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ACUTE TOXICITY OF INDUSTRIAL EFFLUENTS FROM AGBARA ENVIRONS OF OLOGE LAGOON ON EARLY LIFE STAGES OF AFRICAN CATFISH *Clarias gariepinus*.

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ABSTRACT
The acute toxic effect of industrial effluents from Agbara Industrial environs of Ologe lagoon was investigated in a static renewable lethal bioassay using fingerlings and Juveniles of African catfish (*Clarias gariepinus*). 20 fish was stocked per tank and each treatment was in triplicate. Physico-chemical parameters: temperature, pH, DO and conductivity in treatment tanks were monitored for 96hours. Behavioural responses were studied, mortality data recorded, and histopathological analysis was also done. Except for dissolved oxygen, other physicochemical parameters monitored did not show significant differences ($p>0.05$). The lethal bioassay showed that as the concentration of effluent increased, more mortality was recorded; but as the time of exposure increased, mortality reduced. The LC_{50} obtained at all the different time intervals for *C. gariepinus* fingerlings in 24, 48, 72 and 96hrs were: 69.45, 46.39, 40.81and 34.03(%). While in juveniles, the values were: 64.52, 49.21, 32.50, and 19.63(%), respectively. Abnormal behaviour was observed; they showed repeated darting movement within an hour of introduction, darkening in the eye and on the skin, haemorrhage in the gills, spiral swimming and death. Histopathological examination of the gills and liver of the fishes showed lesions which increased progressively with increasing level of toxicants. Observed changes in the gills were mainly: epithelial lifting, swollen lamellae, necrosis and mass degeneration, fatty and vacuolar degeneration, loss of lamellae and marked disorganization in gill structure and arrangement. In the liver, there were vacoulation, portal congestions, pancreatic necrosis, fatty degeneration and severe disruption of the hepatic cord. Fishes in the control treatments showed no visible lesions throughout the experiments. This information confirms that histopathological alterations are good biomarkers for toxic impact assessment of industrial effluent on fish. Therefore, an indiscriminate discharge of this effluent to surrounding water should be discouraged.
Keywords: Acute, toxicity, industrial effluent, and Ologe lagoon.

INTRODUCTION

fish living in rivers/lagoons receiving high discharges of effluent from industries, a range of alterations related to physiological abnormalities have been observed (Vethaak *et al.*, 2002). These effects have been attributed to various estrogenic chemicals known to be present within treated or/and untreated industrial effluents. Indeed, extensive laboratory-based studies have confirmed that chemicals contained in industrial effluents can induce many effects seen in effluent exposed fish (Seki *et al.*, 2002). Furthermore, it has been shown that exposure to industrial effluents can inhibit the reproduction of fish (Thorpe *et al.*, 2007). Although, the concentrations of chemicals typically measured in Waste water, treated or un-treated effluents may be low, compared to those required to affect fish reproduction in short-term laboratory studies, there are still major concerns about long-term exposures to effluents. This is because a prolonged exposure to some of these chemicals increases their level of effect and in some cases; this are shown to have population-level consequences (Kidd et al., 2007). Additionally, industrial effluents contain mixtures of chemicals and it is now well established that they can interact in an additive manner to induce effects on the reproductive physiology of fish at lower concentrations than those required individually (Thorpe *et al.*, 2001, 2003; Brian *et al.*, 2007). Specific studies that directly assess the effects of industrial effluents on population-relevant end-points, such as fish seed production (fingerlings/juveniles), are therefore, required to understand the consequence of exposure; relative to their histopathology, physiology, growth and survival.

