Journal of Biology and Today's World
ISSN 2322-3308
Journal home page: http://journals.lexispublisher.com/jbtw/

Received: 21 June 2013 • Accepted: 28 October 2013

Research

doi:10.15412/J.JBTW.01020602

Effect of Dimethoate on mortality and Biochemical changes of Freshwater fish *Labeo rohita* (Hamilton)

Nagaraju Bantu[1], Rathnamma vakita[2*]

[1] Department of Biochemistry, Acharya Nagarjuna University, Guntur-522510, A.P, India

[2] Department of Zoology, Acharya Nagarjuna University, Guntur-522510, A.P, India

*correspondence should be addressed to Rathnamma vakita, Department of Zoology, Acharya Nagarjuna University, Guntur-522510, A.P, India; Tell: +91; Fax: +91; Email: nagaraju.bantu301@gmail.com.

ABSTRACT

The acute toxicity tests were conducted during certain intervals in various concentrations of Dimethoate. The physical and chemical analyses of water were carried out by following APHA methods. While treating with Dimethoate, the percentage of fish mortality was assessed during 24, 48, 72 and 96 hours. The estimated lethal concentration values along with 95% confidence limits of Dimethoate were found to be 17.532 mg/l (16.781-19.877), 17.321 mg/l (16.521-18.134), 16.721 mg /l (16.063-17.952), 16.350 mg/l (15.388-17.143), mg/l for 24, 48, 72, and 96 h, respectively. In sub lethal and lethal exposure, the glycogen and protein levels was decreased, minimum percentage of protein depletion was (22.21%) in kidney and maximum percentage was (49.80%), in liver, were observed, the depletion of glycogen content minimum in kidney (18.73%), and maximum in liver (80.14%), the fish was exposed to sub lethal and lethal concentrations for 8 days, The decrease in protein content in various tissues might also be due to inhibition of metabolizing enzymes by administration of agro chemicals. Decline protein levels in early periods due to stress in metabolic process and impairment of protein synthesis machinery in fish.

Key words: percentage, sub lethal, protein, liver, enzymes, and synthesis

1. INTRODUCTION

Environmental protection has attracted the attention of the wide cross-section of people all over the world which has now become a global issue amongst scientists and researchers working in this area. Unfortunately several toxic pollutants, few are even unknown or un-identified to the biota, are being regularly introduced in large quantities into the environment, especially into the aquatic environment. Pollution of water by pesticides is an important dimension of environmental degradation. The disposal of the industrial and agricultural wastes directly into the aquatic medium burdens the eco-system and stresses the need to analyze, the concentration of these substances in the medium as well as in the organisms. The pesticides are also found to be highly toxic not only to fish but also to other organisms which constitute food of the fish. It is estimated that approximately 3000000 people are exposed to effects of organophosphates or carbamates every year worldwide, which leads to up to 300000 deaths. Many of research workers have used the acute toxicity tests of pesticides on fish to acquire rapid estimates of the concentrations that caused direct, irreversible harm to test organism. Many of these chemicals are mutagenic (1, 2), linked to the development of cancers (3) or may lead to the developmental deficits. Worldwide pesticide usage has increased dramatically during the past two decades, coinciding with changes in farming practices and increasingly intensive agriculture. Contamination of water by pesticides, either directly or indirectly, can lead to fish kills, reduced fish productivity, or elevated concentrations of undesirable chemicals in edible fish tissue which can affect the health of humans consuming these fish (4). Pesticides were found to adversely affect a number of biological functions, thus causing harm to the non-target organisms. Organophosphate compounds are known for this persistence in the environment and accumulation in the tissues for long periods for controlling the loss of produce due to pest attack and as a consequence of the demand for producing more food, there has been an increasing use of

pesticides in India. Dimethoate is an organophosphate insecticide used to kill mites and insects systemically and on contact. In the present study, an attempt has been made to analyze the toxicity of the Dimethoate on the freshwater fish *Labeo rohita*

2. MATERIALS AND METHODS

The fresh water fish *Labeo rohita* size 4-5 cm and weight 7-9 g were brought from a local fish farm located at Nandivelugu, Guntur district of Andhra Pradesh, India. The fish *Labeo rohita* were fed daily with commercial fish pellets and acclimatized to the laboratory conditions at 28 + 2°C for 15 days. During the acclimatization period daily fed with fish meal. If in any batch, mortality exceeds 5% during acclimatization, that entire batch of fish was discarded. The water used for acclimatization and conducting experiments was clear unchlorinated ground water and the hydrographic conditions of water are shown in the

Table 1. The physical and chemical analyses of the water were carried out (5). The containers of the test media are of 10 liter capacity, where in each test five containers were used and each container consisted of ten fish.

2.1. Procurement of commercial grade

Dimethoate 30% EC (Rogor) manufactured by Rallis India Limited, Mumbai. It was purchased from the local market in Guntur.

2.2. Preparation of stock solution

The stock solutions were made with acetone and concentrations were taken in mg/l. Controls were maintained for each experiment and they were added with the quantity of acetone equal to the highest concentration used in the test. Precaution is taken to minimize the acetone as solvent.

