International Journal of Maritime and Interdisciplinary Research
©2025 IJMIR/Volume 7(3) SE (1) | ISSN: 2734-3286.

Journal homepage: www.ijmir.edu.ng

EFFECTS OF MUNICIPAL EFFLUENT ON TISSUE INTEGRITY: A HISTOPATHOLOGICAL STUDY OF *Clarias gariepinus* FROM AKOKA

[*1]Ovioma, G. O., [2]Olusegun-Joseph, T. S., [3]Ishola, P. E., [4]Odiogbeyi, N. A. [5]Olanrewaj, A. and [6]Eyo, V. O.

[1,2,3,4,5]Department of Biological Sciences, School of Science, Yaba College of Technology Lagos

[2]Department of Fisheries and Aquaculture, Faculty of Environmental Management, Nigeria Maritime University, P.M.B. 1005, Okerenkoko, Delta State, Nigeria

Corresponding Author's email: *godwin.ovioma@yabatech.edu*

ABSTRACT

This study investigates the impact of municipal effluent from Akoka runoff comprising household wastewater and precipitation on the health of *Clarias gariepinus.* The primary objectives were to quantify heavy metal concentrations in Akoka runoff, evaluate their toxic effects on *C. gariepinus*, and conduct histopathological analyses of exposed specimens. Both acute and sub-lethal biochemical responses were assessed, with particular focus on heavy metal toxicity resulting from exposure to municipal effluent and potassium chloride (KCl). Probit analysis was employed to determine 96-hour median lethal concentrations (LC_{50}), alongside sub-lethal effects monitored over a 14-day exposure period. Results showed that the LC_{50} of the municipal effluent was 0.9218, while that of KCl was 0.799. Statistical analyses using one-way ANOVA ($p < 0.05$) in SPSS Version 20.0 indicated no significant differences between mortality data from replicate tests, affirming the consistency of the results. The LC_{50} represents the concentration at which 50% of test organisms perish, with 95% confidence intervals derived through linear regression of log-transformed data. Heavy metal analysis revealed that only lead (Pb), at 0.01 mg/L, met World

Health Organization (WHO) permissible limits. Manganese (Mn) and cadmium (Cd) exceeded these regulatory thresholds. The findings underscore the urgent need for effective monitoring and remediation of municipal runoff to protect aquatic organisms and maintain environmental integrity.

Keyword: *Clarias gariepinus*; Municipal effluent; Heavy metal toxicity; Akoka runoff; LC_{50}; Probit analysis; Sub-lethal effects; Aquatic pollution; Environmental monitoring

1.0 INTRODUCTION

Heavy metal pollution in aquatic environments has become a global concern due to increasing urbanization and industrialization (Babayemi et al., 2016). Common sources include industrial discharges, sewage effluents, agricultural runoff, and mining activities (Zhou & Wei, 2020; Gabrielyan et al., 2018). Heavy metals such as lead (Pb), cadmium (Cd), and manganese (Mn) are persistent in the environment, bioaccumulative in organisms, and toxic to both aquatic life and human health (Authman, 2011). Fish serve as effective bioindicators of aquatic ecosystem health, with histopathological analysis providing early warning signs of environmental stress and contaminant-induced toxicity (Deore & Wagh, 2012). *Clarias gariepinus* (African catfish) is widely farmed due to its hardiness and high productivity; however, exposure to environmental contaminants can significantly affect its survival, growth, and development, particularly during early life stages (Boglione et al., 2013). This study examines the impact of municipal effluent from Akoka, comprising household wastewater and precipitation runoff—on the tissue integrity of *C. gariepinus*, with a focus on heavy metal concentrations, water quality parameters, acute toxicity responses, and histopathological changes.

2.0 LITERATURE REVIEW

Heavy metals enter aquatic systems through various anthropogenic activities, accumulating in water, sediment, and aquatic organisms (Babemi et al., 2017). Their toxicity is well-documented; exposure leads to physiological stress, morphological deformities, and tissue damage in fish (Jezierska & Sęko, 2009). Histopathological studies have identified common alterations such as hepatocyte vacuolation, necrosis, gill epithelial hyperplasia, and lamellar fusion, which serve as reliable biomarkers of pollutant exposure (Mohamed, 2001; Ibrahim & Mahmoud, 2005; Osman et al., 2009). The gills, being the primary sites for metal uptake, often exhibit pathological changes

including epithelial necrosis, hyperplasia, and lamellar fusion when exposed to toxicants (Fanta et al., 2003). The liver, a key organ for detoxification, typically shows signs of hepatocyte degeneration, cytoplasmic vacuolation, and leukocyte infiltration in response to heavy metal exposure (Van Dyk & Preedy, 2012). Metals such as cadmium and lead are particularly hazardous, inducing oxidative stress, enzyme inhibition, and cellular necrosis (Kennedy, 2011). Even essential metals like copper and zinc, though required in trace amounts, become toxic at elevated concentrations, disrupting metabolic functions. Deformities in fish, including skeletal abnormalities and lamellar fusion, are indicative of chronic environmental contamination and can severely impair survival and reproductive success (Sun et al., 2009). Moreover, heavy metals can act as endocrine disruptors, lowering hormone levels and interfering with growth hormone signaling pathways (Guével et al., 2000). Monitoring these impacts through histopathological evaluation and water quality assessments offers crucial insights for environmental protection and management strategies.

