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TOXICOLOGICAL ASSESSMENT OF SOME INDUSTRIAL EFFLUENTS ON *CLARIAS GARIEPINUS*

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ABSTRACT

Industrial effluent discharges are a worrisome phenomenon due to their impact on aquatic life, with direct effects on humans who consume fish and other aquatic life. This study aimed at assessing the acute and chronic toxicity of discharged industrial effluents on fish. 400 fingerlings of the African catfish (*Clarias gariepinus*) were exposed to different effluents (in tanks) from factories that manufacture paint (A), household products (B), feed, food and oil (C), carbonated drinks (D) at concentrations of 25, 50, 75 and 100% volume-in-volume respectively. The effluents were analyzed for trace metal concentration and physicochemical parameters. Acute toxicity testing involved exposing 10 fish per tank for each effluent at the different concentrations for 96 hours. Acute (LC_{50}) and sub-acute toxicity tests were conducted by exposing a different set of fish to different effluent concentrations of each sample for 28 days. The surviving fish were sacrificed, the liver and kidney collected and processed for histopathological analysis. Lead, chromium and cadmium were detected in samples. Lethal bioassay showed 20% and 100% mortality occurring in effluent A and C after 96 hours and effluent B after 24 hours respectively (at 50, 75 and 100% effluent concentrations). Histopathological analysis showed loss of cellular architecture, with fat vacuoles, hepatocellular degeneration and necrosis observed. Effluents were found to cause toxicity in fish and are capable of causing serious health risks to both aquatic and human lives exposed to these toxicants.

KEYWORDS: Acute toxicity, *Clarias gariepinus*, Industrial effluents, Mortality.

INTRODUCTION

In many countries, including Nigeria, environmental pollution from residential and industrial wastewaters is currently of serious concern. Industrial effluent contamination of natural water bodies has emerged as a major challenge in developing and densely populated countries like Nigeria.[1]

Effluent discharged into the environment with enhanced concentration of nutrient, sediment and toxic substances may have a serious negative impact on the quality and life forms of the receiving water body.[2] Estuaries and inland water bodies, which are the major sources of drinking water in Nigeria, are often contaminated by the activities of the adjoining populations and industrial establishments.[3] Industrial wastewaters entering a water body represent a heavy source of environmental pollution in Nigerian rivers affecting both the water quality as well as the microbial and aquatic flora.[4]

Toxicity evaluation is an important and cost effective tool in wastewater quality monitoring as it provides the complete response of test organisms to all the compounds in a cumulative manner.[5] Toxicity evaluation is also useful in regulating toxic chemicals and determining the long and short-term impacts of discharge on the aquatic life of the receiving water body and ground water table.[6] Determining the safe concentration of wastewater to be discharged into aquatic water bodies is also an area where toxicity evaluation will be of importance.[7] This study analyzed effluents discharged from some industries in Jos Metropolis, Plateau State, Nigeria for toxicity and to compare the results with the WHO permissible standards for industrial waste discharge.

MATERIALS AND METHODS

Acute toxicity testing (LC_{50})

Effluent concentrations (100, 75, 50 and 25 % $^{V}/_{V}$) were prepared using the control (borehole water) as diluent in four 10-liter plastic tanks. Ten fish were weighed and placed in each of the four tanks, with another 10 fish in a separate tank containing borehole water as control for 96 hours. Fish were observed at 24, 48, 72 and 96 hours respectively. LC_{50} was determined by plotting the graph of percentage mortality against log concentration. Safe concentration was obtained from the lethal concentration values by calculation.[8] Acute Toxicity unit (TUa) was calculated using the following formula: $TUa = 100/LC_{50}$ % (v/v).

Sub-acute toxicity testing

Twenty fish were placed in different tanks containing effluents at concentrations lower than that used for acute toxicity testing and lower than the LC_{50} (up to 28.25, 89 and 44.7 % $^{V}/_{V}$ for effluents A, B and C respectively) were used for this test to study the effects on the fish for 28 days. The behavior and growth of the fish and appearance were observed and compared against fish grown in the control tank for the same duration. Mortality was expressed as a percentage.

Histological analysis

The fish were sacrificed after exposure to the effluents for 28 days by percussive stunning and dissected to remove the liver and kidney. These organs were then preserved in 10% buffered formalin and processed using a method described by.[9]

Statistical analysis

Data was subjected to Kruskal-Wallis test to find the association of growth rate and weight gain with effluent exposure and also to determine the association of mortality with effluent exposure.

Ethical Approval

Ethical approval to conduct this research was not required since fish was used.

RESULTS AND DISCUSSION

Determination of Effluents' Physicochemical Properties

pH of effluents A-D (paint manufacturing effluent; household products industry effluent; effluent from a food, feed and oil producing industry and effluent from a carbonated drinks bottling industry respectively) was 8.83, 10.46, 8.96 and 7.2 respectively (Table 1). Effluent temperature was 18 °C, 19 °C, 19.5 °C, 18.5 °C and 18.9 °C for effluents A-D respectively. A non-offensive odour was detected in effluents A, D and control; a highly offensive and less offensive odour detected in effluents B and C respectively (Table 1). TDS for effluents A-D and control was 202, 705, 581, 658, and 150 mg/l respectively while TSS for the same effluents and control was 150, 633, 419, 771 and 100 mg/l respectively.

Biological Oxygen Demand (BOD) was determined to be 144, 780, 610, 87 and 15.98 mg/l respectively for paint manufacturing effluent (A); household products industry effluent (B); effluent from a food, feed and oil producing industry (C) and effluent from a carbonated

drinks bottling industry (D) and control respectively; while Chemical Oxygen demand was 894, 998, 960, 127 and 150 mg/l respectively for the same effluents.

