# Responses of Two Life Stages of *Oreochromis niloticus* to Acute Toxicity of Ridoff (Organophosphat Insecticide)

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***Abstract***

*The acute toxicity of Ridoff (Insecticide dichlorvos) on two life stages of Oreochromic niloticus of mean standard length and weight (fingerlings - 4.20 ± 0.60 cm and weight - 7.00±0.20g and juveniles - 12.04 ± 0.20cm and 14.04±0.05g) were investigated under laboratory condition at the concentrations of 0.05, 0.08, 0.10, 0.13 and 0.15ml/l. All the physicochemical parameters (ammonia, dissolved oxygen, pH, temperature and nitrite) under consideration were within acceptable limits. $LC_{50}$ of Ridoff was calculated at 24, 48, 72 and 96 hours using probit test. Mortality was observed in all treatments of the two life stages exposed from 0.08mg/l at 96 hours. In the first life stage (fingerlings), LC50 was 0.058 within 24 and 96hours and the second life stage (post fingerlings) $LC_{50}$ was 0.019 within 24 and 96 hours. At a close watch the two life stages exhibited agitated swimming, loss of equilibrum, air gulping, period of quiescence, and turned on flank and swarm in circles and finally died. Accumulation of mucus also was observed on the gill filaments and body surface of the dead fish on exposure to toxicant. Ridoff (Dichlorvos) indeed is harmful to Oreochromis niloticus, which could be a non-target organism in an aquatic ecosystem.*

***Keywords:*** *Acute toxicity, dichlorvosl, behaviour, Oreochromis niloticus*

## 1.0 INTRODUCTION

Over the last few decades, due to the significant impacts on aquatic flora and fauna, the problem associated with environmental pollution has been a concern worldwide (Alan and Maughan 1993). Toxic organic pollutants that include a large number of agrochemicals, such as pesticides, many of which are non-biodegradable and carcinogenic, are consistently being used in crop fields. As a result, fish and other aquatic biota, exposed to the pesticide-contaminated water are at much higher risk of dying, Elahee and Bhagwant (2007). An organophosphate insecticide is a liquid with a pale yellow to amber color and a garlic-like odor. It was first registered in the United States in 1982. The Environment Centre of National Toxicology declared that, there are a dozen highly dangerous

chemicals including Ridoff (Dichlorvos). Ridoff (Dichlorvos) is a hard insecticide, which has become a matter of concern because of its potentiality and hazardous effect. Seepage of pesticides into rivers and streams can be highly lethal to aquatic life and often might change the bionetwork of a particular area (Ghaffar, *et al*., 2015.). Moreover, repeated exposure to sublethal doses of some pesticides can cause physiological, behavioral, and environmental modifications by endangering fish population, abandonment of nests and broods, decreased immunity to diseases, and decreased predator avoidance (ASTM (1977). Additionally, pesticides can accumulate in water bodies and affect the source of food for young fish by actively altering the trophic levels (Alan, and Maughan, 1993). Pesticides can also abruptly alter the lower trophic levels that instigate the fish to forage further, exposing them to greater risks of predators. Generally, insecticides are more toxic to aquatic life than herbicides and fungicides (Hossain, *et al.*, 1987), therefore, their tremendous use for the domestic sphere should be reconsidered. However, the sooner a pesticide degrades in the atmosphere, the less menace it might cause to aquatic life (Gill *et al*.,1991). Envoy 50 SC is a wide-ranging commonly used organophosphate (OP) insecticide, used commercially to control foliar insects in croplands, ASTM (1977) Accumulation of this OP insecticide in different aquatic organisms, particularly in fish through air drift or surface runoff adversely affects them (Anees,1978).This chemical is a well-known acetyl cholinesterase inhibitor, which plays a crucial role in neurotransmission by rapid hydrolysis of neurotransmitter acetylcholine (ACh) to choline and acetate at cholinergic synapses (Rath and Misra,1981). Therefore, they can alter the neurological responses of non-target organisms even at very low concentrations (Rao, *et al*., 2017.). Acetyl cholinesterase (AChE) is also said to be a functional key enzyme of the nervous system for the termination of the nerve impulses by hydrolyzing the neurotransmitter acetylcholine (Varo, *et al.*, 2003). Inhibitions of AChE results in the accretion of acetylcholine in the central and peripheral synapses and subsequently modify the physiological and neuro endocrine processes (Rath and Misra,1981). Such physiological variations can lead to a succession of behavioral changes that include impeded swimming performances, altered social behavior, reduced foraging, and greater predation risks. Therefore, AChE is also a widely used biomarker to give insight to the environmental and pathological perspectives (Rath and Misra,1981 and Lotti, 2001).