Ologe lagoon is used for fishery, waste disposal, sand mining and transportation. Fish farms are located along the banks of the Ojo axis of the Badagry creek which brings in marine water from the Lagos harbour and also drain the water from the lagoon. The lagoon is essentially a fishing point for the people of Lagos and Ogun states. As with the Lagos lagoon, it is also regarded as the 'large septic tank' in the region. The main anthropogenic pressure on the lagoon is from the adjacent Agbara industrial estate. According to Adeboyejo, *et al* (2011) over 20 factories belonging to the food and beverages, pharmaceutical, breweries, metal finishing industries, chemical and pulp and paper companies presently occupy the industrial area. The effluents of these industries are discharged in the lagoon all year round, with their immediate impacts on the ecology of the lagoon system. Ologe lagoon drains into the Atlantic Ocean through the Badagry creek and through the Lagos harbour. Ologe lagoon is of great importance not only to Lagos state and Nigeria, but to the entire West-African sub-region as ecological catastrophes occurring upstream, could have severe consequences downstream if unchecked. This study investigate: the acute toxicity of different concentrations of industrial effluent from Ologe Lagoon on the fingerlings and juveniles stage of *Clarias gariepinus*, evaluate the physical and chemical composition of the effluents and examine the effects of the effluents on the tissues of kidney, liver and gills using histological techniques.

MATERIALS AND METHODS

Study Area: Lagos, being the economic and industrial hub of Nigeria, is located in the south-western part of the country within the latitudes 6° 23′ N and 6° 41′ N and longitudes 2° 42′ E and 3° 42′ E on the west coast of Africa. The state is flanged from the north and east by Ogun State, in the south by Republic of Benin and the Atlantic Ocean/Gulf of Guinea (figure 1). The total landmass of the State is about 3,345km^2, which is just about 0.4% of the total land area of Nigeria. It is the physically smallest; but the most highly populated state in the country with an estimated population of about 10 million inhabitants which is about 10% of the total population of Nigeria, Africa's most populous country. Lagoons are ecologically and economically important aquatic ecosystems in South-western Nigeria. They provide natural food resources rich in protein which includes an array of fish and fisheries. They are also important in water transportation, energy generation, exploitation and exploration of some mineral resources including sand (Onyema *et al.,* 2003, 2007). Lagoons also inadvertently serve as sinks for the disposal of domestic, municipal and industrial wastes in the region. Ologe lagoon is one of the nine lagoons in South-western Nigeria. (Nwankwo, 2004). It is presumably the smallest of the lagoons with a surface area of 9.4km2. The climate is of the equatorial type with two peak rainy periods which are generally May-July and September -October. The water of Ologe lagoon at this period is very dilute with pH being neutral. This may be due to low tidal influence since the lagoon is more inwardly situated.

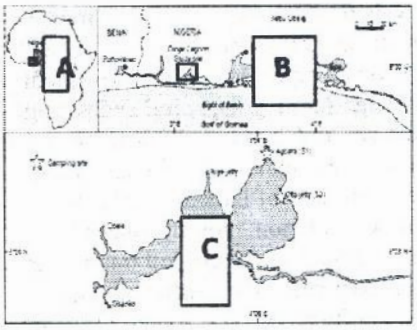

Figure 1: The administrative map of Africa inset Nigeria in [A], [B] shows the Lagos lagoon complex with the Atlantic Ocean, and [C] shows the map of Ologe lagoon as described.

Construction of Research Tanks: Thirty (30) rectangular glass tanks of 50 litres were constructed using glass of thickness 5mm and silicone. The glass tanks (50cm x 80cm) were filled with well aerated dilution water (pH 7.8 – 8.0) and placed in the laboratory. The tanks were transparent and evenly spaced for proper observation of specimen. White paper tapes with black markers were used to tag each of the tanks. Water aerators and net were provided in the tanks.

Test Organism: The test organism is fingerlings and juveniles of African Catfish, *Clarias gariepinus*. A total of 500 healthy specimens were randomly selected and purchased from a reputable fish pond. The fish were visibly free of any deformities, lesions or disease.

Feeding: The fish were fed with 56% protein Coppens feed. The composition and proximate analysis of the feed were carried out using Aitken *et al* (2001). Fixed feeding regime of 4% of the body weight per day was employed during the period of acclimatisation.

Experimental Design: The glass tanks were constructed [30tanks] in 40cm x 80cm x 40 cm tri-dimensional.The experimentation expresses the entire set-up: from the dilution water for each mixing vessels to the replicate exposure tank for each graded effluent treatment (0% [DWC], 20%, 40%, 60%, 80% and 100% effluent).