2.3. Selection of sub-lethal concentrations

The lethal concentrations ensure death even before noticing the behavioral abnormalities. Even when the animal is exposed to low concentrations continuously, many behavioral abnormalities and physiological alterations would be observed. In the present study 1/10th of 96 hr. LC_{50} value (1.635 mg/l) was selected as sub lethal concentration to study the behavioral alterations and biochemical alterations (As per the recommendations of committee on toxicity studies – OECD. The concentrations of pesticides, which may normally be sub-lethal during short-term exposure, may prove to be lethal, if the exposure time is extended. Since the toxicity of the poison is a function of time, it is customary to expose the test organisms over a fixed period of time to the toxicant usually for 24, 48, 72 and 96 h. The acute toxicity (96 h LC50) of Dimethoate for the freshwater fish, *Labeo rohita* was determined in the laboratory using the semi-static method in OECD (6).The mortality rate was taken into consideration and while taking the data, dead fish was removed immediately. Pilot experiments were conducted to choose the mortality range between 10% and 90%.

Basing on the pilot experiments, the experiments were conducted to determine the toxicity in five different concentrations for 24, 48, 72, and 96 hours with organophosphate compound Dimethoate in semi- static system, in this method, the test solution and test organisms are kept in the test chambers for a specific duration of the experiment. The data of each concentration was pooled up to calculate the LC50 values by using the un-weighted regression method of probit analysis (7). The exposure was continued for 8 days at 28±1°C with photoperiod of 12D:12L. The water was renewed freshly every day to produce constant effect of Dimethoate on fish. At the end of 8 days exposure, the tissues such as liver, muscle, kidney, brain, and gill were collected by dissecting the animal and stored at – 20°C for biochemical studies.

2.4. Estimation of Glycogen

The total glycogen was estimated, employing the method of (8). Each tissue was homogenized in 80% Methanol and centrifuged at 3000 rpm for 10 minutes. The tissue residue was suspended in 5 ml of 5% TCA (Trichloro acetic acid), and boiled for 15 minutes at 100°C and then cooled in running water. The solution was made up to 5 ml with 5% TCA to compensate for the evaporation and then centrifuged, from this, 2 ml of supernatant was taken into the test tube, 6 ml of conc. H_2SO_4 was added and the mixture was boiled for 6 minute. The mixture was cooled and the optical density was measured of 520 nm.

2.5. Estimation of Proteins

Each tissue was homogenized in 5% TCA and centrifuged at 3000 rpm for 10 minutes. The suspended protein residue was dissolved in 1 ml of in NaOH; 0.2 ml of the extract was taken into the test tube and 5 ml of alkaline copper solution (50 ml of 2% Na_2CO_3 in 0.1 N NaOH and 1 ml of 0.5% $CuSO_4$ $5H_2O$ in 1% of sodium or potassium tartrate) was added. The contents were mixed well and allowed to stand for 10 minutes; 0.5 ml of diluted Folin phenol reagent was added. After 30 minutes the optical density was measured spectrophotometrically at 540 nm (9).

Table 1. Chemical analysis of water used for experiment

Turbidity	:	8 silica units
Electrical conductivity at 28oC	:	816 micro ohms/cm
pH value at 28oC	:	8.1
Alkalinity: i. Phenolphthalein ii. Methyl orange	 : :	 Nil 472
Total Hardness (as CaCO3)	:	232
Non-carbonate Hardness (as CaCO3)	:	Nil
Calcium Hardness (as N)	:	Nil
Sulphate (as SO4)	:	Trace
Chloride (as Cl)	:	40
Fluoride (as F)	:	1.8
Iron (as fe)	:	Nil
Dissolved oxygen	:	8-10 ppm
Temperature	:	28 + 32oC

3. RESULTS AND DISCUSSION

In the present investigation the test species, *Labeo rohita* has shown differential toxicity level with the function of period. The 96 hours LC50 concentration is less than the 24 h, 48, and 72 h concentration, which shows that the more is the duration period the less is the concentration required. The observed percentage of mortality of *Labeo rohita* for Dimethoate in static tests for 24, 48, 72 and 96 hours were shown in (Table 2). The observed and estimated LC50 values of *Labeo rohita* for Dimethoate in static test for 24, 48, 72 and 96 h, parameter estimation, were shown in Table 2 and

Table 3, estimated median lethal concentration values from 1 to 99 were shown in (Table 4 and Figure 1) respectively. The changes in the amount of glycogen and protein content in the fish *Labeo rohita* exposed to sub-lethal and lethal concentrations of Dimethoate for 8 days along with standard deviation was given in the
Table 5,

Table 6 and also graphically represented in the (Figure 2, Figure 3, Figure 4 and Figure 5) respectively. The values were expressed as mean ± S.D. Statistical analysis was performed by one-way analysis of variance (ANOVA) followed by using the computer package SPSS 20.0 v and the significance of difference was set up at ($p< 0.05$).

