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Histopathology of the Gills, Livers and Kidney of *Clarias gariepinus* (Burchell, 1822) Exposed to Sniper 1000EC Under Laboratory Conditions

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ABSTRACT

Indiscriminate use of Sniper 1000EC has become a serious problem among local fishermen in the Northern parts of Niger state. Juveniles of *Clarias gariepinus* (mean body weight 23.34 ± 0.05g; mean standard length, 20.00 ± 0.45cm) were subjected to 5 treatment levels of 0, 5, 10, 15 and 20mg/L. The striking tissue damages were observed majorly at 15 and 20mg/L of sniper 1000EC exposed fish species. Lesions such as oedema and hyperplasia were observed in the gills of the exposed groups. Hepatocellular steatosis and vacuolations were observed in the livers. Tubular nephrosis and hyperplasias of epithelial cells were also observed in the kidney. It is concluded that alterations in gills, livers and kidney of the exposed fish species were consequences of exposure to the toxicant (Sniper 1000EC). It is recommended that the use of Sniper 1000EC by local fishermen be banned to save the aquatic environment from destruction.

Keywords: Sniper 1000EC, Histopathology, *Clarias gariepinus,* Gills, Livers and Kidney.

INTRODUCTION

Histopathological changes have been widely used as biomarkers in the evaluation of the health of fish exposed to contaminants, both in the laboratory (Wester and Canton, 1991; Thophon *et al.,* 2003) and field studies (Hinton *et al.*, 1992; Schwaiger *et al.*, 1997; Tech *et al.*, 1997). One of the great advantages of using histopathological biomarkers in environmental studies is that this category of biomarkers allows the examination of the target organs (Gernhofer *et al.*, 2001). Further more, the alterations found in these organs are normally easier to identify than functional ones (Fanta *et al.*, 2003), and serve as warning signs of damage to animal health (Hinton and Lauren, 1990). The gills and other accessory

respiratory organs of fish (Banerjee, 2007) being constantly in contact with fish's external environment, are vulnerable to aquatic toxicants (Abubakar, 2013). Studies have been conducted on histopathological changes in the gills, liver and kidney of fish exposed to various substances (Auta, 2001) including pesticide which have been reported to cause pathological alteration in the exposed *C. gariepinus* (Auta, 2001). The liver of fish can be considered as a target organ to pollutants, alterations in its structure can be significant in the evaluation of fish health (Kolbasi *et al.,* 2009).

Widespread application of various pesticides has aggravated the problem of pollution to aquatic environment. Due to these synthetic chemicals, environment has failed to keep its healthy characteristics. The insecticides of proven economic potentialities could not do well in the ecosystem when viewed on extra fronts since these revenue poisons, in a residual form or as a whole, get into the aquatic ecosystem. They cause a series of problems to aquatic organisms (Mastan and Ramayya, 2010).

Sniper 1000EC (2, 3-dichlorovinyl dimethyl phosphate), a brand of dichlorvos, is contact acting and fumigant insecticide (Abubakar, 2013). Like all organophosphates, it kills insects and other target organisms because of its toxicity to the nervous system. This is achieved by inhibition of enzyme acetylcholinesterase (AchE) that breaks down acetycholine at the receptor site for partial uptake into the nerve terminal. Without functioning AchE, accumulation of acetylcholine results in depolarizing block of muscle membrane, producing rapid twitching of involuntary muscles, convulsions, paralysis and early death. Indiscriminate use of Sniper 1000EC is common among local fishermen from Northern parts of Niger state.

The African catfish *(Clarias gariepinus)* is an important food fish in Nigeria, which is also good for research work (Abubakar, 2012). *Clarias gariepinus* is not only the most predominant fish species raised in aquaculture in Nigeria, but has also served as an experimental model of aquatic vertebrate for two decades (Cavaco, *et al.*, 2001). Despite the indiscriminate use of Sniper 1000EC by local fishermen, there is a paucity of information on its toxicity.

The aim of the present study was to evaluate histological alterations in gills, liver and kidney of *Clarias gariepinus* (Burchell, 1822) exposed to sniper 1000EC under laboratory conditions.