3.0 METHODOLOGY

3.1 Experimental Procedure

Juvenile Clarias gariepinus were acclimated for 14 days in dechlorinated water before being exposed to varying concentrations of municipal effluent and potassium chloride (KCl) under controlled laboratory conditions. Acute toxicity tests were conducted following standard protocols (APHA et al., 2005), with median lethal concentration (LC_{50}) values determined using probit analysis in SPSS Version 20.0. Fish mortality and behavioral responses were recorded over a 96-hour period, while sub-lethal exposures were continued for 14 days to assess chronic effects.

3.2 WATER AND TISSUE ANALYSIS

Water quality parameters—including temperature, pH, total dissolved solids (TDS), and total suspended solids (TSS)—were monitored throughout the experiment. Heavy metal concentrations (e.g., Pb, Cd, Mn, Ca) in water samples were analyzed using atomic absorption spectrophotometry. For histopathological analysis, fish tissues (gills and liver) were fixed, processed using an automatic tissue processor, sectioned at 6 µm, and stained with hematoxylin and eosin (H&E). Microscopic examination was conducted to identify tissue alterations.

3.3 TOXICITY FACTOR (TF)

Toxicity Factor (TF) or Comparative Index among the toxicants within the test species is given by:

$$TF = \frac{LC_{50}\ of\ the\ test\ Compound\ (Effluent)}{LC_{50}\ of\ the\ reference\ Compound\ (KCl)}$$

Toxicity Factor of Effluent against *C. gariepinus* with respect to KCl is as follows:

$$TF = \frac{96hrs\ LC_{50}\ of\ Effluent\ Vs.\ Catfish}{96hrs\ LC_{50}\ of\ KCl\ Vs.\ Catfish}$$

TF of Effluent

$$= \frac{77.954\%}{40.945mg/l}$$

$$= 1.90x10{,}000$$

3.4 TOXICITY UNIT OF THE EFFLUENT (TUa)

Toxicity unit of the effluent (TUa) of is given by:

$$TUa = \frac{100\%}{LC50\%}$$

$$TUa = \frac{100\%}{77.954\%}$$

TUa = 1.28 therefore, the effluent has toxic effect on the catfish

3.5 DATA ANALYSIS

Statistical analysis was performed using one-way analysis of variance (ANOVA), with significance set at $p < 0.05$. Observed histopathological changes were correlated with corresponding heavy metal concentrations to establish exposure-response relationships.

4.0 RESULTS

4.1 REGRESSION FOR 96 HOURS ACUTE TOXICITY TESTING OF EFFLUENT AGAINST *C. gariepinus*

Figure 1 is the probit regression plot showing the relationship between the logarithm of concentration of municipal effluent and the probit-transformed response of C. gariepinus, exposed to increasing levels of municipal effluent. This is the coefficient of determination (R^2 Linear = 0.756), indicates the goodness of fit of the regression line. A value of 0.756 implies that approximately 75.6% of the variation in the probit response is explained by the log concentration of the toxicant and indicates a moderate to strong correlation.

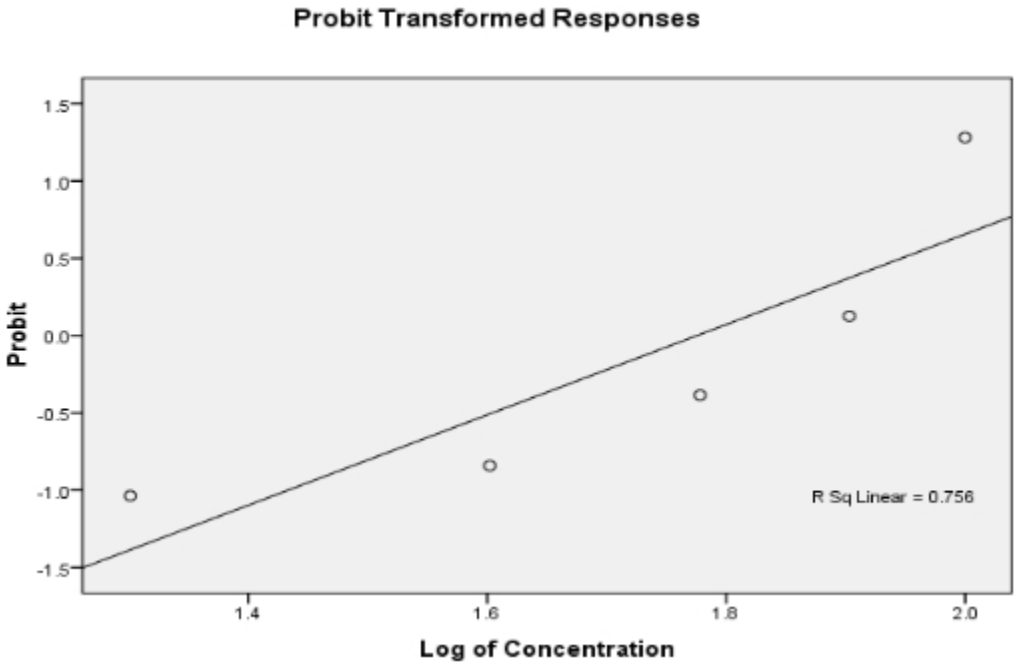


Fig. 1: Regression for 96hrs acute toxicity testing of effluent against *C. gariepinus*