Range Finding Test: Twenty (20) specimens were randomly distributed into the appropriate tanks and exposed to five widely spaced toxicant concentrations and a control for 24 hours (Reish and Oshida, 1987). The concentration of the effluent in which some organisms survived after 24 hours to the lowest concentration that no organism survived were used for the actual test (Vincent-Akpu. 2001). Six dilutions of the toxicant were made in geometric progression in similar ratios. The concentrations were prepared in the following order: 0% (Control), 20%, 40%, 60%, 80%, 100%. Healthy fish were randomly selected and placed in appropriate sized tanks and covered with mosquito nets that were fastened with rubber band to prevent fish escape. One hour after the preparation of the test solutions, the fish were carefully placed into each replicate tank containing test solutions for the different test concentrations. The fish were starved during the bioassay. The range finding test lasted for 96hours and was observed initially at 3-4hours interval followed by 12hour intervals. The mortality and behaviour was used as the measure of toxicity. *Determination of Physical/Chemical Parameters of dilution water and test solutions:* Water quality parameters of the test tanks were monitored daily for the replicates using standard methods (APHA, 1998). The dilution water and the test solutions were tested for the following parameters: temperature, Conductivity, pH and dissolved oxygen (DO).

Histopathological examination: Six fish from each group were subjected to histological procedure, the abdominal cavities of the sacrificed fish were opened and organs (liver and gills) were removed. The organs were fixed in 10% buffered formalin, processed in automatic tissue processor, embedded in paraffin wax and sectioned at 5mm on a rotary microtome. Sections were stained with Haematoxylin and Eosin (Roberts, 2001).

Statistical Analysis: Data obtained were analyzed by a two-way ANOVA appropriate to each experiment and any statistical significance of difference between means was tested at 95% confidence level by the Student's t test. Differences were considered significant at $P < 0.05$ (Zar, 1996). According to FAO (2007 and 2009), mortality data were analysed using probit method to calculate 50% lethal concentration (LC_{50}), and median lethal time (LT_{50}).

RESULTS

Physicochemical parameters of Treated tanks. The summary of data on the physico-chemical parameters from Industrial effluents treated tanks with fingerlings and juveniles are presented in Tables 1 and 2. Minimum mean temperature was 28.13±0.31 for 20% effluent treated tank, and 29.13±0.25 was recorded for 100%. The Pearson's correlation matrix analysis showed that temperature had strong/positive inter-correlation with Dissolved oxygen (0.818, $p<0.05$) and Conductivity (0.61) at 0.05 significance, but showed no significant correlation with pH.

Table 1: Summary of physicochemical parameters of industrial Effluents treated tanks during Acute Toxicity test for *Clarias gariepinus* Fingerlings

Stages	Conc. (%)	Temp. (^{0}C)	DO (mg/l)	pH	Conductivity (μS/cm)
Fingerlings	0	28.5 ± 0.2	16.4 ± 0.36	7.6 ± 0.2	80.2 ± 0.1
	20	27.73 ± 0.31	6.27 ± 0.32	7.7 ± 0.2	80.2 ± 0.1
	40	28.37 ± 0.25	3.47 ± 0.31	7.6 ± 0.2	80.2 ± 0.1
	60	28.43 ± 0.25	3.40 ± 0.3	7.6 ± 0.1	80.2 ± 0.1
	80	28.27 ± 0.25	3.37 ± 0.25	7.8 ± 0.1	80.2 ± 0.1
	100	29.4 ± 0.3	3.07 ± 0.15	7.43 ± 0.06	80.2 ± 0.1

Table 2: Summary of physicochemical parameters of industrial Effluents treated tanks during Acute Toxicity test for *Clarias gariepinus* Juveniles

