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Original article

Histoarchitectural alterations induced by Monocrotophos in the gills and liver of *Heterobranchus longifilis*

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ABSTRACT

Monocrotophos (MCP) is an insecticide whose use in pest control has been banned in developed countries due to its toxicity on non-target organisms. However, in developing countries such as Nigeria, its use still continue because it is cheap and readily affordable. Monocrotophos intoxication can cause oxidative stress and alter the structural integrity of fish. Therefore, this study is aimed at investigating the effects of MCP on the structural integrity of gills and liver in *Heterobranchus longifilis*. *H. longifilis* juveniles were exposed to lethal (0, 0.67, 0.72, 0.77, 0.82 and 0.87 µg/l) and sub-lethal (0, 0.20, 0.25, 0.30 and 0.35 µg/l) concentrations of MCP in a semi-static renewal bioassay for 96 hours and 28 days, respectively. At the end of the bioassays, the gill and liver were excised for histological analysis. Light microscopy analyses revealed that in both exposures, the branchial and hepatic tissues lost their characteristics architecture. Initial response of MCP intoxication was mucus secretion, followed by cellular degeneration, epithelial hyperplasia, chloride cells hypertrophy, necrosis, cell erosion, vacuolation/fatty degeneration and lamellae fusion in gills. The liver showed congestion of blood sinus, focal fibrosis and nuclear degeneration in both lethal and sub-lethal exposures with total loss of architecture at higher concentrations. These structural deformities could affect the metabolism of the fish and thus diminish its fitness for survival.

Keywords: *Heterobranchus longifilis*, Monocrotophos, Gill, Liver, Histopathological alterations.

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INTRODUCTION

Human population increase and the quest to reduce food shortage through the production of agrochemicals such as pesticides and herbicides have led to continuous introduction of harmful pollutants into the environment with its attendant impacts on man's ecosystem. Pollution of water bodies due to anthropogenic activities has so much bearing on the health status of aquatic organisms including fish. Among the different pollutants introduced into aquatic ecosystems, pesticides constitute one of the most potentially harmful pollutants. According to the Stockholm Convention on Persistent

Organic Pollutants, nine out of twelve most dangerous and persistent chemicals are pesticides (Gilden, *et al*., 2010), hence the growing concern on their indiscriminate use worldwide.

Organophosphate pesticides (OPs) are the most commonly used pesticides in the world and due to their toxic effects on non-target organisms, adequate information concerning their toxicity is pertinent (Aktar *et al*., 2009). The exposure of non-target organisms such as fish to a very low level of chemical insult may result in various biochemical, physiological and histological alterations in their tissues (El-Shenawy *et al*, 2009, Dash *et al*., 2011). Fish being the intermediate organisms in the ecological food chain, any debilitating effects of chemicals on them and/or the accumulation of toxic substance in their tissues could cause disequilibrium of the ecosystem in which they inhabit. PAN Europe (2010) and Rathore and Nollet (2012) opined that large scale use of pesticides would not only contribute a huge economic loss but would also cause a lot of undesirable side effects on the environment, biodiversity, food quality and human health. It has been observed that less than 0.1 % of the applied pesticide reaches the target organisms, while the rest 99.9 % has the potential to drift/move into the environment, including surface water and ground water (Racke, 2003).

In North central Nigeria, monocrotophos, Dimethyl (E) -1-methyl-2- (methyl-carbamoyl) vinyl phosphate (MCP) is one of the major pesticides widely used to eradicate insect pests of vegetables, sugar cane, sorghum and rice. It is a broad spectrum OP pesticide and one of the most toxic agrochemicals with wide variation in toxicity between different species of fishes, depending on the rate of absorption, detoxification and inhibition of enzyme acetylcholinesterase (AChE) that break down the neurotransmitter acetylcholine so that impulses can be transmitted across the synapse. Inhibition of AChE by OP drastically affects growth, feeding and reproductive behaviours and can lead to death of fish (Sparling and Fellers, 2007). Although, MCP has been banned in many countries of the world due to its high toxicity (Maniyar, *et al*., 2011), its importation into Nigerian markets still continues due to the perception that it is cheap and readily affordable. MCP may become toxic to beneficial and non-target organisms. In the cotton regions of Paraguay, MCP has been identified as a cause of paralysis in children after three weeks of exposure to the toxicant (Dinham, 1993). In agricultural areas, large amounts of pesticides can be drained and discharged into water bodies as run-off, jeopardizing, not only the survival of exposed organisms, but also the quality of these waters (Monkiedje *et al*., 2004).

There have been several reports on the toxicity of MCP to different fish species including *Oreochromis mossambicus* (Rao, 2006), *Clarias gariepinus* (Yaji and Auta, 2007), *Gambussia affinis* (Kavitha and Rao, 2007), *Channa punctatus* (Agrahari *et al*., 2007), *Cyprinus carpio* (Maniyar *et al*., 2011), *Clarias batrachus* (Narra *et al*., 2011), *Oreochromis niloticus* (Thangnipon *et al*., 1995, Vroumsia *et al*., 2014), *Puntius filamentosus* (Nair and Rathod, 2013) and *Labeo rohita* (Muthukamaravel *et al*., 2013) but most of these information were confined to reporting haemato-chemical changes in fish exposed to varying concentrations of MCP and very little attention has been paid to the effects of MCP on fish histopathology.

