Effect of Exposure to Sub-Lethal Potassium Cyanide on Growth Rate, Survival Rate, and Histopathology in Juvenile *Heteroclarias* (*Heterobranchus longifilis* x *Clarias gariepinus*)

Donald Chukwudi Aniche[1*], Femi Victor Oluwale[2] and Bright Ogechi Ogbolu[3]

ABSTRACT

Experiments were conducted to determine the toxicity of potassium cyanide (KCN) on juvenile *Heteroclarias* following a 96-h static renewal bioassay. Test specimens were acclimatized for 21 days prior to exposure; the acute toxicity (LC_{50}) was determined to be 0.96 $mg \cdot L^{-1}$. Fractions of LC_{50} (⅕, ¼, ⅓ and ½) were estimated in triplicate for sub-lethal studies in a 21-day exposure/ recovery period. Toxicity of KCN was assessed by examining the behavioural responses, growth parameters, survival rate, and histopathological alterations. Potassium cyanide induced behavioural responses such as irregular swimming activity, rapid jerk-like movement, loss of orientation, and subsequent mortality during lethal toxicity, and reduced or erratic feeding behaviour during sub-lethal exposure. Relative growth rate (RGR) and specific growth rate (SGR) were reduced during exposure, and there was no significant difference ($P>0.05$) in daily length increase (DLI). Condition factor ($K \geq 1$) across the treatments indicated the specimens were in good condition, and this could be due to cytochrome oxidase in catfish. Survival rate was 100%. Histopathological alterations in gills included lesions, severe stunting and multiple foci of erosion of the secondary lamellae. Fatty infiltration of the hepatic parenchyma and severe necrosis in liver were also observed. During the recovery period, improvements in the affected growth parameters (RGR and SGR), and tissues (gill and liver) were observed. Incidence of sub-lethal concentration of KCN in aquatic environments may induce physiological dysfunctions as indicated by reduced growth, and gill and liver alterations in such organisms.

Keywords: Histology, Hybrid catfish, Physiology, Sublethal test, Toxicity

INTRODUCTION

The increasing levels of toxic elements in the aquatic environment can expose fish and other organisms to both lethal and sub-lethal effects. The discharges of contaminants such as cyanide into aquatic habitats are involved in massive mortality of fish and other resident fauna (Kori-Siakpere *et al*., 2006; Okonji *et al*., 2011).

Potassium cyanide (KCN) is important in industrial processes such as in the electroplating of metals, chemical synthesis, and extraction of gold and silver. It is also used in photography; in plastic, paper and textile processing; for metal coating, cleaning or polishing, etc. (Gurbuza *et al*., 2009; Pandey and Govind, 2013; CDC, 2015). In most developing countries, cassava processing also releases large amounts of cyanoglycosides. The peel, fibre, cassava juice, and the residual water produced after the separation of starch and fibre during fermentation contribute to cyanide

[1] Department of Biology, Nigeria Maritime University, Okerenkoko, Delta State, Nigeria
[2] Fisheries and Hydrobiology Research Unit, Department of Zoology, University of Ibadan, Ibadan, Oyo State, Nigeria
[3] Department of Chemistry, Nigeria Maritime University, Okerenkoko, Delta State, Nigeria
* Corresponding author. E-mail address: donaniche@yahoo.co.uk
Received 23 July 2018 / Accepted 5 February 2019

concentrations in wastewater. This concentration is reported to be as high as 200 $mg \cdot L^{-1}$ (Oboh *et al*., 2003; Oseni, 2015). The practice of potassium cyanide fishing causes irreversible damage of the coral reefs and fish mortality, with negative impacts on fish diversity and biomass (Glaser *et al*., 2015), as well as on non-target fish and other invertebrates along with their eggs and larvae, and other microorganisms (Authman *et al*., 2013).

Freshwater fish are the most cyanide-sensitive group of aquatic organisms that have been tested. However, varied sensitivities to cyanide have been reported in different species of fish (Dzombak *et al*., 2005; Akinsiku *et al*., 2010; Ramzy, 2014). Substantial interspecies variability exists in sensitivity to free cyanide; however, the juvenile and adult stages are the most sensitive, whereas the embryos and sac fry are the most resistant (Eisler and Wiemeyer, 2004). Free cyanide, which results from the dissociation of cyanide salts such as potassium cyanide is the primary toxic agent in the aquatic environment due to its high metabolic inhibition potential on organisms (Gurbuz *et al*., 2009; Okonji *et al*., 2011). However, the low absorption, rapid volatilization, and rapid metabolism of cyanides limit its potential for bioaccumulation in fish (ECETOC, 2007).