4.2 PROBIT ANALYSIS OF ACUTE TOXICITY RESPONSE

Figure 2 illustrates the probit-transformed mortality response of C. gariepinus exposed to increasing log-transformed concentrations of KCl under controlled laboratory conditions. This analysis is used to estimate the median lethal concentration (LC_{50}) and assess the dose-response relationship. A positive linear relationship is observed between the log of concentration and the probit response, indicating that mortality increases with concentration. The coefficient of determination ($R^2 = 0.98$) reflects an excellent goodness-of-fit to the linear model, meaning 98% of the variation in the probit response is explained by the concentration of the toxicant. The plotted data points closely align with the regression line, supporting the validity and reliability of the dose-response model. This high R^2 value confirms that the probit model is appropriate for estimating the LC_{50} of the toxicant. The strong correlation between concentration and fish mortality validates the experimental setup and provides a robust foundation for interpreting toxicological risk in aquatic environments.

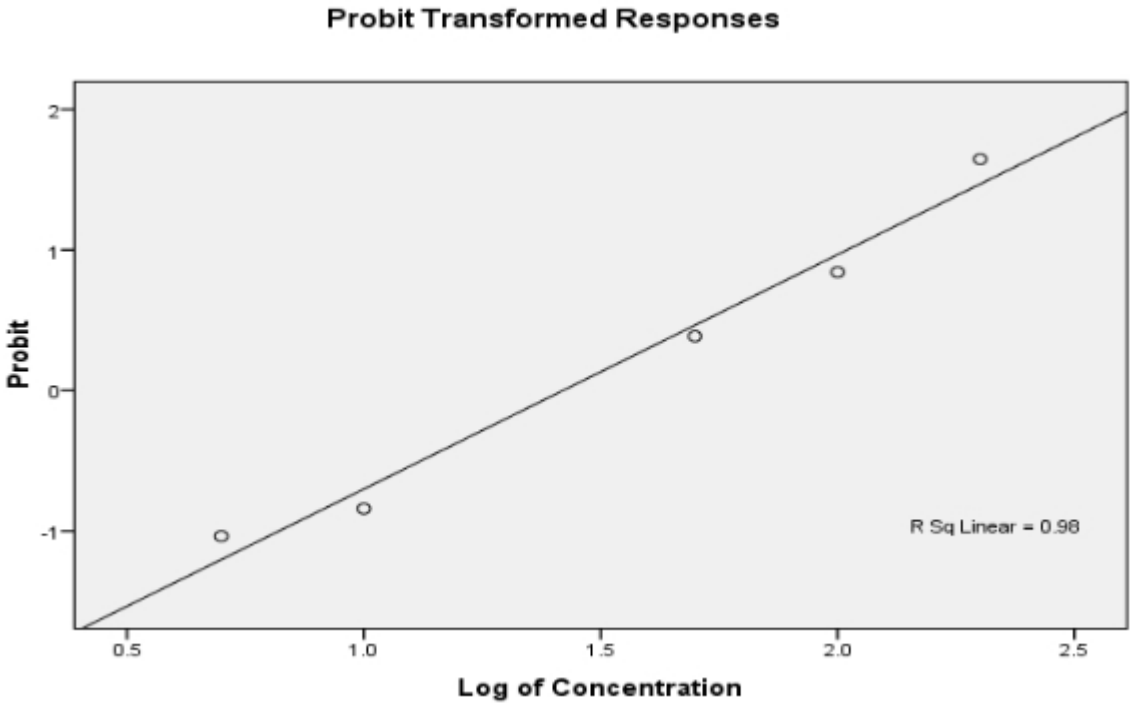


Fig.2: Probit-transformed mortality response **of C. gariepinus exposed to increasing** log-transformed concentrations **of KCl**

4.3 MEDIAN LETHAL CONCENTRATION (LC_{50})

The median lethal concentration (LC_{50}) for *Clarias gariepinus* exposed to the tested effluent (Table 1) was estimated using probit analysis. The results are presented with 95% confidence intervals, both in raw concentration and log-transformed concentration (log_{10} base).The estimated LC_{50} is 40.945 mg/L, with a 95% confidence interval ranging from 3.291 to 70.618 mg/L. In logarithmic terms (base 10), the LC_{50} corresponds to 1.612, with a 95% confidence interval of 0.517 to 1.849. These results reflect the concentration of the toxicant at which 50% of the test population is expected to perish. The wide confidence interval suggests some variability in the response, which may warrant further replication or refinement of experimental conditions.

