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Effects of Dimethoate Insecticide on Serum Biochemistry of African Catfish (*Clarias gariepinus*) Juveniles

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ABSTRACT: The present study was designed to investigate acute toxicity of dimethoate on juvenile Clarias gariepinus. Catfish with mean weight and length of 24.01 ± 11.60 g and 13.98 ± 5.66 cm respectively were exposed to sub-lethal levels of dimethoate in static bioassay system for 28 days and mortality data was statistically evaluated using Finney's Probit analysis. The 96-hour LC_{50} value for C. gariepinus was estimated as 29.05 mg/l. The value for the LC_{50} was used in deducing the sub-lethal concentrations (0.01, 0.15and 0.29 mg/l). Biochemical changes in the serum such as glycogen, total protein, cholesterol, urea and creatinine were analyzed after the exposure period (28 days). The glycogen level decreased in all sublethal exposures. An increase in cholesterol at lower exposure concentration, followed by slight increase in urea and creatinine among treated groups was recorded. Behavioural changes which includes changes in skin colour, jerking, restlessness, erratic swimming, among others were observed. Based on the result of this study, it was concluded that C. gariepinus juveniles are very sensitive to low concentration of dimethoate. Dimethoate is highly toxic to juvenile catfishes and as such measures must be taken to avoid its run-off into our aquatic environment thus jeopardizing the recruitment of juvenile catfishes into the fishery.

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Ecological concerns arising from the high persistence of organochlorines have favored extensive use of organophosphates for controlling household, agricultural and public health pests due to their less persistence and high efficacy (Oscan Oruç *et al;* 2006). This shift has resulted into increased occurrence of organophosphates into water bodies causing acute and chronic toxicity to fish fauna (Pandey *et al;* 2009). Sometimes pesticides are directly applied into water bodies but most often, their residues reach aquatic ecosystems through surface run off. Aquatic ecosystem is thus faced with the threat of biodiversity loss due to extensive use of pesticide Several physiological and behavioral dysfunctions occur in animals after exposure to low concentrations of organophosphate pesticide (Ambali *et al;* 2011). Synthetic organophosphate pesticides are mostly non-selective but highly effective chemicals (Breckenridge and Stevens, 2008). The widespread use of pesticides in agriculture and health and hygiene programs are the major causes of aquatic pollution (Cope, 2004). Aquatic organisms including fish accumulate pollutants directly from contaminated water and indirectly via the food chain. Fishes are among the group of non-target organisms in the aquatic environment and they represent the largest and most diverse group of vertebrate in this environment. A number of characteristics make them excellent targets especially for the contaminants which are likely to exert their impact on aquatic systems (Raisuddin and Lee, 2008.). In fishes, pesticides can damage vital organs (Ansari and Ansari, 2013), reduce reproductive ability (Ansari and Ansari, 2011) and cause various biochemical alterations (Sharma *et al*; 2012). The possibility that dimethoate can affect humans and wildlife in their natural habitats is of great concern. Dimethoate has been reported to cause developmental toxicity as well as reproductive failures in organisms upon repeated exposures (verma, R. and, Mohanty, B. 2009). Developmental toxicity of dimethoate includes decreased number of implantations and live foetuses, incidences of resorptions and decreased foetal body weights (Farag *et al*; 2006). In present scenario of environmental pollution, the increasing contamination of aquatic environ is a matter of great concern for environmentalists. This study was conducted to determine the toxicity effect of dimethoate on freshwater African catfish (*Clarias gariepinus*). Alterations in behavioral activities as well as biochemical components are important indicators of stress in exposed fishes to toxicants, therefore changes in behaviour and biochemical components was studied.

MATERIALS AND METHODS

Test substance: Formulated pesticide product with trade name Action 40 which contained 40% EC Dimethoate was purchased from Jiangsu Tenglong

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Biological and Medicine Company Limited, China, represented by African Agro Products Limited; Kano, Nigeria.