The basic mechanism of cyanide toxicity is through its potency as a respiratory poison in aerobic organisms; it is readily absorbed across the gill membranes in fish, and it acts as an asphyxiant by inducing tissue anoxia and cytotoxic hypoxia through the inhibition of cytochrome oxidase (Eisler and Wiemeyer, 2004; Okonji *et al*., 2011). Significant toxic effects in fish include hypoxia, susceptibility to predation, growth inhibition, behavioural alteration and reproductive impairment (Authman *et al*., 2013). It is also reported to induce histopathological alterations in physiological tissues such as the gill and liver, as well as altering enzymatic activities in fish species (Ramzy, 2014; Oseni, 2015).

The use of cyanide compounds as fish poisons have been prohibited in Nigeria, but artisanal fishermen still use it in Nigerian inland waters (Eyo and Ahmed, 2005). Potassium cyanide fishing is employed as a fast method to stun and collect fish (Mak *et al*., 2005; Surleva *et al*., 2012), and the compound is used as a fumigant in agriculture and as a poison against rats and pests (Pandey and Govind, 2013). Consumption of cyanide-caught fish or fish exposed to cyanide poses health problems to humans (Oseni, 2015). The hybrid catfish *Heteroclarias* as well as other species of catfish play an important economic role in Nigeria's aquaculture as fish food. Hence, it is very popular with both fish farmers and consumers (Yisa and Olufeagba, 2005). Furthermore, *Heteroclarias* is observed to have superior growth, improved survival, hardiness and ability to adapt to adverse water conditions compared to other catfish species (Solomon *et al*., 2013; Afia and Ofor, 2016). Studies have been carried out on cyanide's effects on different catfish species. Thus, the toxic effect on this hybrid is investigated for selected parameters.

MATERIALS AND METHODS

Fish specimen collection and acclimatization

Healthy specimens of juvenile *Heteroclarias* (*Heterobranchus longifilis* and *Clarias gariepinus*) were obtained from a commercial fish farm in Ibadan, Oyo State, and transported in well-aerated plastic containers between 7.00 h and 9.00 h (to avoid heat stress) to the laboratory in the Department of Zoology, University of Ibadan. The specimens were acclimatized for 14 days in well-aerated holding containers containing 150 L of water, and fed once daily on 1.8 mm commercial feed (Skretting fish feed) containing 52% crude protein, at 2.5% of total body weight (Sadati *et al*., 2013). The water was renewed daily. Feeding was stopped 24 hours before the commencement of the experiment.

Range finding test and acute toxicity

Range finding tests were carried out in duplicate using five fish per concentration to determine the concentrations for acute toxicity tests. Exposure time ranged from 1 h to 6 h until minimal mortality was observed from concentration

of 2.0 mg·L^{-1} down to 1.10 mg·L^{-1}. The standard protocol as described by OECD (2014) was employed. The following concentrations: 0.90, 0.95, 1.00, 1.05, 1.10 mg·L^{-1} were made into 10 L, with three replicates each. Ten average size fish specimens (7.18 ± 1.26 g of weight and 8.53 ± 0.89 cm standard length) were used for each replicate (including the control without the toxicant KCN). The LC_{50} of 96 h of exposure was obtained using the Static renewal bioassay method. The treatments (including control) were renewed every 24 h and the fish were unfed during the 96 h period of exposure.

Sub-lethal test

Sub-lethal concentrations of one-fifth (⅕), one-fourth (¼), one-third (⅓), and one-half (½) of 96 h LC_{50} were used for the test. The corresponding sub-lethal concentrations were 0.19, 0.24, 0.32 and 0.48 mg·L^{-1} respectively. The experiment lasted for a period of 21 days, and fifteen aquaria were used with three replicates per treatment. Ten fish of fairly uniform size (7.49 ± 1.05 g weight and 8.57 ± 1.07 cm standard length) were exposed to sub-lethal concentrations of potassium cyanide in each replicate as in Rejeki *et al*. (2006). Test solutions were renewed every 24 h in each of the treatments, and specimens were fed at 2.5% of total body weight.

Recovery test

The exposed fish were transferred to clean dechlorinated tap water (without KCN) for 21 days in all treatments. Weight and length measurements were taken for three consecutive weeks, while samples for haematological and histopathological examinations were collected at the end of the recovery period.

Determination of growth parameters

The weight (g), standard length (cm) and total length (cm) of the fish were measured after each week, and the following growth parameters were determined using the corresponding formulae:

Relative Growth Rate (%)

$$RGR\ (\%) = \frac{Wf - Wt}{t_2 - t_1}\ x\ 100$$

(Lugert *et al*., 2014)

Specific Growth Rate (%)

$$SGR\ (\%) = 100 \left(\frac{\ln Wf - \ln Wt}{t_2 - t_1} \right)$$

(Rejeki *et al*., 2006)

Condition factor (*K*)

$$K = \frac{100\ xW}{L^3}$$

(Jin *et al*., 2015)

Daily Length Increment (DLI)

$$DLI = \frac{Lf - Li}{t_2 - t_1}$$

(Panase and Mengumphan, 2015)

where W = Total wet body weight of fish in grams; Wf = Final weight; Wi = Initial weight; L = Standard length of fish in centimeters; Lf = Final length; L_i = Initial length; t_1 and t_2 = Difference in time (duration of experiment in days).