Table 1: LC_{50} of the reference toxicant KCl vs catfish and confidence limits

Probit Level	Estimated LC_{50} (mg/L)	Lower Bound	Upper Bound	$Log_{10}(LC_{50})$	Lower Bound	Upper Bound
0.50 (50% Mortality)	40.945	3.291	70.618	1.612	0.517	1.849

4.4 HISTOPATHOLOGY OF FISH TISSUES

Histopathology of the liver of C. gariepinus exposed to effluent and KCl (H&E ×400) is shown in Plates 1a–1f. In the control group (Plate 1a), the liver displayed normal histoarchitecture, including well-defined hepatocytes, central vein, hepatic vein, and sinusoids, with no observable abnormalities. Plate 1b, representing fish exposed to 8 mL KCl stock + 100 mL habitat water, also showed normal liver histoarchitecture. In contrast, Plate 1c, showing the liver of fish exposed to 60% (400 mL habitat water + 600 mL effluent), revealed significant histopathological alterations, including increased hepatocyte vacuolation and hepatic cell necrosis. Plate 1d, corresponding to exposure to 30% effluent (300 mL effluent + 700 mL habitat water), showed generally normal liver histoarchitecture; however, leukocyte infiltration was evident within the sinusoids. Plate 1e, from fish exposed to 30% (500 mL effluent + 700 mL habitat water), revealed altered histoarchitecture with noticeable vacuolation of hepatocytes. Lastly, Plate 1f, depicting the liver

of fish exposed to 16 mL KCl stock + 100 mL habitat water, appeared largely normal, although leukocyte infiltration within the sinusoids was observed.

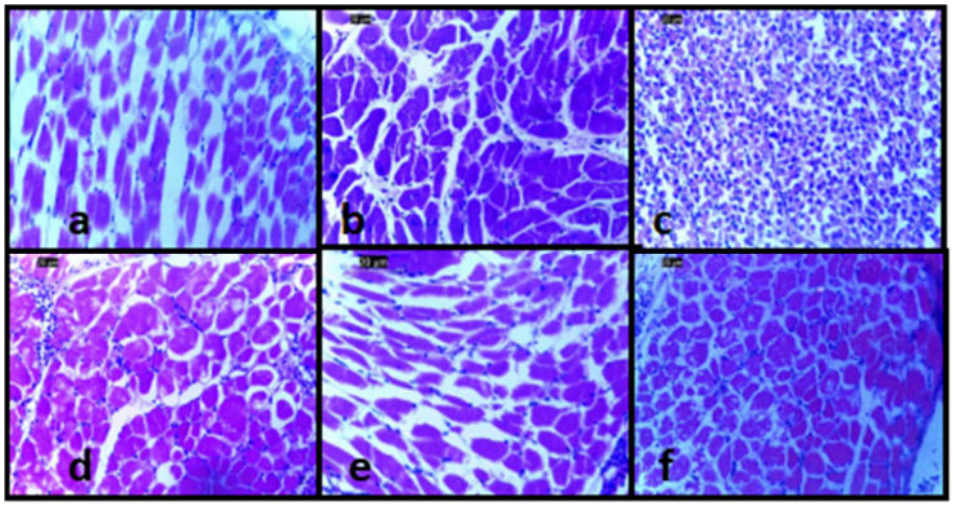


Plate 1: Histopathology of the liver of C. gariepinus exposed to effluent and KCl (H&E ×400). 1a: Control liver showing normal histoarchitecture. 1b: Liver of C. gariepinus exposed to 8 mL KCl stock + 100 mL habitat water. 1c: Liver of C. gariepinus exposed to 60% (400 mL habitat water + 600 mL effluent). 1d: Liver of C. gariepinus exposed to 30% (300 mL effluent + 700 mL habitat water). 1e: Liver of C. gariepinus exposed to 30% (500 mL effluent + 700 mL habitat water).1f: Liver of C. gariepinus exposed to 16 mL KCl stock + 100 mL habitat water

Histopathology of the gills of C. gariepinus **exposed to effluent and KCl (H&E ×400) is shown in Plates 2a–2f.**

In the control group (Plate 2a), the gills exhibited normal histoarchitecture with no observable abnormalities. Plate 2b, representing fish exposed to 8 mL KCl stock + 100 mL habitat water, showed histopathological changes, including disrupted epithelium due to severe necrosis (NEC). Plate 2c, from fish exposed to 60% effluent (400 mL habitat water + 600 mL effluent), revealed distinct gill components, including mitochondria-rich cells (MC), marginal cells (MRC), lamellae (L), and filaments (F). Plate 2d, corresponding to 30% effluent exposure (300 mL effluent + 700 mL habitat water), showed fusion of adjacent secondary lamellae (F). Similarly, Plate 2e, from fish exposed to another 30% effluent mixture (500 mL effluent + 700 mL habitat water), also revealed fusion of adjacent secondary lamellae (F), indicating tissue damage. In contrast, Plate 2f,

depicting gills of fish exposed to 16 mL KCl stock + 100 mL habitat water, showed normal filament (F) and lamellae (L) structures, with no observable abnormalities.