## 2.0 MATERIALS AND METHODS

**2.1. Experimental site:** University of Port Harcourt demonstration farm. Fisheries Department.

**2.2. Experimental fish:** Tilapia fish (*Oreochromis niloticus)*.

**2.3 Procurement of the pesticide:** Agriculture grade organo phosphorus pesticide compound, Ridoff in original sealed container was procured from an authorized dealer in Rivers state, Nigeria. It was in liquid form and white in colour. The expiry date of the test pesticide was checked prior to start of the experiment.

**2.4** Definitive test for the two life stages of *Oreochromis niloticus* exposed to different concentrations of Ridof (Organophosphate insecticide) from 24 hours to 96 hours were properly carried out using serial dilution method.

## 3.0 Physico-chemical parameters assessed.

**3.1. pH**: The effluent was collected in clean glass bottle. The pH of the samples was measured on getting to the laboratory, using model 291 Mk 2 pH meter produced by Hannah INC. LTD, China. The pH meter was first calibrated using standard buffer solutions of pH7. This was carried out by pouring small amount of the buffer, pH 7 into a clean beaker and a magnetic stirrer bar dropped into it and the beaker placed on magnetic stirrer to get a homogenous mixture. The pH meter electrode was lowered into the beaker, so that the tip was immersed in the buffer solution and the magnetic stirrer started. The meter was adjusted to read the pH value of the buffer. The electrode was removed, and washed with distilled water and dried with soft tissue paper.

### 3.2 Ammonia - Nitrogen ($NH_3$ - N)

The phenate method of ammonia determination (APHA, 1998) was adopted for the study. To 10.0ml sample in a 50m1 beaker, one drop $MnSO_4$ solution was added. This was then placed on a magnetic stirrer and 0.5m1 hypochlorous acid reagent was added. Thus reagent was added without delay using a burette. The set up was stirred vigorously during addition of reagent. The procedure was carried out for blank and standard, followed by measurement of absorbarice at 630nm using reagent blank to zero the spectrophotometer.

### 3.3. Dissolved Oxygen (DO)

The Winkler's method (APHA, 1998) was used in detecting the dissolved oxygen in each of the aquarium. Water samples for dissolved oxygen were collected and to these were added, 1ml of potassium fluoride solution, 2ml alkaline iodine solution, 2ml manganese sulphate solution and 2ml concentrated sulphuric acid. Then 200ml of the treated sample was used in the titrimetry and the liberated iodine in the treated sample was titrated with a prepared standard of 0.025N sodium through sulphate titrant. Two milliliters starch indicator was added to the sample, when its colour turned pale straw in the process of titration, the value was recorded and used in the calculation.

**Calculation**

Average volume of 0.025N used = 5.65ml

Volume of treated sample = 200ml

Collected sample aliquot = $\frac{200 \times 300}{300 - T}$

The 200ml sample aliquot for the reagents was corrected to 300ml.

T = total volume of reagents added to the sample bottle

Thus DO = $\frac{A \times N \times 8000}{B}$ (APHA 1998)

Where; A = Millimeters of titration sample

B = Millimeters of corrected sample aliquot

C = Normality of sodium throsulphate

**Temperature**

Temperature measurements were made with a mercury-filled Celsius thermometer. Sufficient time was allowed for the thermometer to reach a constant reading. The thermometer readings were recorded in 0c for the samples.

**Nitrite**

The nitrite test was conducted by using a nitrite reagent spoon to collect a levelled spoon full of the reagent. This was put into a 5mls water sample calibrated test tube then stirred. At this point, there was a color change which matched with the reagent color chart.