Table 2. Mortality of *Labeo rohita* exposed to Dimethioate

Conc. *(24 h)	% mortality	conc. *(48 h)	% mortality	conc. *(72 h)	% mortality	conc. *(96 h)	% mortality
15	20	14	20	13	20	12	20

15.5	40	14.5	40	13.5	30	12.5	40
17	50	16	60	15	50	13	60
17.5	70	16.5	70	15.5	70	14.5	80
18	90	17	90	16	90	15	100
Control	0	0	0	0	0	0	0

*Estimated LC50 values in semi-static test for 24 h: 17.532 mg/l (16.781-19.877) 48 h: 17.321 mg/l (16.521-18.134), 72 h: 16.721 mg/l (16.063-17.952), and 96 h: 16.350 mg/l (15.388-17.143).

Table 3. Parameter estimates for the probit analysis

Probit a/Parameter	*Estimate*	*Std. Error*	*Z*	*Sig.*	*L. B*	*U.B*
concentration of pesticide	20.571	6.425	3.202	.001	7.979	33.164
Intercept	-24.963	7.830	-3.188	.001	-32.793	-17.134

a. PROBIT model: PROBIT(p) = Intercept + BX (Covariates X are transformed using the base (10.000logarithm.)L.B=Lower bound, U.B= Upper bound at 95% confidence intervals.

Table 4. Estimated lethal concentration values and confidence limits

Probability	*95% Confidence Limits for concentration of pesticide*			*95% Confidence Limits for log(concentration of pesticide)a*		
Probit	Estimate	Lower Bound	Upper Bound	Estimate	Lower Bound	Upper Bound
.010(LC1)	12.602	8.168	14.043	1.100	.912	1.147
.020	12.992	8.831	14.320	1.114	.946	1.156
.030	13.246	9.278	14.500	1.122	.967	1.161
.040	13.440	9.629	14.638	1.128	.984	1.165
.050	13.600	9.923	14.752	1.134	.997	1.169
.060	13.738	10.181	14.850	1.138	1.008	1.172
.070	13.860	10.412	14.937	1.142	1.018	1.174
.080	13.970	10.623	15.016	1.145	1.026	1.177
.090	14.071	10.818	15.089	1.148	1.034	1.179
.100(LC10)	14.165	11.001	15.156	1.151	1.041	1.181
.150	14.559	11.786	15.444	1.163	1.071	1.189
.200	14.880	12.442	15.686	1.173	1.095	1.196
.250	15.161	13.026	15.907	1.181	1.115	1.202
.300	15.418	13.563	16.121	1.188	1.132	1.207
.350	15.660	14.065	16.339	1.195	1.148	1.213
.400	15.893	14.538	16.572	1.201	1.163	1.219
.450	16.121	14.981	16.835	1.207	1.176	1.226
.500(LC50)	16.350	15.388	17.143	1.214	1.187	1.234
.550	16.581	15.754	17.515	1.220	1.197	1.243
.600	16.820	16.077	17.966	1.226	1.206	1.254
.650	17.070	16.363	18.506	1.232	1.214	1.267
.700	17.338	16.625	19.144	1.239	1.221	1.282
.750	17.632	16.878	19.899	1.246	1.227	1.299
.800	17.965	17.136	20.808	1.254	1.234	1.318
.850(LC85)	18.361	17.419	21.949	1.264	1.241	1.341
.900	18.872	17.761	23.500	1.276	1.249	1.371
.910	18.997	17.842	23.894	1.279	1.251	1.378
.920	19.134	17.930	24.331	1.282	1.254	1.386
.930	19.286	18.027	24.821	1.285	1.256	1.395
.940	19.458	18.134	25.381	1.289	1.259	1.405
.950	19.655	18.257	26.038	1.293	1.261	1.416
.960	19.889	18.401	26.832	1.299	1.265	1.429

.970	20.181	18.577	27.844	1.305	1.269	1.445
.980	20.575	18.813	29.251	1.313	1.274	1.466
.990(LC99)	21.213	19.187	31.621	1.327	1.283	1.500

a. Logarithm base = 10.