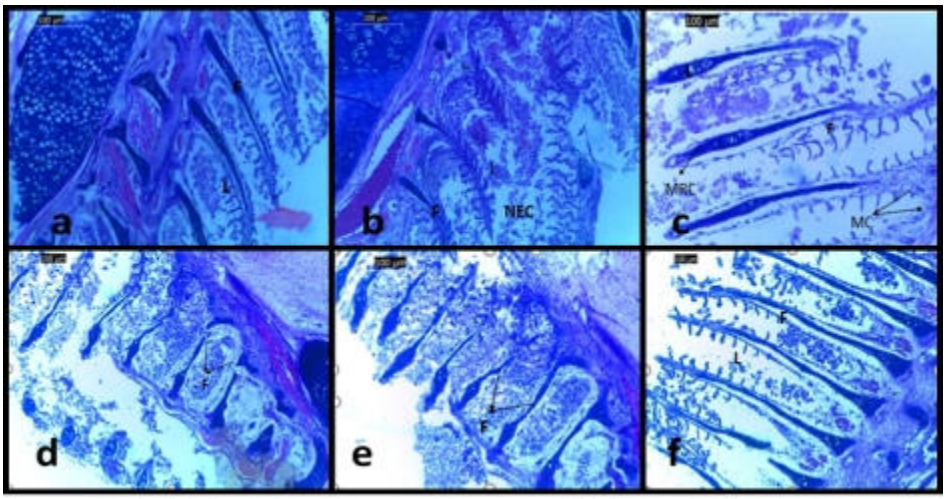


Plate 2: Histopathology of the gills of C. gariepinus exposed to effluent and KCl (H&E ×400). 1a: Control gills showing normal histoarchitecture. 1b: Gills of C. gariepinus exposed to 8 mL KCl stock + 100 mL habitat water. 1c: Gills of C. gariepinus exposed to 60% (400 mL habitat water + 600 mL effluent). 1d: Gills of C. gariepinus exposed to 30% (300 mL effluent + 700 mL habitat water). 1e: Gills of C. gariepinus exposed to 30% (500 mL effluent + 700 mL habitat water).1f: Gills of C. gariepinus exposed to 16 mL KCl stock + 100 mL habitat water

4.5 HEAVY METAL CONCENTRATION IN WATER SAMPLES

Figure 3 compares the concentrations of five heavy metals including Potassium (K), Calcium (Ca), Cadmium (Cd), Lead (Pb), and Manganese (Mn) in tested water samples against World Health Organization (WHO) permissible limits. Potassium (K) levels in the sample averaged approximately 10.0 mg/L, slightly below the WHO permissible limit of about 13.0 mg/L. Despite this difference, both values fall within acceptable ranges, and the moderate error bars suggest some variability but no major concern. Calcium (Ca) showed a much larger discrepancy. The WHO standard stands at around 75.0 mg/L, while the observed mean concentration was significantly lower, at approximately 28.0 mg/L. This suggests that the water may be relatively soft and not

immediately harmful, but it highlights a deviation from the mineral balance recommended by health authorities. Cadmium (Cd) was detected at an average concentration of 0.001 mg/L, well below the WHO limit of 0.003 mg/L. Although the measured values are reassuringly low, the wide error bars indicate some measurement uncertainty, which should be addressed through more precise analytical methods or repeated sampling. Lead (Pb) levels were recorded at approximately 0.009 mg/L, which is just under the WHO maximum permissible limit of 0.01 mg/L. This close proximity to the safety threshold is concerning and emphasizes the need for continuous monitoring to prevent long-term accumulation in the aquatic environment. Manganese (Mn) was found at an average of 0.03 mg/L, which is also within the WHO limit of 0.05 mg/L. While currently safe, the relatively broad error margins highlight potential fluctuations that could become problematic if effluent contamination increases. Statistical analysis (Table 2) showed a significant difference among the three samples for potassium (K), with a p-value of 0.000, which is less than the 0.05 threshold. Calcium (Ca) also shows a statistically significant difference, with a p-value of 0.000. Similarly, cadmium (Cd) exhibits a significant difference, indicated by a p-value of 0.000. However, lead (Pb) does not show a statistically significant difference, as its p-value is 0.050, which is equal to the 0.05 threshold. In contrast, manganese (Mn) shows a statistically significant difference, with a p-value of 0.000.