MATERIALS AND METHODS

Experimental Fish and Test Chemical

Juveniles of *Clarias gariepinus* (mean body weight 23.34 ±0.05g) were purchased from a reputable fish farm in Minna, Niger State. The samples were transported to the laboratory in plastic container of 100L capacity filled with water to two-third volume between 07:00 hours and 09:00 hours. They were held in large water baths of 160L capacity and acclimated for 14days to laboratory conditions. The top of water bath was covered with netted material to prevent jumping out of the fish. A slit was made at middle of the net to allow for feeding fish and cleaning of the bath. Feeding commenced two days after the arrival and stopped twenty-four hours before the commencement of the experiment. During acclimation, fish were fed twice daily (08:00 and 16:00 hours) with formulated feed (35% crude protein) at 3% body weight.

The fishes were accepted as well as adapted to laboratory conditions when less than 5% death was recorded for the 14 days. The water in the bath was changed daily and uneaten food and faecal matters were siphoned out. Dead fish were also removed to minimize contamination of water.

Test chemical (2, 3-dichlorovinyl dimethyl phosphate), a brand of Dichlorvos with the trade name Sniper 1000EC was obtained from Minna central market and was used for the study. The test concentrations were prepared with reference to the Manual of Method in Aquatic Environment Research.

Experimental Design

The experimental design was a complete randomized design. A total of one hundred and fifty (150) juvenile of *Clarias gariepinus* were randomly distributed into the tanks at a stocking rate of 10 fish per tank. The fifteen (15) tanks were assigned to 5 treatments (control inclusive). Dead fish from experimental groups were removed and dissected immediately for histological analysis while specimens from control were vivisected without anesthesia and subjected to histological analysis at the end of one- week experiment.

Histological Procedures

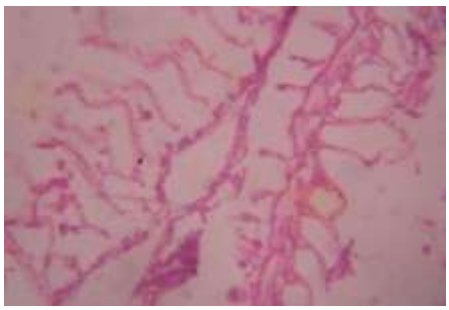
Samples of fish from both control and experimental groups were excised, rinsed in physiological saline and fixed in aqueos Boulin's fluid for 6, 12 and 8 hours respectively. The gills, livers and kidneys were subjected to micro techniques using the method of Bucke (1989). The

tissues were dehydrated in an ethyl alcohol series of ascending concentrations, embedded in paraffin and sectioned at 5µm thickness. The tissues sectioned were stained with haematoxilin-eosin (HE). Stained slides were cleared in xylene and mounted in synthentic resin medium. The slides were examined under low power (x4) objective and high power (x10) objective with Tension Binocular microscope. Under extreme low power, the condenser was moved into a very low position to prevent the image of the lamp bulb from obscuring the image of the micrographs. Photomicrographs were then taken at low power (x40) objective with Samsung ES25 4X Zoom Lens digital Camera and downloaded into a computer. Photomicrographs of control groups were compared with those of exposed groups under the guidance of a pathologist.

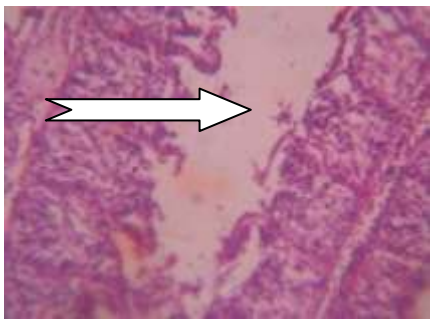
RESULTS

Histopathology of the Gill

No alterations were observed in the gills of the control (plate 1A). The most common pathological changes in the gills of exposed fish species were fusion of lamellae, oedema and hyperplasia of the secondary lamellae (plate 1B).