The following metals were detected in all the effluents: calcium, magnesium, iron, lead, arsenic, chromium and cadmium, as well as chloride, nitrate and nitrogen. Alkalinity of the effluents (A-D) and control was determined to be 400, 939, 604, 170 and 140 respectively.

The total viable count was $1.0x10^3$ for paint manufacturing effluent (A); household products industry effluent (B); effluent from a food, feed and oil producing industry (C) and effluent from a carbonated drinks bottling industry (D) respectively, while the coliform count was 3, 15, 5 and 3 for the effluents respectively. Yeast, mould and Salmonella were detected in all effluents and none in the control (Table 1).

Table 1: Physicochemical Properties of the different effluent samples.

PARAMETER	EFFLUENT SAMPLE					WHO
	A	B	C	D	Control	max std
pH	8.83	10.46	8.96	7.2	7.1	6.5-8.5
Temp (°C)	18.0	19.0	19.5	18.5	18.9	40.0
Colour	Whitish	Grey	Turbid	clear	Clear	Clear
Odour	-	++	+	-	-	Odourless
TDS (mg/L)	202	705	581	658	150	500
TSS (mg/L)	150	633	419	771	100	150
BOD(mg/L)	144	780	610	87	15.98	30
COD(mg/L)	894	998	960	127	150	250
Ca(mg/L)	1.030	1.175	1.056	1.110	0.075	200
Mg (mg/L)	0.022	0.081	0.035	0.109	ND	0.20
Cl^- (mg/L)	0.13	0.46	0.95	0.15	0.09	0.2-0.25
Na (mg/L)	1.265	6.226	4.051	1.121	0.05	200
Nitrate (mg/L)	45	78	61	16	2.44	20
Nitrogen (mg/L)	15	30	24	5	2	100
Fe (mg/L)	1.763	11.858	2.68	16.36	0.00	1
Pb (mg/L)	0.009	0.019	0.004	0.016	0.004	0.01
Ar (mg/L)	0.011	0.065	0.046	0.021	ND	0.01
Cr (mg/L)	0.003	0.022	0.001	0.003	ND	0.05

Cd(mg/L)	0.000	0.008	0.012	0.003	0.01	0.003
Alkalinity	400	930	604	170	140	100
Coliform count	3	15	5	3	0.03	0
Total viable count	$1.0x10^3$	$1.0x10^3$	$1.0x10^3$	$1.0x10^3$	-	-
Yeast and mould	present	present	present	present	nil	nil
Salmonella	present	present	present	present	nil	nil

Key: A- Effluent from a paint manufacturing industry; B- Effluent from a household products industry; C- Effluent from a food, feed and oil producing industry; D- Effluent from a carbonated drinks bottling industries; BOD- Biological Oxygen Demand; COD-Chemical Oxygen Demand.

- not offensive, + offensive, ++ very offensive

Effect of Different Effluent Concentrations on *Clarias gariepinus* Mortality

Percentage mortality was 0% (at concentration of 25% v/v) and 20% (at concentration of 50, 75, 100% v/v) 96 hours post-exposure for effluent from paint factory, with 10% mortality observed at 72 and 96 hours post-exposure (at 50 and 75% v/v), while for 100% v/v, 0% mortality and 10% mortality respectively was recorded at 24 and 96 hours post exposure making a total of 20% mortality at the end of the 96hours (table 2).

Mortality was highest at 25, 50, 75, 100% v/v concentration with 10 deaths each observed only at 24 hou0rs post-exposure to effluents from the factory producing household products (B) and food, feed and oil (C) respectively. Percentage mortality was 100% at these concentrations and 70% at the lowest concentration of 5% v/v for effluent B and 0% for effluent C at 10% v/v.

No mortality was recorded at 100% v/v up to 96 hours post-exposure to the effluent from the soft drink bottling factory (D).

Table 2: Effect of Different Effluent Concentrations on *Clarias gariepinus* Mortality.

Effluent	Conc (%) $^v/_v$	% Mortality at different times post exposure				Mortality at 96hrs
		24hrs	48hrs	72hrs	96hrs	
Control	100	0	0	0	0	0
A	25	0	0	0	0	0
	50	0	0	10	20	20
	75	0	0	10	20	20

	100	10	10	10	20	20
B	5	30	30	40	70	70
	10	20	50	60	100	100
	25	100	0	0	0	100
	50	100	100	100	100	100
	75	100	100	100	100	100
	100	100	100	100	100	100
C	10	0	0	0	0	0
	25	100	100	100	100	100
	50	100	100	100	100	100
	75	100	100	100	100	100
	100	100	100	100	100	100
D	100	0	0	0	0	0

n=10

Key: A- Effluent from a paint manufacturing industry; B- Effluent from a household products industry; C- Effluent from a food, feed and oil producing industry; D- Effluent from a carbonated drinks bottling industries.

4.3 Determination of Acute Toxicity of the Different Effluents

LC_{50} (Median Lethal concentration) for the effluent from the paint factory (A) at 24 and 48 hours post-exposure was 31622.78, which then decreased to 3548.13 and 354.80 at 72 and 96 hours post-exposure (Table 3). Acute toxicity unit (TUa) however increased from 0.003 at 24 post-exposure, to 0.028 72 hours after exposure and 0.282 after 96 hours. LC_{50} for the effluent from the factory producing household products (B) decreased from 25.12 after 24 hours to 10.00 at 48 hours post exposure and down to 1.12 at 96 hours post-exposure. An increase in TUa was observed from 3.981 after 24 hours, 10.00 after 48 hours and 89.286 at 96 hours post-exposure.

Table 3: Relative Acute Toxicity of the Different Effluents.