Stages	Conc. (%)	Temp. (0C)	DO (mg/l)	pH	Conductivity (μS/cm)
Juveniles	0	28.4 ± 0.2	6.2 ± 0.1	7.53 ± 0.25	80.2 ± 0.1
	20	28.13 ± 0.31	3.77 ± 0.15	7.63 ± 0.15	80.33 ± 0.21
	40	29.03 ± 0.21	3.6 ± 0.1	7.7 ± 0.2	80.17 ± 0.15
	60	28.73 ±0.25	3.4 ± 0.1	7.4 ± 0.1	80.43 ± 0.15
	80	28.43 ± 0.15	3.2 ± 0.15	7.4 ± 0.26	80.43 ± 0.12
	100	29.13 ± 0.25	2.3 ± 0.2	7.7 ± 0.1	80.6 ± 0.1

Table 3: Behavioral Responses of *Clarias gariepinus* to different percentage (%) concentration of Industrial Effluent from Agbara Environs of Ologe lagoon, Lagos.

Parameter	Fingerlings (%)						Juveniles (%)					
	0	20	40	60	80	100	0	20	40	60	80	100
Erratic Swimming	-	-	+	++	+++	+++	-	-	+	++	+++	+++
Loss of Reflexes	-	-	-	+	+++	+++	-	-	+	+	+++	+++
Discolouration	-	-	-	+	++	+++	-	-	+	+	++	+++
Change in Behaviour	-	-	+	+	++	+++	-	-	+	+	++	+++
Gill Movement	-	-	-	+	++	+++	-	-	+	+	++	+++
Frequent Surfacing	-	-	+	++	+++	+++	-	-	+	++	+++	+++
Lethargy	-	-	-	+	++	+++	-	-	+	++	++	+++

+ indicates an increase in responses; -indicates no response;

Mortality in treated tanks. The summary of results for the mean lethal concentrations (LC_s) and the 95% confidence limits of industrial effluent are shown in Table 4 .

Table 4: Mean Lethal Concentrations (LC_s) and 95% Confidence Limits of Industrial effluent on the on the different life stages of *Clarias gariepinus*

LC	Life stages	Time (hrs)			
		24hrs	48hrs	72hrs	96hrs
LC_1	Fingerlings	8.01 (1.15 – 15.86)	3.84 (0.25 – 9.55)	4.51 (0.5 – 9.95)	4.46 (0.71 – 9.42)
	Juveniles	17.67 (7.60 – 25.92)	2.97 (0.07 – 8.64)	3.81 (0.46 – 8.61)	0.74 (0.0 – 4.03)
LC_5	Fingerlings	15.09 (3.94 – 24.58)	7.97 (1.09 – 15.66)	8.60 (1.84 – 15.63)	8.09 (2.01 – 14.39)
	Juveniles	25.82 (14.04 – 34.38)	6.76 (0.45 – 14.79)	7.14 (1.44 – 13.39)	1.94 (0.0 – 7.21)
LC_{10}	Fingerlings	21.14 (7.53 – 31.25)	11.76 (2.38 – 20.47)	12.13 (3.43 – 19.96)	11.12 (3.48 – 18.1)
	Juveniles	31.61(19.40 – 40.12)	10.48(1.23 – 19.78)	9.98(2.63 – 16.99)	3.24(0.02 – 9.86)
LC_{15}	Fingerlings	26.54(11.60 – 36.94)	15.29(4.01 – 24.60)	15.30(5.22 – 23.60)	13.77(5.04 – 21.16)
	Juveniles	36.23(24.06 – 44.66)	14.09(2.39 – 24.17)	12.51(3.94 – 19.99)	4.57(0.04-12.20)
LC_{50}	Fingerlings	69.45(54.21 – 99.72)	46.39(31.81 – 61.9)	40.81(28.17 – 52.4)	34.03(22.70 – 43.4)
	Juveniles	64.52(54.46 – 77.09)	49.21(32.57 – 69.4)	32.50(20.55 – 42.1)	19.63(3.01 – 31.46)

Table 5: Mean Lethal times (LT_{50}) of industrial effluents on the different life stages of *Clarias gariepinus*.