Survival rate

$$\text{Survival rate} = \left(\frac{N_f\ x100}{N_t} \right)$$

(Rejeki *et al*., 2006)

where N_f and N_t = Number of fish at the end of the experiment and the initial number of fish stocked at the start of the experiment, respectively.

Histopathological examinations

The gills and livers were isolated from sacrificed test organisms (two from each replicate) each week during exposure, and at the end of recovery period (two from each replicate). These were transferred into sterile bottles and fixed in Bouin's fluid. Pathological studies were performed at the Histopathology Laboratory of the University College Hospital (UCH), University of Ibadan, Ibadan.

Statistical analyses

The quantal response (mortality) was analyzed by probit analysis for LC_{50} of 96 h of exposure at the 95% confidence interval (Finney, 1952). Data were subjected to descriptive statistics and one-way analysis of variance (ANOVA) using SPSS version 20 software. Duncan's Multiple Range Test (DMRT) was used to separate the significantly different means at 0.05 level of probability.

Physico-chemical parameters

Temperature (ºC) and pH were determined using the Eutech Multi-Parameter PCSTestr 35 by dipping the device into water (treatment solutions) to a depth of about 3 cm until a stable reading was observed and recorded. Dissolved oxygen (DO: $mg{\cdot}L^{-1}$) values were determined using a dissolved oxygen meter.

RESULTS

Toxicity studies

There was no mortality observed in the control, whereas mortality increased with increase in concentration of the toxicant (KCN) and duration of exposure. Mortality rates for acute exposure to potassium cyanide are presented in Table 1. The estimated 96 h LC_{50} was 0.96 $mg{\cdot}L^{-1}$, and the linear equation for selected concentrations from the probit analysis was $y = 45.644x - 131.1$ with $R^2 = 0.9545$ (Figure 1).

Behavioural responses

Observed behavioural changes during acute toxicity compared to the control treatment included irregular swimming activity, hyper-activity, rapid jerk-like movement, loss of orientation, and ultimately death. Test specimens tried to jump out

Table 1. Mortality of *Heteroclarias* exposed to 96 h acute concentrations of potassium cyanide.

Conc.	6h	12h	24h	48h	72h	96h	Average mortality (n = 10)	% mortality
0.00 $mg{\cdot}L^{-1}$	-	-	-	-	-	-	0	0
0.90 $mg{\cdot}L^{-1}$	-	-	-	-	-	1	1	10
0.95 $mg{\cdot}L^{-1}$	-	-	-	1	2	2	5	50
1.00 $mg{\cdot}L^{-1}$	-	-	-	1	3	4	8	80
1.05 $mg{\cdot}L^{-1}$	-	-	1	2	3	3	9	90
1.10 $mg{\cdot}L^{-1}$	1	2	4	3	-	-	10	100

h = hour; n = total number per replicate; (-) = no mortality

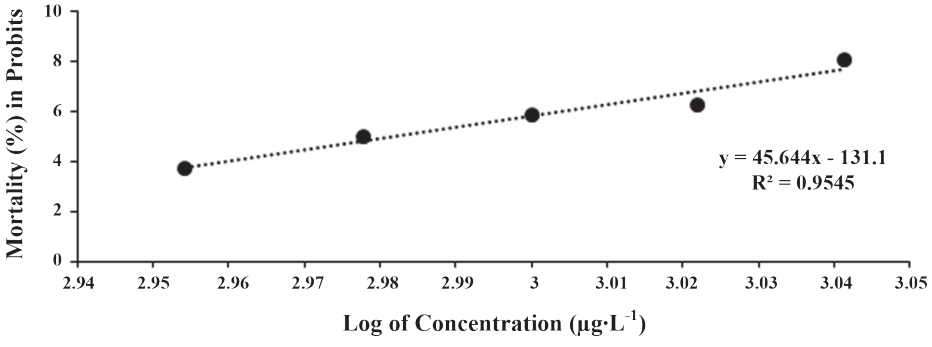

Figure 1. Lethal Concentration (LC_{50}) for 96 h potassium cyanide exposure on *Heteroclarias* probit analysis.

of the solution, and with increase in the period of exposure, most of the specimens aligned vertically with their heads at the surface of the solution. Sub-lethal exposure resulted in reduced, erratic feeding behaviour and nesting at the bottom with increase in exposure period.

Physico-chemical parameters

The total average of the parameters observed each day of experiment and presented in Table 2 did not vary significantly.

Effect of sub-lethal concentrations of potassium cyanide on growth parameters of juvenile *Heteroclarias*

The observed changes in the mean values of different growth parameters during the exposure and recovery periods are shown in Table 3. The toxicant (KCN) significantly induced reduced growth in RGR and SGR. The condition factor (*K*) was ≥ 1 in all treatments.