## 4.0 RESULTS

**4.1 Physico-chemical parameters of the experimental water:** The mean physic-chemical water parameters from the experiment after 96 hours are presented in Table 4.1. The temperature value ranges from 26.00±0.0°C in 0.15ml/l and 0.13 ml/l concentrations to 27±0.0°C in 0.05, 0.08 and 0.10 ml/l concentration respectively. Nitrite did not differ significantly (P>0.05) across the five different concentrations (0.01±0.0µg/l).The Ammonia values ranged between 0.4±2.78 µ/dL to 1.5±0.0 µ/dL. The highest value (1.5±0.0 µ/dL) was observed at 27±0.0°C in 0.05 ml/l 0.08 ml/l and 0.10 ml/l respectively while the least value (0.4±2.78 µ/dL) was recorded in 0.15ml/l concentration of Organophosphate insecticide with significant variations at 26.00±0.0. The Dissolve Oxygen (mg/l) varied significantly (P < 0.05) with the highest value (4±0.0 mg/) recorded in 0.05ml/l concentration while the least value (4±0.0 mg/l) was recorded in 0.15 ml/l of the test chemical. The highest pH value (7.2±0.0) was recorded in 0.05 ml/l, 0.08 ml/l and 0.10 ml/l concentrations while the least value (6±0.0) was at 0.15ml/l and 0.13 ml/l concentrations respectively.

**Table 4.1:** Mean water parameters after exposure for 96 hours.

| **WATER PARAMETERS** | | | | | |
|---|---|---|---|---|---|
| **Conc. (ml/l)** | **Ammonia (µ/dL)** | **Dissolve Oxygen (mg/l)** | **pH** | **Tempt. (°C)** | **Nitrite (µg/l)** |
| **0.05** | 1.5±0.0[a] | 4±0.0 [a] | 7.20±0.0 [a] | 27.00±0.0 [a] | 0.01±0.0 [a] |
| **0.08** | 1.5±0.0 [a] | 2±0.0 [b] | 7.00±0.0 [a] | 27.00±0.0 [a] | 0.01±0.0 [a] |
| **0.10** | 1.5±0.0 [a] | 1.7±0.24 [b] | 6.80±0.0 [ab] | 27.00±0.0 [a] | 0.01±0.0 [a] |
| **0.13** | 1.3±0.14 [ab] | 1±0.0 [c] | 6.60±4.4 [ab] | 26.00±0.0[b] | 0.01±0.0 [a] |
| **0.15** | 0.4±2.78 [b] | 1±0.0 [c] | 6±0.0 [b] | 26.00±0.0[b] | 0.01±0.0 [a] |

*Means with different superscripts across the rows are significantly different.
*Means with the same superscript across the rows are not significantly different

The number of mortalities recorded for the first life stage of *Oreochromis niloticus* exposed to different concentrations of Organophosphate insecticide is presented in Table 4.2. There was statistical significance ($P<0.05$) in the number of mortalities observed in the five concentrations from 24 hours to 96 hours and higher mortality recorded were from the higher concentrations of the test chemical. There was an increase in the percentage of mortality after 96hours of exposure to the test chemical with increase in concentrations of exposure as seen in the cumulative mortality (Figure 4.1).The Median lethal concentration of $LC_{50}$ was 0.058ml/l after 96 hours of exposure of the test fish samples. The $LC_{50}$ and the acute toxicity test after exposing *Oreochromis niloticus* to Organophosphate insecticide recorded the linear and regression equation with lower 95% and upper 95% values of 0.047 % and 0.071% respectively (Table 4.2).The cumulative death rate recorded after exposure of *Oreochromis niloticus* to Organophosphate insecticide was time-dependent and increased with exposure time (Figure 4.1). The Plot of Log of Concentration Versus Probit at 96Hrs exposure to Ridoff (Organophosphate insecticide) is Figure 4.2.

**Table 4.2: Mean values of mortality recorded after 24 to 96 hours of exposure of the first life stage of *Oreochromis niloticus* to Organophosphate insecticide.**

| Conc. (ml/l) | Mean mortality | | | | % Mortality |
|---|---|---|---|---|---|
| | 24hrs/day 1 | 48hrs/day 2 | 72hrs/day 3 | 96hrs/day 4 | |
| **0.05** | 1.33±0.57$^{b}$ | 1.33±0.0$^{b}$ | 2.47±2.1$^{c}$ | 2..5±2.0$^{d}$ | 78.57 |
| **0.08** | 1.67±1.0$^{b}$ | 3.67±1.0$^{a}$ | 4.50±0.1$^{b}$ | 4.55±0,0$^{c}$ | 79.28 |
| **0.10** | 2.33±0.0$^{a}$ | 3.33±1.1$^{a}$ | 4.33±0.0$^{b}$ | 5.6±0,01$^{b}$ | 80 |
| **0.13** | 2.33±1.0$^{a}$ | 3.33±1.15$^{a}$ | 4.67±1.0$^{ab}$ | 5.67±.06$^{ab}$ | 81 |
| **0.15** | 2.67±0.2$^{a}$ | 3.67±2.61$^{a}$ | 5.0±0.57$^{a}$ | 6.0±0.00$^{a}$ | 100 |

*Means with the same superscript down the column are not significantly different
**Means with different superscripts down the column are significantly different.