1A

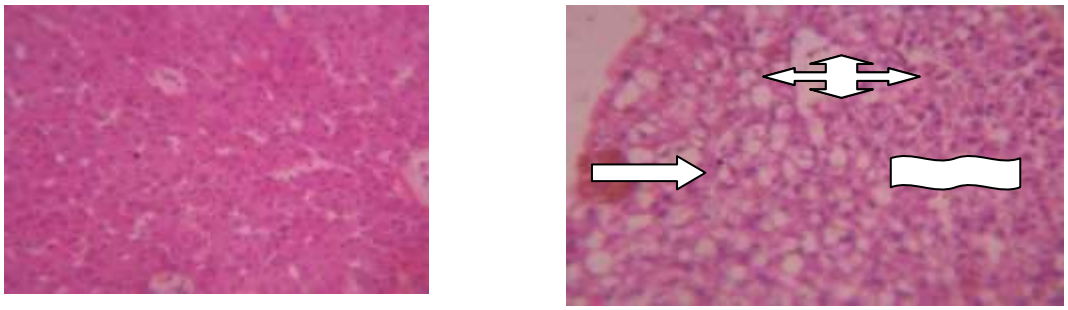


1B

Plate 1: Photomicrograph of a Section of the Gill from a Control Clarias *gariepinus* Showing Normal Appearance of the Gill Lamellae (A) and Photomicrograph of Sections of the Gill of *Clarias gariepinus* Exposed to 20mg/L of Sniper 1000EC (B) Showing Fusion of Lamellae, Oedema and Hyperplasia of Interlamellae ⟹

Histopathology of the Liver

There were no abnormalities in liver of the control groups (Plate 2A. The pathological changes in the liver tissues of the exposed fish species showed distortions with steatosis, vacuolations and necrosis. (Plate 2B).

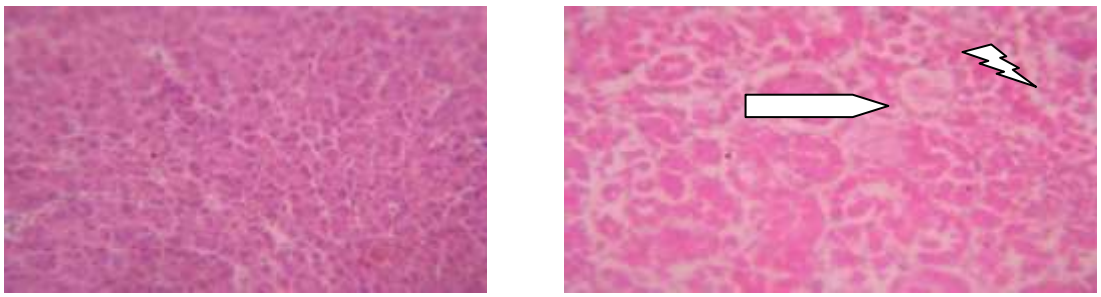


2A 2B

Plate 2: Photomicrograph of a Section of the Liver from a Control *Clarias gariepinus* Showing Normal Appearance (A) and Photomicrographs of Sections of the Liver of *Clarias gariepinus* Exposed to 15mg/L of Sniper 1000EC (B) Showing Steatosis ⇨ Vacuolation ⇦⇨ and Necrosis ⌇

Histopathology of the Kidney

There were no observed distortions in the kidneys of the control group (Plate 3A). Exposed kidneys had marked cellularity of tubules infiltrated by lymphocytes and neutrophils. The interstitial were heavily infiltrated by inflammatory cells. The tubular cells were hypertrophic and the lumina contained armorphous eosinophilic materials (Plate 3B).



3A 3B

Plate 3: Photomicrograph of Section of Anterior Kidney from a Control *Clarias gariepinus* Showing Homogeneous Parenchyma Tissues (A) and Photomicrograph of Sections of the Kidney of *Clarias gariepinus* Exposed to 20mg/L of Sniper 1000EC (B) Showing Tubular Nephrosis ⇨ and Hyperplasia ↯

DISCUSION

Gills are the primary corridor for molecular exchange between the internal milieu of fish and their external environment (Olson, 1996), such as gas transfer, acid-base regulation and ionic regulation (Eddy, 1982). The filament of gills and their secondary lamellae represent two general types of epithelium (Laurent