Effluent	Exposure Duration (hrs)	LC_{50} % $^{v}/_{v}$	Acute Toxicity Unit (TUa)
A	24	31622.78	0.003
	48	31622.78	0.003
	72	3548.13	0.028
	96	354.80	0.282
B	24	25.12	3.981
	48	10.00	10.000
	72	18.62	5.371
	96	1.12	89.286
C	24	22.39	4.466
	48	22.39	4.466
	72	22.39	4.466
	96	22.39	4.466

Key: A- Effluent from a paint manufacturing industry; B- Effluent from a household products industry; C- Effluent from a food, feed and oil producing industry; D- Effluent from a carbonated drinks bottling industries.

4.5 Effects of Effluents on Mortality 28 days post-exposure

Percentage mortality was 5% (at concentration of 25% v/v) and 25%, 10% and 90% (at concentration of 50, 75, 100% v/v) 28 days post-exposure for effluent from paint factory (Table 5). For effluent from the factory producing household products (B), % mortality after sub-acute toxicity test was 30, 40, 40 and 70% for 0.4, 0.6, 0.8, and 1.0% v/v respectively. An increase in percentage mortality with increase in concentration of the effluent was observed. Effluent from the food, feed and oil factory (C) recorded low mortality of 10, 0, 10 and 30% for 1.0, 2.5, 5.0 and 10.0% $^{v}/_{v}$ respectively. No mortality was recorded at 100% v/v up to 28 days post-exposure to the effluent from the soft drink bottling factory (D).

Table 5: Effect of Effluents on Mortality 28 days post-exposure.

Effluent	Concentration (% v/v)	% Mortality at 28 days
Control	100	0
A	25	5

	50 70 100	25 10 90
B	0.4 0.6 0.8 1.0	30 40 40 70
C	1.0 2.5 5.0 10.0	10 0 10 30
D	100	0

$P<0.05$

Key: A- Effluent from a paint manufacturing industry; B- Effluent from a household products industry; C- Effluent from a food, feed and oil producing industry; D- Effluent from a carbonated drinks bottling industries.

4.7 Histopathological Analysis of the liver and kidney after Sub-acute Toxicity Testing

A histopathological assessment of the liver and kidney of *Clarias gariepinus* was conducted, with the slides from the control group showing normal architecture, while fish exposed to the different concentrations of effluents however showed varying degrees of loss of cellular architecture and tissue damage (Plates 2 - 9).

The liver of fish in the control group showed a typical compact architecture with characteristic distribution and morphology of the hepatocytes. The test groups however showed varying degrees of loss of cellular architecture. Liver tissues of fish exposed to effluent A showed severe hepatocellular degeneration, necrosis and diffuse macrovesicular fatty change which occurred to different degrees depending on the the concentration of effluents they were exposed to (Plate 2). With effluent B there was severe macrovesicular fatty change, hepatocellular damage, cytoplasmic vacoulation (Plate 4). Effluent C showed liver tissues with moderate to severe macrovesicular fatty change, hepatocellular damage and sinusoidal congestion all to different degrees (Plate 6) and Effluent D showed severe diffuse macrovasicular fatty change (Plate 8)

The kidneys of fish in the control group showed no visible lesion, while those exposed to the paint, soap/detergent and feed, food and oil as well as soft drink effluents showed different degrees of renal tubular atrophy, necrosis and individualization (Plates 3, 5, 7, 9). Kidney

tissues of fish exposed to effluent A showed renal tubular atrophy, necrosis and individualization (Plate 3) seen with all concentrations of effluent A at varying degrees and stages of damage. Sample B showed random tubular degeneration with severe effects seen with higher concentrations (Plate 5). Sample C showed moderate to severe random tubular degeneration with lymphositic and histolytic infiltrates (Plate 7). With sample D, which was 100% $^{v}/_{v}$ of effluent from a Soft drink bottling company there was severe diffuse tubular degeneration with necrosis and individualization (Plate 9).

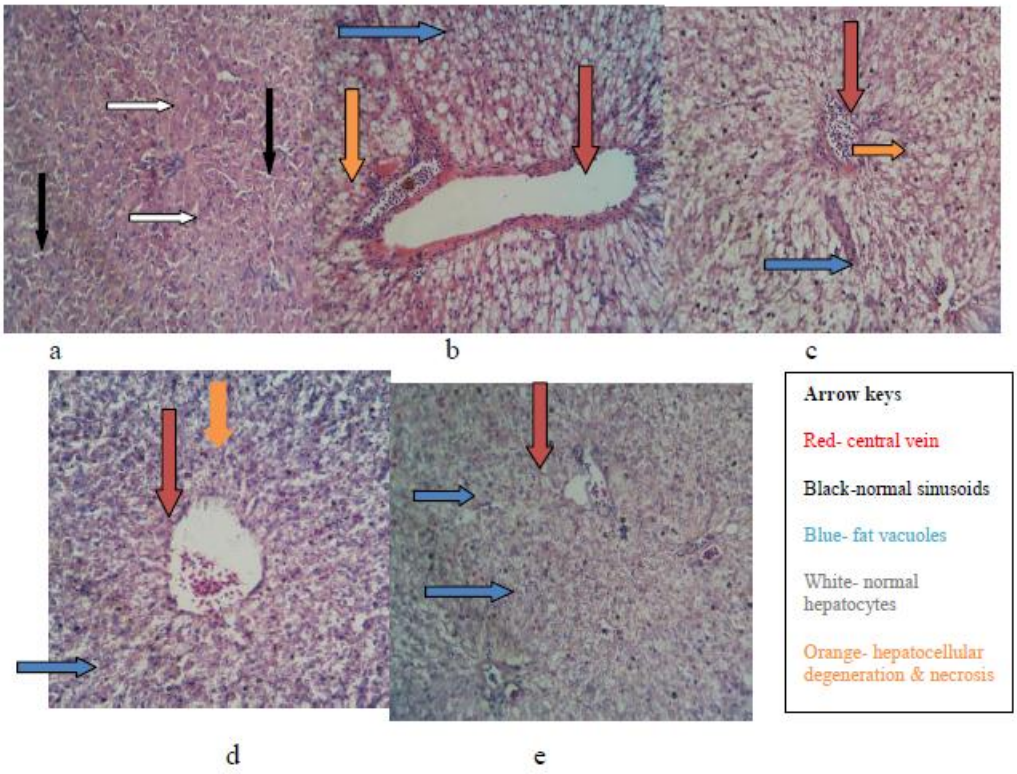

Plate 2. Photomicrograph of the liver of fish exposed to different effluent concentrations from a paint-manufacturing factory

a: Exposed to borehole water (Control). No visible lesion observed.