Experimental Animals and Acclimatization: 300 Catfish juveniles with mean weight and length of 24.01 ± 11.60 g and 13.98 ± 5.66 cm respectively procured from Michael Okpara University of Agriculture Fish Farm were acclimated for 14 days (two weeks) in 30 liters' plastic basins in the laboratory prior to the start of the experiment. During this period the fish were fed with commercial feed (Coppens) twice dailyat 5 % body weight and the water changed daily before the start of the experiment. Temperature and pH of the aquaria water during acclimatization were 26 ± 2^{O}C and 6.82 ± 0.56 pH respectively.

Acute Toxicity Test: Feeding was stopped 24 hours before starting the experiment. The range finding test was conducted to establish the range of concentrations used for the main test. Ten *C. gariepinus* juveniles were exposed in triplicates to nominal dimethoate concentrations of 0, 20, 30, 40, 50 and 60 mg/l for 96 hours and mortalities were recorded at 24, 48, 72 and 96 hours and lethal concentration at these time intervals were estimated.

Sub Lethal Toxicity Test: To evaluate the biochemical effects of dimethoate, 84 catfish (three per group) were randomly divided into one control group (A) and three experimental groups - B, C and D which were treated with 0.01mg/l ,0.15 mg/ and 0.29 mg/l respectively for 28 days each having three replicate.

Table 1 Terms Suitable for Describing Fish Appearance and Behaviour

Term	Definition
INTEGUMENT	**The Epithelial Covering of the Body, Including the Gills**
Shedding	Peeling or loss of portions of the integument
Mucous	Excessive secretions of mucous; especially evident at the gills
Hemorrhaging	Bleeding (e.g, form the gill, anal opening, eyes)
PIGMENTATION	**Color of skin due to Deposition or Distribution of Pigment**
Light	Color lighter than usual for the species (as evident under the test conditions exclusive of the test solution)
Dark	Colour darker than usual for the species (as evident under the test conditions exclusive of the test solution)
Mottled	Colour of individual dish abnormally varied
GENERAL BEHAVIOUR	**Observable Response of the Test Fish, Individually or in Groups, to their Environment**
Quiescent	Marked by a state of inactivity or abnormally low activity; motionless or nearly so
Hyperexcitable	Reacting to stimuli with substantially greater intensity than control fish
Irritated	exhibiting more or less continuous hyperactivity
Surfacing	rising and remaining unusually long at the surface
Sounding	diving suddenly to the bottom; remaining unusually long at the bottom
Twitching	Sudden jerky movements (muscle spasms) for part or all of the body
Tetanic	in a state of tetany, marked by intermittent tonic spasms of the voluntary muscles
Normal	Apparently unaffected by (or not exposed) the test solution, conforming to the normal appearance e and behvioural characteristics of the species under the defined test conditions.
SWIMMING	**Progressive Self-propulsion in Water by coordinated Movement on the Tail, Body and Fins**
Ceased	No longer evident
Erratic	Characterized by lack of consistency, regularity, or uniformity, fluctuating, uneven
Gyrating	Revolving around a central point, moving spirally about an axis
Skittering	Skimming hurried along the surface with rapid body
Inverted	movements
On side -±	Turned upside down (or approximately)
	Turned 90 degrees laterally, more or less, from the normal body orientation
RESPIRATION	**Physical Exchange of Water at the Gill Surface, Evident by Movement of the Opercula**
Rapid	Faster than normal (obviously exceeding respiratory rate for
Slow	control)
	Slower than normal (obviously exceeding respiratory rate for
Coughing	control)
	Increased (relative to control) rate of coughing (back-flushing
Surface	of gills, evident by marked flairing of opercula)
	Swimming at surface with mouth open and pumping surface
Irregular	water or air through gills
	falling to occur at regular (rhythmic) intervals