Species	Stages	24hrs	48hrs	72hrs	96hrs
Clarias gariepinus	fingerlings	70.79	54.95	47.86	38.02
	juveniles	83.18	56.23	39.81	25.12

HISTOLOGICAL ANALYSIS On theGILLS OF Clarias gariepinus AFTER ACUTE EXPOSURE

Control: There was no visible or discernible changes from that of the control in the gill structure of *C. gariepinus* at the fingerlings and juveniles stages; on gross examination, the colour of the gills was red with no visible mucus or lesions. Microscopic examination of the gills of untreated *tanks* revealed normal parallel arrangement of the gill filament consisting of primary lamellae and other arrays of delicate secondary lamellae with epithelial cells intact.

Fingerlings: The gills of *C. gariepinus* at fingerlings stage exposed to different concentrations of industrial effluents (I.E) showed various pathological changes ranging from mild epithelial lifting, swollen lamellae to slight hyperplasia especially between concentrations of 20% and 40% I.E (plate). However, at higher concentrations, the impact/effect of I.E was more severe. There was loss of lamellae and mass degeneration at 60% treatment, necrosis and serious lamellae distortion at 80% and at 100%; there was hyperplasia and general fusion (plate).

Juveniles: Sections of gill filaments of *C. gariepinus* juveniles after 96hrs exposure to industrial effluent (x100) showed slight epithelial lifting of the primary lamellae (see plate 20%), also there was swollen lamellae and slight necrosis. Plate 2.0 C-gill exposed to 40%, shows that lamellae were degenerated and slightly swollen]. Plate 2.0 D-(gill exposed to 60% I.E) reveals that there was loss of lamellae and mass degeneration. And at 80% and 100% ; lamellae were seriously distorted and necrotic. Hyperplasia, massive lamellae necrosis and fussion were also consistent findings at this level.

Histological analysis of livers of Clarias gariepinus AFTER ACUTE EXPOSURE

Control: Microscopic examination of the controlled experiment for *C. gariepinus* showed normal liver appearance, normal hepatocytes morphology including minimal vacoulation, lipid and glycogen storage, and normal arrangement of hepatic cord (plate 3.0 & 4.0).The organs were intact and had no visible gross lesions,

Fingerlings: Present of irregularity in cell shape and size, at 20% treatment, there was infiltration of the hepatocytes and vacoulation. Portal congestion at 40% exposure, necrosis of the hepatic cells at 60%, widespread pancreatic degeneration and at 100%, there were cases of pancreatic necrosis, fatty degeneration and presence of melano-macrophages.

Juveniles: Photomicrograph histology of liver of *Clarias gariepinus* juveniles exposed to industrial effluents (I.E.) in 96hrs [x400] showed the following responses: Plate 20 B- At 20% I.E, there was infiltration of hepatocytes and vacoulation. C- At 40% treatment, there were portal congestions. D- At 60% exposure, necrosis of the hepatocytes and infiltration. E & F- At 80% & 100% exposure, there were widespread pancreatic necrosis, fatty degeneration and hepatocytes hydrophic vacoulation.