Table 2. Mean values of physico-chemical parameters.

Parameter	0.0 mg·L^{-1}	0.19 mg·L^{-1}	0.24 mg·L^{-1}	0.32 mg·L^{-1}	0.48 mg·L^{-1}
Temp (ºC)	27.0 ± 0.14	26.99 ± 0.21	26.75 ± 0.25	26.90 ± 0.38	26.70 ± 0.37
pH	7.63 ± 0.20	7.61 ± 0.21	7.78 ± 0.20	7.65 ± 0.26	7.80 ± 0.26
DO (mg·L^{-1})	4.44 ± 0.01	4.43 ± 0.02	4.43 ± 0.02	4.41 ± 0.02	4.40 ± 0.02

Means (±SD) with different superscript letters across rows are significantly different (P<0.05).

Table 3. Changes in growth parameters of juvenile *Heteroclarias* during 21 days of exposure / recovery periods.

Parameter	Conc. (mg·L^{-1})	Weeks					
		1	2	3	1(R)	2(R)	3(R)
Relative growth rate (RGR)	0.00	22.43 ± 5.32	18.99 ± 4.11^{a}	15.06 ± 3.33^{a}	12.35 ± 2.43^{a}	14.18 ± 2.81^{a}	17.29 ± 2.81^{a}
	0.19	26.14 ± 7.51	19.59 ± 6.11^{a}	12.95 ± 4.02^{ab}	9.90 ± 4.32^{ab}	13.12 ± 4.72^{ab}	15.58 ± 4.56^{ab}
	0.24	17.03 ± 6.37	14.39 ± 5.55^{ab}	10.35 ± 3.13^{ab}	7.98 ± 3.71^{ab}	10.32 ± 2.24^{ab}	13.42 ± 2.34^{ab}
	0.32	7.13 ± 1.60	6.44 ± 0.71^{b}	5.92 ± 2.93^{b}	7.20 ± 2.72^{ab}	8.85 ± 0.40^{ab}	12.14 ± 1.38^{ab}
	0.48	13.14 ± 3.77	7.79 ± 0.92^{b}	5.83 ± 0.80^{b}	5.45 ± 1.27^{b}	8.42 ± 1.54^{b}	11.67 ± 0.32^{b}
Specific growth rate (SGR)	0.00	6.04 ± 4.29^{a}	6.87 ± 1.60^{a}	5.40 ± 1.14^{a}	4.38 ± 0.75^{a}	4.54 ± 0.60^{a}	4.7 ± 0.41^{a}
	0.19	6.21 ± 6.82^{a}	6.73 ± 3.12^{a}	4.34 ± 2.37^{ab}	3.33 ± 1.77^{ab}	4.24 ± 1.00^{ab}	4.41 ± 0.66^{ab}
	0.24	1.34 ± 5.53^{ab}	4.30 ± 3.97^{ab}	3.30 ± 2.40^{ab}	2.61 ± 1.69^{ab}	3.63 ± 0.59^{ab}	4.09 ± 0.44^{ab}
	0.32	1.02 ± 3.19^{b}	0.77 ± 0.80^{bc}	0.58 ± 2.66^{c}	2.31 ± 1.49^{ab}	3.20 ± 0.13^{b}	3.87 ± 0.28^{b}
	0.48	1.64 ± 4.42^{ab}	0.59 ± 0.82^{c}	0.93 ± 0.68^{c}	1.44 ± 0.88^{b}	3.09 ± 0.48^{b}	3.78 ± 0.06^{b}
Condition factor (*K*)	0.00	1.16 ± 0.06	1.28 ± 0.0.5^{a}	1.22 ± 0.41^{a}	1.18 ± 0.06^{a}	1.14 ± 0.03	1.12 ± 0.63
	0.19	1.21 ± 0.14	1.21 ± 0.14^{ab}	1.19 ± 0.97^{ab}	1.15 ± 0.08^{a}	1.18 ± 0.15	1.15 ± 0.01
	0.24	1.09 ± 0.19	1.10 ± 0.06^{bc}	1.10 ± 0.05^{bc}	1.08 ± 0.06^{ab}	1.09 ± 0.11	1.12 ± 0.05
	0.32	1.12 ± 0.07	1.13 ± 0.04^{abc}	1.10 ± 0.02^{bc}	1.13 ± 0.03^{ab}	1.03 ± 0.05	1.07 ± 0.08
	0.48	1.04 ± 0.06	0.96 ± 0.12^{c}	1.06 ± 0.00^{c}	1.04 ± 0.02^{b}	1.08 ± 0.06	1.06 ± 0.02
Daily length increase (DLI)	0.00	0.16 ± 0.03	0.09 ± 0.02	0.07 ± 0.02	0.06 ± 0.01	0.06 ± 0.01	0.06 ± 0.01^{a}
	0.19	0.14 ± 0.08	0.08 ± 0.04	0.06 ± 0.02	0.05 ± 0.02	0.06 ± 0.02	0.06 ± 0.01^{ab}
	0.24	0.08 ± 0.12	0.06 ± 0.06	0.04 ± 0.04	0.03 ± 0.03	0.04 ± 0.02	0.04 ± 0.01^{ab}
	0.32	0.07 ± 0.10	0.04 ± 0.05	0.04 ± 0.02	0.04 ± 0.02	0.05 ± 0.01	0.05 ± 0.01^{ab}
	0.48	0.06 ± 0.05	0.06 ± 0.05	0.03 ± 0.03	0.03 ± 0.02	0.03 ± 0.02	0.04 ± 0.01^{b}