Despite the ban on the use of MCP in many countries of the world, in Nigeria it is still being used against birds in rice fields, particularly in North central region. However, there is a dearth of scientific information regarding its toxicity effects on local fish species.

Heterobranchus longifilis, a giant African clariid catfish, is a major source of animal protein for the rural populace and widely distributed in Nigerian waters. It is a very hardy fish that can tolerate a harsh condition and easily adaptable to a varying environment. In Nigeria and, specifically in the North central region, *H. longifilis*, whose seeds and broodstock more often than not are obtained from the wild, occupies an important place in aquaculture production. Water pollution by MCP may therefore pose a serious threat to the fish population and other aquatic animal communities. MCP has been detected in ground, surface, rain, urban and rural water (White, *et al*., 1992) and it may exert harmful effects on the organs of aquatic organisms (Nemcsok *et al*., 1997). Given the growing use of MCP in Nigeria and lack of knowledge about its potential toxicity in local fish fauna, the present study was carried out to examine the toxicity effects of MCP on the histopathology of gills and liver of *H. longifilis*.

MATERIALS AND METHODS

Test chemicals

Analytical grade of monocrotophos with 98% purity was purchased from Sigma-Aldrich (St Louis, USA) and used for the experiment without further purification.

Experimental set up/ Animal maintenance

Juvenile *Heterobranchus longifilis* of average weight (9.46 $\pm$ 0.77 g) and length (11.12 $\pm$ 0.80 cm) and of the same brood stock were obtained from the hatchery of National Institute for Freshwater Fisheries Research (NIFFR), Kainji, Nigeria and transported in oxygenated plastic bags containing water from the hatchery to the laboratory. Prior the trip, no food was administered to the specimen as well as on arrival at the laboratory until the next day; so as to minimize mortality. In the laboratory, the water from hatchery was replaced by well aerated borehole water and fish were held in a large tank of 800 litres capacity and maintained at temperature ranging between 24^{o}C and 27^{o}C using a 300 Watt AZOO submersible thermometer with thermostat, and acclimatized for 14 days. Fish were fed twice daily at 9.00 am and 4.00 pm with commercial feed (pellets) at 3% body weight. Unconsumed food and faecal wastes were siphoned out regularly and water was changed every two days to reduce the risk of mortality due to accumulation and contamination of waste materials. Feeding was stopped 24 hours before the commencement of the experiment. The fish were checked for any infectious disease by monitoring their swimming activities and observing any physical changes.

Water quality parameters

Water quality parameters such as dissolved oxygen (DO), temperature and pH were monitored throughout the experimental period using a digital CS-C933T Electrochemistry multimetre (Topac Instrument, Inc., USA). Conductivity was measured using conductivity meter (Eutech EC Testr 11 Pocket tester) and BOD was determined using a BOD metre (Aqualytic Sensor System, AL606). These probing instruments were dipped into water in

the aquaria and the reading taken accordingly.

Behavioural response, lethal toxicity test and determination of 96 h LC_{50} value of MCP to *H. longifilis*

A range-finding test was conducted to determine the concentrations of toxicant to be used in the experiment using standard procedure following the methods of APHA (1998). Based on the result of the test, five varying definitive concentrations (0.67, 0.72, 0.77, 0.82, and 0.87µg/l) for acute assay and four concentrations (0.20, 0.25, 0.30 and 0.35µg/l) for chronic assay of the MCP and a control (0.00 µg/l) were prepared in glass aquaria in triplicates. Ten acclimatized fishes each of equal weight and size were randomly introduced into each aquarium. The fishes were monitored for 96 hours and 28 days for acute and chronic assay respectively. The test was carried out using a static renewal method to keep the toxicants concentration constant (FAO, 1986). The behaviour of the fishes was monitored and those which did not respond to gentle prodding were considered dead (Rand and Procelli, 1985). After treatment, both the experimental and control fish were sacrificed at the end of 96 hours and 28 days respectively for histopathological studies.

The gills and liver were extirpated and fixed immediately in aqueous Bouin's fluid for 24 hours at room temperature. After fixation, the tissues were washed in running tap water to remove excess picric acid and dehydrated in graded series of alcohol (i.e. 70, 80, 90 and 100%) consecutively and left for one hour, while in the 100% alcohol; it was left for two hours. Tissues were then cleared in xylene and infiltrated in the paraffin wax. Sections of 4-6 µg/l were prepared from paraffin wax using a rotary microtome. The sections were then stained with Haematoxylin-Eosin and histopathological lesions examined and photomicrographic impressions taken, using Carl Zeiss binocular microscope (Axiophot, Germany). Histopathological alterations were assessed using a score ranging from – to +++ depending on the degree and extent of the alteration: - (none), + (mild), ++ (moderate), and +++ (severe) as proposed by Peebua *et al*. (2008). A total of 5 slides were observed per treatment.