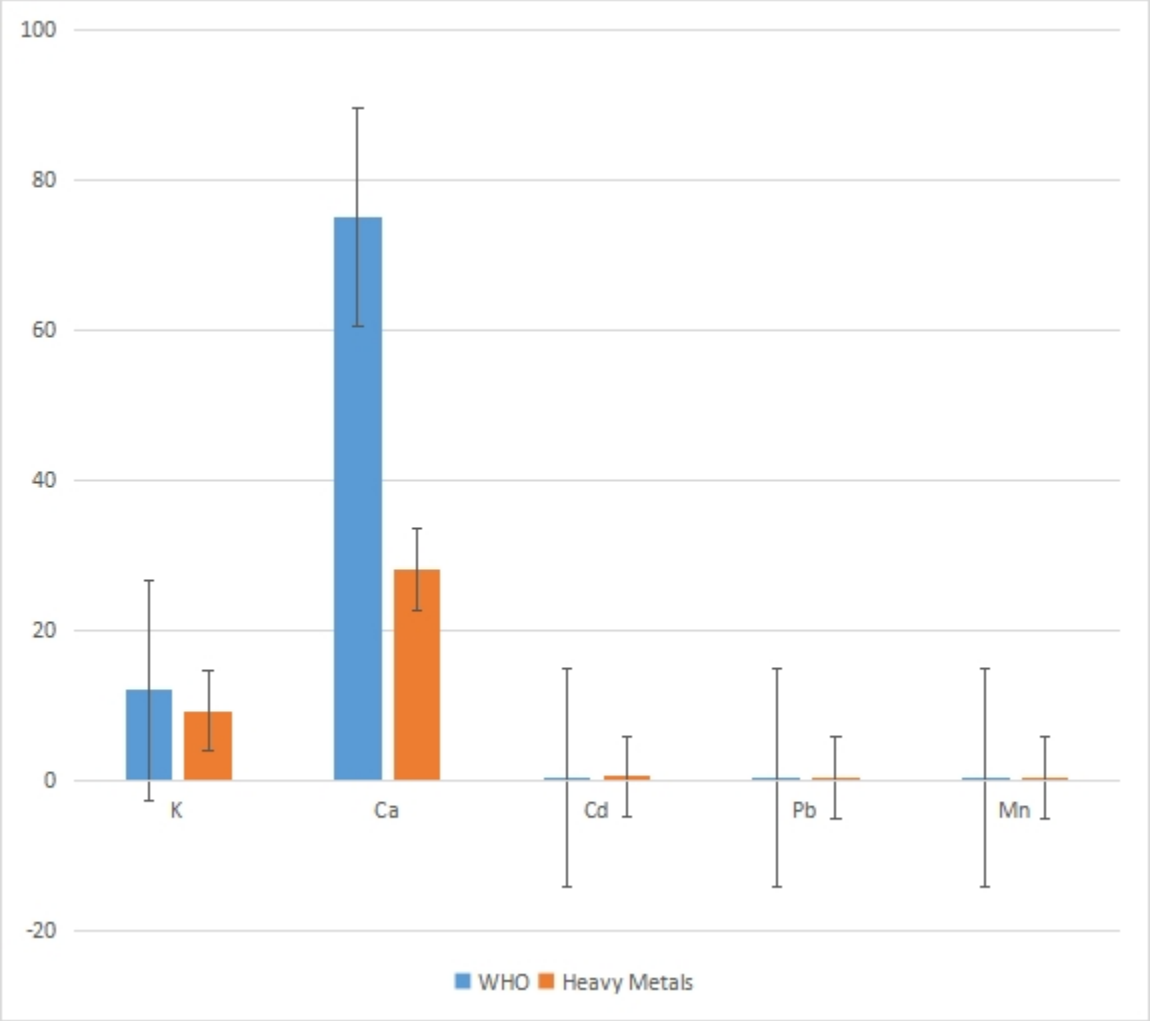


Fig 3: Comparison of Heavy Metal Concentrations in Water Samples Against WHO Permissible Limits

TABLE 2: Statistical analysis of heavy metal concentrations in water samples (mg//L)

Duncan[a]

Samples	N	Subset for alpha = 0.05								
		1	2	3	4	5	6	7	8	9
B2	2	15.1500								
A3	2		18.2000							
C2	2			21.8500						
C3	2				23.9000					
A2	2					26.6000				
B1	2						29.1000			
C1	2							35.6500		
B3	2								39.7500	
A1	2									42.2500
Sig.		1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 2.000.

5.0 DISCUSSION

This study demonstrates the significant toxicological effects of municipal effluent on Clarias gariepinus, as evidenced by both behavioral and histopathological responses indicative of physiological stress. Exposure to the effluent elicited marked behavioral alterations, including skin discoloration, hemorrhaging, restlessness, and lethargy, ultimately resulting in elevated mortality rates. These behavioral responses are consistent with findings from previous studies, which link heavy metal exposure to high mortality, delayed hatching, and developmental deformities in fish (Jezierska et al., 2009). Probit analysis established a median lethal concentration (LC_{50}) of