Means (±SD) with different superscript letters across rows are significantly different (P<0.05), R = Recovery

Histopathology alterations in gill and liver of juvenile *Heteroclarias* from sub-lethal potassium cyanide exposure

Tissues of gill and liver of *Heteroclarias* were observed and present in Figure 2 and 3. Several parts of gill and liver tissue were damaged after exposure to different concentration of KCN. For example, multiple foci of erosion of the secondary lamellae was observed in gill tissue by exposure to 0.48 mg·L^{-1} of KCN (Figure 2D). A few foci of vacuolar degeneration of hepatocytes and fatty infiltration was found in the hepatic parenchyma of specimen exposed to 0.48 mg·L^{-1} of KCN (Figure 3B).

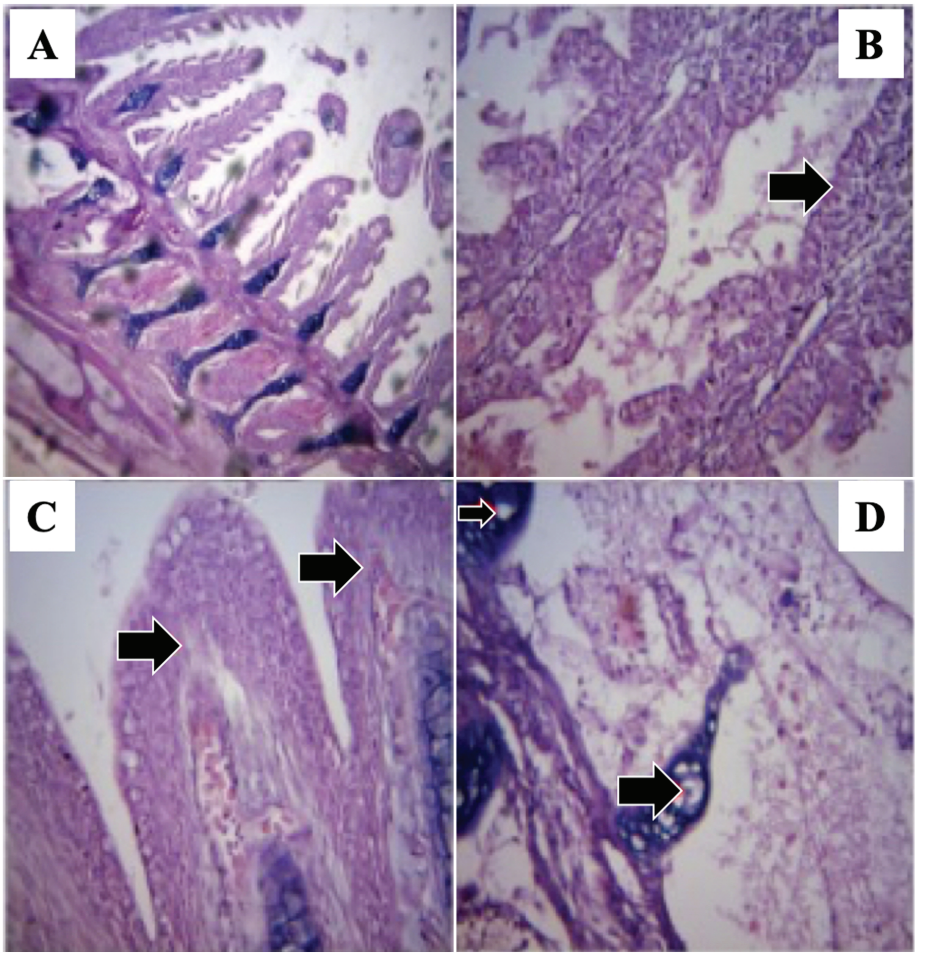

Figure 2. Gill tissue of *Heteroclarias*. (A) Normal gill of the control specimen with no visible lesions. (B) Gill of specimen exposed to 0.32 mg·L^{-1} of KCN with mild congestion of the submucosa area (arrow). (C) Gill of specimen exposed to 0.48 mg·L^{-1} of KCN with moderate to severe stunting of the secondary lamellae (arrows). (D) Multiple foci of erosion of the secondary lamellae (arrows) observed in specimen exposed to 0.48 mg·L^{-1} of KCN.