b: Exposed to 100% $^{v}/_{v}$ paint effluent; Severe diffuse hepatocellular degeneration and necrosis observed.

c: Exposed to 75% $^{v}/_{v}$ paint effluent; Diffuse severe macrovesicular fatty change seen.

d: Exposed to 50% $^{v}/_{v}$ paint effluent. Diffuse severe diffuse macrovesicular fatty change and sub-acute severe diffuse hepatocellular degeneration and necrosis observed.

e: Exposed to 25% $^{v}/_{v}$ paint effluent. Diffuse severe macrovesicular fatty change seen.

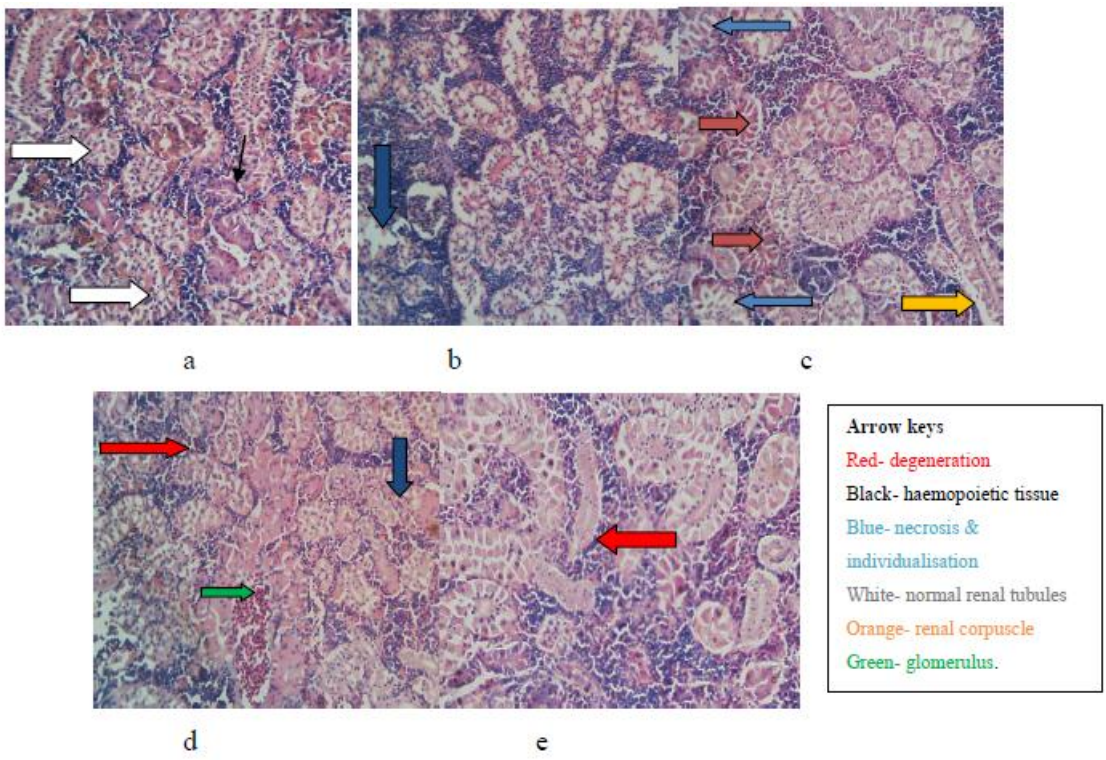

Plate 3. Photomicrograph of the kidney of fish exposed to different effluent concentrations from a paint-manufacturing factory.

a: Exposed to borehole water (Control). Atrophy of tubular epithelia observed.

b: Exposed to 100% $^{v}/_{v}$ paint effluent. Sub-acute severe diffuse tubular degeneration and necrosis observed.

c: Exposed to 75% $^{v}/_{v}$ paint effluent. Renal tubular epithelial atrophy, degeneration and necrosis observed.

d: Exposed to 50% $^{v}/_{v}$ paint effluent; Renal tubular epithelial atrophy and individualization observed.

e: Exposed to 25% $^{v}/_{v}$ paint effluent. Renal tubular epithelial atrophy, degeneration and necrosis observed.