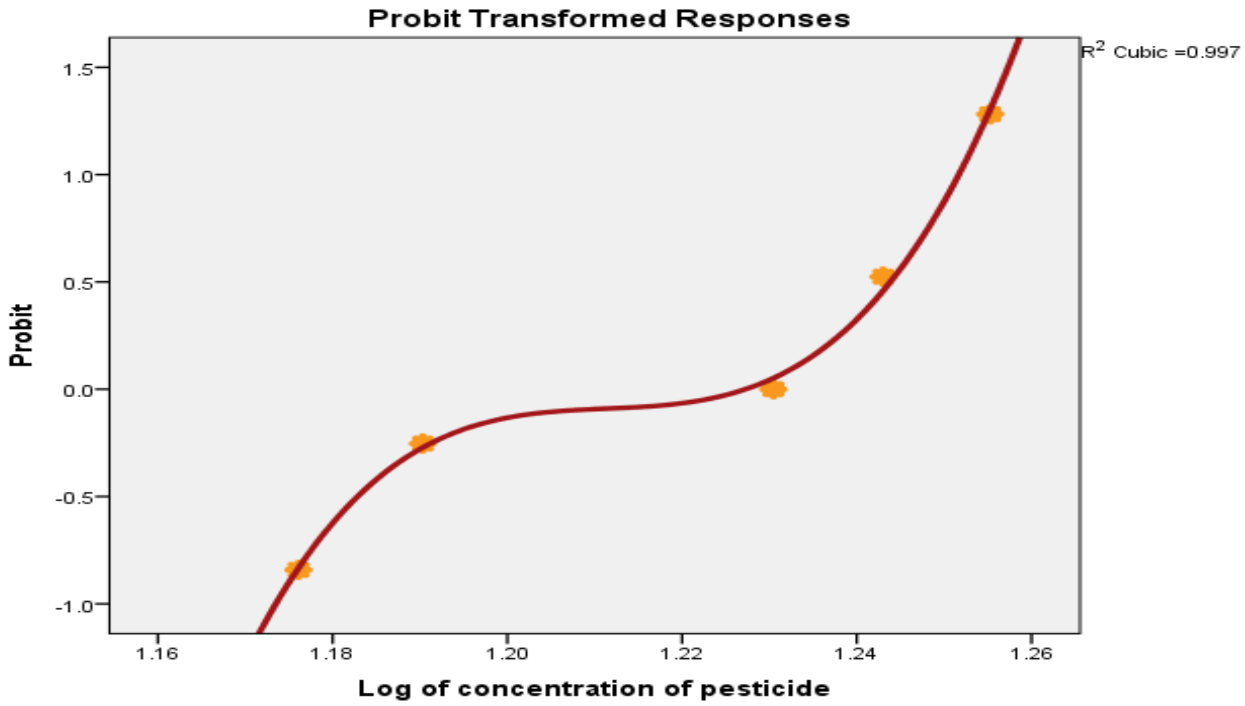

Figure 1. Probit line graph of acute toxicity of Dimethioate on fish *Labeo rohita*

Table 5. Effect of Dimethioate on Glycogen content in different tissues of fish *Labeo rohita* in sublethal and lethal concentrations– mg/g wet weight of tissue

Tissues	Control	Sub-lethal	Lethal
Muscle	19.15± 0.416	7.18±0.316 (62.50%)	5.29± 0.341(72.37%)
Kidney	9.18± 0.316	7.46 ± 0.347 (18.73%)	6.34± 0.373(30.93%)
Gill	12.50± 0.314	8.15± 0.316(34.08%)	3.38± 0.374(72.96%)
Liver	27.14± 0.316	8.29± 0.344(69.45%)	5.39± 0.377(80.14%)
Brain	10.29± 0.341	6.46± 3.47(37.22%)	5.39± 0.371(47.61%)

Values represent mean of five individual observations, mean ± S.D and the mean difference at the $P < 0.05$ level.