Modified from USEPA 1985

Water Quality Analysis: Water quality parameters (Dissolved oxygen, pH, Temperature, Total dissolved substance, Electrical conductivity and Alkalinity), in the aquaria were checked bi-weekly for the 28 days' study period.
Dissolved oxygen: Dissolved oxygen was measured by using DO meter. (Milwaukee MW 600, SN:60015532)
pH: pH values were measured with a pH meter. (PH-008(1))
Water temperature: Water temperature was recorded using the same DO meter while measuring dissolved oxygen.
Electrical conductivity: Electrical conductivity was measured using an electrical conductivity meter.
Total alkalinity: Total alkalinity was determined by titration with standard sulfuric acid in the presence of methyl orange indicator in the solution, (APHA, 1985).
Total dissolved solid: Total dissolved solid was measured using a TDS meter. (Model: EC-1)

Behavioral Indices For Fish: Terms Suitable for Describing Fish Appearance and Behaviour Modified from (U.S.E.P.A. 1985) (as presented in Table 1).

Biochemical analysis: After the 28 sublethal study period, the fish samples from all groups where stunned and blood collected from their caudal vein. Blood plasma was obtained from blood samples by centrifugation (4o, 800xg, 10min) was used in determination of selected biochemical indices (glycogen and cholesterol). Checking of the biochemical indices was done the same day the fish were sacrificed.

Serum Total Protein Determination: Total protein content was determined using Randox test kit for protein (Randox Laboratories Limited, United Kingdom). Three test tubes were set up for each sample and labeled test, blank and standard. 1.0 ml of the total protein reagent was introduced into each of the test tubes. 20 µl of the test sample was collected and introduced into the test tube labeled test. The same volume of the standard reagent was added to the test tube labeled standard while the same volume of distilled water was added to the blank test tube. The mixtures were incubated at 25^0C for 30 minutes before absorbance of both the standard and test were read on a spectrophotometer after zeroing with the blank at 560nm. Total protein content of sample was obtained using the formula below: The organ (liver) were chilled by immersing into ice-cold 1.15% solution of KCL and immediately processed for biochemical analysis. fresh tissue samples were weighed and haemoginate (10% w/v) were prepared using a glass homogenizer with ice-cold 1.15% KCL. The adequate tissue homogenate was centrifuged (10mins x 1500rpm) after which aliquots were used in d spectrophotometer.

$$\mathrm{TP} = \frac{\mathrm{At\ x\ CS}}{\mathrm{As}}$$

Where concentration of standard = 5.95mg/dl; TP = Total protein; At = Absorbance of test; CS = Concentration of standard; As = Absorbance of standard

Urea determination: Total urea in each sample was determined using Randox commercial kits for urea (Randox Laboratories, United Kingdom). Three test tubes were set up and were labeled test, standard and blank. 100 µl of urea reagent 1 was pipette into each of the test tubes. 10 µl of the sample was pipette into the test tube. The same volumes of standard reagent and distilled water were added to the standard and blank respectively. The contents of the test tubes were adequately mixed and incubated at 37^0C for 10 minutes. Reagents 2 and were then added (2.5 ml each) to the test tubes and properly mixed and also incubated at 37^0C for 15 minutes before absorbance of the test and standard were read using a spectrophotometer (722N, Mindray Company, India) at 560nm against the blank. Urea concentration in mg/dl was the calculated using the formula:

$$\mathrm{UC} = \frac{\mathrm{At\ x\ CS}}{\mathrm{As}}$$

Where UC = Urea concentration; At = Absorbance of test; CS = Concentration of standard; As = Absorbance of standard