RESULTS

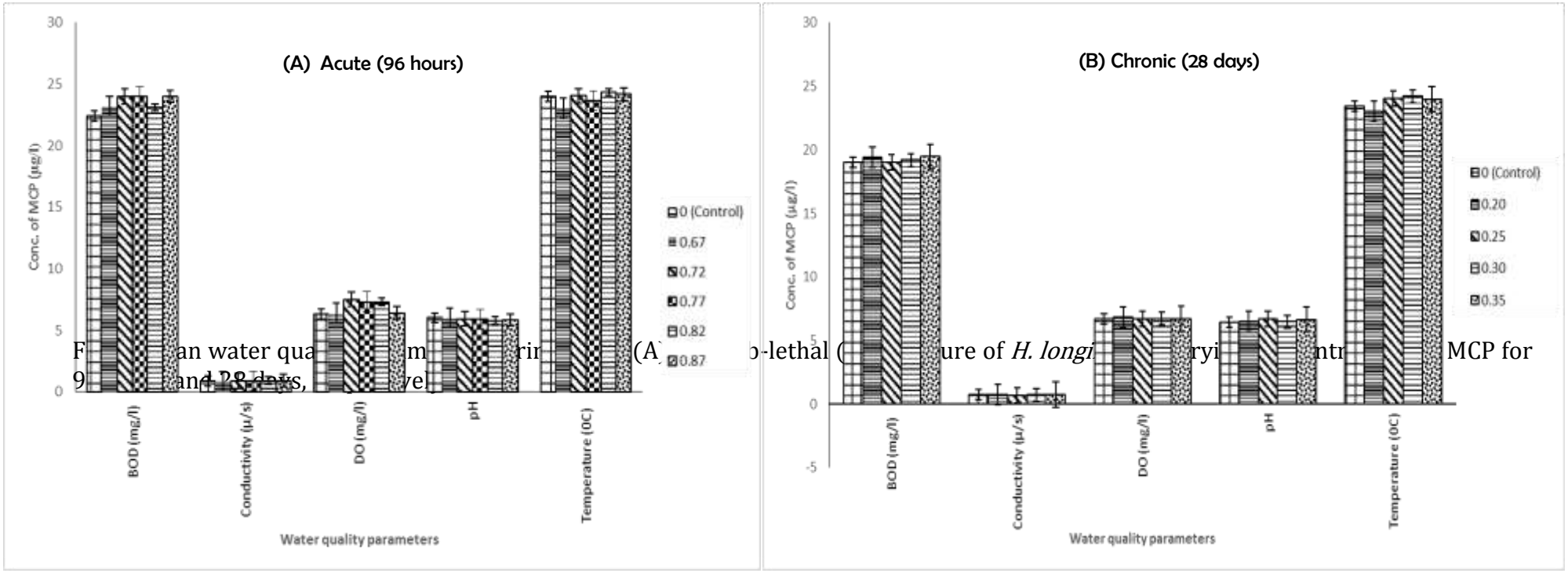
The physicochemical parameters of the test media during the acute and chronic exposure of *H. longifilis* to varying concentrations of MCP are detailed in Figure 1. The parameters did not show any significant difference ($P > 0.05$) between the various concentrations of MCP-contaminated water. All the parameters were within the NESREA (2011) standard except dissolved oxygen (6.30-7.50 µg/l) that was higher.

In both acute and chronic exposures, there were no histological changes in the gills of fish in the control experiment (Figs 2a and 3a). The primary gill lamellae showed laterally compressed leaf-like structures arranged in alternate positions on either side of the interbranchial septum. From each lamella arouse secondary lamellae on both sides perpendicular to its long axis. In MCP-treated fish, the initial response of the gill was the secretion of copious amount of mucus covering almost the entire gill surface. Pathological features such as necrosis, cellular degeneration, epithelial hyperplasia, chloride cells hypertrophy, cell erosion, lamellae fusion and fatty degeneration were

generally observed in gills of fish in both exposures (Figs 2 and 3). The most common gill alterations at all concentrations during acute and chronic assays were cellular degeneration, vacuolation/fatty degeneration and necrosis. These alterations were however, more pronounced at higher concentrations of the toxicant (Table 1).

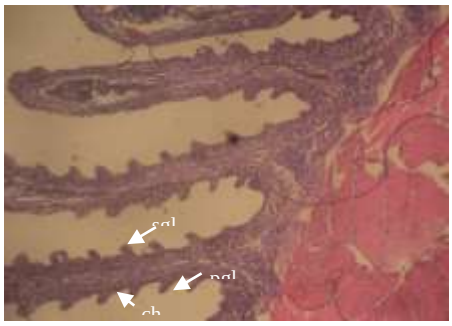
The gills showed mild to moderate alterations in fish exposed to 0.67-0.77µg/l, while at higher concentrations (0.82 and 0.87 µg/l respectively), the alterations were more severe (Table 1). In the acute exposure, fish exposed to 0.67 µg/l exhibited cellular degeneration, epithelial hyperplasia, necrosis and fusion of lamellae while those exposed to 0.72, 0.77, 0.82 and 0.87 µg/l respectively showed massive necrosis in addition to cellular degeneration as compared to the control. Vacuolation made its first appearance in fish exposed to 0.77µg/l (Fig 2) and became severe in fish exposed to 0.87 µg/l, while chloride cell hypertrophy was only observed in fish exposed to the highest concentrations (0.87µg/l). The gills of MCP-intoxicated fish for 28 days, revealed cellular erosion and fatty degeneration in 0.20 µg/l and 0.25µg/l respectively. However, this was in addition to other degenerative changes like cellular degeneration and necrosis found in fish exposed to other concentrations (Fig 2). The changes were more marked in higher concentrations (0.30 and 0.35 µg/l respectively) at the end of 28 days intoxication (Table 1).