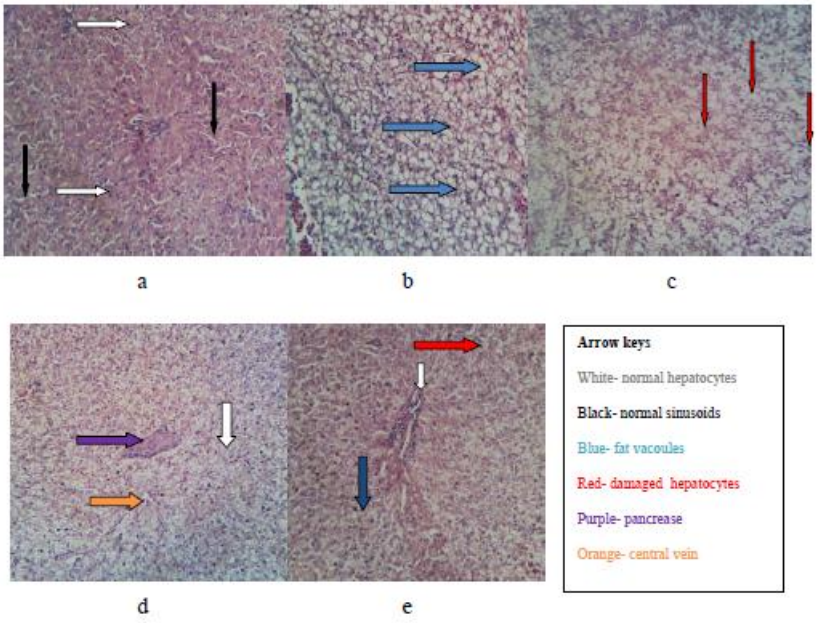

Plate 4. Photomicrograph of the liver of fish exposed to different effluent concentrations from a soap and detergent factory.

a: Exposed to borehole water (Control). No visible lesion observed.

b: Exposed to 1.0% $^{v}/_{v}$ effluent from Soap and Detergent factory. Diffuse severe macrovesicular fatty change observed.

c: Exposed to 0.8% $^{v}/_{v}$ effluent from Soap and Detergent factory. Severe macrovesicular fatty change, with centrilotubular hepatocellular degeneration and necrosis.

d: Exposed to 0.6% $^{v}/_{v}$ effluent from Soap and Detergent factory. Moderate diffuse centrilobular macrovesicular fatty change observed.

e: Exposed to 0.4% $^{v}/_{v}$ effluent from Soap and Detergent factory. Mild macrovesicular fatty change with centrilobular hepatocellular degeneration seen.

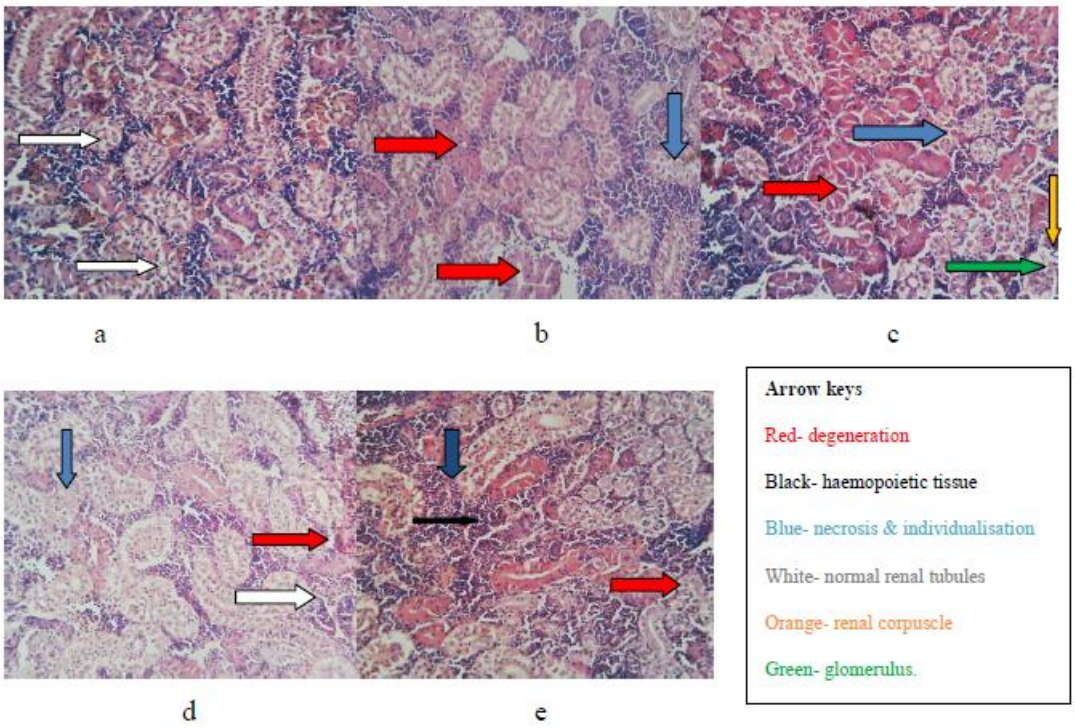

Plate 5. Photomicrograph of the kidney of fish exposed to different effluent concentrations from a soap and detergent factory.

a: Exposed to borehole water (Control). Atrophy of tubular epithelia observed.

b: Exposed to 1.0% $^{v}/_{v}$ effluent from a soap and detergent factory. Moderate random tubular degeneration observed.

c: Exposed to 0.8% $^{v}/_{v}$ effluent from a soap and detergent Factory; Moderate random tubular degeneration, with lymphocytic and histiocytic infiltrates and multifocal tubular necrosis seen.

d: Exposed to 0.6% $^{v}/_{v}$ effluent from a soap and detergent factory. Renal tubular epithelial atrophy, degeneration and necrosis observed.

e: Exposed to 0.4% $^{v}/_{v}$ effluent from Soap and Detergent Factory; Moderate random tubular degeneration, with lymphocytic and histiocytic infiltrates.

