residues less than 2µg/g. In the United States, Se is normally presented at 0.47 ppm (wet weight) in fish, but whole-body levels of about 1 ppm are common (Pakkala *et al*., 1972; Nielsen and Nielsen, 1978).

For whole-body fish, most researchers have proposed a toxicity threshold of approximately 4 µg/g beyond which adverse effects are recognized (USDOI, 1998: cited in Hamilton, 2004). In 2004, the USEPA proposed replacing its water-borne total selenium criterion for the protection of freshwater organisms with a whole-body fish tissue concentration of 7.91 µg/g on a dry weight basis (USEPA 2004: cited in Van Kirk and Hill, 2006).

Fish flesh content of selenium content in the current experiment ranged between 24.1 and 44.11 µg/g on a dry weight basis, which were well above the USEPA proposed safe whole-body fish tissue concentration of 7.9 µg/g on a dry weight basis (USEPA, 2004). Consequently, it could be concluded that fish farming activities that use polluted water with selenium at 30 to 60 µg/l, would produce fish that is not good for human consumption and the harvested fish would be heavily polluted with selenium that

presents a hazard to human health. This is in accordance with Fordyce (2013).

Growth rate of Nile tilapia

The biological function of selenium depends on its dose. High concentration of selenium inhibited the mitochondria metabolism (Li *et al*., 2000), which inhibited the activity of enzymes and other functional proteins.

Growth of Nile tilapia followed a dose-dependent response, being negatively affected as selenium dose increased from 30 µg/l to 60 µg/l, within each source of pollution (i.e. selenite or selenate). Growth rates of Nile tilapia in fish exposed to the lowest selenium input (i.e. 30 µg/l), were more than fish exposed to the highest selenium input (i.e. 60 µg/l).

Selenium can bind to hemoglobin and causes impaired respiratory capacity, rendering it incapable of carrying oxygen. A decrease in respiratory capacity can quickly lead to metabolic stress (Lemly, 1993). Body weight, total length and condition factor significantly decreased at concentrations of 12.0 mg/kg-dw and greater (Rigby *et al*., 2010).

Teh *et al*. (2004) observed that fish fed diets containing 26.0 and 57.6 µg Se/g diet had significantly lower body weight and higher mortality. The authors attributed the impaired growth to the long-term energy trade-off from somatic growth to tissue maintenance and repair. Hamilton *et al*. (1990) reported that Chinook salmon exposed to 18.2 and 35.4 µg Se/g diet as SeMet for 8 weeks grew to only 78 and 37% of control fish weight, respectively.

Selenite toxicity had more adverse effects on growth of Nile tilapia than that of selenate. Daily weight gains of tilapia were significantly lower in the selenite treatments (0.006-0.062 g/fish/day) than those of the selenate treatments (0.044-0.088 g/fish/day). This may be due to the higher rate of selenomethionine formation under selenite toxicity compared to that of selenate toxicity as shown by (Neumann *et al.*, 2003). Selenomethionine is the most toxic form of selenium to aquatic animals including fish.

High accumulation of Se in fish had caused a stress effect and thus impaired energy retention (Deng *et al*., 2007).The presence of Se either directly or indirectly affects hemoglobin formation (Sorensen, 1991a, b).

Survival rate of Nile tilapia

Survival in the current experiment depended on the type of selenium pollution (selenite vs selenate) and its dose. Survival followed a dose-dependent response according to the initial selenium concentration in tank water at the start of the experiment. The lowest survival was observed in fish with the highest selenium inputs (i.e. 60 µg/l), while higher survival was obtained in fish with the lowest selenium input (i.e. 30 µg/l) within each source of pollution (i.e. selenite and selenate).

Selenite toxicity had more adverse effect on survival of fish than did selenate toxicity. Fish survival was significantly higher in the 60 µg-selenate treatment (80%) than that of the 60 µg-selenite treatment (65%), with significant differences among treatments ($p<0.05$). Equal survival rates of fish (90%) were obtained when selenium pollution was applied at the 30 µg/l level in both the selenite and selenate treatments. Increasing the selenium pollution to 60 µg/l, decreased survival to the 65-80 % level. Several studies indicated that both organic and mineral forms of selenium reduce survival (Pieterek and Pietrock, 2012; Gledhill and Van Kirk, 2011; Srivastava and Srivastava, 2010).