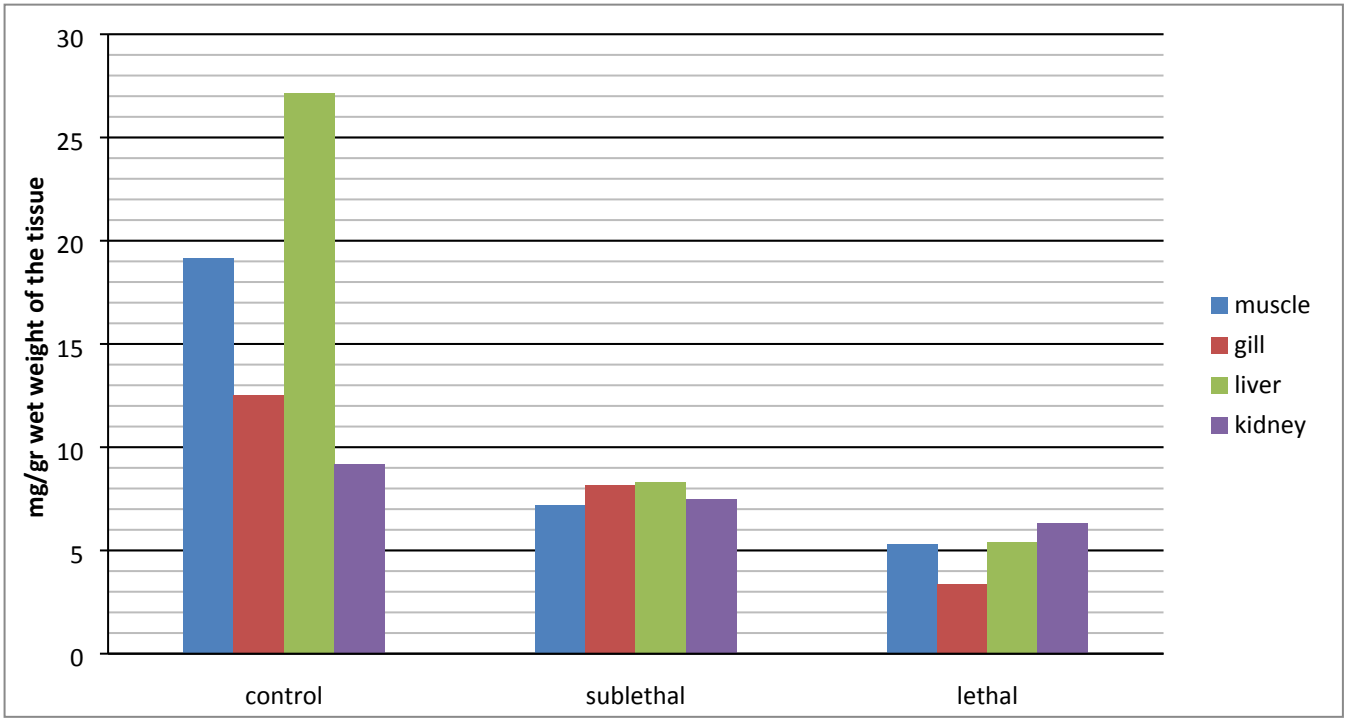

Figure 2. Changes in the glycogen (mg/g wet weight of tissue) in different tissues of *Labeo rohita* on exposure to lethal and sub-lethal concentrations of Dimethoate for 8 days. (mean±S.D, n=5 observations, $p < 0.05$.

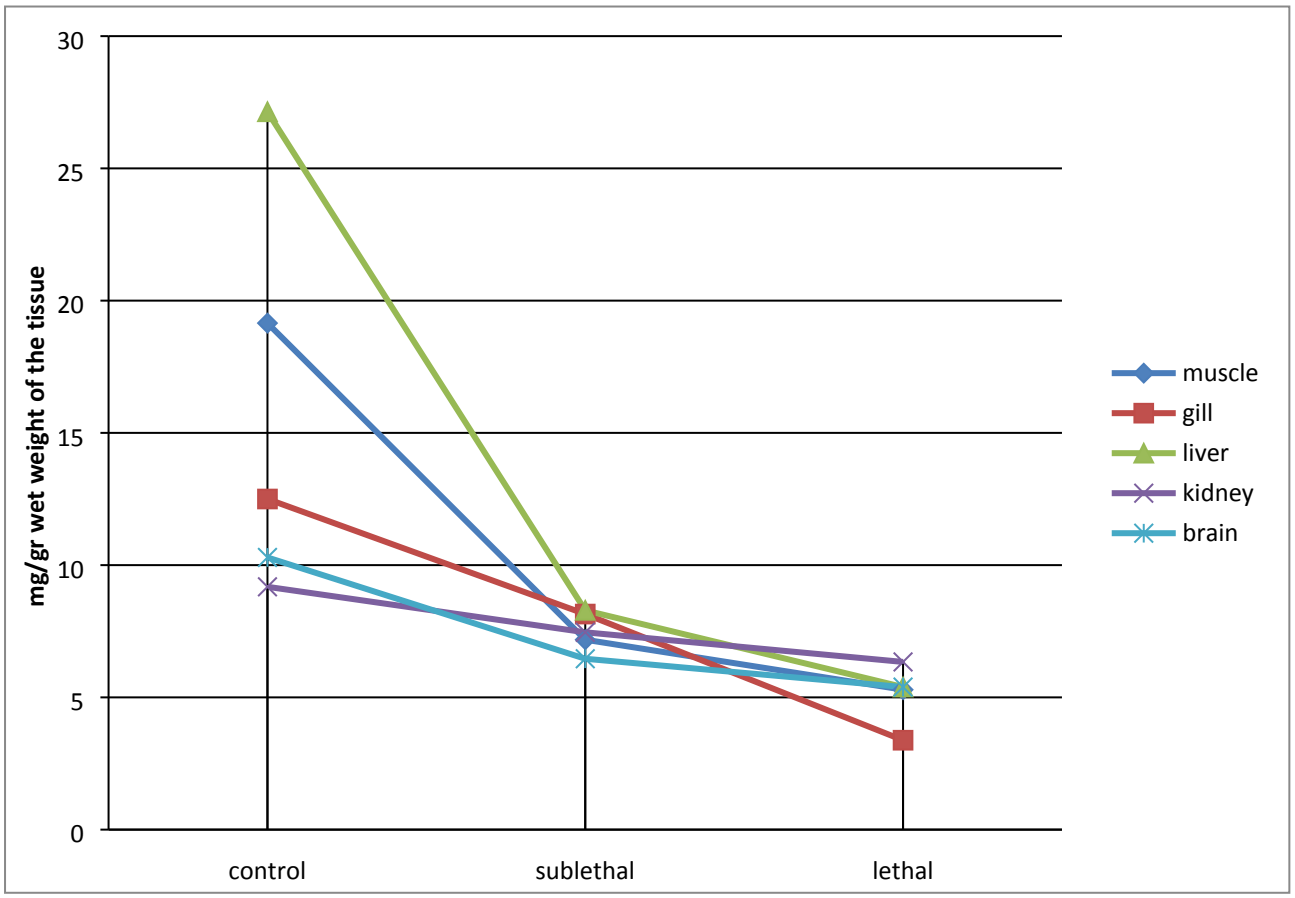

Figure 3. Changes in the glycogen (mg/g wet weight of tissue) in different tissues of *Labeo rohita* on exposure to lethal and sub-lethal concentrations of Dimethoate for 8 days in a steep slope and a flat slope direction, (mean ± S.D), n=5 observations, $p < 0.05$.