Creatinine determination: This was determined according to the method of Burtis (2008). Set of three test tubes labeled S- sample, T- test and ST- standard were set up. The tissue haemoginate and control were pipette into the test tube labeled S- sample and ST-standard respectively, followed by adding 0.5 ml of sample and mixed thoroughly. Tungstic acid 4.5 ml was added into the test tubes and mixed carefully and allowed to stand for 10minutes. It was centrifuged at 1500 x g for 10minutes into another set of test tubes labeled T- test and ST- standard were pipette 3.0 ml of the supernatant and standard respectively. A reagent blank was also set up into which 3.0 ml of distilled was pipette. Picric acid (0.1 ml) was added to each test tube and mixed thoroughly. After 30 seconds, 0.5 ml of sodium hydroxide was added into each of the test tubes and mixed thoroughly. They were allowed to stand for 15 minutes and absorbance read at 550nm against blank, concentration of the creatinine in serum was calculated thus;

$$\mathrm{CR} = \frac{\mathrm{A-sample}}{\mathrm{A-standard}} \times 177\ (\mu\mathrm{mol/l})$$

$$\mathrm{CR} = \frac{\mathrm{A-sample}}{\mathrm{B-standard}} \times 2\ (\mathrm{mg/dl})$$

Cholesterol and glycogen determination: This was determined using Randox commercial kits (Randox Company, UK). For each sample, 3 test tubes were set up and labeled test, standard and blank.10 µl of the serum was pipetted into the test tube, while the same volume of the standard reagent and distilled water was pipette into the standard and blank test tubes respectively. 1000 µl of the Randox cholesterol reagent was then added to each test tube. The mixture in each test tube was mixed and incubated for 10 minutes at 25^0C. Absorbance at wavelength 546nm were taken on a spectrophotometer (Model 722N, Mindray, India) against the reagent blank. The absorbance of the standard was made equivalent to 200.65mg/dl of cholesterol as prescribed by the producer and was used to obtain the test result. Glycogen content was analyzed by using Anthrone method (De Zwaan *et al;* 1972).

*Statistical analysis***:** Lethal concentrations were estimated using Probit analysis. The data collected were analyzed using one-way analysis of variance(ANOVA) while Turkey's posthoc test was used to separate significant means at $p<0.05$. All analysis was performed with Statistical Package for Social Sciences (SPSS) version 22.

RESULTS AND DISCUSSION

Mortality and Acute Lethal Concentration of Dimethoate: The results of the mortality of *C. gariepinus* fingerlings and the estimated 24, 48, 72 and 96hour lethal concentrations are presented in Table 2 and 3.

Table 2: Mortality of *Clarias gariepinus* exposed to dimethoate for 96 hours

Pesticide Conc. (mg/l)	Total exposed	Mortality count at			
		24h	**48h**	**72h**	**96h**
20	30	0	4	8	8
30	30	0	9	14	16
40	30	9	16	17	19
50	30	18	23	25	25
60	30	22	30	30	30
0(Control)	30	0	0	0	0

Table 3 Lethal Concentrations (LC_x) of dimethoate at 24 and 48 hours

Duration	**LC_x**	**Estimate**	**Lower Bound**	**Upper Bound**	**Slope ± SE**	**df**	**Probit line equation**
24h	LC_{10}	34.44	28.89	38.03	8.705 ± 1.467	3	y= -14.661+8.705x
	LC_{20}	38.69	34.05	41.90			
	LC_{30}	42.08	38.13	45.17			
	LC_{40}	45.21	41.72	48.50			
	LC_{50}	48.34	45.03	52.24			
	LC_{60}	51.69	48.20	56.71			
	LC_{70}	55.54	51.50	62.35			
	LC_{80}	60.40	55.34	70.06			
	LC_{90}	21.11	7.38	27.94			
48h	LC_{10}	25.17	11.71	31.86	5.757 ± 0.819	3	y= -8.907+5.757x
	LC_{20}	28.57	16.12	35.49			
	LC_{30}	31.85	20.78	39.65			
	LC_{40}	35.24	25.63	45.24			
	LC_{50}	39.00	30.40	53.66			
	LC_{60}	43.47	34.95	67.25			
	LC_{70}	49.35	39.64	90.91			
	LC_{80}	58.84	45.69	142.67			
	LC_{90}	34.44	28.89	38.03			