Liver of control fish comprised a continuous mass of hepatocytes which are arranged in an irregular cord. The hepatic cells were large, polygonal in shape and contained spherical nuclei. Blood sinuses were observed among the hepatocytes (Figs 3a and 4a). In the liver tissue of fish exposed to varying concentrations of MCP for 96 hours, the hepatocytes depicted various histopathological features such as congestion and dilation of blood sinuses, dense focal fibrosis within the matrices, fatty and nuclear degenerations (Figs 3a-f). Similar pathological features were also found in fish after 28 days exposure. At higher concentrations in both exposures (Figs 3e and f and Figs 4d and e), there was a total loss of liver architecture with more fibrosis, necrosis and vacuolation of the hepatocytes as evident by fatty degeneration. At the lowest concentrations, observed changes were mainly congestion of blood sinuses and cellular degenerations (Figs 3b and 4b). The semi-quantitative histological lesions noticed in the liver of the control and MCP-exposed fishes are detailed in Table 2.

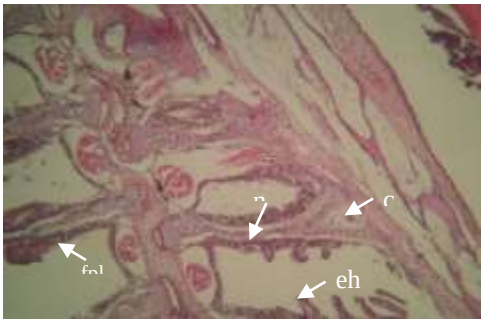


Values are means ± SEM of three replicates. BOD: Biological Oxygen Demand, DO: Dissolved Oxygen **(NESREA Standard: BOD (mg/l) - 30.00; Conductivity (µ/s) - Nd; DO (mg/l) 4.00; pH – 6.00 – 9.00; Temperature^{0}C - 40.00)** Nd: Not determined, NESREA: National Environmental Standards and Regulations Enforcement Agency

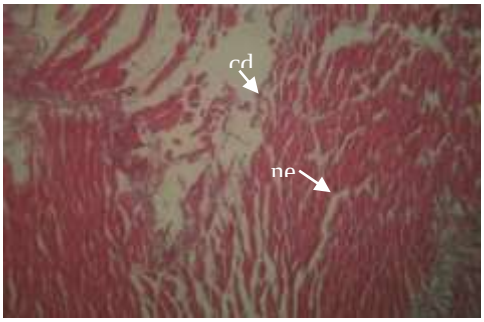
Fig 1: Mean water quality parameter during lethal (A) and sub-lethal (B) exposure of *H. longifilis* to varying concentrations of MCP for 96 hours and 28 days, respectively



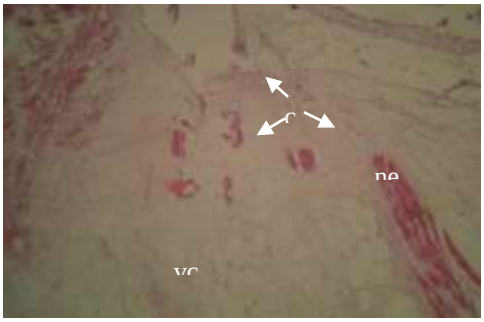
(a)



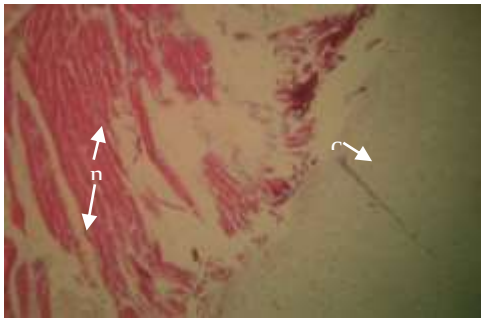
(b)



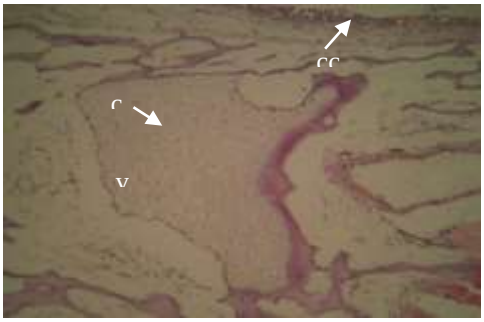
(c



(d)



(e)



(f)

Fig 2: Histopathology of the gill tissue of *H. longifilis* exposed to different concentrations of MCP for 96 hours. (a) Normal cellular architecture in the gill of fish in control experiment showing PGL-primary gill lamella, SGL-secondary gill lamella, CHC-chloride cell X400; b-f-gill with alterations: (b) fusion of primary lamellae (fpl), fusion of secondary lamellae (fsl), necrosis (ne), cellular degeneration (cd), chloride cell hypertrophy (cc), epithelial hyperplasia (eh), in the fish exposed to 0.67µg/l of MCP for 96 hours (c, d, e & f) cellular degeneration (cd), chloride cells hypertrophy (cc), vacuolation (vc) and necrosis (ne) in fish exposed to 0.72µg/l, 0.77µg/l, 0.82µg/l and 0.87µg/l of MCP respectively.