Table 6. Effect of Dimethoate on Protein content in different tissues of fish *Labeo rohita* in sub-lethal and lethal concentrations – mg/gm wet weight of tissue

Tissues	Control	Sub-lethal	Lethal
Muscle	53.4± 0.286	52.0± 0.301(26.21)	51.15± 0.032(42.13)
Kidney	55.4±0.286	47.4±0.344(34.29)	43.1±0.036(22.21)
Gill	94.3±0.371	69.4±0.347(26.40)	56.5±0.323(40.08)
Liver	51.3±0.328	25.6±0.325(49.80)	49.4±0.377(39.33)
Brain	56.2±0.340	58.10±0.316(33.29)	54.4±0.281(32.02)

Values represent mean of five individual observations, ± S.D and the mean difference at the $P< 0.05$ level.

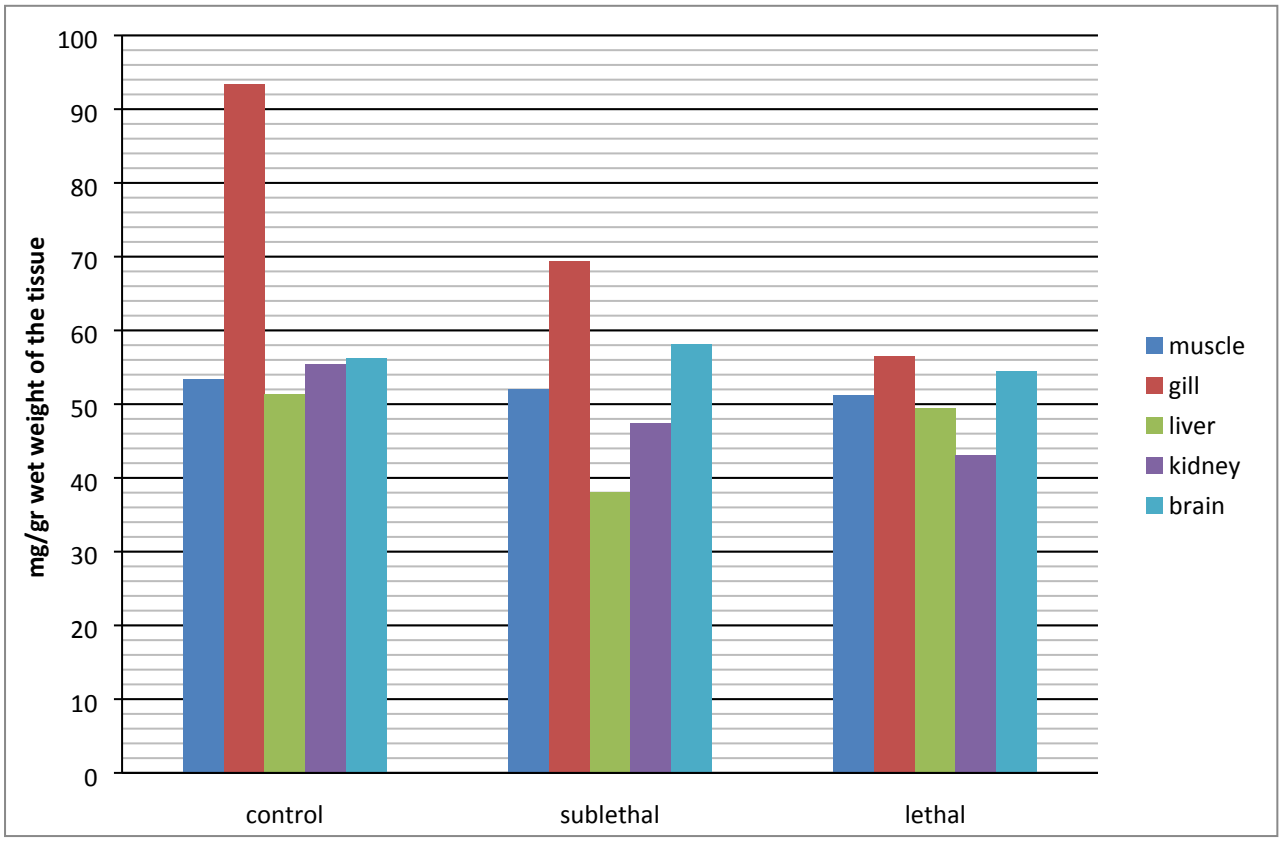

Figure 4. Change in the Total protein content (mg/gr wet weight of the tissue) in different tissues exposed to sub-lethal, lethal concentrations of Dimethoate for 8 days (mean±S.D), n=5 observations, $p < 0.05$