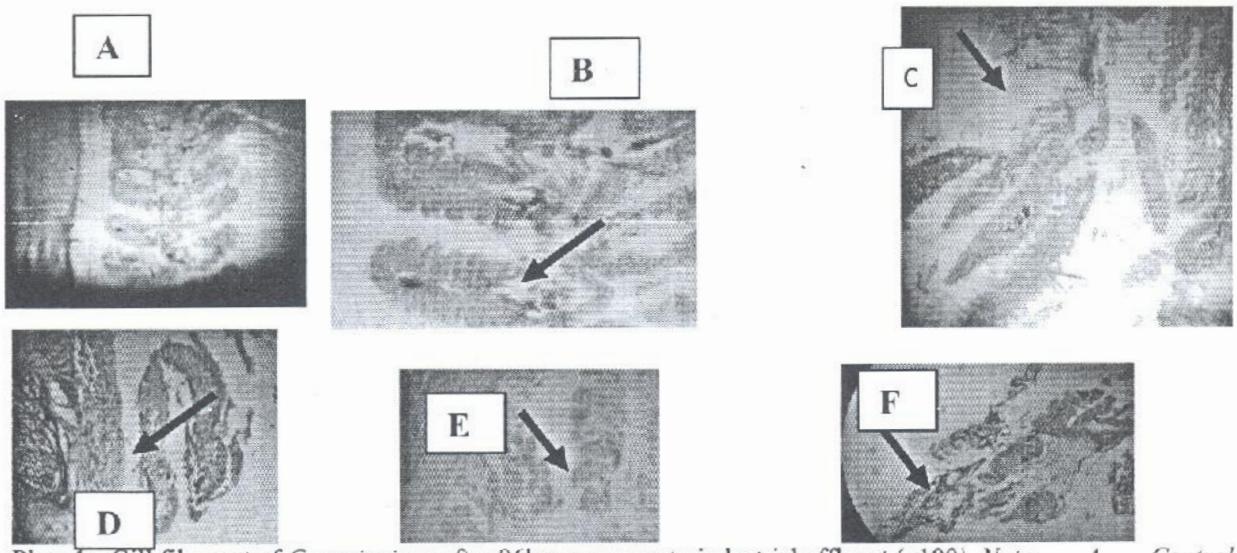

Plate 1: Gill filament of *C. gariepinus* after 96hrs exposure to industrial effluent (x100). *Note: A- Control [normal with no visible lesion], B-gill exposed to 20% I.E.[showing swollen lamellae and slight hyperplasia], C-gill exposed to 40% I.E [showing distruption of the filament arrangement and marked disorganization], D-gill exposed to 60% I.E [loss of lamellae and mass degeneration], E-exposure at 80% [lamellae seriously distorted and necrotic] and F-gill exposure to 100% I.E [hyperplasia, massive lamellae necrosis and fussion of lamellae]. (Arrow showing specific areas).*

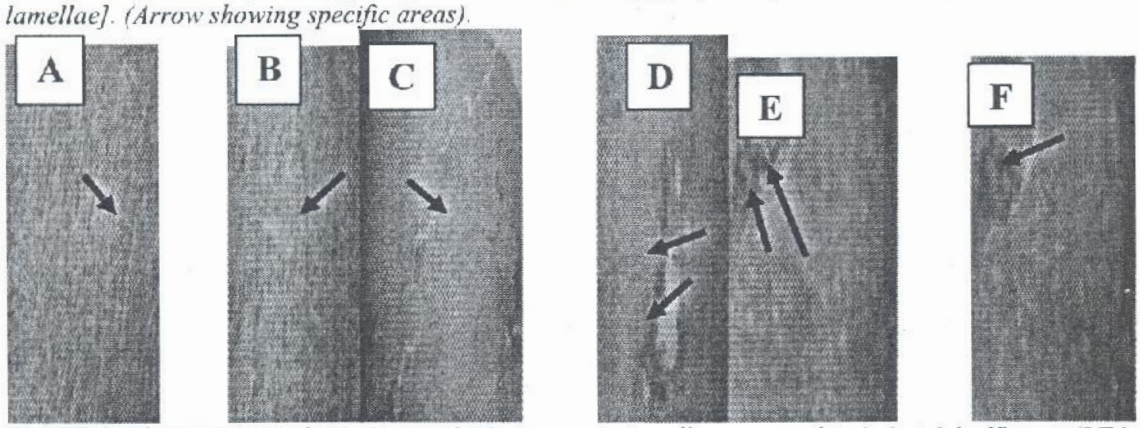

PLATE 2: Photomicrograph histology of *Clarias gariepinus* liver exposed to industrial effluents (I.E.) in 96hrs [x100]. *Note: A- The control showing normal liver cells. B- At 20% I.E, there was infiltration of hepatocytes and vacoulation. C- At 40% treatments, there were portal congestions. D- At 60% exposure, necrosis of the hepatocytes and infiltration. E & F- at 80% & 100% exposure, there were widespread pancreatic necrosis, fatty degeneration and hepatocytes hydrophic vacoulation (arrow).*

DISCUSSION

Metabolic rate and physiological processes are controlled by water temperature. As metabolic activities increase with an increase in temperature, fish demand for oxygen increases. Grand mean temperature (10.25±1.18) recorded in this study falls within the range of (6-9^{0}c) suggested by the Federal Ministry of Environment and the Federal Environmental Protection Agency (FEPA, 1999), that DO level of 3-4mg/l is required for fish growth. This is also supported by Agboola, *et al.* (2008) who reported the mean DO level of 4.81mg/l in Badagry creek, Nigeria. Comparatively, the DO level in Ologe lagoon is well above the minimum 5.0mg/l. However, Dissolved Oxygen in this study remained slightly stable throughout sampling period.