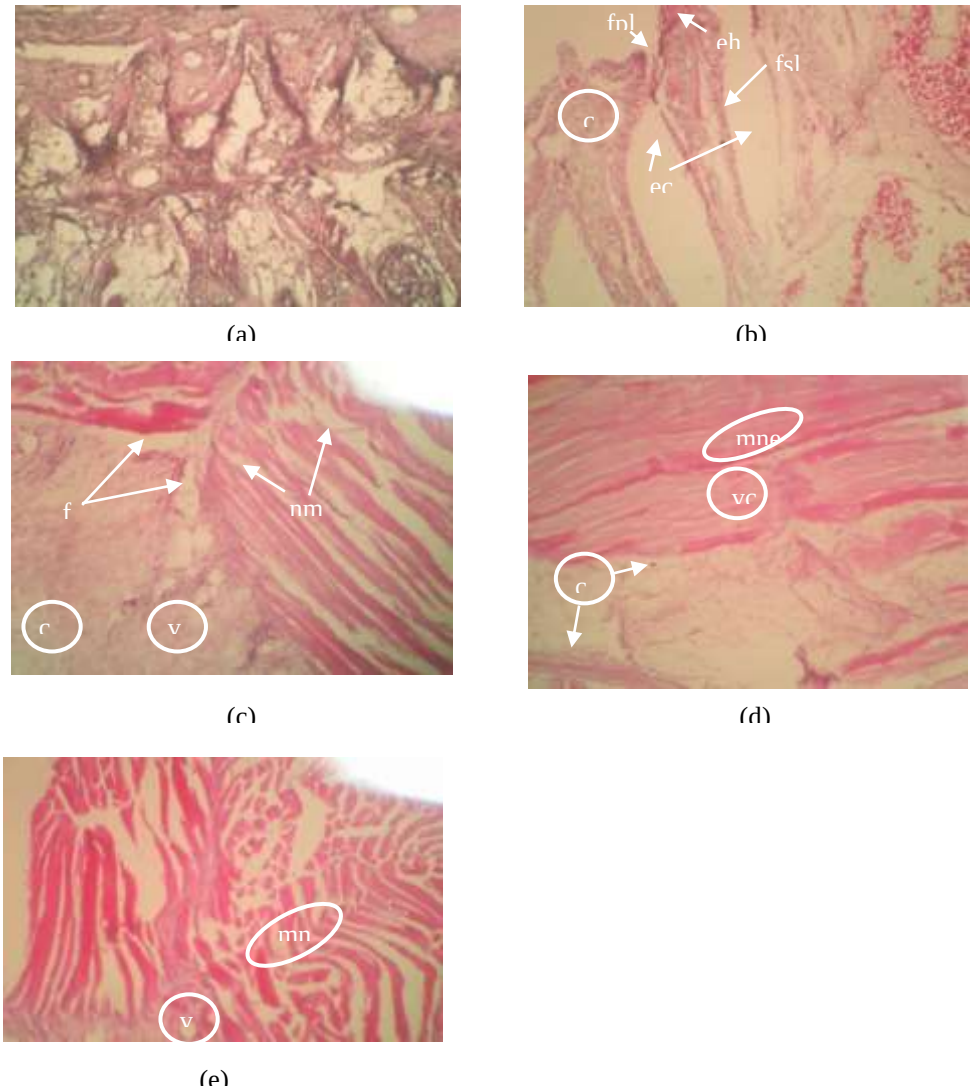


Fig 3: Histopathology of the gill tissue of *H. longifilis* exposed to different concentrations of MCP for 28 days. (a) Normal gill architecture in control experiment X400, b-e gill with alterations: (b) fusion of primary lamellae (fpl), fusion of secondary lamellae (fsl), cellular degeneration (cd) and erosion of cells (ec) in fish exposed to 0.20µg/l of MCP (c) fatty degeneration (fd) massive necrosis (mne), cellular degeneration (cd) and vacuolation (vc) in fish exposed to 0.25 µg/l of MCP, (d) massive necrosis (mne), cellular degeneration (cd), vacuolation in fish exposed to 0.30 µg/l of MCP (e) massive necrosis (mne) and vacuolation (vc) in fish exposed to 0.35 µg/l of MC