Y= a+ bX (Y= response (mortality); X= concentration

Table 4: Lethal Concentrations (LC_x) of Dimethoateat 72 and 96 hours

Duration	LC_x	Estimate	Lower Bound	Upper Bound	Slope ± SE	Df	Probit line equation
	LC_{10}	15.99	1.19	23.88			
	LC_{20}	19.93	2.96	27.67			
	LC_{30}	23.36	5.63	31.19			
	LC_{40}	26.75	9.58	35.26			
72h	LC_{50}	30.37	15.13	41.11	4.599 ± 0.742	3	y=-6.817+4.599x
	LC_{60}	34.48	22.05	51.97			
	LC_{70}	39.49	28.93	76.14			
	LC_{80}	46.29	35.13	134.70			
	LC_{90}	57.70	42.24	323.49			
	LC_{10}	15.30	10.20	19.08			
	LC_{20}	19.06	13.98	22.73			
	LC_{30}	22.34	17.48	25.88			
	LC_{40}	25.59	21.05	29.07			
96h	LC_{50}	29.05	24.86	32.64	4.601 ± 0.749	3	y=-6.732+4.601 x
	LC_{60}	32.97	29.02	37.09			
	LC_{70}	37.76	33.64	43.28			
	LC_{80}	44.26	39.16	52.94			
	LC_{90}	55.16	47.31	71.54			

Y= a+ bX (Y= response (mortality); X= concentration

Table 5: Physicochemical of test solution during the sub lethal toxicity test

	Test solutions			
	Control	**0.01 mg/l**	**0.15 mg/l**	**0.29 mg/l**
DO (mg/l)	2.09 ± 0.28	2.22 ± 0.16	2.41 ± 0.24	2.47 ± 0.24
pH	5.91 ± 0.24	5.43 ± 0.14	5.44 ± 0.13	5.50 ± 0.14
Temp. (°C)	27.25 ± 0.72	26.06 ± 0.43	26.31 ± 0.48	26.25 ± 0.49
TA (mg/l)	19.25 ± 0.38	18.31 ± 0.22	17.88 ± 0.18	30.31 ± 0.58
TDS (ppm)	19.75 ± 3.95	17.13 ± 2.01	17.63 ± 2.01	19.93 ± 3.05
EC (µs/cm)	38.75 ± 7.74	35.00 ± 4.09	34.81 ± 4.11	40.81 ± 6.15

Values are expressed as mean and Standard error (mean ± SE), DO: Dissolved Oxygen, TA: Total alkalinity, TDS: Total dissolved solid, EC: Electrical conductivity; pH: Hydrogen ion concentrate

Biochemical Effects of Dimethoate in Clarias gariepinus: The result of the effect of dimethoate after 28 days of exposure on serum glycogen, total protein, cholesterol, urea and creatinine are present in Figures 1 to 5 respectively, while table 5 shows the physicochemical parameters of the test solution during the experiment.

