Digital Innovations & Contemporary Research in SCIENCE, ENGINEERING & TECHNOLOGY

Vol. 7. No. 4, December, 2019

Article Citation Format
Olaifa, Flora. E. & Ewutanure, S.J. (2019): Acute Nickel Toxicity and Effects on Liver and Kidney of Clarias gariepinus Juvenile
Journal of Digital Innovations & Contemp Res. In Sc., Eng & Tech. Vol. 7, No. 4 Pp 99-108

Article Progress Time Stamps
Article Type: Research Article
Manuscript Received: 24th October, 2019
Review Type: Blind Final
Acceptance: 17th December, 2019
Article DOI: dx.doi.org/10.22624/AIMS/DIGITAL/V7N4P9

Acute Nickel Toxicity and Effects on Liver and Kidney of *Clarias gariepinus* Juvenile

[1]Olaifa, Flora. E. & [2]Ewutanure, S.J.
[1]Department of Aquaculture and Fisheries Management, University of Ibadan, Ibadan, Nigeria.
[2]Department of Fisheries & Aquaculture Management, Nigeria Maritime University, Okerenkoko, Nigeria.
E-mail: ewutanure@gmail.com; floraolaifa@yahoo.com;
Phone: +2348101634482; +2348035509342

ABSTRACT

A study was carried out to evaluate the impact of nickel toxicity on selected physiological parameters, liver and kidney of Clarias gariepinus juvenile in an acute bio – assay for 7 days. The experiment was set up in a completely randomised design comparising six treatments and two replicates each. The concentrations were: 10 mg.L-1 – T1WFa; 7.5 mg.L-1 – T2WFa; 5.6 mg.L-1 – T3WFa; 3.2 mg.L-1 – T4WFa; 1.8 mg.L-1 – T5WFa and 0 mg.L-1 – T0WFa (control) for the test solution were prepared according to standard procedures. The volume of the test solution used per replicate for the experiment was 20 litres of water containing nickel (toxicant). Blood samples were collected according to standard methods. Parameters analysed for were Packed cell volume (PCV, %), Red blood cells (RBC, 106mm-3), Platelets (g.L-1), Aspartate transaminase (AST, IU-1), Alanine transaminase ALT (IU-1) and Creatinine (mg.dL-1). Data were analysed by using descriptive statistics and ANOVA at α0.05. Significantly highest and least PCV were 18.97±0.50 and 4.09±0.20 in T0WFa and T1WFa, while RBC ranged from 6.02±0.55 to 15.17±0.30, Platelets (3.51±0.05, 4.50±0.50) in T1WFa and T0WFa, respectively. Mean AST ranged from 1.17±0.75 to 7.57±0.16, ALT (3.43±0.20, 9.80±0.18) and Creatinine (1.60±0.11, 2.70±0.31), respectively. The patterns of haematological and serum biochemical indices indicated that Clarias gariepinus was under stress during the experimental period as a result of exposure to acute nickel toxicity.

Keywords: Nickel toxicity, Haematology, Serum biochemistry, Toxicant.

1. INTRODUCTION

Pollution of the aquatic environment and the bioaccumulation of heavy metals by aquatic fauna are major global causes of death (Olaifa and Ewutanure, 2018). Heavy metals have the ability to alter the normal activities of aquatic fauna through the interference with their neurological functions and metabolic processes (Olaifa and Ewutanure, 2019). Heavy metals could enter into the body of fish through ingestion and skin absorption thereby inducing impairment and dysfunction in the blood, liver, kidneys, heart, intestine, skin, endocrine glands, nervous systems, enzymatic and reproductive pathways (Olowu et al., 2012).

Nickel is an abundant element in the earth crust but found majorly mixed with oxygen or sulphides (FAO/SIDA, 1983). It forms alloys with copper, iron, zinc, copper and chromium. These alloys are used in making coins, jewelry, heat exchanger and valves in manufacturing industries. Nickel is used in making stainless steel, batteries, electroplating and serves as a catalyst in speeding up the rate of chemical reactions. It could be released into the environment through oil spillages, nickel mining and factories that process nickel into alloys (Ololade and Oginni, 2010). Major sources of nickel in aquatic organisms are linked with the exposure to oil spillages, industrial wastes and testing of nuclear devices (Olaifa and Ewutanure, 2017). Symptoms of nickel toxicity in fish are itching, redness and erratic swimming behaviour. Prolonged exposure of fish to nickel toxicity could cause damage to the lungs, liver, kidney and heart (Korisiakpere and Obogu, 2008).

Nickel has the ability to destroy a developing embryo of fishes and its consumption along with food substances had been reported to cause stomach, blood, liver, kidneys and lungs diseases in Oreochromis niloticus (Korisiakpere et al., 2006). Nickel has been shown to cause kidney and liver damages in C. gariepinus (Olaifa and Ewutanure, 2018). Nickel and its compounds have high acute and chronic toxicity on flora and fauna (Olaifa and Ewutanure, 2019). Acute toxic effects may include the death of animals, birds, fish or poor growth rate in plants (Olaifa and Ewutanure, 2018; 2019). Water hardness affects nickel toxicity to aquatic organisms (Gupta, 2001). Nickel concentration in fish tissues could be higher than the average concentration of nickel and its compounds in the water from which the fish was taken. Reports had it that, nickel induced various physiological and histopathological alterations in the organs of different fish species (Olaifa and Ewutanure, 2018; Gupta et al., 2006).