When the behaviours of the fish were examined, both the fingerlings and the juveniles showed similar reactions. The juveniles exposed to different concentrations of industrial effluents moved more rapidly, lost equilibrium in water and began to show sideways swimming. They made attempts to jump out of the tanks. The fingerlings, on the other hand, showed increase in their movements, hyperventilation and death with convulsions, spiral swimming and efforts to swallow air from the surface of water. The mouth and the gills of the dead fish were

gaping. The behaviour of intoxicated fish was similar to that described by other authors. Ogeleka *et al* (2009) observed that *C. gariepinus* fingerlings swim erratically prior to death. Samuel (2008) recorded coughing, hyperventilation followed by sporadic ventilation, twisting, loss of equilibrium, spiral swimming, convulsions and death following a short period in a coma-like state, in which there were no body motions except weak movements of the gills. At death, the mouth was usually gaping and the gills were widely extended. Samuel (2008) determined that increasing the unionized concentrations in water increased the oxygen consumption. Similar reactions were also observed for Siberian sturgeon (*Acipencer baeri*): increased ventilation, loss of equilibrium, swimming on the back and finally a very violent tendency followed by death. These actions subsided and the fishes became calm as the experiments progressed.

These reactions to the effluents/toxicants were most pronounced in tanks containing the highest concentration (100%). This conformed with the submission of Donalson and Dye (1975), who are of the opinion that, fish exposed to low concentration of toxicant do not reach the stage of exhaustion, rather they quickly become adapted to the stressor. The stressful and erratic behavior of the fish in this investigation gives a signal to respiratory impairment, and this may be as a results of the effect of the detergent effluent on the gills, this is in agreement with the opinion of Ayoola, (2008) and Ogundiran, *et al.*, (2009). At increased lethal concentrations, the behavioral responses of the test organisms greatly increased and the organisms later inactive and this is a normal situation in acute and sub-acute toxicity test (Kulakkattolickal and Kramer, 1997). Hyperactivities observed in this study are attributed probably due to the disturbances in the metabolic state resulting in the depletion of energy. It is possible that animals which have higher metabolic activities could require higher level of oxygen and thus would embark on higher respiratory activities (Canli and Kargin, 1995). Lethargies and loss of equilibrium observed in this study may be due to depletion of energy in the body of the exposed animals (Anderson *et al.*, 1988).

Histopathology is widely accepted as a useful method for the assessment of injury in fish to the adverse short term and chronic effect of industrial effluents. Several live lesions have been established as tissue bio-makers consistent with the exposure of fish to effluent. These include pigmented macrophage aggregation (Patino, *et al.* 2003., Fournie, J.W., 2001). Hepatocytes vacoulation (Stehr, *et al. 1998*), multi foci coagulative necrosis and liver fatty degeneration. These biomarker have been conclusively linked with certain factors: increase in age (Blazer, *et al. 2007)*, stress (Fournie, J.W., 2001) and degeneration relative to the level of exposure to effluents (Stehr, *et al. 1998*). The preponderance of these lesions in fish from contaminated waters bordering urban locations similar to our test location has been firmly established and described in details for Winter flounder *Pleuronectes americanus* (Augspurger, *et al. 1994.*, Blazer, *et al.* 2007., Murchelano, *et al. 1985)* and conclusively establish a direct relationship between pervasiveness and severity of this lesion, hepatic neoplasm and levels of site contamination. There is a consensus however, that vacuolated hepatocytes are frequently found proximal to neoplasm and that tumour is usually associated with increasing numbers of vacuolated liver cells. The extent of deformities and cell injuries is also consistent with hepatotoxicant actions (Augspurger, *et al. 1994)*.