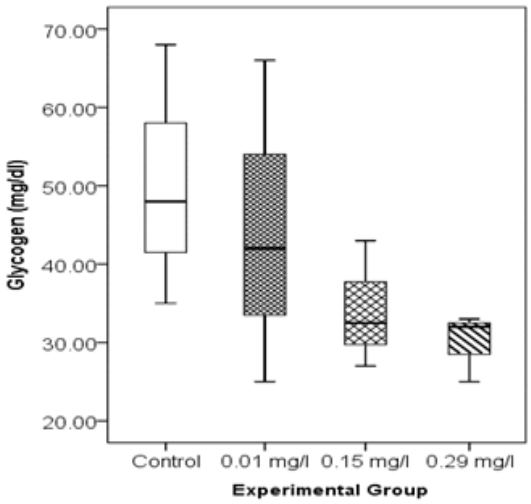

Fig 1: Boxplot of serum glycogen of exposed fishes compared to control, n=4,

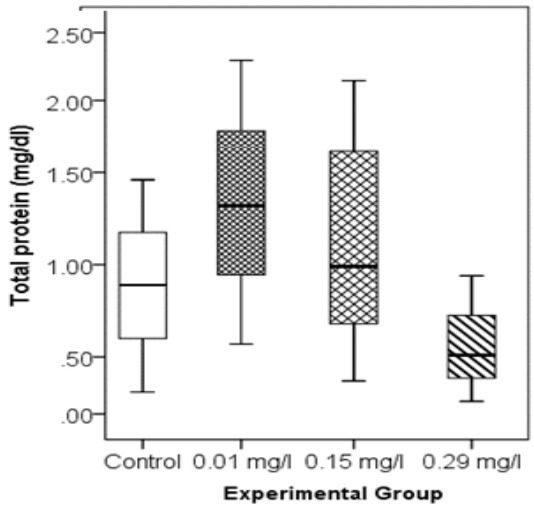

Fig 2: Boxplot of total protein of exposed fishes compared to control, n=4,

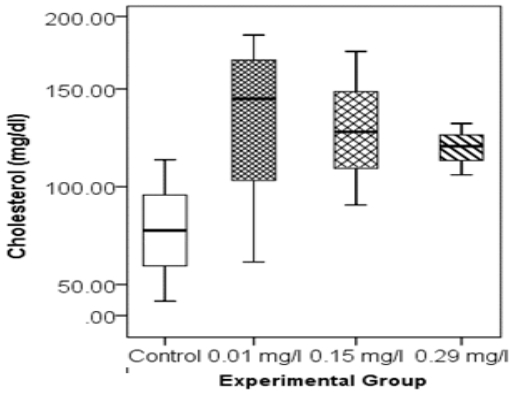

Fig 3: Boxplot of the level of cholesterol of exposed fishes compared to control, n=4, * indicates significant difference compared to control ($p < 0.05$)

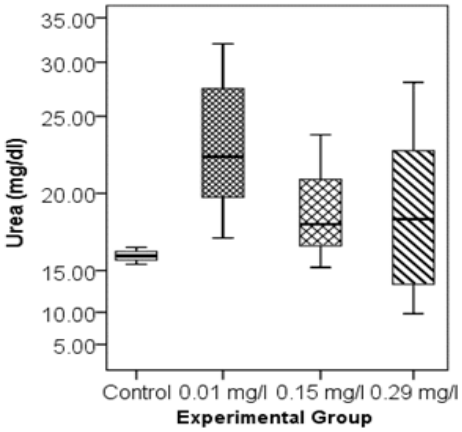

Fig 4: Boxplot of urea level in exposed fishes compared to control, n=4,

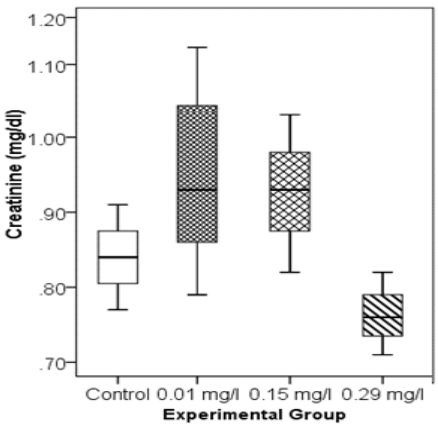

Fig 5: Boxplot of creatinine of exposed fishes compared to control, n=4,

Changes in the glycogen in fishes exposed to the pesticide were not statistically significantly different