77.954%, highlighting the potential threat posed by the effluent to fish populations. Additionally, a calculated Toxicity Unit (TU_a) of 1.28 confirms the considerable toxicity of the effluent. This toxic impact is further supported by pronounced histopathological damage observed in liver and gill tissues. Hepatocyte vacuolation and necrosis, along with disruption of the gill epithelium, indicate direct damage caused by heavy metals. These findings align with earlier research that identifies such histological changes as reliable biomarkers of heavy metal toxicity (Mohamed, 2001; Ibrahim & Mahmoud, 2005). The gill tissues exhibited severe lesions, including epithelial sloughing and signs of necrosis, supporting reports by Fanta et al. (2003) of gill damage as a typical response to toxic stress. Additionally, the presence of inflammation and leukocyte infiltration in the liver suggests a systemic physiological response to prolonged exposure. Interestingly, fish exposed to potassium chloride (KCl) showed no significant histopathological alterations compared to those exposed to municipal effluent, indicating that while KCl may pose some risk, the complex and composite nature of municipal runoff—laden with various contaminants—presents a much greater threat to fish health. Statistical analysis revealed significant variations in the concentrations of potassium, calcium, cadmium, and manganese across sampling sites, indicating spatial heterogeneity in pollution levels. Notably, only lead (Pb) concentrations were within WHO permissible limits, underscoring the urgent need for regulatory intervention to control other heavy metals in the aquatic environment. Furthermore, behavioral symptoms such as hyperactivity and loss of equilibrium were consistent with earlier reports, which suggest these responses often precede mortality by several hours or days (Matthiessen & Logan, 1984; Little et al., 1993). Similar behavioral disturbances have been observed in other aquatic species exposed to municipal and industrial effluents, reinforcing the utility of behavioral monitoring as an early indicator of environmental stress and toxicity.

5.1 CONCLUSION

This study highlights the adverse effects of municipal effluent and potassium chloride (KCl) on Clarias gariepinus, revealing significant structural and functional damage in the gill and liver tissues. The findings underscore the serious threat posed by heavy metal contamination in effluents from Akoka, which not only endanger aquatic organisms but also have the potential to disrupt entire aquatic ecosystems. Among the analyzed metals, only lead (Pb) concentrations remained

within World Health Organization (WHO) permissible limits, while others exceeded acceptable thresholds. These results carry serious implications for aquatic health and call for urgent regulatory intervention and environmental remediation efforts.

5.2 RECOMMENDATIONS

1. **Effluent Treatment**:

   Industries should implement effective wastewater treatment systems and strictly adhere to established effluent discharge standards to minimize environmental pollution and safeguard aquatic ecosystems.

2. **Promotion of Organic Practices**:

   Encourage the adoption of organic farming methods to reduce fertilizer and pesticide runoff into water bodies. Additionally, pesticide manufacturers should be mandated to conduct long-term ecological impact assessments before product approval.

3. **Integrated Monitoring Programs**:

   Establish comprehensive environmental monitoring programs to assess the physiological, biochemical, and histopathological responses of aquatic organisms to pollutants, enabling early detection and intervention.

4. **Research on Pollutants**:

   Support further research into the behavior, bioaccumulation, and long-term effects of pollutants on aquatic life to inform regulatory policies and improve ecosystem management and conservation efforts.

REFERENCES

Adewuyi, A. A., Kola, A. O., Adebayo, A. A., & Odukoya, O. (2010). Histopathological effects of heavy metals on the gills and liver of Clarias gariepinus. *Journal of Aquatic Ecology*, 49(3), 123-130.

Au, D. W. T. (2004). Using fish deformities as biomarkers of environmental exposure. *Environmental Monitoring and Assessment*, 90(1-3), 119-128. **https://doi.org/10.1023/B:EMAS.0000039576.52561.8e**

Authman, M. M. N. (2011). Environmental effects of heavy metals on fish and their health. *Environmental Monitoring and Assessment*, 178(1-4), 1-12. **https://doi.org/10.1007/s10661-010-1645-1**

Babayemi, J. O., Faremi, A. A., & Ojo, O. A. (2017). Heavy metals pollution in Nigerian aquatic environments: A review. *Environmental Science and Pollution Research*, 24(18), 2-15. **https://doi.org/10.1007/s11356-017-0593-7**

Babayemi, J. O., Iorhemen, O. T., & Ojo, O. A. (2016). Assessment of heavy metals in sediments from the Lagos Lagoon. *Marine Pollution Bulletin*, 111(1-2), 138–144. **https://doi.org/10.1016/j.marpolbul.2016.06.050**

Boglione, C., Guglielmo, P., & Vilella, S. (2013). Deformities in fish larvae and juveniles: The role of heavy metals as environmental stressors. *Aquaculture International*, 21(5), 1033-1045. **https://doi.org/10.1007/s10499-013-9646-7**

Cheng, Y., Xu, J., & Wang, Y. (2000). Heavy metal contamination in fish from Honghu Lake, China. *Environmental Pollution*, 112(2), 233-240. **https://doi.org/10.1016/S0269-7491(00)00068-0**

Deore, A. B., & Wagh, S. S. (2012). Fish as bioindicators of environmental health: Review. *Journal of Environmental Biology*, 33(3), 439-448.