and Dunel, 1980; Laurent, 1984) that contain three cell epithelia, the pavement, the chloride (ionocyce or mithochondria- rich) cells and the mucous cells, which are most prevalent. In some fish species, the gills contain microvillous or smooth surface lamellar pavement cells (Hossler *et al.,* 1986). Study of the gills in the control fish showed a typical structural organization of the lamella while gills from the fish exposed to the toxicant had several histological distortions, namely clumping/fusion, oedema and hyperplasia of the lamellae. The gill abnormalities observed in this study were similar to previous studies on fish gill morphology, which showed separation of the epithelial layers of secondary gill lamellae, deformation of secondary lamellae and degeneration of chloride cells accompanied by hyperplasia of undifferentiated cells in the primary lamellae (Daye and Garside, 1976; Chevailer *et al.*, 1985). The oedema and hyperplasia in the gills of the exposed fish might be due to acute inflammatory nature of the lesion induced in such gills by the sniper 1000EC. Eller (1970) reported hyperplasia of gill lamella epithelium as findings suggestive of toxicity in several fish species following exposure to several toxicants. Oedema with epithelial separation was reported by (Eller, 1971 and Vanvalin *et al.* 1968) as additional gill change in fish exposed to toxicants. Epithelia sodium pump failure might be responsible for gill oedema (Mitchel and Cotran, 2004). The hyperplasia might in addition be an attempt to increase blood supply to compromised gill in order to increase blood oxygenation. (Gabriel *et al.,* 2007). This could be a compensatory maechanism in the exposed fish to counter hypoxia sequel to edema and obliterated inter-lamellae space, which impeded gaseous exchange across such gills (Gabriel, *et al.,* 2007). Oedema has been reported to increase the diffusion distance between gill capillaries and epithelial cells, thereby increasing internal diffusion resistance, which is a major factor that inhibits gaseous exchange across the affected gills (Turala, 1983). The lamellar fusions are defense mechanisms that reduce the branchial superficial area in contact with the toxicant. These mechanisms also increase the diffusion barrier to the pollutant (Lauren and Mc Donald, 1985; Van Heerden *et al.*, 2004).

Similarly, fusion of adjacent secondary lamellae as a consequence of edema could lead to telangiectasis, which is a characteristic pathologic feature of compromised gills associated with physical or chemical damage (Robert, 2001). However, such structural changes are reported to be non-toxicant specific (Meyer and Hendricks, 1985), which could be

mere stress response of fish, to toxicants exposures (Gabriel*, et al*, 2007). The effect of sniper 1000EC that brought about edema, hyperplasia and laceration of gill lamella explains why fishermen used sniper 1000EC indiscriminately causing heavy fish kills as it destroys the gills, the major respiratory and food filtering organ in fish (Abubakar, 2013). It was recognized that liver is difficult to analyse due to its high histological variability (Kotin, 1967).

However, Myers *et al.*, (1991) and Myers *et al.,* (1992) have described the effects of different toxicants on the livers of various fish species. They showed that the liver distortions developed gradually over the time of exposure and that this varies from individual fishes. They also showed that it is possible that liver could regenerate and recover from those distortions. In this study, the histopathologies of the liver of *C. gariepinus* were carried out with a view to explaining the toxic mechanism of sniper 1000EC. The toxicant caused alterations of the liver parenchyma, such as vacuolization and necrosis. These alterations were often associated with a degenerative- necrotic condition (Myers *et al.,* 1987). This is in agreement with the reports of Omoregie and Ufodike (1990) and Auta, (2001) who separately reported reduced liver vacoulation in *Oreochromis niloticus* exposed to actellic 25EC, paraquat and dimethoate respectively. The reduced vacoulation of hepatocytes may be due to fatty degeneration (Thiyagarajah and Grizzle, 1985; Auta, 2001 and Langiano and Martinez, 2008) Vacuolation of hepatocytes with pycknotic (condensed) nuclei in the liver was most likely due to deposition of glycogen and lipid (Myers *et al.,* 1987) as a result of hepatotoxicity induced by the presence of the toxicant and /or reduced food intake (Khan and Kiceniuk, 1988).