(p>0.05) compared to the control after 28 days of exposure. There were no significant changes (p>0.05) in the total protein in fishes exposed to the dimethoate compared to the control after 28 days of exposure. After 28 days of exposure, the level of cholesterol increased significantly (p<0.05) in all the treatment groups compared to the control. Changes in the level of urea of exposed fishes was not statistically different (p>0.05) from the control after 28 days of exposure. Changes in the creatinine levels of fishes exposed to the pesticide was not significantly different (p>0.05) compared to the control after 28 days of exposure. Water quality parameter showed no significant difference (p>0.05) between the control and the treated groups with regard to pH, dissolve oxygen, temperature, electrical conductivity, total alkalinity and total dissolve solid (TDS). The current study investigated the lethal, behavior and biochemical toxicity of dimethoate on the *C. gariepinus.* The estimated 96h LC_{50} value of 29.05 mg/l indicated that dimethoate is slightly toxic to *C. gariepinus* juvenile and the fish species may be tolerant to dimethoate. The $LC_{50}$of 29.05 mg/l recorded in the current study was lower than 65 mg/l for *Clarias batrachus* (Svoboda *et al;* 2001) due to the adult fishes used in the study. It was however higher than 2.98 mg/l for *Clarias batrachus* (Begum and Vijayaraghavan, (1995) and 0.14 mg/l for *Pteronarcys californica* after 48hours exposure (Menzie, 1969) in which utilized fingerlings had reduced capacity to metabolise and eliminate toxicant from the body. Behavioural characteristics can serve as biosensor of biochemical and physiological response of fish under the influence of a toxicant (Radhaiah *et al*; 1987; Chindah *et al;* 2004). In the current study, the intensity of the behavior changes was both time and concentration dependent which suggests that *C. gariepinus* juveniles may be tolerant of dimethoate (40%EC). The behavioural signs elicited by dimethoate were similar to signs seen withother insecticides such as malathion (Ahmad, 2012), endosulfan (Shao *et al*; 2012), diazinon (Ahmad, 2011) and atrazine (Nwani *et al*; 2011). The behavioural changes may be attributed to the neurotoxic effect of dimethoate by inhibition of ACHE. The inhibition interferes with normal neurotransmission in cholinergic synopses and neuromuscular junctions of the nervous system thus affecting normal functioning of the nerves (dos Santos Miron *et al*; 2005). The continuous accumulation of acetylcholine leads to loss of balance, Jerking, paralytic symptoms and eventually death (Table 6).

Table 6: Behavioural and Morphological Changes in Exposed *C. gariepinus*

	Time (h)	Mucous secretion	Haemorrhagic patches	Uncoordinated movement	Body lighten intensity	Surfacing/ sounding	Quiescent
Control	12	-	-	-	-	-	-
	24	-	-	-	-	+	-
	48	-	-	-	-	-	-
	72	-	-	+	-	-	+
	96	-	-	-	-	-	+
20 mg/l	12	-	-	++	-	+++	-
	24	+	-	++	-	+++	-
	48	+	-	+	+	++	+
	72	+	+	-	+	+	++
	96	+	+	-	++	-	+++
30 mg/l	12	-	-	+++	-	+++	-
	24	+	-	++	+	++	-
	48	++	-	+	+	-	++
	72	++	+	+	+	-	++
	96	++	++	-	+++	-	+++
40 mg/l	12	-	-	+++	-	+++	-
	24	+	-	+++	-	+++	-
	48	+	++	++	-	-	++
	72	++	++	-	++	-	++
	96	++	++	-	+++	-	++++
50 mg/l	12	++	-	+++	++	++++	-
	24	+++	+	+++	++	+++	-
	48	++++	+++	+	++	-	++
	72	-	-	-	++++	-	+++
	96	-	-	-	++++	-	++++
60 mg/l	12	+++	+++	++++	+++	++++	-
	24	++++	+++	+++	++++	++	+++
	48	-	+++	++	++++	-	++++
	72	-	-	-	-	-	-
	96	-	-	-	-	-	-