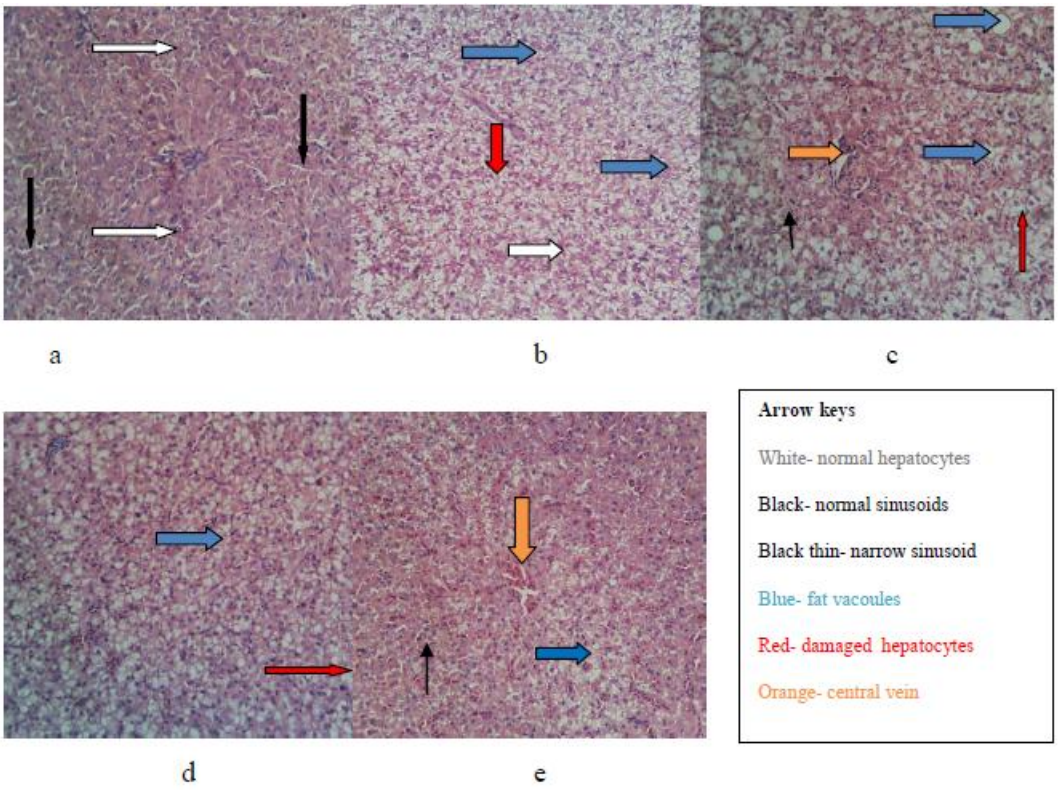

Plate 6. Photomicrograph of the histology of the liver of fish exposed to effluents from a food and feed factory.

a: Exposed to borehole water (Control). No visible lesion observed.

b: Exposed to 10% $^{v}/_{v}$ of effluent from a food and feed factory. Severe diffuse macrovesicular fatty change observed.

c: Exposed to 5% $^{v}/_{v}$ of effluents from a food and feed factory. Moderate diffuse mid-zonal macrovesicular fatty change.

d: Exposed to 2.5% $^{v}/_{v}$ effluents from a food and feed factory. Severe diffuse macrovesicular fatty change, vascular and sinusoidal congestion observed.

e: Exposed to 1% $^{v}/_{v}$ of effluent from a food and feed factory. Mild multifocal, periportal microvesicular fatty change less than 10% observed.

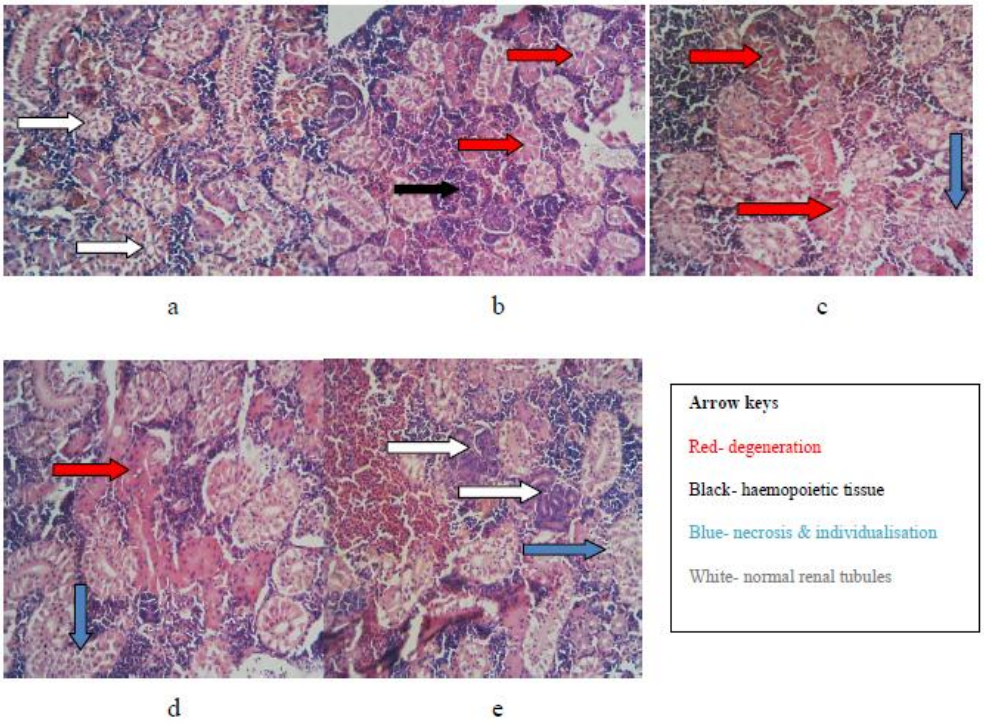

Plate 7. Photomicrograph of the kidney of fish exposed to effluents from a food and feed factory.

a: Exposed to borehole water (Control). Atrophy of tubular epithelia observed.

b: Exposed to 10% $^{v}/_{v}$ of effluent from food and feed factory. Moderate to severe random tubular degeneration seen.

c: Exposed to 5% $^{v}/_{v}$ effluent from a food and feed factory. Moderate to severe random tubular degeneration, with lymphocytic and histiocytic Infiltrates observed.

d: Exposed to 2.5% $^{v}/_{v}$ of effluent from a food and feed factory. Severe acute tubular degeneration, individualization and multifocal necrosis seen.

e: Exposed to 2.5% $^{v}/_{v}$ of effluent from a food and feed factory. Moderate acute tubular degeneration characterized by tubular epithelia swelling and focal necrosis observed.