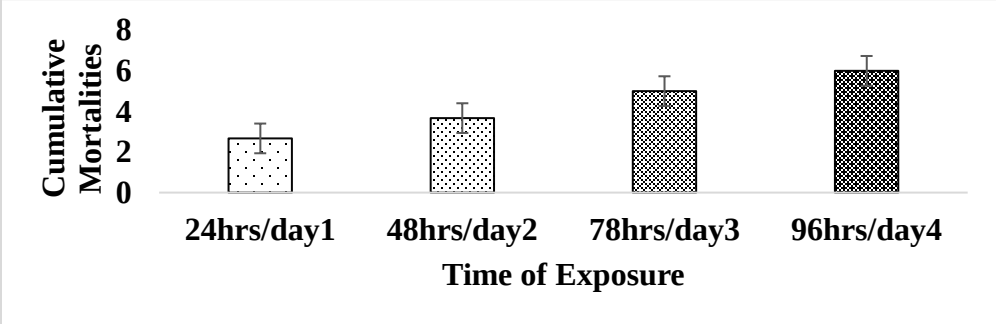


**Figure 4.1:** Cumulative Mortalities of the first life stage of *Oreochromis niloticus* exposed to different concentrations of Organophosphate insecticide.

**Table 4.3:** The $LC_{50}$ and the Acute Toxicity Test After exposing *Oreochromis niloticus* to Organophosphate insecticide.

| Life stages | Time (hrs.) | $LC_{50}$ | Lower 95% | Upper 95% | Regression Equation |
|---|---|---|---|---|---|
| First Stage | | | | | y = 5.3674x + 11.645 |
| | 72 | 0.058 | 0.047 | 0.071 | $R^2$ = 0.9612 |
| Second Stage | 96 | | | | y = 1.9159x + 8.3048 |
| | | 0.019 | 0.010 | 0.034 | $R^2$ = 0.9218 |

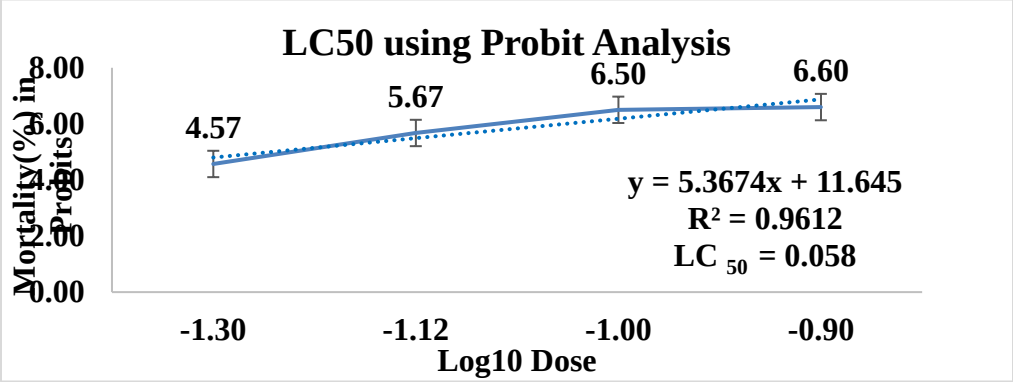


**Figure 4.2**: The Plot of Log of Concentration Versus Probit at 96Hrs exposure of the first life stage of *Oreochromis niloticus* to Ridoff (Organophosphate insecticide).