Various pathological changes were observed in the experimental organisms at different exposure levels of the effluent. An evaluation of the clinical symptoms showed an increase in mucus secretion in the gills and on the body surface, haemorrhage in the gills and darkening in the eye and on the skin. Increase in mucus secretion in the gills and on the body surface also appeared as a symptom of gill necrosis and was observed by Salin and Williot (1991) on Siberian sturgeon. Darkening on the eye and on the skin was thought as a reaction of fish to the toxicant. Gill haemorrhage was also observed in acute toxicity study by Daud et al. (2008) on red tilapia and a sub-lethal toxicity study by Kucuk (1999) on blue tilapia (*Oreochromis aureus*). Gills were studied histologically and some lamella deformations were observed. Gill hyperplasia and lamella fusion were also reported. Similar results were also confirmed by Mitchell and Cech (1985), who studied channel catfish. This result was corroborated by the work of Adeyemo (2005) who observed similar symptoms.

The liver of the exposed organisms revealed slight vacuolated cells which is an indication of fatty degeneration of hepatocytes. Cellular necrosis as observed in this work probably resulted from excessive work required by the fish to get rid of the toxicants from its body during the process of detoxification. High accumulation of several components of the industrial effluent in the liver is a pointer to the fact that, liver plays a major role in the accumulation and detoxification. Fishes are known to possess sequestering agent (metallothionein), the bioaccumulation of these trace elements in the liver tissue reaches a proportion in which the function of the liver is impeded, thus resulting in a progressive degeneration of the liver cells synctial arrangement. Necrosis became evident as the concentration increases and this may be due to the inability of fishes to regenerate new liver cells. It was also observed that the histopathological changes in the liver caused metabolic problems; this is evident and more pronounced as observed in the edematous cells (which is an indication of bile stagnation in the liver of exposed fish). The histological alterations identified within the hepatocytes in this study may have been the results of various biochemical lesions. Anomalies such as irregular shaped central vein, cellular vacoulation and infiltration may be attributed to the accumulation of lipids and glycogen due to liver dysfunction as a result of exposure to the toxicants. Therefore, the histological changes observed in the liver of the *C. gariepinus* in the

present study indicate that the fish were responding to the direct and the additive effects of the contaminants as much as other effects such as stress. Such information confirms that histopathological alterations are good biomarkers for both field and laboratory assessment, particularly in tropical areas that are naturally subjected to a multiplicity of environmental variations or depletion due to chemical contamination.

Whole effluent toxicity tests were carried out to determine the actual impacts of effluents on organisms residing in receiving waters where the effluents were discharged. For both stages, fingerlings and juvenile, the LC_{50} became progressively smaller as the duration of exposure increased from 24hrs to 96hrs. The value of 96-h LC_{50} recorded in this study was close to the range of the 96-h LC_{50} reported by Chung-Min Lao *et al* (2003) for Tilapia [28.68mgL^{-1}; 95% CI: (24.92 – 32.44)] and Adewuyi *et al* (2010) for *C. gariepinus* [38.00mgL^{-1}; 95% CI]. Several studies have reported acute toxicity of industrial effluents on various other fish species. For instance, *O. niloticus* by Ikpi *et al* (2003) and *Sarotherodon spp* by Wong (1989). Thus this result indicated that *Clarias gariepinus* found in effluent-contaminated lagoons are sensitive to toxic substances, as was also observed by Adewuyi *et al.* (2010). The effluent was found to have a LC_{50} lower than 100%. This means that at full strength without dilution, the effluents are capable of causing mortality of 50% or more in the population of the experimental organisms. This study has established to some degree, the destructive and pathological potentials of industrial pollution on the Lagos lagoon complex vis a vis Ologe lagoon on the resident aquatic fauna and corroborate earlier work done by other researchers. It is therefore recommended that a toxicity investigation evaluation (TIE) which will result in identification of the compounds responsible for toxicity be carried out.

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