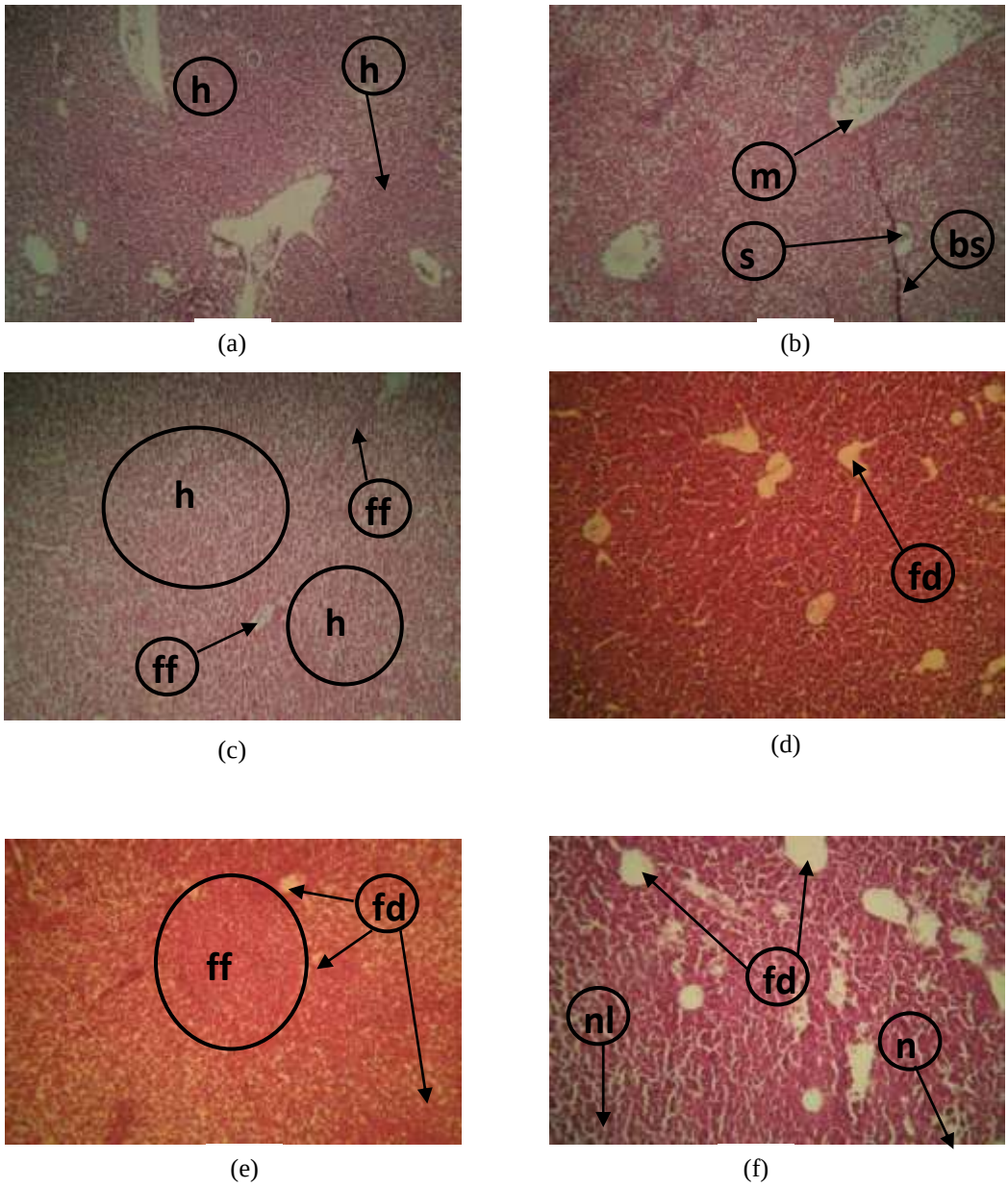


Fig 4: Histopathology of liver tissue of *H. longifilis* exposed to varying concentrations of MCP for 96 hours.
(a) Normal cellular architecture in the liver of fish in control experiment, (hp) hepatocytes, (hv) hepatic vein, b-f liver with alterations X400: (b) congestion of sinus(s), blood sinus (bs), melanomicrophage centre (mc) in liver of fish exposed to 0.67µg/l of MCP for 96 hours, (c) focal fibrosis (ff); hepatocytes (hp) in liver of fish exposed to 0.72µg/l of MCP for 96 hours, (d-f) fatty degeneration (fd) in liver of fish exposed to 0.77µg/l, 0.82 µg/l and 0.87 µg/l of MCP, respectively. (e) focal fibrosis (ff) in the liver of fish exposed to 0.82 µg/l of MCP, (f) necrosis of liver tissues (nl), nuclear degeneration (nd) in liver of fish exposed to 0.87 µg/l of MCP.