**4.2 Mean values for** the **second life stage of *Oreochromis niloticus* exposed to different concentrations of Organophosphate insecticide from 24 hours to 96 hours.**

The number of mortalities recorded for the second life stage of *Oreochromis niloticus* exposed to different concentrations of Organophosphate insecticide is presented in Table 4.2. In this table, the highest mortalities were recorded in the highest concentrations of the test chemical with change in time. There was significance ($P<0.05$) in the mortalities recorded in the five concentrations during the 96 hours assay. There was also an increase in the percentage of mortality after 96hours of exposure to the test chemical with increase in concentrations of exposure (Figure 4.3).The Median lethal concentration of $LC_{50}$ was 0.019ml/l after 96 hours of exposure of the test second samples. The $LC_{50}$ and the acute toxicity test after exposing this stage of *Oreochromis niloticus* to Organophosphate insecticide recorded the linear and regression equation with lower 95% and upper 95% values of 0.010 % and 0.034 %respectively (Table 4.3).The cumulative death rate recorded after exposing the second stage of *Oreochromis niloticus* to Organophosphate insecticide was also time-dependent and increased with exposure time (Figure 4.4). The Plot of Log of Concentration Versus Probit at 96Hrs exposure to Ridoff (Organophosphate insecticide) - Figure 4.4.

**Table 4.2:** Mean values of the mortality recorded after 24 to 96 hours of exposure of the second life stageof *Oreochromis niloticus*to Organophosphate insecticide.

| Conc. (ml/l) | Mean mortality | | | | % Mortality |
|---|---|---|---|---|---|
| | 24hrs/day 1 | 48hrs/day2 | 72hrs/day 3 | 96hrs/day 4 | |
| **0.05** | 1.33±0.57$^{c}$ | 2.330.57$^{c}$ | 3.33±0.6$^{d}$ | 5.67±0.6 | 81 |
| **0.08** | 1.33±2.6$^{c}$ | 1.33±2.6$^{c}$ | 3.33±1.0$^{d}$ | 6.20±0.0 | 85.71 |
| **0.10** | 1.66±2.1$^{c}$ | 2.67±2.2$^{c}$ | 4.33±1.5$^{c}$ | 6.33±0.0 | 90.42 |
| **0.13** | 3.67±1.0$^{b}$ | 4.33±0.0$^{b}$ | 5.33±1.5$^{b}$ | 6.67±2.1 | 95.28 |
| **0.15** | 4.00±1.5$^{a}$ | 5.00±2.2$^{a}$ | 6.33±2.6$^{a}$ | 7±0.0 | 100 |

*Means with the same superscript down the column are not significantly different
**Means with different superscripts down the column are significantly different.

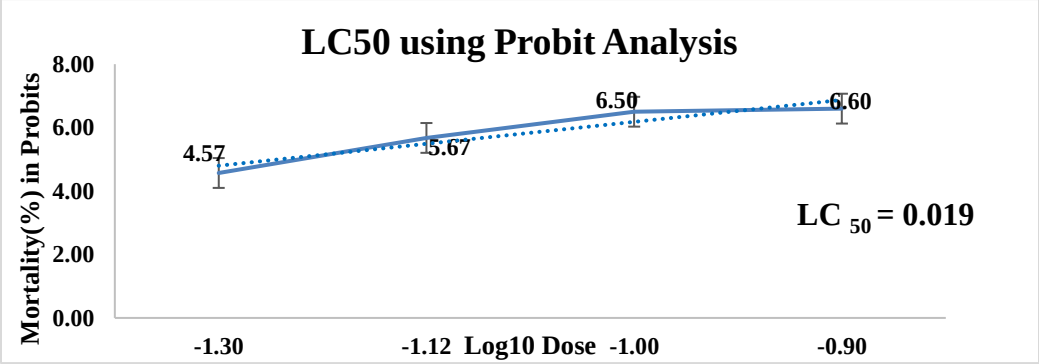


**Figure 4.3**: The Plot of Log of Concentration Versus Probit at 96Hrs exposure of the second life stageof *Oreochromis niloticus*to Ridoff (Organophosphate insecticide).

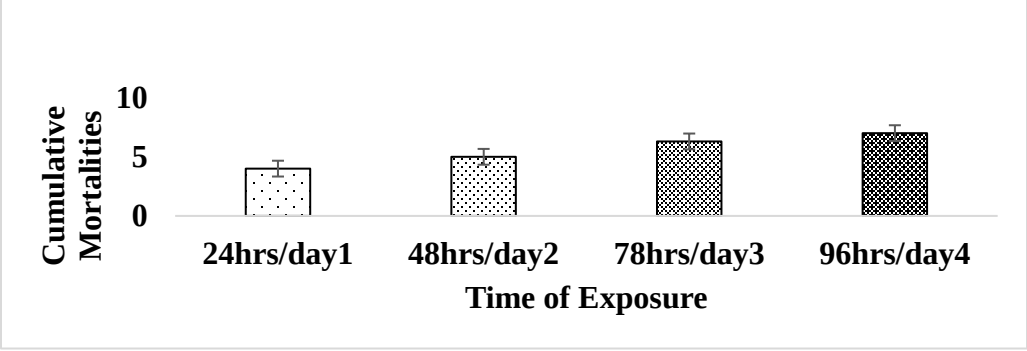


**Figure 4.4:** Cumulative Mortalities of the second life stage of *Oreochromis niloticus* exposed to different concentrations of Organophosphate insecticide.