The increasing concern on the assessment and bio – monitoring of the aquatic ecosystems has brought about the importance of the application of appropriate biological indices in in assessing pollution stress (Odiete, 1999). Fish diseases and histopathology with a very wide range of causes are being used as indicators of environmental stress because they provide a definite biological end – point of historical exposure (Stentiford et al., 2003). This study aims at investigating the effects of acute nickel toxicity on liver and kidney of Clarias gariepinus juveniles in a static bio – assay for 7 days. The selected physiological parameters were packed cell volume (PCV), Haemoglobin (Hb), Red blood cells (RBC), White blood cells (WBC), Platelets, mean cell volume (MCV), while serum indices were Aspartate Transaminase (AST), Alanintransaminase (ALT), Sodium ion (Na+), Potassium ion (K+), Creatinine, Total protein (TP), Albumin (Alb) and Globulin (Glo), while organs were liver and kidney.

2. MATERIALS AND METHODS

Acute Bio – Assay (7 days LC50)

An acute static bio – assay comprising six treatments with two replicates each was set up. The concentrations (10 mg/L, 7.5 mg/L, 5.6 mg/L, 3.2 mg/L, 1.8 mg/L and T0WFa (control) for the test solution were prepared according to the procedure described by Reish and Oshida, (1987). The volume of the test solution used per replicate for the experiment was 20 litre of water containing nickel (toxicant).

Acclimatisation of Test Fish

Initial acclimatization of the test fish was done for two weeks before introducing them into the experimental aquaria tanks. Feeding was done two times daily (morning and evening) to satiation during the acclimatization periods, while stressed and dead fish were removed.

Handling of Collected Blood Samples of Experimental Fish

Blood samples for haematology (PCV, Hb, RBC, WBC, Platelets, MCV) were collected, preserved and analysed as described by Culling, (1974); Blaxhall and Daisey, (1973).

Blood samples for blood biochemistry were collected and preserved following Dacie and Lewis, (1991) method, while blood biochemical indices (AST, ALT, Na+, K+, Creatinine, TP, Albumin and Globulin) were determined by using commercial kits (Randox Laboratory Ltd., United Kingdom). Heavy metals were determined by using Atomic Absorption Spectrophotometer (AOAC, 1990).

Results of Histopathology on Liver and Kidney Samples of C. gariepinus juvenile

Histopathological examination of the juvenile of C. gariepinus liver and kidney was as described by Drury et al., (1967); Alaa et al., (2010). The photomicrograph of the sections were taken by using electrical microscope (Model: B – 350 – Optika, Italy) and compared with the aid of atlas of fish histology.

Statistical Analysis

Data collected were subjected to descriptive statistics and ANOVA at α0.05 by using Statistical Package for Social Sciences (SPSS) version 20.0.

Results and Discussion

It has been reported that nickel has the ability to destroy a developing embryo of fishes (Korisiakpere et al., 2006). Nickel has been shown to cause kidney and liver damages in C. gariepinus (Olaifa and Ewutanure, 2018). There were significant ($P > 0.05$) increases in the concentration of Ni present in water and in fish compared with control (Table 1). The PCV and MCV reduced significantly ($p < 0.05$), while the concentrations of WBC, Hb and Platelet were significantly ($p > 0.05$) elevated in comparison with control (Table 2). The AST, ALT, Creatinine and total protein were significantly ($p > 0.05$) elevated in nickel – exposed C. gariepinu, while the concentrations of Na+, K+, Albumin and Globulin diminished significantly ($p < 0.05$) in C. gariepinus exposed to sublethal concentration of nickel in comparison with control data (Table 3). Histopathological results showed mild to severe diffused vacuolar degeneration and congestion in blood sinusoids in the liver and kidney of the experimental C. gariepinus exposed to nickel toxicity compared with control (Plates 1 and 2).

Table 1: Means of nickel concentration in water and C. gariepinus

	Samples	
Treatments	Water (mg/L)	Fish (mg/Kg)
T0WFa	< 0.001	< 0.001
T1WFb	0.77±0.10^{a}	0.45±0.01^{a}
T2WFc	0.61±0.02^{b}	0.28±0.73^{b}
T3WFd	0.49±25^{bc}	0.19±0.02^{c}
T4WFe	0.35±0.03^{c}	0.11±9^{d}
T5WFf	0.15±0.01^{d}	0.09±0.01^{e}

Note: Means with the same super script are not significantly different from each other at p<0.05 level of significance.