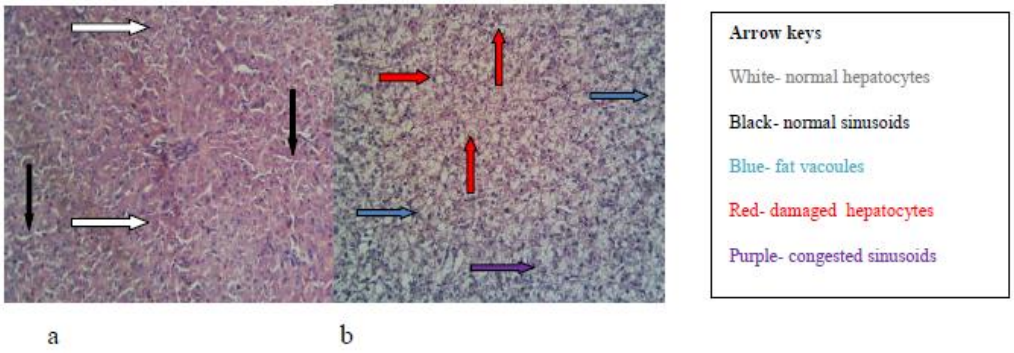

Plate 8. Photomicrograph of the liver of fish exposed to different effluent concentrations of effluents from a soft drink bottling factory.

a: Exposed to borehole water (Control). No visible lesion observed.

b: Exposed to 100% $^{v}/_{v}$ of effluent from a soft drink bottling factory. Severe diffuse macrovesicular fatty change observed.

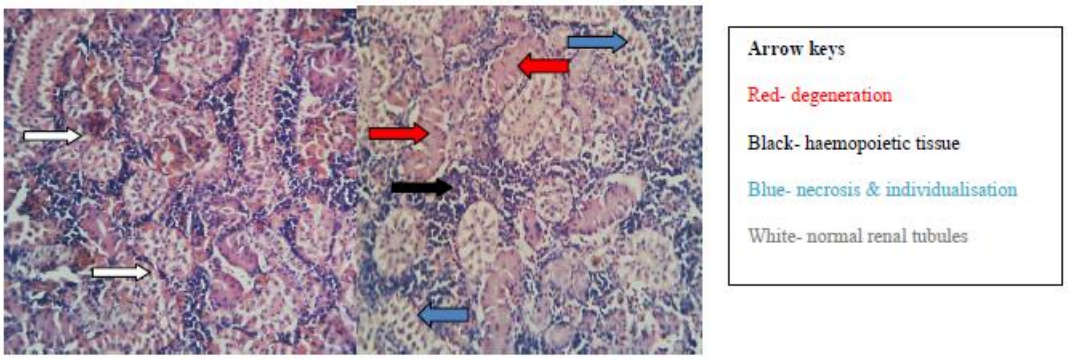

Plate 9. Photomicrograph of the kidney of fish exposed to different effluent concentrations from a food and feed factory.

a: Exposed to borehole water (Control). Atrophy of tubular epithelia observed.

b: Exposed to 100% $^{v}/_{v}$ of effluent from a soft drink bottling factory. Severe diffuse tubular degeneration and necrosis observed.

DISCUSSION

Aquatic toxicity testing may be used to predict the environmental effects of chemical wastes, to compare toxicants, or as a regulatory tool for control of industrial and municipal discharges. Each effluent is unique in its composition and may be different from other samples in its physicochemical and toxic properties. This is due, in part, to the variability over time in the presence and concentrations of different effluent constituents that can contribute to acute lethality. It is essential, therefore, to collect and test many samples to understand the variability of an effluent.

The physicochemical parameters of the effluents analyzed are presented in Table 1. The pH of water or effluents can alter the biological characteristics of aquatic organisms. Generally, the direct toxic effects of pH as a lethal factor become more severe and rapid with a deviation in either direction from neutrality.[10] The pH range 6.5 to 8.5 meets the requirement for indefinite survival of aquatic life.[11] The pH of the effluent collected from the soap and detergent factory was detected to be above the upper stipulated limit, possibly because soaps are basic. Specifically, mortality was highest in the *Clarias gariepinus* fish exposed to soap

and detergent effluents at pH 10.46, an observation supported by[12] who reported no mortality in *Clarias gariepinus* fish exposed to effluents at pH 6.0-8.0.

Increased water temperature is an important consideration when toxic substances are present in effluents. This is because temperature denatures proteins and also affects enzymatic reactions from hormonal and nervous control, digestion, respiration and osmoregulation to all aspects of an organism's performance and behavior.[9] This is particularly important in this study as samples were collected early in the day when temperatures were low and may thus not reflect conditions for the rest of the day.[13] reported that when water is highly heated, much energy, oxygen and vapour is released into the air, leaving behind a high concentration of CO_2, which makes the water more acidic. This buttresses the point that the temperature of effluents disposed into our water bodies from the industrial sectors be regulated and properly monitored since the resultant temperature fluctuation can completely destroy the entire flora and fauna in extreme cases.

Colour is a qualitative characteristic that can be used to assess the general condition of wastewater. The blackening of wastewater is often due to the formation of various sulphides, particularly, ferrous sulphide (a metal detected in the effluents in this present study). These colour observations are similar to those made in this study (Table 1), where effluent color ranged from whitish, grey and turbid A, B and C respectively. Based on the earlier observation by[14] effluents from paint (A); Soaps and detergents (B); food, feed and oil (C) would be unsuitable for aquaculture and would most likely result in increased mortality, compared to the control. In fact, the high mortality observed in fish exposed to effluents from the soaps and detergents factory may be attributed to the effluent undergoing decomposition, with the resultant harmful effect.