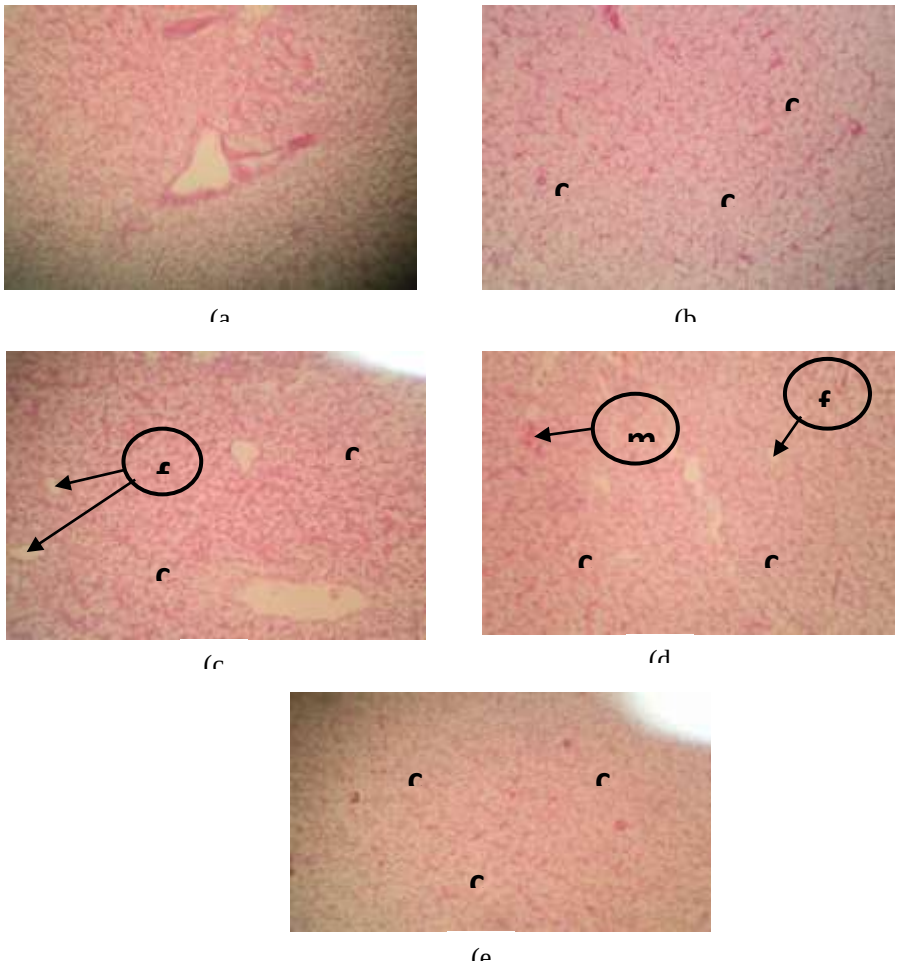


Fig 5: Histopathology of liver tissue of *H. longifilis* exposed to varying concentrations of MCP for 28 days

(a) Normal cellular architecture in the liver of the fish in control experiment X400, b-e liver with alterations: (b) cellular degeneration (cd) in liver of fish exposed to 0.20 µg/l of MCP, (c) fatty degeneration (fd), cellular degeneration (cd) in the fish exposed to 0.25 µg/l of MCP, (d) fatty degeneration, melanomicrophage centre (mc), cellular degeneration(cd) in the fish exposed to 0.30 µg/l of MCP and (e) cellular degeneration (cd) in the fish exposed to 0.35 µg/l of MCP.

Table 1: Semi quantitative scoring of gills lesions in *Heterobranchus longifilis* during acute (96 hours) and chronic (28 days) MCP exposure

Lesion	Exposure								
	Acute (96 h)					Chronic (28 d)			
	Concentration (µg/l)								
	0.67	0.72	0.77	0.82	0.87	0.20	0.25	0.30	0.35
Cellular degeneration	+	+	++	+++	+++	+	++	+++	+++
Cell erosion	-	-	-	-	-	+	++	+++	-
Epithelial hyperplasia	+	-	-	-	-	+	-	-	-
Fusion of primary lamellae	+	++	+++	+++	+++	+	++	+++	+++
Fusion of secondary lamellae	+	++	+++	+++	+++	+	++	+++	+++
Necrosis	+	++	++	+++	+++	+	++	+++	+++
Hypertrophy of chloride cells	+	-	-	-	+++	-	-	-	-
Vacuolation/Fatty degeneration	-	-	+	-	+++	-	+	++	+++

- = none, + = mild, ++ = moderate, +++ = severe

Table 2: Semi quantitative scoring of liver lesions in *Heterobranchus longifilis* during acute (96 hours) and chronic (28 days) MCP exposure

Lesion	Exposure								
	Acute (96 h)					Chronic (28 d)			
	Concentration (µg/l)								
	0.67	0.72	0.77	0.82	0.87	0.20	0.25	0.30	0.35
Cellular degeneration	-	+	++	+++	+++	+	++	+++	+++
Cell erosion	_	_	_	_	_	-	_	_	_
Congestion of sinus	+	++	++	+++	+++	-	++	++	+++
Epithelial hyperplasia	+	_	_	_	_	_	_	_	_
Necrosis	+	++	++	+++	+++	+	++	+++	+++
Hypertrophy of chloride cells	_	_	_	_	+++	_	_	_	_
Fatty degeneration	_	+	+	++	+++	_	+	++	+++
Focal fibrosis	-	+	++	++	++	-	++	++	+++
Melanomicrophage centre	+	-	-	-	-	-	-	-	++
Nuclear degeneration	-	-	-	++	++	-	++	++	+++

- = none, + = mild, ++ = moderate, +++ = severe

DISCUSSION

As an indicator of contaminant MCP has been reported to be very toxic to fish (Rao, *et. al.*, 2005, Rao, 2006). The present study clearly demonstrated that even trace level of the toxicant induces pathological lesions in gills and liver of *H. longifilis*. Histological alterations brought about by waterborne pollutants can easily be observed in fish because the gills come into immediate contact with the

environment. The survival of *H. longifilis* throughout the duration of exposure in both acute and chronic assays may be attributed to the quality of water in the experimental set up, good enough to be tolerated by the fish. The non-significant values recorded for the physico-chemical parameters attest to the fact that the values are normal for toxicity test (FAO, 1977). All the parameters were within the standard range recommended for fish culture and/or survival (Alabaster and Lloyd, 1980, NESREA, 2011). However, the likely accumulation of MCP in the tissues of *H. longifilis* might have produced the pathological responses observed in this study. The gill covers more than 60% of fish surface area and its external location and high pesticide absorption makes it the most vulnerable target organ for pollutants (Roberts, 1978). Fish internal environment is separated from the external one by only a few microns of delicate gill epithelium and thus the branchial function is very sensitive to environmental contaminants.