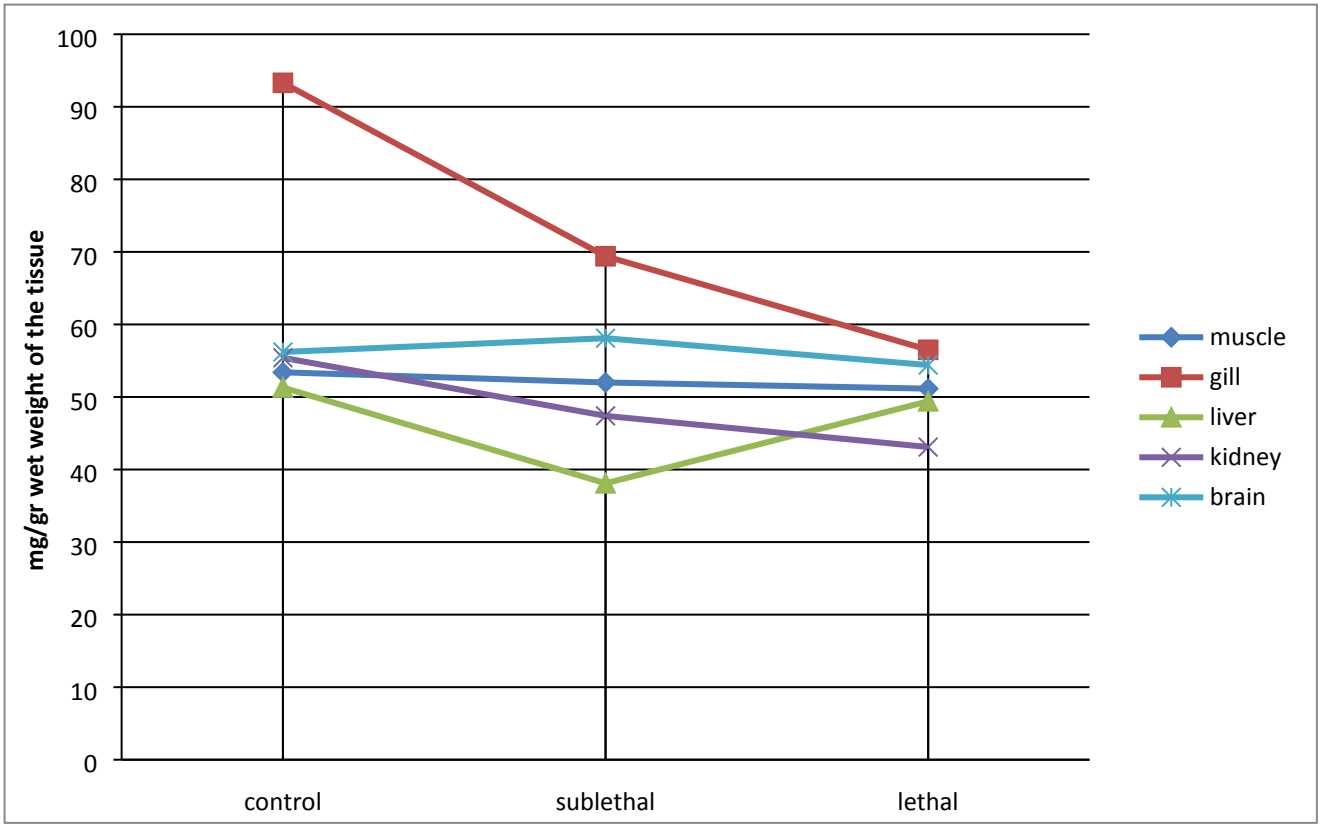

Figure 5. Change in the Total protein content (mg/gr wet weight of the tissue) in different tissues exposed to sublethal, lethal concentrations of Dimethioate for 8 days , in a steep slope and a flat slope direction.(mean±S.D),n=5 observations , $p < 0.05$.

The observed percentage of mortality of *Labeo rohita* for Dimethoate in static tests for 24, 48, 72 and 96 hours were 17.532 mg/l (16.781-19.877), 17.321 mg/l (16.521-18.134), 16.72 mg /l (16.063-17.952), 16.350 mg/l (15.388-17.143), mg/l , respectively. It is evident from the results that Dimethoate can be rated as toxic to fish. In the present study the control fish showed normal behavior, they were active with well-coordinated movements, jerky movements, hyper secretion of mucus, opening mouth for gasping, losing scales, hyperactivity were observed experimental group but in the toxic environment the fish exhibited irregular, erratic and darting swimming movements and loss of equilibrium due to inhibition of AChE activity, leading to accumulation of acetylcholine in the cholinergic synapses, leading to hyper stimulation (10). In which acute chemical toxicity is determined as a 96 h LC50 value. However the environmental significance of death of individuals after short term exposure to high concentration is questionable (11). The varying degree of mortality reported in this study is consistent with the earlier reports. It has been suggested that differences in an organism's biological adjustment and behavior response to change in water chemistry (12, 13). The toxicity of a pesticide could vary from species to species. These results have important implications for ecological risk assessments, particularly those that focus on the toxicity of individual chemicals as the basis for estimating impacts to imperiled aquatic species. Although the importance of multiple stressors is widely recognized in aquatic ecotoxicology (14), pesticide mixtures continue to pose major challenges for natural resource agencies (15). In the present study of test organism showed normal behavior in control group but jerky movements, hyper secretion of mucus, opening mouth for gasping, losing scales, hyperactivity were observed experimental group. Behavioral characteristics are obviously sensitive indicators of toxicant effect. In toxic medium of Dimethoate the fish *Labeo rohita* sank to bottom of the test chamber and independency in swimming. Subsequently fish moved to the corners of the test chambers, which can be viewed as avoidance behavior of the fish to the toxicant. In the toxic environment fish exhibited irregular, erratic, darting swimming movements and loss of equilibrium followed by hanging vertically in water. The above symptoms are due to inhibition of AChE activity leading to accumulation of acetylcholine in cholinergic synapses ensuing hyper stimulation. And inhibition of AChE activity is a typical characteristic of organophosphate compounds (16, 17). Gulping air and swimming at the water surface (surfacing phenomenon) were observed also with mucus secretion on the body in both the lethal and sub-lethal exposure periods (18). In sub-lethal and lethal exposure periods there is a significant decrease glycogen content in liver, kidney and followed by other tissues of freshwater fish *Labeo rohita*, relative to control group. Maximum decrease glycogen level is observed at lethal and sub-lethal exposure in all tissues but maximum in liver (80.14%), muscle (62.50%) and minimum in kidney (18.73%) as compared to other tissues were observed. Glycogen is found in all the tissues of the body but in varying amounts. Carbohydrates are stored in liver, muscle as glycogen as such that contain more energy. It has both functional and storage significance. The results indicated that the liver is vital organ of carbohydrate metabolism and were drastically affected by Dimethoate.