## 5.0 DISCUSSION

The acute toxicity of dichlorvos (the active substance in Ridoff) to fish has been previously determined by a number of researchers. The 96h-LC50 value of dichlorvos obtained for juveniles of European sea bass (*Dicentrarchus labrax)* was 3.5 mg/L.

**5.1 Water quality for 24 to 96 hours:** The physicochemical values reported in this work were within the recommended values (30ºC) by FAO: Food and Agricultural Organization (2001). The reported value agrees with the temperature values recorded in other studies which generally varied between 25 ºC to 35 ºC (Uedeme-Naa and George, 2017). The values were not significantly different ($P<0.05$) from the control group of fish and the other concentrations. Mean while Sharma. *et al* 2019 reported a lower value (25.3ºC) in their findings and this could
be related to the different climatic conditions at that particular geographical location and period. The pH values agrees with the range (6.5-8.9) recommended by APHA, 1985.. Although the values indicated slight acidity as the concentration increased, this agreed with what was reported in similar studies (De Lemos et al., 2008). Low pH is linked to increased solubility and toxicity of chemicals (Sharma. *et al* 2019). It is also a generally acceptable fact that concentration of toxicants influences the elevation and or reduction of test water in an experimental set up (Sprague,1971). coupled with other activities of the fish which might have also affected the physico-chemical parameters while trying to survive (Oladimeji and Ologunmeta, 1987). The mean values of dissolved oxygen, ammonia and nitrite obtained were within the acceptable range as reported by Uedeme-Naa and George (2017). The observed reduction in the DO of water may suggest that some fractions of snipper which became bio-available were sufficient to deplete the oxygen level in the water ((Oladimeji and Ologunmeta, 1987)). There have been many research works on the extent of damage posed by several industrial activities such as oil spillage etc. (Sturve, *et al.,* 2016) .Toxicity of chemicals has been reported to vary depending on species, developmental stages (Ugwu *et al.,* 2006) and testing protocols (Jones *et al.,* 2009). Environmental pollution resulting from industrial effluents and other anthropogenic activitieshas become a global issue because of the extent of damage caused to the aquatic ecosystems and the disruption in the natural food chain Uedeme-Naa and John-Amadi (2021).

**5.2 Mechanism of toxicity:** Dichlorvos exerts its toxic effect by irreversibly inhibiting neural acetylcholinesterase (Lotti, 2001), the inhibition provokes the accumulation of acetylcholine in synapse with disruption of nerve function Wang *et al*., 2004).In an animal study, Aditya *et al*., 2012 reported activation of the microglia cells resulted in microgliosis manifested by increased damage in the manifested by increased damaged in the affected regions. The study reported that the microglial cells undergo cell death after 48hours of dichloruss treatment. In another study, Lotti, (2001) reported Nigrostriatal neuronal death. In another study, Sogbesan, *et al*., 2012 reported significant reduction in testosterone levels of animal fed water contaminated with dichlorvos. It also reported the levels of distortions in the cells of the semininia cells (Sogbesan, *et al*., 2012)

**5.3 Behavioural Alterations:** Behavioural changes in any fish species are very sensitive parameters to measure in an organism's response to stresses associated with aquatic environmental contaminants Ufodike, and Omoregie, (1990). Several authors have reported the bahavioural alterations induced by dichlorvos as well as other pesticides on different fish species at different developmental stages. Onusiriuka,(2002) reported abnormal behavioural responses such as rolling