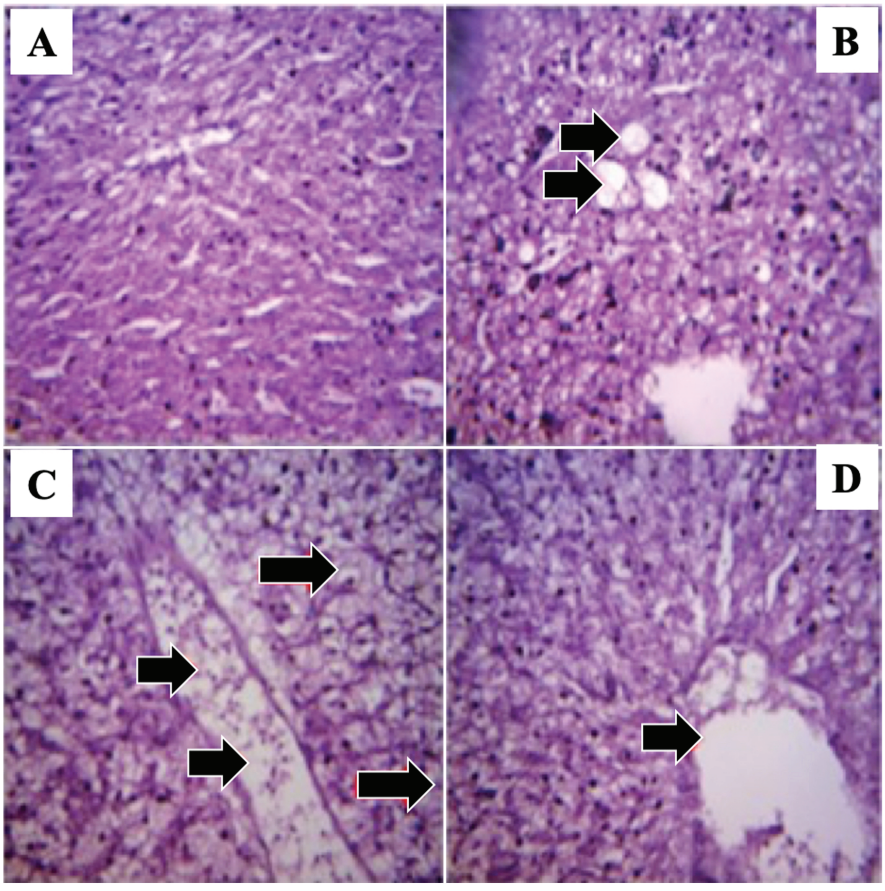

Figure 3. Liver tissue of *Heteroclarias*. (A) Normal liver tissue with no visible lesions. (B) Few foci of vacuolar degeneration of hepatocytes and fatty infiltration (arrows) present in the hepatic parenchyma of specimen exposed to 0.48 $mg{\cdot}L^{-1}$ of KCN. (C) Moderate congestion of the central vein and portal area (small arrows), with diffuse vacuolation of hepatocytes (large arrows) observed in specimens in 0.24 and 0.32 $mg{\cdot}L^{-1}$ concentrations over longer exposure. (D) Moderate diffuse vacuolation of the hepatocytes (arrow) after recovery period in specimen exposed to 0.48 $mg{\cdot}L^{-1}$ of KCN.

DISCUSSION

Behavioural responses

There were marked changes in the behaviour of test specimens in the lethal concentrations. Observed changes included irregular swimming activity, hyper-activity, rapid jerk-like movements, restlessness, loss of orientation, and ultimately death. Test specimens tried to jump out of the test solution, and most of the specimens aligned vertically with their heads at the surface of the solution. Similar erratic behaviours were reported by Oseni (2015) for *Clarias gariepinus* exposed to KCN. Al-Ghanim and Mahboob (2012) expressed that the behavioural changes in fish on exposure to toxicant may be due to the inhibition of the brain's cytochrome c oxidase activity induced by cytotoxic hypoxia from cyanide exposure. This results in changes in electrical activity of the specimens, and causing damage to the region of the brain associated with maintenance of equilibrium. Mortality of test specimens was observed to increase, with increasing lethal concentration of KCN.