Table 2: Mean haematological parameters of C. gariepinus exposed to nickel in water

	Treatments					
Parameters	T0WFa	T1WFa	T2WFa	T3WFa	T4WFa	T5WFa
PCV (%)	18.97±0.50^{e}	4.09±0.20^{a}	10.50±0.23^{c}	9.55±0.01^{d}	11.80±0.12^{b}	9.20±0.21^{d}
Hb (g.dL^{-1})	3.49±0.12^{c}	4.40±0.10^{a}	3.61±0.05^{d}	3.52±0.50^{d}	4.00±0.50^{b}	3.55±0.53^{d}
RBC (10^{6}mm^{-3})	6.02±0.55^{b}	15.17±0.30^{a}	5.90±0.30^{b}	6.15±0.31^{b}	6.70±0.07^{b}	6.02±1.13^{b}
WBC (10^{3}mm^{-3})	2.95±0.35^{c}	15.50±0.10^{a}	3.80±0.10^{b}	7.65±0.21^{b}	5.70±0.42^{b}	7.94±0.11^{b}
Platelets (g.L^{-1}	3.51±0.05^{b}	4.50±0.50^{a}	3.51±0.20^{b}	3.49±0.70^{b}	4.50±0.35^{a}	4.23±0.55^{b}
MCV (Fl)	30.15±0.31^{c}	6.62±0.30^{b}	17.80±0.50^{d}	15.53±0.50^{e}	7.52±0.17^{a}	15.28±0.50^{c}

Note: Means with the same super script along rows are not significantly different from each other at p<0.05 level of significance. PCV= Pack cell volume; Hb= Haemoglobin; RBC= Red blood cell; WBC= White blood cell and MCV= Mean cell volume. T0WFa = Control, T1WFa = Treatment 1, T2WFa = Treatment 2, T3WFa = Treatment 3, T4WFa = Treatment 4 and T5WFa = Treatment 5.

Table 3: Means of serum biochemistry in C. gariepinus juvenile

	Treatments					
Parameters	T0WFa	T1WFb	T2WFc	T3WFd	T4WFe	T5WFf
AST(IU.L^{-1})	1.17±0.75^{c}	7.57±0.16^{a}	1.41±0.31^{b}	1.57±0.09^{bc}	2.73±0.61^{b}	3.7±1.27^{b}
ALT (IU.L^{-1})	3.43±0.2^{c}	9.80±0.18^{a}	5.50±0.16^{b}	3.95±0.19^{c}	4.97±0.48^{b}	4.46±1.71^{b}
K^{+} (meq.L^{-1})	4.90±0.4^{ab}	6.09±0.41^{a}	3.04±0.25^{b}	3.71±0.90^{b}	3.70±0.16^{ab}	2.57±0.26^{b}
Na^{+} (meq.L^{-1})	11.65±0.01^{a}	7.57±0.13^{a}	4.39±0.17^{c}	4.40±0.16^{c}	5.59±0.58^{c}	3.89±0.16^{c}
Cre (mg.dL^{-1})	1.60±0.11^{b}	2.70±0.31^{a}	1.07±0.21^{b}	1.19±0.1^{ab}	1.90±0.40^{b}	1.75±0.5^{ab}
Tp (g.dL^{-1})	0.99±0.10^{a}	1.15±0.03^{b}	1.03±0.6^{bc}	1.40±0.40^{b}	0.16±0.1^{c}	1.05±0.1^{b}
Alb (g.dL^{-1})	1.78±0.4^{a}	1.01±0.02^{a}	0.75±0.2^{b}	0.53±0.4^{b}	0.69±0.4^{b}	0.91±0.6^{a}
Glo (g.dL^{-1})	1.40±0.13^{a}	1.28±0.11^{b}	1.14±0.3^{b}	1.55±0.3^{b}	1.27±0.42^{b}	1.11±0.05^{c}
Alb:Glob	1.27±0.02^{a}	0.79±0.57^{b}	0.66±0.05^{b}	0.34±0.01^{c}	0.54±0.32^{b}	0.82±0.43^{b}

Note: Means with the same super script along rows are not significantly different from each other at p<0.05 level of significance. AST = Aspartate transaminase, ALT = Alanine aminotransferase, K^{+} = Potassium ion, Na^{+} = Sodium ion, Cre = Creatinnine, Tp = Total protein, Alb = Albumin, Glob = Globulin. T0WFa = Control, T1WFa = Treatment 1, T2WFa = Treatment 2, T3WFa = Treatment 3, T4WFa = Treatment 4 and T5WFa = Treatment 5.