on the back, quick circular movements, restlessness and excessive mucus productions on the body surface in *Clarias gariepinus* exposed to sublethal concentrations (0.1, 0.05, 0.025 and 0.0125) of dichlorvos for 96h. Dichlorvos induced severe behavioural changes such as erratic and spiral swimming, sudden quick movement/jumping, lateral and upward bending, respiratory distress and calmness and spontaneous air gulping at different rates were induced by dichlorvos in *Clarias gariepinus* fingerlings and post fingerlings (Omoniyi *et al.,* 2013). The report of Ashade *et al.* (2011) revealed that the behavior of exposed fishes is a function the concentration of the media and the duration of exposure. They studied the behavior of *Clarias gariepinus* exposed to dichlorvos (0.1, 0.05, 0.025 and 0.0125ml/L) for 96h. At 0.16ml/L, swimming was normal in the first 24h. However, fingerlings became agitated and restless with time they swam to the surface for air, assumed vertical position and death. Some fingerlings were reported to be active even after 96h. At 0.0125ml/L after 48h, fish showed increased opercula activity, erratic movement, and sudden quick movement after 48h. Mucus secretion from gills was observed after 96h. The fishes showed a spectacular response at 0.1ml/L, they showed quick sudden movements tried to jump out of the test medium, decreased opercular movements and loss of equilibrium with time. They sank to the bottom of the tank looking week and sluggish with excessive mucus secretion from the gills and finally died. At the highest concentration, the fishes exhibited loss of equilibrium, quick and fast swimming movement, incessant jumping and swimming to the surface for air, and as exposure time increased they showed signs of weakness, assumed vertical position and died with excessive mucus secretion. Matsumura (1975) reported that the primary and principal sign of nervous system failure could be due to pesticide poisoning which affects physiological and biochemical activities in non target organisms. This is in agreement with this work in that on exposure of two life stages of *Oreochromis niloticus* (fingerlings and post fingerlings) to lethal concentrations of Snipper (dichlorvos), it was observed that specimens exhibited agitated swimming, loss of equilibrum, air gulping, period of quiescence, and the fish turned on their flank and swarm in circles and finally died. Hyper activities were the most common responses noticed on *O. niloticus* and were dose dependent. Pal and Koner (1987) opined that disruption of the functioning of the nervous system of fish might be the cause of slow and agitated swimming, erratic movement and loss of equilibrum. Accumulation of mucus also was observed on the gill filaments and body surface of the dead fish after their exposure to the lethal concentration of Snipper (dichlorvos). Hossain *et al.* (1987) noted that increase in production of mucus over the body due to the effect of toxicant may interfere with the gaseous exchange, secretions, waste products and osmoregulation. Similar observations were made by Shafiel and Costa (1990), Babatunde (1997), Auta (2001) and Uedeme-Naa and George,(2017) who studied the effects of toxicants on different species of fish. The buildup of mucus may result from a boost in the activity of mucus cells due to exposure to pesticides. This caused an increase in the production of mucus over the body of the specimen. It seems that the pesticides tend to precipitate or coagulate mucus protein on the gill epithelium. This may affect the gaseous exchange, secretion of waste products and osmoregulation. The toxic action of the toxicants appeared to have caused a negative effect on the mucus, gills and osmoregulatory resulting in stress and death due to suffocation. Blood was also observed around the gill coverings of the dead fishes. This suggests that the fishes might have suffered from gill haemorrhage. Similar findings were reported by Shafiel and Costa (1990) when fry and fingerlings of *Oreochromis mossambicus* were exposed to some pesticides like Ronstar, Elsan, Endosulfan, Basfapon, Rogor 40 (dimethoate) and Azodrin 60. The reduction of respiratory rate implies that the fish had become stressed due to several attempts to escape from the toxic

medium to facilitate more oxygen intake. These behavioural changes are indicative of respiratory impairment, due to the effect of the toxicant on the gills and general metabolism (Chindah *et al.*, 2004).

**5.4 Morphological Alterations**: Pal, and Konar, (1987). observed discoloration in fish exposed to dichlorvos. Similarly, Omoniyi *et al*., (2013) observed bleached body with lesions in *Clarias gariepinus* exposed to dichlorvos at different concentrations (fingerlings: 0.1, 0.05, 0.025 and 0.0125mg/L; post fingerlings: 0.1, 0.05, 0.025 and 0.0125mg/L). These external changes were more pronounced in the fingerlings at higher concentrations. Foll *et al*., (1966). exposed fish to dichlorvos and reported the greying of its natural skin colour, while discoloration and caudal bending was observed by Ashade *et al*. (2011) in *Clarias gariepinus* exposed to dichlorvos (0.16, 0.32, 0.4 and 0.52ml/L). This is in agreement with this in that morphological alterations and death were observed with increase in concentration at 0.05 (78.57%), 0.08 (79.28%), 0.10 (80%), 0.13 (81%) and 0.15 (100%) mg/l.

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