Survival followed selenium concentration response: the lowest survival was in fish larvae with the highest selenium residue (9.7 µg/g), intermediate in fish larvae with the intermediate

selenium residue (7.7 µg/g), and highest in fish larvae with the lowest selenium residues (3.3–3.6 µg/g) (Hamilton *et al*., 2005a). Bryson *et al*. (1984) reported that juvenile bluegill (*Lepomism acrochirus*) collected from Hyco Reservoir, NC, experienced 97% mortality within a week after being fed plankton, which contained 45µg/g selenium. Hamilton *et al*. (1996) reported rapid mortality in 5- day-old razorback sucker larvae 5–10 days after feeding zooplankton containing selenium concentrations of 3.5–25.7 µg/g. (Hamilton *et al.*, 2005b). Food-chain organisms that contain 5-15 µg se/g are toxic in the diet of fish (Lemly, 2004). Selenium concentrations in Belews Lake were approximately 10 micrograms/l in the main lake, where adverse effects were measured in fish (Hamilton, 2004; Browne and Lutz, 2010).

Selenium poisoning in fish can induce edema-induced exopthalmus, or protruding eyeballs (Lemly, 2002a).The excess fluid can create pressure sufficient to force the eyes to protrude from their sockets (Lemly, 2002a). All harvested fish from the selenate and selenite treatments in the current study exhibited marked exophthalmos. Acute Selenium toxicity results in congestion and hemorrhages in several organs (Kumar *et al*., 2013) as well as hematological disorders (Serian*i et al*., 2011).

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تأثير التلوث بالسيلنيوم على جودة المياه وتلوث لحوم البلطى النيلى

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اجريت التجربة الحالية لمقارنة تأثير مستويين من تركيزات تلويث السيلينيوم فى المياه (٣٠، ٦٠ ميكروجرام /لتر) على البلطى النيلى. وقد تم استخدام السيلنيت والسيلينات كمصدر للتلوث . وفى هذه التجربة تم تقييم جودة المياه فى أحواض الرعاية وكذلك مدى تلوث لحوم البلطى النيلى. أما بالنسبة لتكرراية المعاملات فقد تم تخصيص حوضين لكل معاملة . وقد اشتملت التجربة على ٥ معاملات : معاملة الكنترول بدون تلوث سيلينيوم – معاملتين للتلوث بالسيلينيت- معاملتين للتلوث بالسيلينات. ولقد تم تسميد وتغذية الأسماك فى جميع الأحواض بنفس المعدلات لمدة ٧٥ يوم . وقد لوحظ انخفاض نمو أسماك البلطى النيلى فى معاملات السيلينيت بالمقارنة بمثيلاتها فى معاملات السيلينات . وعند بداية التجربة كان تركيز السيلينيوم فى الماء ٣٠-٦٠ ميكروجرام/ لتر حسب المعاملة. وقد انخفض هذا التركيز فى نهاية التجربة الى مستوى يتراوح بين ٠,٢٥ الى ٠,٣٦٥ ميكروجرام/لتر . وهذا يرجع الى التراكم البيولوجى للسيلينيوم فى السلسلة الغذائية (البلانكتون) وكذلك فى المادة العضوية المتحللة .وقد لوحظ تضاعف متوسط التراكم البيولوجى للسيلينيوم من المياه الى الكائنات المائية التى تمثل غذاء الأسماك بين ٣٤٣-٤١١ ضعف . أما بالنسبة لتلوث لحوم البلطى النيلى فقد تراوح بين ٢٤,١-٤٤,١ميكروجرام/ لتر . وهذا المستوى يعتبر أعلى من مستوى الأمان فى لحوم الأسماك الصالحة للإستخدام الآدمى .

الكلمات الدالة: السيلنيوم – السيلنيت – السيلينات- تنفس العشائر – السمية – تلوث اللحوم – البلطى النيلى