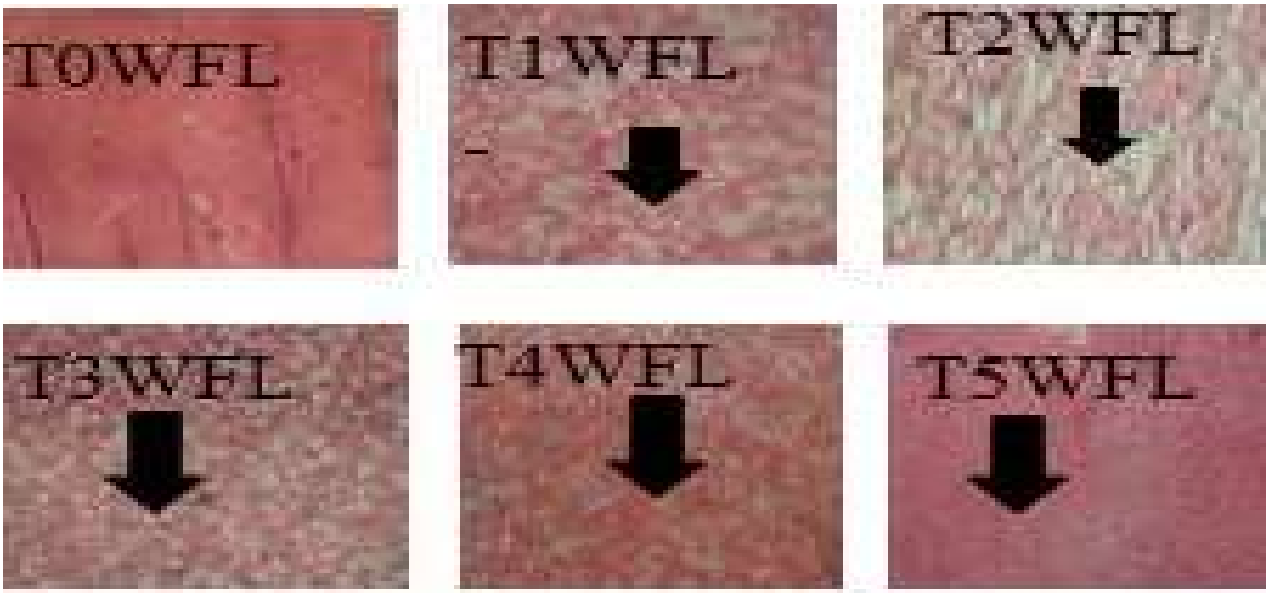

Plate 1: T0WFL (Control) to T5WFL liver samples of Clarias gariepinus subjected to nickel toxicity. Control (T0WFL) with normal condition of the fish liver (X400). T1WFL to T4WFL showed severe diffused vacuolar degeneration in liver of the fish (X400), while T5WFL showed mild vacuolar degeneration in liver of the fish (X400). Note: T0WFL = Control Treatment for liver; T1WFL = Treatment 1 for liver; T2WFL = Treatment 2 for liver; T3WFL = Treatment 3 for liver; T4WFL = Treatment 4 for liver and T5WFL = Treatment 5 for liver.

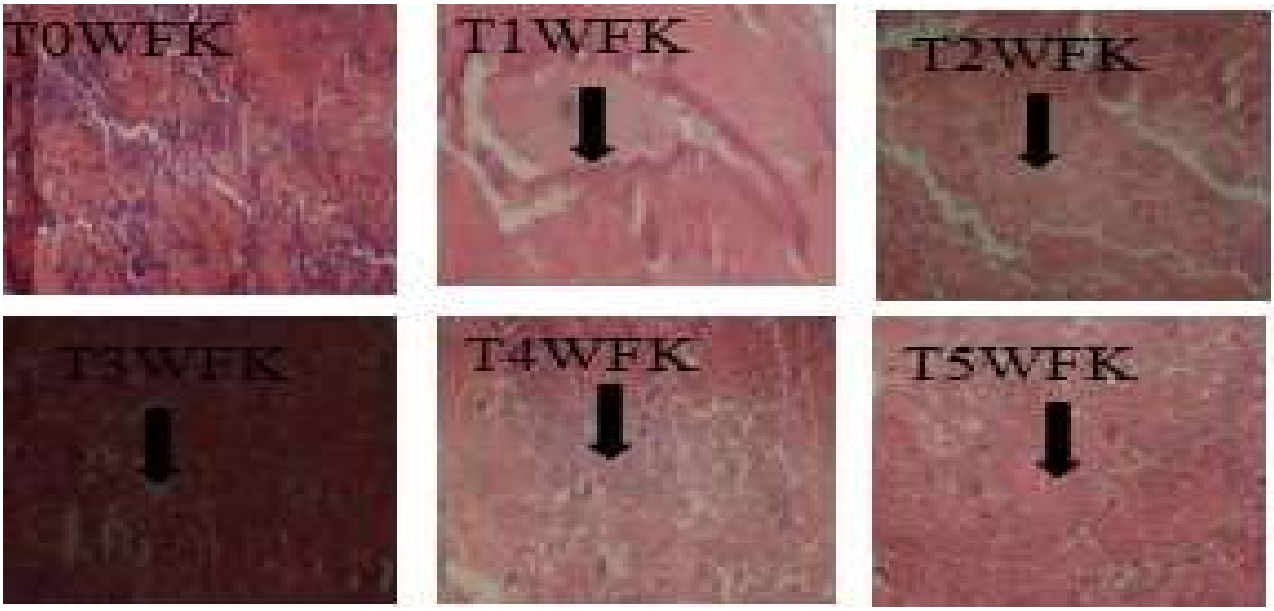

Plate 2: T0WFK (Control) to T5WFK of kidney samples of Clarias gariepinus subjected to nickel toxicity. Control (T0WFK) with normal condition of the fish kidney (X400). T1WFK and T2WFK showed severe vacuolar degeneration in the renal tubule, congestion in blood sinusoids (x400), while T3WFK to T5WFK showed mild necrotic degeneration in the renal tubules (x400). Note: T0WFK = Control Treatment for kidney; T1WFK = Treatment 1 for kidney; T2WFK = Treatment 2 for kidney; T3WFK = Treatment 3 for kidney; T4WFK = Treatment 4 for kidney and T5WFK = Treatment 5 for kidney.