Fish and other aquatic animals depend on dissolved oxygen (the oxygen present in water) to live. BOD in effluents from all the industries analyzed in this study was found to be higher than the WHO requirement, with those from the soaps/detergents and food/feed/oil factories being 20 and 26 times higher respectively. The greater the decomposable matter present, the greater the oxygen demand and the greater the BOD values.[15] [16] and [17] had earlier reported that indiscriminate deposition of effluent into an aquatic system might decrease the dissolved oxygen concentration, which stand to impair respiration leading to asphyxiation (which is an indication of unconsciousness or death produced by failure of the blood to become properly

oxygenated in the lungs) and may ultimately results into organ architectural degradation in fish.

COD is also one of the most common measures of pollutant organic material in water, and is similar in function to BOD, in that both measure the amount of organic compounds in water. The presence of high concentrations of these pollutants, as seen in the COD for effluents from paint, soaps/detergents and food/feed/oil factories above the critical values stipulated by WHO maximum standard (Table 1) are considered unacceptable in receiving water bodies. This is because, apart from causing a major drawback in wastewater treatment systems, they also lead to eutrophication and various health impacts in humans and animals.[18]; [19]; [20]

In a study investigating metal toxicity to *Ceriodaphnia dubia*[21], demonstrated that suspended solids could have additional adverse effects by way of physical forces or ingestion. This is of concern, as results from this present study (Table 1) show concentration of Total Suspended Solids (TSS) to be 3-5 times higher than WHO stipulation of 150 mg/L, except in the effluent from the paint industry. This can be dangerous to both aquatic life and humans as suspended solids can harbor pathogens such as bacteria and protozoa.[22,23] reported that if ingested, suspended solids may act as a secondary stressor by reducing food assimilation or increasing body weight and requiring cladocerans to expend more energy to stay at the desired depth within the water column.

Wastewater effluents from several industries, such as bottling, soaps and detergents, paint, metal plating processes, and plastic industries, generally contain high total dissolved solid (TDS) concentration[24], a situation similar to results from this study (Table 1), where TDS concentration for the different effluents from the soaps and detergents (B); food/feed/oil (C); and bottling (D) industries were higher than the WHO maximum of 500 mg/L, a situation that is unacceptable. The major component of solutes in TDS is inorganic, including cations (e.g., sodium, calcium, magnesium, and potassium), and anions (e.g., carbonates, nitrates, bicarbonate, and chloride, sulfate;[25], similar to those detected in the different effluents analyzed in this study. Ideally, TDS of industrial effluents should be reduced to its standard level before the effluent is discharged into rivers, other water bodies, or municipal sewers[26], though this was clearly not done in effluents from 3 out of the 4 factories/industries analyzed in this study.

Chromium, an essential element for humans, has been found to be toxic at levels as low as 0.1 mg/L[27] and in excess leads to growth retardation, cancer and kidney/liver injury.[28] Cadmium that fell within or below tolerable limit, detected in effluents from the paint and bottling factories in this study, could also by way of their additive effect become toxic to all forms of aquatic lives and this result conforms with the submission of[17] which opined that if concentration of metals fell within or below[29] specifications, it could also be biomagnified in the water, the resultant effect could be gradual accumulation of the metals in water which in turn become toxic to aquatic organisms.

Results from this study showed that lead detected in the effluents from the soap/detergents and bottling factories showed slightly higher levels stipulated by the World Health Organization. Lead bioconcentrates in the skin, bones, kidneys, and liver of fish, however, people who eat the whole fish, can potentially be exposed to high concentrations of lead.[30]

Iron, an essential element in many physiological processes[31], causes cellular injury to induce oxidative stress in excess.[32] After an iron-enriched diet, lipid peroxidation (LPO) was observed in the liver and heart of the African catfish *Clarias gariepinus*[33], the same species of fish used in this study. This is a cause for concern, as results from this study show that iron levels in all effluents analyzed (Table 2) were higher than the WHO requirement, thus the possibility of toxicity in fish exists. Ultimately, humans who consume these fish are exposed to toxic levels of iron. This is supported by the report of[34], which stated that in many aquaculture systems, the presence of iron at concentrations above 0.1mg/L will damage the gills of the fish.

Contamination of water by arsenic and consequent toxicity in aquatic organisms has now emerged as a global environmental problem. In the present study, concentration of arsenic ranged from 0.011 to 0.065 mg/L, higher than the WHO maximum standard. Arsenic is known to cause adverse effects in aquatic biota and is a major concern to human health.[35]

The liver of fish can be considered a target organ to pollutants; thus alterations in its structure can be significant in the evaluation of fish health[36], and exhibit the effects of a variety of environmental pollutants.[37] In the present study, a histopathological assessment of the liver and kidney of *Clarias gariepinus* was conducted and showed varying degrees of loss of necrosis, as well as tubular and hepatocellular degeneration (Plates 2-9). This is indicative of

injury or damage and also supports other results from this study that show the toxic effect of the effluents from the different industries.

CONCLUSION

The discharged effluents were shown to be toxic in the animal model used. This has the potential to cause toxicity in humans who consume fish present in such water bodies. Hence, the need to constantly monitor effluents discharged into the environment.

Conflict of interest

Authors have declared that no competing interests exist.

CRediT author statement

LA: Conceptualization, Methodology, Software, Data curation, Writing- Original draft preparation, Visualization, Investigation. SSG: Supervision. KIA: Writing- Reviewing and Editing.

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Supplementary material

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