Growth parameters

There was reduction in relative growth rate (RGR) and specific growth rate (SGR) of the test specimens with increase in exposure to potassium cyanide, when compared to the control group. This could be attributed to the suppressive effect on food consumption or increased activity of the fish in an attempt to avoid polluted water, or due to reduced oxygen-carrying capacity via erythropoiesis of the blood, resulting from the reduction in cellular haemoglobin. Similar observations on growth were reported by Abdulkareem and Owolabi (2014) on the sub-lethal exposure of *Heteroclarias* to monocrotophos (pesticide), and by Dixon and Leduc (1981) on effects of cyanide on juvenile rainbow trout (*Oncorhynchus mykiss*). Reduction in growth parameters improved in the post-exposure period, but were significantly different ($P<0.05$) from the control. Condition factor of greater than one ($K\geq1$) across the treatments showed the well-being (good condition) of the specimens during exposure and recovery periods. The cyanide-detoxifying enzyme (cytochrome oxidase) could have resulted in high tolerance of test specimens to KCN. The enzyme helps to inhibit reduction in metabolic rate or enhance anaerobic metabolism in species of teleosts (Eisler and Wiemeyer, 2004). However, there was a decreasing trend in condition factor with increase in exposure concentration, and Jin *et al*. (2015) acknowledged that reduced feeding rate or health problems may induce decreasing *K* values in fish. Daily length increase (DLI) did not vary significantly throughout the exposure and post-exposure periods, implying that the exposure to KCN did not induce significant negative effect on length increase.

Survival rate

Survival rate of test specimens was 100% during the exposure and recovery periods. Reports on different cyanide toxicities by Okonji *et al*. (2011), Al-Ghanim and Mahboob (2012), and Ramzy (2014) also did not indicate mortality of test fish to sub-lethal exposures.

Histopathology

The gills of the exposed fish showed lesions in the higher concentrations, and these were also observed with increased period of exposure. Severe sub-mucosal congestion of the gills observed was reversible after 21 days of post-exposure. The hepatocytes were more affected than the gills, with the presence of fatty infiltrations, diffused vacuoles and severe hepatic necrosis in the exposed concentrations. Velmurugan *et al*. (2009) reported that this could be because the liver is the primary organ for metabolism, detoxification and excretion of harmful substances, after observing similar results in *C. gariepinus* exposed to cypermethrin. The regulating mechanisms of the liver can be overwhelmed by elevated concentrations of contaminants, and subsequently result in structural damage. The variation in cyanide's effects on the liver and gills was also reported by Eisler and Wiemeyer (2004); that sub-lethal exposure of fish to cyanide (HCN) was sufficient to induce extensive necrosis in the liver, but without damage to the gill tissue.

CONCLUSION

Potassium cyanide (KCN)-induced effects were observed in the behavioural responses, growth rates (RGR and SGR), and tissues (gill and liver) of juvenile *Heteroclarias*. Sub-lethal concentrations of toxicants such as potassium cyanide in aquatic environment may not ultimately cause mortality, but may induce severe physiological dysfunctions such as reduction in growth, anaemia, and several lesions in fish.

LITERATURE CITED

Abdulkareem, S.I. and O.D. Owolabi. 2014. Toxicity of sub-lethal concentrations of Monocrotophos (MCP) on the haematological, biochemical and growth

responses of hybrid catfish, *Heteroclarias* and contaminated-*Heteroclarias* fed rats. I**nternational Journal of Current Microbiology and Applied Sciences** 3(6): 917–931.

Afia, O.E. and C.O. Ofor. 2006. Haematological indices of the *Clarias gariepinus* X *Heterobranchus longifilis* (hybrid catfish - *Heteroclarias*) reared at different feeding levels. **Nigerian Journal of Agriculture, Food and Environment** 12(3): 6–11.

Akinsiku, O.T., F.K. Agboola, A. Kuku and A. Afolayan. 2010. Physicochemical and kinetic characteristics of rhodanese from the liver of African catfish *Clarias gariepinus* Burchell in Asejire Lake. **Fish Physiology and Biochemistry** 36(3): 573–86.

Al-Ghanim, K.A. and S. Mahboob. 2012. Effect of sodium cyanide on the activities of some oxidative enzymes and metabolites in *Clarias gariepinus*. **African Journal of Biotechnology** 11(41): 9849–9854.

Authman, M.M.N., W.T. Abbas, I.M.K. Abumourad and A.M. Kenawy. 2013. Effects of illegal cyanide fishing on vitellogenin in the freshwater African catfish, *Clarias gariepinus* (Burchell, 1822). **Ecotoxicology and Environmental Safety** 91: 61–70.

Centers for Disease Control and Prevention (CDC). 2015. **Cyanide.** Atlanta: Office of Public Health Preparedness and Response (OPHPR). p. 2.

Dixon, D.G. and G. Leduc. 1981. Chronic cyanide poisoning of rainbow trout and its effects on growth, respiration, and liver histopathology. **Arch. Environm. Contam. Toxicology** 10: 117–131.

Dzombak, D.A., R.S. Ghosh and G.M. Wong-Chong. 2005. **Cyanide in water and soil: chemistry, risk, and management.** Florida: CRC Press. pp. 22–23.

Eisler, R. and S.N. Wiemeyer. 2004. Cyanide hazards to plants and animals from gold mining and related water issues. **Reviews of environmental contamination and toxicology** 183: 21–54.

European Centre for Ecotoxicology and Toxicology of Chemicals (ECETOC). 2007. Cyanides of hydrogen, sodium and potassium, and acetone cyanohydrin. **ECETOC Joint Assessment of Commodity Chemicals (JACC) programme.** 1(53): 39–185.