Heavy metals could be responsible for mortality, delay in hatching and deformities in fishes (Olaifa and Ewutanure, 2018). Heavy metals have been reported to cause spinal deformities in fish, while deformities in fishes could be used as biomarkers of environmental contamination (Gupta and Sriavastava, 2006). The heavy metals are considered most toxic to humans, fishes and environment and possess the ability to destabilize ecosystems because of their bioaccumulation and harmful effects in aquatic biota (Gupta, 2001). In comparison with control, the sharp decrease in PCV and fluctuation in Hb could be associated with the excess accumulation of Nickel, while Nickel has been shown to cause kidney and liver damages in C. gariepinus (Olaifa and Ewutanure, 2018). Higher concentration of RBC (polycythemia vera) could lead to abnormal blood clot. A similar result was also obtained by Brix et al. (2006). A rise in the concentration of WBC indicated its ability to fight infection due to Ni contamination. The findings of the work agreed with Olaifa and Ewutanure, (2018). Mean cell volume is a value that explains the mean size of red blood cells in a blood sample. It helps in diagnosing different types of anemic conditions (Lichtnfels et al., 2006). Lower MCV than the control, indicates that red blood cells produced are too few as a result of Ni toxicity (Brix, 2004). This condition is known as microcytic anemia which may be caused by iron deficiency due to poor feed intake by the fish during the experimental period. Sigel and Sigel, (2007) reported that when a fish is subjected to stress, its blood composition could be altered as a result of decrease in appetite.

Exposure of C. gariepinus to heavy metals could cause various degrees of ion regulatory disruption, while plasma ion levels can be used to quantify toxic effects of metals intoxication (Olaifa and Ewutanure, 2019). Gupta and Srivastava, (2006) reported that during an exposure of fish to water contaminated with heavy metals, the active uptake of ions from the water may be initially altered, resulting in ionic homeostasis disturbances in fishes. Olowu et al. (2012) reported that the exposure of Oreochromis niloticus to copper, cadmium and mercury led to a significant reduction in blood sodium and chloride concentrations. Osmotic water influx in freshwater fish and rapid decrease in sodium and chloride are controlled by the excretion of large volumes of dilute urine and active uptake to replace ions lost by the gills. Plasma values of C. gariepinus decreased with varying concentration of Nickel. Previous reports showed significant increase in the levels of blood glucose in fishes treated with nickel, lead, copper, cadmium and mercury (Olaifa and Ewutanure, 2018; Al-Attar, 2005). Generally, heavy metals pollution has been reported to stimulate plasma, corticosteroid, glucose concentrations and inter renal activity in fishes (Olaifa and Ewutanure, 2018). Excessive secretion of cortisol and adrenalin hormones are considered major stress responses.

In comparison with control, the observed hyperproteinaemia and hyperalbuminaemia in the C. gariepinus at the end of the experiment could be possibly due to liver damage. This result supports Korisiakpere and Ubogu, (2008) who stated that an increase in the level of muscle glycogenolysis due to blood hyperlacticaemia and highly stressed conditions could lead to histopathological changes in the liver and kidneyof C. gariepinus. The accumulation of nickel sulphate in the liver could result into a loss of normal structural architecture, fatty changes, extensive vacuolization in hepatocytes and Kupffer cell hypertrophy (Das et al., 2006). Elevated total protein level in fishes is directly related to the level of the toxicant it is being subjected to (Gupta et al., 2006). Exposure of animals to various environmental stressors could cause pancreatic damage, hyperamylasemia and hyperlipasemia (Cosen – Binker et al., 2006).

Albumin is a protein produced in liver which prevents fluid from leaking from bloodstream into other tissues (Olowu et al., 2012), while a decrease in its concentration is an indication of liver or kidneys problem (Olaifa and Ewutanure, 2019). High level of globulin could indicate the presence of infection, inflammatory disease or immune disorders, while an abnormal result could be due to dehydration as a result of stress (Korisiakpere and Ubogu, 2008). A low albumin/globulin ratio could be associated with liver and kidney diseases (Olowu et al., 2012). It has been reported that a decrease in sodium concentration could indicate a reduction in sodium influx rate (Hoang et al., 2004). The Na+/K+-ATPase is a major crux of ion movement across cellular membranes.