Srivastav *et al*. (1997) reported that a high rate of pesticide absorption is through the gills, hence the severe histopathological changes noticed in the gills of *H. longifilis* in the present study. Several other studies have shown similar branchial responses to OP pesticides on fish. Degenerative changes in gills such as cellular degeneration, necrosis, coverage of gill filaments with mucus, dilation and congestion of blood sinuses, hypertrophy and hyperplasia observed in this study have been reported in many species exposed to MCP and other OP pesticides (Santhakumar, *et. al.*,2001, Rao, *et al.*, 2005, 2006; Velmurugan, *et al.*, 2007). Patil and David (2000) remarked that the gills are the major respiratory organs and all metabolic pathways depend on the efficiency of the gills for their energy supply and damage to these vital organs causes a chain of destructive events, which may ultimately lead to respiratory stress. The proliferative thickening (hyperplasia) of epithelial cells, cellular degeneration, cell erosion, necrosis and hypertrophy of chloride cells observed in this study might result in hypoxic condition and reduction in the ion-transport capacity of the gill leading to respiratory failure. Although hyperplasia could be a defensive mechanism employed by the fish to wade off MCP toxicity by decreasing the available respiratory surface area, the adoption of such mechanism could also lead to an increase in toxicant-blood diffusion distance leading to a decreased oxygen-uptake capacity of the gills. The histological lesions observed in the gills of *H. longifilis* as a result of waterborne toxicity of MCP is an indication of impending gill damage prior the death of the experimental fish and these might have been responsible for respiratory stress as evident from the erratic behaviour of the fish (Owolabi and Adesida, 2015).

The secretion of mucus as initial response of the fish to MCP-intoxication also appears to be a coping strategy toward the toxicant. Mucous cells could be of advantage in trapping the entry of toxicant into the sensitive gill epithelium, thus avoiding erosion and damage of respiratory surface which could affect the reduction in gaseous exchange between blood and water (Rao *et al.*, 2005). Although, mucous cells proliferation may be helpful in reducing toxicant entry, the end result could still be an increase in the distance for gaseous exchange along the

secondary lamellae; thus reducing the efficiency of gas exchange and causing hypoxic condition. Increased mucus secretion by the gills is a common response to aquatic pollutants (Shepard, *et al*., 1994, Scheckat, *et al*., 2002). However, the likely negative effects this may have on the respiratory process may be minimised by the species ability to resort to atmospheric air to fulfil its oxygen requirements, making its gill an oxy-regulator (Jumawan, *et al*., 2010). It has been shown that catfishes generally have accessory breathing organs with which they supplement deficiency of oxygen. Fish can alter branchial cell turn-over and gill dimensions in order to adapt to increased metabolic demands during pollutant exposure (Dang, *et al*., 1998, Handy, *et al*., 2002).

The liver is the primary organ responsible for accumulation, biotransformation and excretion of xenobiotics (Hinton, *et al*., 2001) and hence undergoes different levels of damage as a consequence of these processes. Any structural changes in the liver may therefore be useful as biomarkers of exposure to environmental stressors. In this study, fatty and nuclear degenerations exhibited by the liver were consequences of highly vacuolated cells and may probably be attributed to the accumulation of the toxicant in the cytoplasm of hepatocytes and their nuclei. Necrosis of the liver tissue might have resulted from the gross inability of the fish to regenerate new liver cells and/or the inability to meet the overwhelming energy required to detoxify the toxicant from its body. This is consistent with the observations of Ezemonye and Ogbomida (2010). Liver changes affect the metabolism of fish, diminishing its fitness and the health becomes weakened, thus making the fish more vulnerable to predation. The reproductive capacity of the fish may also be reduced and they may lose competition for space or food in their habitat.

Qualitatively, almost a uniform pattern of pathological response were noticed in the gills and liver of *H. longifilis* and few tissue-specific damages were observed, though the organs showed more damages at higher concentrations as the magnitude of damage was directly related to the concentration of MCP and exposure period. However, the present study suggests that a period of 96 hours of exposure to MCP was just enough to generate reactive oxygen species (ROS) which alters the normal structural architecture of the gills and liver of the fish and this could lead to loss of normal physiological functions of the fish. The environmental implication of this study is obvious. The levels of MCP in water bodies close to rice farms where MCP is mostly used, which, unfortunately, dearth of information exist in Nigeria; raises the concern of possible bioaccumulation of this toxicant not only in *H. longifilis* but also among other fishes. This could pose serious danger to the health of fish and consumers. Effort to ascertain the levels of MCP in waters close to rice farms where the pesticide is mostly used should therefore be a future concern for risk assessment and biomonitoring studies. Bioaccumulation studies of this toxicant in the tissues of this fish and other fishes with which they cohabit, along with other specific parametric analysis for stress response in field studies would be required to further support laboratory investigation of this nature.

Conclusion

This study has shown that exposure to varying concentrations of MCP caused destructive effect in the gills and liver of *H. longifilis*. Gills and liver alterations such as these as well as those observed in previous studies, could directly affect the metabolism of fish and therefore diminish their fitness for survival. This might have informed the ban of MCP in most developed countries of the world and same should be extended to developing countries where MCP is still being used.

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