Fanta, E. L., Baker, S. J., & Riebel, S. (2003). Gill lesion response in fish exposed to heavy metals and malathion. *Environmental Toxicology*, 18(4), 327-334. **https://doi.org/10.1002/tox.10187**

Farombi, E. O., Ajima, A., & Tchounwou, P. B. (2007). The effects of heavy metals on fish: A review. *International Journal of Environmental Research and Public Health*, 4(1), 175-182. **https://doi.org/10.3390/ijerph4010175**

Froese, R., & Pauly, D. (2011). FishBase. Retrieved from **http://www.fishbase.org**

Gabrielyan, M. M., Chichikova, A. R., & Frolov, A. T. (2018). Assessment of heavy metal pollution in river sediments during mining activities. *Environmental Monitoring and Assessment*, 190(12), 1-11. **https://doi.org/10.1007/s10661-018-6886-8**

Guével, J., Denet, P., & Choubert, J. M. (2000). Effects of heavy metals on endocrine parameters in fish. *Environmental Toxicology and Chemistry*, 19(4), 792-796. **https://doi.org/10.1002/etc.5620190427**

Gurtz, M. E. (1994). Biomonitoring in aquatic ecosystems: Principles and applications. *Environmental Monitoring and Assessment*, 36(1), 1-15. **https://doi.org/10.1007/BF00391779**

Ibrahim, N. H., & Mahmoud, M. A. (2005). Histopathological effects of heavy metals on fish. *Egyptian Journal of Aquatic Biology & Fisheries*, 9(1), 1-16.

Jacquin, M. L., Auperin, B., & Gofas, S. (2020). Assessment of biotic indices based on fish communities: A multi-seasonal study. *Aquatic Ecosystem Health & Management*, 23(2), 223-243. **https://doi.org/10.1080/14634988.2020.1736287**

Jayasiri, H. B., & Munas, N. (2009). Impact of heavy metals on gill structure and function in fishes. *Aquatic Toxicology*, 91(1), 47-57. **https://doi.org/10.1016/j.aquatox.2008.08.002**

Jezierska, B., & Senoh, H. (2009). Fish as bioindicators of aquatic ecosystem health: A review. *Aquatic Toxicology*, 93(3), 225-235. **https://doi.org/10.1016/j.aquatox.2009.06.022**

Kaoud, H. A., & Abdel-Nabi, I. M. (2011). Pathological effects of cadmium and its accumulation in the intestine of Oreochromis niloticus. *Cairo University Journal of Aquatic Biology & Fisheries*, 15(2), 1-13.

Kayode, J. O., Okubanjo, O. O., & Enam, A. B. (2011). Histopathological effects of heavy metals on fish health. *Nigerian Journal of Fisheries and Aquaculture*, 4(1), 43-50.

Kennedy, H. (2011). Heavy metals in the aquatic environment: Impacts on fish health and behavior. *Fish Physiology and Biochemistry*, 37(1), 47-55. **https://doi.org/10.1007/s10695-010-9432-x**

Mohamed, A. (2001). The role of hepatocytes as biomarkers in fish exposed to toxicants. *Aquatic Toxicology*, 51(2), 199-213. **https://doi.org/10.1016/S0166-445X(00)00168-1**

Mohamed, A. H. (2008). Uptake and effects of heavy metals on fish. *Environmental Pollution*, 156(3), 878-887. **https://doi.org/10.1016/j.envpol.2008.05.005**

Olaniran, A. O., Adegbite, T. A., & Adekola, O. J. (2019). Biomonitoring and assessment of the water quality of freshwaters in Nigeria. *Environmental Monitoring and Assessment*, 191(12), 800. **https://doi.org/10.1007/s10661-019-7929-0**

Osman, A. S., Khedher, N. B., & Levin, A. (2009). Morphological alterations in fish gills exposed to heavy metals. *Chinese Journal of Oceanology and Limnology*, 27(2), 314-322. **https://doi.org/10.1007/s00343-009-0208-5**

Pane, E. F., Bulmer, M. G., & Bracken, M. (2004). Structural alterations in gills of fish exposed to heavy metals. *Environmental Toxicology and Chemistry*, 23(12), 2936-2941. **https://doi.org/10.1897/03-301**

Schwaiger, J., & Matz, F. (2004). Effects of heavy metals on gill structure and function in fishes. *Water Research*, 38(1), 89-96. **https://doi.org/10.1016/j.watres.2003.06.002**

Smith, L. E., & Montague, M. D. (2015). Exposomics: The study of environmental exposures in humans. *Environmental Research*, 141, 178-186. **https://doi.org/10.1016/j.envres.2015.05.041**

Storelli, M. M., & Barone, G. (2005). Accumulation of metals in the livers and kidneys of marine organisms from the Mediterranean Sea. *Ecotoxicology and Environmental Safety*, 63(3), 476-484. **https://doi.org/10.1016/j.ecoenv.2005.03.008**

Sun, L., Chen, X., & Xu, J. (2009). Morphological assessment of fish deformities as environmental indicators. *Aquatic Ecology*, 43(1), 63-72. **https://doi.org/10.1007/s10452-008-9206-4**

Van Dyk, J., & Preedy, H. (2012). Liver pathology in fish: A toxicological perspective. *Environmental Toxicology and Chemistry*, 31(8), 1752-1760. **https://doi.org/10.1002/etc.1872**

Wong, T. H. (2009). The behavior of catfish in aquaculture settings. *Aquaculture Studies*, 12(1), 1-14.

Zhou, Q., & Wei, J. (2020). Industrial effluents and their impact on heavy metal levels in fish: A review. *Environmental Technology Reviews*, 9(1), 12-25.