Finney, D. J. 1952. Probit Analysis (2nd ed). **Journal of the Institute of Actuaries** 78(3): 388–390.

Glaser, M., A. Breckwoldt, R. Deswandi, I. Radjawali, W. Baitoningsih and S.C.A. Ferse. 2015. Of exploited reefs and fishers - a holistic view on participatory coastal and marine management in an Indonesian Archipelago. **Ocean and Coastal Management** 116: 193–213.

Gurbuza, F., H. Ciftci and A. Akcilb. 2009. Biodegradation of cyanide containing effluents by *Scenedesmus obliquus*. **Journal of Hazardous Materials** 162: 74–79.

Jin, S., X. Yan, H. Zhang and W. Fan. 2015. Weight–length relationships and Fulton's condition factors of skipjack tuna (*Katsuwonus pelamis*) in the western and central Pacific Ocean. **PeerJ** 3: e758.

Kori-Siakpere, O., J.E.G. Ake and U.M. Avworo. 2006. Sublethal effects of cadmium on some selected haematological parameters of *Heteroclarias* (A hybrid of *Heterobranchus bidorsalis* and *Clarias gariepinus*). **International Journal of Zoological Research** 2(1): 77–83.

Lugert, V., G. Thaller, J. Tetens, C. Schulz and J. Krieter. 2014. A review on fish growth calculation: multiple functions in fish production and their specific application. **Reviews in Aquaculture** 8: 30–42.

Mak, K.K.W., H. Yanase and R. Renneberg. 2005. Cyanide fishing and cyanide detection in coral reef fish using chemical tests and biosensors. **Biosensors and Bioelectronics** 20: 2581–2593.

Oboh, G., A.A. Akindahunsi, and A.A. Oshodi. 2003. Dynamics of phytate-Zn balance of fungi fermented cassava products (flour and garri). **Plants Foods for Human Nutrition** 58: 1–7.

Okonji, R.E., H.A. Adewole, A. Kuku and F. K. Agboola. 2011. Physicochemical properties of Mudskipper (*Periophthalmus Barbarus* Pallas) liver rhodanese. **Australian Journal of Basic and Applied Sciences** 5(8): 507–514.

Organization for Economic Co-Operation and Development (OECD). 2014. **OECD guideline for testing of chemicals: Fish, acute toxicity test.** Paris: OECD. pp. 1–6.

Oseni, K. 2015. Acute and sub lethal effect of potassium cyanide on the behaviour and ATPase enzyme activity in the freshwater fish, *Clarias gariepinus* (Catfish). **International Letters of Natural Sciences** 49: 50 –57.

Panase, P. and K. Mengumphan. 2015. Growth performance, length-weight relationship and condition factor of backcross and reciprocal hybrid catfish reared in net cages. **International Journal of Zoological Research** 11: 57–64.

Pandey, G. and P. Govind. 2013. Toxicity of cyanide in fishes: an overview. **Universal Journal of Pharmacy** 2(2): 23–26.

Ramzy, E. M. 2014. Toxicity and stability of sodium cyanide in fresh water fish Nile tilapia. **Water Science** 28: 42–50.

Rejeki, S., D. Desrina, and R. A. Mulyana. 2006. **Chronic effects of detergent surfactant (linear alkylbenzene sulfonate / LAS) on the growth and survival rate of sea bass (*Lates calcalifer* Bloch) larvae.** Aquaculture Study Program, Fisheries Department, Faculty of Fisheries and Marine Science, Diponegoro University, Semarang, Indonesia. p. 4.

Sadati, F., D. Shahsavani and H. Baghshani. 2013. Biochemical alterations induced by sublethal cyanide exposure in common carp (*Cyprinus carpio*). **Journal of Biodiversity and Environmental Sciences** 7(20): 65–69.

Solomon, S.G., V.T. Okomoda and L. Ochai. 2013. Growth responses of pure bred *Heterobranchus bidorsalis*, *Clarias gariepinus* and their intergeneric crosses fed commercial diet. **Banat's Journal of Biotechnology** IV(8): 71–72.

Surleva, A., R. Gradinaru. and G. Drochioiu. 2012. Cyanide poisoning: from physiology to forensic analytical chemistry. **International Journal of Criminal Investigation** 2(2): 79–101.

Velmurugan, B., T. Mathews and E.I. Cengiz. 2009. Histopathological effects of cypermethrin on gill, liver and kidney of fresh water fish *Clarias gariepinus* (Burchell, 1822), and recovery after exposure. **Environmental Technology** 30(13): 1453.

Yisa, M. and S.O. Olufeagba. 2005. An exposition on field identification of clariid catfishes as an important tool in fish breeding and genetics. In: Araoye, P. A. (ed.). **Proc. of the 19th Annual conference held at Ilorin on 29th Nov-3rd December 2004.** pp. 185–192.