It regulates ion levels in the body of marine and estuarine teleost. It has been reported that Na+/K+-ATPase is a useful indicator of pollution stress in aquatic organisms (Kim and Kang, 2004). Creatinine is a by -product of creatine phosphate from muscle and protein metabolism (Cengiz, 2006). Olaifa and Ewutanure, (2019) reported that damage to the kidney will lead to the retention of creatinine in the blood and thereby making it difficult for its excretion in the urine of fish. The ALT and AST are the most sensitive tests for diagnosis of liver diseases, while the degree of hepatic damage is assessed by increased serum level of cytoplasmic enzymes, ALT and AST (Ololade and Oginni, 2010). Olaifa and Ewutanure, (2018) observed several histological alterations in the liver and kidney of C. gariepinus under a 21 – day chronic exposure of the fish to nickel toxicity. Thealterations were focal necrosis and altered bile ducts. The present study showed a high level increase in ALT and AST after 7 days of exposure to nickel, indicating a visible hepatocellular damage. Similar observations and suggestions were stated by many experimental investigations on animals treated with heavy metals (Kim and Kang, 2004; Al-Attar, 2005).

The present study also revealed that the liver and kidney of C. gariepinus juvenile exposed to nickel toxicity during the 7 days presented a higher occurrence of histopathological lesions in the liver and kidney. Similar results were observed in Oreochromis niloticus, Colsia fasciatus; in Oncorhynchus mykiss subjected to nickel (Pane et al., 2004); in Cyprinus carpio and Prochilodus scrofa exposed to copper (Mazon et al., 2002); in mercury – administrated to freshwater Chana punctatus (Gupta and Dua, 2002; De Boeck et al., 2001). Histopathology is a veritable tool in fish pathology, physiology and aquatic toxicology because it provides changes which could indicate essential clues in an investigation of the toxic effects of heavy metals (Olaifa et al., 2018). In constant contact with heavy metals, the liver and kidney are very sensitive primary target for a variety of pollutants (Stentiford et al. 2003). The liver being the major site of detoxification and blood supply in fish, any physiological changes in it could results in distortion in osmotic and ionic concentration (Thophon et al., 2003).

Degeneration in the epithelial cells of renal tubule and reduction in the tubular lumen has been shown in kidney of fishes exposed to deltamethrin (Cengiz, 2006). Fish found in polluted aquatic environments are very prone to negative impact of pollutants capable of damaging liver and kidney structures (Olaifa et al., 2004). Heavy metals are readily detoxified absorbed and excreted through the liver and kidney (Lichtenfels et al., 2006). Accumulation of pollutants in high concentration could result in negative changes in cellular structures (Pane et al., 2004). Hampel et al. (2008) reported tubular retraction in the kidney of Scophthalmus maximus L. exposed to the sodium dodecyl sulphate. The liver is organ most associated with the detoxification and biotransformation process as a result of its position, blood supply and function. It is also one of the organs most affected by aquatic pollutants (Martenez et al. 2004). Changes in the liver could be an essential bio – marker that indicate stress due to exposure to environmental stressors (Monteiro, 2005). Congestion and dilation of sinusoid, hepatocyte and vacuolar degeneration were observed in C. gariepinus subjected to Ni toxicity (Ransatavatron et al. 2004).

Previous studies have shown that alterations in number, size and shape of the hepatocyte nucleus can be due to contaminants (Naeemi, et al., 2013; Hampel et al., 2008). Vacuolar degeneration, shrinkage of hepatocytes, congestion of sinusoid and slight atrophy were reported in liver of fishes after chronic exposure to a toxicant by Hampel et al. (2008) and Rejeki et al. (2008). Congestion is a blood circulation disturbance due to the increased volume of the blood in the blood capillary. In fisheries, vacuolar degeneration is an acute swelling of the fish organs (Naeemi, et al., 2013). In this study, the extent of the liver and kidney damages in C. gariepinus were dependent on the concentrations of Ni across all treatments. In comparison with control (T0WFL – liver; T0WFK – kidney), histopathological changes (T1WFL, T2WFL, T3WFL; T1WFK, T2WFK) showed severe damages than T4WFL; T3WFK, T4WFK, while T5WFL and T5WFK had the least distortion. Therefore, the higher level of Ni resulted into more and severe damage compared with the lower ones. The tissue alteration of the fish subjected to Ni could be as a result of accumulation of the toxicant.

From the current investigation, it could be concluded that nickel induced severe toxic effects on the blood, serum biochemical compositions, liver and kidney functions of C. gariepinus juvenile. It can also be stated that these physiological and histopathological changes observed in C. gariepinus revealed that the fish is a good species to be used as a biological indicator of aquatic pollution.