Ferguson (1989) observed that a common morphologic response of fish liver to toxicity is the loss of hepatic glycogen and/or lipid. This condition according to Wolfe and Wolfe (2005) might have occurred by direct intoxication or it might have occurred secondarily to deceased body condition caused by stress or concurrent disease. Furthermore, the authors noted paradoxically that toxic exposure might also result in the accumulation of fats as recorded in this study. The steatosis and necrosis were similar to those reported for fish caught in contaminated water or those exposed to various chemicals under laboratory conditions (Brand

et al., 2001; Rudolph *et al.*, 2001; Marty *et al.*, 2003; Fafioye *et al.,* 2004; Koehler, 2004; Olojo *et al.*, 2005; Camargo and Martinez, 2007; Wahbi and El- Greishy, 2007 and Anilade*vi et al.,* 2008). Fatty degeneration of the liver (haemosiderosis/steatosis) recorded in the liver of the exposed fish might be suggestive of metabolic disorders and it was commonly associated with dietary deficiency in response to xenobiotic (Myers *et al.*, 1987). These changes are normally reported in disease organisms or those exposed to toxicants (Khan and Kiceniuk, 1988; Hawkins *et al.,* 1988; Ogbulie and Okpkwasili, 1999). It might have resulted from disturbances in any of the steps in the sequence of the events from fatty acid entry to lipoprotein exit (Mitchell and Cotran, 2004). The degree of fatty changes in the exposed species exceeded that of the control implicating the toxicant as being responsible for the change. Lipid or glycogen vacuolation suggested the accumulation of triglycerides usually within the hepatocytes and may be responsible for the hepatocyte enlargement/hyalination (Wolf and Wolf, 2005). Steatosis has been correlated with neoplasms; however, its role in the progression of lesions towards neoplasm formation in fish was not well understood (Mc Cain *et al.,* 1982). Similar changes have been reported in the liver of *Astyanax sp* exposed to WSFs of crude oil (Akaishi *et al.*, 2004). *Perca fluviatilis* and gold fish exposed to oil seed process affected water (Nero *et al.*, 2006), centrolobular necrotic change was the main change in the species exposed to the toxicant and agrees with the observation of Popp (1991) who suggested that the distribution of the interstitial system in the liver resulting in a higher concentration of the toxicant in the centrolobular region account for the occurrence and frequency of centrolobular toxicity. The necrosis of the liver tissue might be due to inability of the fish to regenerate new liver cells due to the effect of Sniper 1000EC. Several studies have reported that chronic accumulation of some toxicant in fish livers causes hepatocytelysis, cirrhosis and eventually death (Pourahamad and O'Brien, 2000; Varanka *et al.,* 2001. The kidney has been established as one of the principal site of erythropoietin production (Gordon and Zanjani, 1970).

As in higher vertebrates, the kidneys of fish perform an important function related to electrolyte and water balance and maintenance of a stable internal environment. Thurston *et al.* (1978) reported mild hydrophobic degeneration in renal tubule. The hydrophobic degeneration observed in the kidneys of the exposed fish

species might be due to an increase in the permeability of fish tissues to water, and increased urine out put as reported by Lloyd and Orr (1969) for rainbow trout.

Following the exposure of the fish to the toxicant, histological alterations have been found at the level of tubular epithelium (Teh *et al.,* 1997). Ortiz *et al.* (2003) found kidneys of expoed fish to have received the largest proportion of post-branchial blood, and therefore renal lesions may be good indicators of environmental pollution. Any effects on the kidney are likely to lead to major problems of anaemia and a lack of new blood cells formation. In this study, there was marked diffused tubular nephrosis, hyperplasia of interstitial haemopoietic tissues. The changes recorded in the kidney section from exposed fish were similar to those caused by petroleum in *English sole* (McCain *et al*., 1978 and rats (Dede and Kagbo, 2001) and *C.gariepinus* exposed to plant extracts (Onusiruka and Ufodike, 2000; Fafioye *et al*., 2004) for various periods. Peripheral blood and cephalic kidney of turbot, *Scophthalmus maximus* and Atlantic cod, *Gadus morus* had micronuclei and severe nuclei abnormality such as nuclear buds, binucleated and nonylphenol (Barsiene *et al*., 2006). Exposure, such as changes in kidney tubules particularly at the highest concentration recorded in this study might greatly impair the infiltration functions of the kidney with grave consequences for the exposed fish. However, the onset development and extent of these changes might be toxicant concentration and exposure time dependent.

CONCLUSION

In this study, alterations in gills, livers and kidney in the exposed fish species were associated with the effects of different concentrations of sniper 1000EC. By this context, the toxicant has to be taken into more consideration as an environmental contaminant.

RECOMMENDATION

The use of sniper 1000EC by fishermen should be banned to save the aquatic ecosystem and more studies recommended for further evaluation of this toxicant.

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