- None +, feeble; ++, progressive; +++, intense; ++++, severe. -, not present

During stress, an organism needs sufficient energy which may be supplied from reserve material that includes protein, glycogen and cholesterol (Ganeshwade *et al*; 2011). In the current study, insignificant decrease in levels of protein and glycogen of exposed fishes suggests its provided sufficient store of energy to meet energy demand warranted by toxic conditions. Begum and Vijayaraghavan (1995)

reported decrease in liver protein during dimethoate exposure and attributed it to increased metabolism. Sobha *et al*; (2007) reported decrease in glycogen. Decrease in liver glycogen was observed in *Tilapia mossomibica* exposed to heptachlor (Radhaiah *et al;* 1987) and *Catla catla* exposed to fenvalerate (Susan *et al.* 1999). Radhaiah *et al.* (1987) stated decreased liver glycogen may be due to the rapid utilization of carbohydrate by the tissue possibly to overcome the pesticide induced stress. Begum and Vijayaraghavan (1995) suggested that the synthesis of detoxifying enzymes by the liver requires high energy which contributes to a reduction in hepatic glycogen. A fall in the glycogen level clearly indicates its rapid utilization to meet the enhanced energy demands in fish exposed to toxicants through glycolysis or hexose monophosphate pathway. Thus, depletion of protein fraction in liver may have been due to their degradation and possible utilization of amino acids in the various catabolic reactions and metabolic purposes (Nelson and Kumar, 1996). Lipid metabolism in fishes is seriously impaired by exposure to organophosphate as attested to by most studies. However, this present study showed a slight deviation in cholesterol level after exposure of *C. gariepinus* to dimethoate for 28 days. This evident that organophosphate induces toxicity in fish thereby impairing lipid metabolism in them. This study was in agreement with work done by Singh and Singh (1980) who reported no significant variations in cholesterol level of *Heteropneustes fossilis* after been exposed to cythion and hexadrin. Lai and Singh (1987) reported an elevation in liver lipid content in respond to malathion exposure. Alteration in the cholesterol in the liver of fish exposed to dimethoate may be associated with toxicity stress which suppressed the activity of some enzymes responsible for lipid transformation thereby disrupting lipid metabolism (Ganeshwade *et al*; 2012). The underutilization in cholesterol content in the liver might have occurred as a result of malfunctioning in enzymes (Cytochrome P450) responsible for cholesterol biosynthesis and metabolism. Cytochrome P450 are involved in a diverse array of biological processes that includes lipid, cholesterol and steroid metabolism as well as conversion of cholesterol to bile acids. This enzyme has metabolic functions with great physiological significance and is closely associated with detoxification mechanisms in tissues. Enzymes activities in fishes are severely disrupted due to pesticide exposure (Joseph and Raj, 2011). An elevated level of urea and creatinine (the end product of protein) in tissue haemoginate is considered an indicator of renal function and it indicates the degenerative changes in kidney and surrounding tissues. The creatinine test has always been used to diagnose impaired kidney function and detect renal damage (Toffaletti, J. G. and McDonnell, 2008; Banaee *et al*; 2011). Creatinine is a breakdown product of creatinine kinase in muscle. It is solely considered a waste product and is then diffused into the bloodstream from the muscles and enters to the renal parenchyma where it is filtered by the glomerulus and excreted in the urine. Creatinine is then found in the blood when it is not being reabsorbed by the renal tubules. An observation of increase in serum creatinine and blood urea in this present study agrees with result of prior studies reporting subtle nephrotoxic changes in fish exposed to pesticide. Common cause for increase in blood urea is atypical nitrogen excretion usually due to kidney damage and urinary obstruction (Goel, S. 1996). Increased urea in the experimental fish was also reported to be due to the inability of the damaged kidney to filter the urea up to the normal levels (Deka and Mahanta, 2015).

Conclusion: From this study, it may be concluded that the behavior and biochemical indices are important biomarkers in deducing effects of xenobiotic on organisms in the environment. Hence, proper care should be given to application and handling of chemicals in order to avoid harmful effects to non-target organisms.

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