REFERENCES

1. Alaa, G. M. Osman, Abd – El – Baset, M. Abd El Reheem, Khalid Y. AbuelFadl, Ali G. GadEl-Rab, 2010. Enzymatic and histopathologic biomarkers as indicators of aquatic pollution in fishes. Journal of Environmental Health, 2.11: 1302-131. http://dx.doi.org/10.4236/ns.2010.211158
2. Al-Attar, A.M., 2005 The physiological response of the fish, Cyprinion mhalensis to mercury intoxication. J. Egypt. Ger. Soc. Zool., 51: 123-137.
3. Association of Official Analytical Chemists (AOAC), 1990. Journal of Official Method of Analyses 8 (1): 551 – 573.
4. Blaxhall, P. C. and Daisley, K. W. 1973. Routine haematological methods used in fish blood. Journal of Fish Biology, 5: 77.
5. Brix, K.V., J. Keithly, D.K. DeForest and J. Laughlin, 2004. Acute and chronic toxicity of nickel to rainbow trout (Oncorhynchus mykiss). Environmental Toxicology and Chemical, 23: 2221-2228.
6. Cengiz, E.I., 2006. Gill and kidney histopathology in the freshwater fish Cyprinus carpio after acute exposure to deltamethrin. Environmental Toxicology and Pharmacology, 22, 200–204.
7. Culling, C.F.A. 1974. Handbook of histopathological and histochemical techniques. Butterworth and company publisher, Britain. 1 – 197.
8. Dacie, J.V. and Lewis, S.M. 1991. Practical haematology. 6th Edition, New York: Churchill. 22.
9. Drury, R.A.M., Wallington, E.A. and Roy, C. 1967. Carleton histological techniques, Oxford University Press. 10 – 150.
10. FAO/SIDA 1983. Manual of Methods in Aquatic Environmental Research, part 9. Analyses of metals and organochlorines in fish. FAO fisheries Technical Paper, 212. Federal Environmental Protection Agency !991). Guidelines and Standards for Environmetal Pollution Control in Nigeria pp 1- 238.
11. Gupta, A.D., A.M. Patil, J.G. Ambekar, S.N. Das, S.A. Dhundasi and K.K. Das, 2006. L-ascorbic acid protects the antioxidant defense system in nickel-exposed albino rat lung tissue. J. Basic Clin. Physiol. Pharmacol., 17: 87-100.
12. Gupta, N. and A. Dua, 2002. Mercury induced architectural alterations in the gill surface of a fresh water fish, Channa punctatus. Journal of Environmental Biology, 23: 383-386.
13. Gupta, P. and N. Srivastava, 2006. Effects of sublethal concentrations of zinc on histological changes and bioaccumulation of zinc by kidney of fish Channa punctatus (Bloch). Journal of Environmental Biology 27: 211-215.
14. Gupta, P. K. 2001. Methods in environmental analysis: Water, Soil and Air. AGROBIOS (India) Publisher. Pp. 1- 408.
15. Hampel, M., Ortiz-Delgado, J.B., Sarasquete, C. and Blasco, J., 2008. Effects of sediment sorbed linear alkylbenzene sulphonate on juveniles of the Senegal sole, Solea senegalensis: toxicity and histological indicators. Histology and histopathology, 23, 87-100.
16. Health, A.G., 1987. Water Pollution and Fish Physiology. 1st Edn., CRC Press, Boca Raton, FL, USA, pp: 51-58.
17. Hoang, T.C., J.R. Tomasso and S.J. Klaine, 2004. Influence of water quality and age on nickel toxicity to fathead minnows (Pimephales promelas). Environmental Toxicology and Chemistry, 23: 86-92.

18. Kim, S.G. and J.C. Kang, 2004. Effect of dietary copper exposure on accumulation, growth and hematological parameters of the juvenile rockfish, Sebastes schlegeli. Mar. Environ. Res., 58: 65-82.
19. Kori – Siakpere, O. and Ubogu, E. O., 2008: Sublethal haematological effects of zinc on the freshwater fish, Heteroclarias sp. (Osteichthyes: Clariidae). African Journal of Biotechnology. 7(12): 2068 – 2073. www.academicjournals.org/AJB.
20. Kori – Siakpere, O.; Ake J.E.G.; Avworo, U.M. 2006. Sub-lethal effects of some selected haematological parameters of Heteroclarias (A hybrid of Heterobranchus bidorsalis and Clarias gariepinus). International Journal Zoological Resources, 2: 77-83.
21. Lichtenfels, A.J.F.C., G. Lorenzi-Filho, E.T. Guimaraes, M. Macchione and P.H.N. Saldiva, 2006. Effects of water pollution on the gill apparatus of fish. J. Comp. Pathol., 115: 47-60.
22. Martinez, C.B.R., M.Y. Nagae, C.T.B.V. Zaia and D.A.M. Zaia, 2004. Acute morphological and physiological effects of lead in the neotropical fish Prochilodus lineatus. Braz. J. Biol., 64: 797-807.
23. Mazon, A.F., E.A.S. Monteiro, G.H.D. Pinheiro and M.N. Fernandes, 2002. Hematological and physiological changes induced by short-term exposure to copper in the freshwater fish, Prochilodus scrofa. Braz. J. Biol., 62: 621-631.
24. Monteiro, S.M., J.M. Mancera, A. Fontinhas-Fernandes and M. Sousa, 2005. Copper induced alterations of biochemical parameters in the gill and plasma of Oreochromis niloticus. Compa. Biochem. Physiol. Part C: Toxicol. Pharmacol., 141: 375-383.
25. Naeemi, A.; Jamili, S.; Shabanipour, N.; Mashinchian, A. and Shariati Feizabadi S. R: 2013: Histopathological changes of gill, liver and kidney in Caspian kutum exposed to Linear Alkylbenzene Sulfonate. Iranian Journal of Fisheries Sciences, 12(4): 887 – 897.
26. Odiete, W.O. (1999). Environmental Physiology of Animals and Pollution. First Edition. Diversified Resources Limited. Lagos.
27. Olaifa, F.E. and Ewutanure, S.J. 2018. Haematological Response of Clarias gariepinus (Burchell, 1822) juveniles to Nickel toxicity. In: Emerging Issues in Sustainable Forest Management: Experiences and Lessons for Nigeria. Proceedings of the 40th Annual Conference of the Forestry Association of Nigeria held in Lagos, Lagos State on 12-16th March, 2018. Editors: Akinwole, A.O., Adekunle , V.A.J.,Ogunsanwo, O.Y. pp 807-817.
28. Olaifa, F.E., Olaifa, A.K., Adelaja, A.A., Owolabi, A.G. 2004. Heavy metal contamination of Clarias gariepinus from a lake and fish farm in Ibadan, Nigeria. African journal of biomedical research, vol. 7 (2004); 145 - 148 ISSN 1119 – 5096 © Ibadan Biomedical Communications Group. Available online at http://www.bioline.org.br/md Full Length Research Article.
29. Olaifa, Flora. E & Ewutanure, Somorhire J. (2019): Phytoremediation potential of Pteridium aquilinum and the toxicity of Nickel on Clarias gariepinus juvenile. Advances in Multidisciplinary Research Journal. Vol. 5. No. 4, Pp 67–80. Available online at https://www.isteams.net/aimsjournal Article DOI: dx.doi.org/10.22624/AIMS/V5N4P7 1.
30. Ololade and Oginni, 2010. Toxic stress and haematological effects of Nickel on African catfish, Clarias gariepinus, fingerlings. Journal of Environmental Chemistry and Ecotoxicology, 2(2): 014 – 019.
31. Olowu, R.A., C.T. Onwordi, A.A. Denloye, M.O Osundiya, N.O. Adebayo, M.S. Owolabi, O.O. Tovide, B.A. Moronkola, O.A. Omoyeni, Ajuwon, O. R. 2012. Heavy Metals in Oreochromis niloticus (Linnaeus, 1758) (Persiformes: Cichlidae), Ictalurus punctanus (Rafinesque, 1818) (Suliriformes: Ictaluridae) and Bottom Sediments from Lagos Lagoon Proximal to Egbin Power Plant, Ijede, Ikorodu, Lagos Nigeria". Research Journal of Environment and Earth Sciences 4(3): 237 – 243.

32. Pane, E.F., A. Hague and C.M. Wood, 2004. Mechanistic analysis of acute, Ni-induced respiratory toxicity in the rainbow trout (Oncorhynchus mykiss): An exclusively branchial phenomenon. Aquat. Toxicol., 69: 11-24. Ptashynski, M.D., R.M. Pedlar, R.E. Evan, C.L. Baron and J.F. Klaverkamp, 2002. Toxicology of dietary nickel in lake whitefish (Coregonus clupeaformis). Aquat. Toxicol., 58: 229-247
33. Pane, E.F., C. Smith, J.C. McGeer and C.M. Wood, 2003. Mechanisms of acute and chronic waterborne nickel toxicity in the freshwater cladoceran, Daphnia magna. Environ. Sci. Technol., 37: 4382-4389.
34. Rangsayatron, N., P. Pokethitiyook, E.S. Upatham, G.R. Lanza and S. Singhakaew, 2004. Ultrastructural changes in various organs of the fish Puntius gonionotus fed cadmium-enriched cyanobacteria. Environ. Toxicol., 19: 585-593.
35. Reish, D. L. and Oshida, P. S. (1987). Manual of methods in Aquatic Environment Research, part10. Short term Static Bioassays. FAO. Fisheries Technical paper NO.247. Rome. Pp 1 - 62Pp.
36. Rejeki, S., Desrina, D. and Mulyana, A.R., (2008). Chronic affects of detergent surfactant (Linear Alkylbenzene Sulfonate / LAS) on the growth and survival rate of sea bass (Lates calcalifer Bloch), larvae Journal of Coastal Development.213P.
37. Roberts, R.J. and Rodger, H.D., 2002. The pathophysiology and systematic pathology of teleosts. in fish pathology (3eds.). Ronald J. Roberts. Harcourt Publishers Limited, London.
38. Sigel, H., and A. Sigel 2007. Metal ions in biological systems. Nickel and its role in biology. Marcel Dekker, New York. Environmental Pollution, 23: 400 - 488. 80 Vol. 5 No. 4, December, 201
39. Stentiford, G.D., M. Longshaw, B.P. Lyons, G. Jones, M. Green and S.W. Feist, 2003. Histopathological biomarkers in estuarine fish species for the assessment of biological effects of contaminants. Mar. Environ. Res., 55: 137-159.
40. Thophon, S., M. Kruatrachue, E.S. Upatham, P. Pokethitiyook, S. Sahaphong and S. Jaritkhuan, 2003. Histopathological alterations of white seabass, Lates calcarifer, in acute and subchronic cadmium exposure. Environ. Pollut., 121: 307-320.