In almost all the tissues of fish brain, gill, kidney, liver, and muscle tested at sub-lethal and lethal concentrations of Dimethoate, a decrease in glycogen levels were noticed during the exposure periods (
Table 5). Carbohydrate metabolism is mainly concerns to fulfill energy demand of animals by its aerobic and anaerobic segment. Among various organs, higher glycogen levels were observed in liver, because involvement of liver in glycogen synthesis and utilization. Glycogen is the major storage form of energy in liver and muscle tissues. Liver glycogen is largely concerned with storage and export of hexose units for maintenance of blood glucose levels. The function of muscle glycogen is to act as a readily available source of hexose units for glycolysis within the muscle itself. In almost all the vital organs brain, gill, kidney, liver and muscle at lethal and sub-lethal concentrations of Dimethoate, showed a decrease in glycogen levels were noticed during the exposure periods. There is a significant decrease proteins in liver, kidney and followed by other tissues of freshwater fish *Labeo rohita* after 8 days exposure relative to control. Maximum decrease protein content is observed at lethal and sub-lethal exposure in all tissues but mainly in liver (49.80%), and muscle (42.13%) as compared to kidney (22.21%) and brain (32.02%) tissue. The proteins of the body like carbohydrates and lipids also serve as a source of energy. There is no storage form of protein. The reserve protein appears to be drawn from the tissues themselves and organs such as liver, kidney and blood during the time of fasting. During the exposure periods decrease in protein content were observed in kidney, liver, muscle, gill and brain tissues. An alteration of protein metabolism was observed in fish exposed to various types of environmental stresses like metals and pesticides, we are also agreeing with these authors,

Table 6 (19, 20). Stress proteins are considered to be general indicators of sub lethal cellular protein damage. The quality of protein is dependent on the rate of protein synthesis, or on rate of its degradation. The quality of the protein may also be affected due to impaired incorporation of amino acids into polypeptide chain (21-23) suggested that the fish exposed to pesticides may compensate any possible protein loss by increasing its protein synthesis (24), concluded that compensatory production of enzymes lost as result of tissue necrosis or to meet increased demand to detoxify the pesticides might have necessitated enhanced synthesis of enzyme proteins. Increase in free amino acid levels were the result of breakdown of protein for energy and impaired incorporation of amino acids in protein synthesis. The changes in total protein content of various tissues of *Channa punctata* when exposed to carbaryl and 1-Napthol were reported by (25, 26) reported that there is marked decrease in the protein content of various tissues like kidney, liver, muscle, exposed to sub-lethal and lethal concentrations of Cypermethrin. The survival ability of animals exposed to stress mainly depends on their protein synthetic potential. The degradation of protein suggests the increase in proteolytic activity and possible utilization of their products for metabolic purposes and cause damage to tissues (27), similar decreasing trend was also observed by (26, 28), reported that the depletion of glycogen content when exposed to sub-lethal concentration of Rogor to freshwater fish, *Heteropneustis fossils* (29) reported that chlordecone induced changes in carbohydrate metabolism of the freshwater cat fish *Heteropneustis fossils* were studied, both hepatic and muscle glycogen concentrations decreased significantly (30, 31) concluded that Malathion is highly toxic to fish and due to its toxicity protein content was reduced in the kidney, reported that exposure of fish to sub-lethal concentration of Malathion decreased the protein content in the gill (-22 to –39%) over the control group during the experimental period of 30 days (31, 32) reported that fish *Channa punctata* exposed to insecticide Dimethoate exhibited a significant dose-dependent decrease in total protein, glycogen, pyruvate level and cytochrome oxidase activity in liver, muscle and gonad tissues (33) reported that the decline in protein level indicates an acceleration of protein catabolism during Cypermethrin and fenvalerate intoxication (34) reported that exposed to sub-lethal concentration of fenvalerate both technical grade and 20% EC, the total protein content was decreased in gill, liver, kidney, brain and muscle of *Channa punctatus* (35) reported that the muscle protein of *Lepidocephalecthyes thermalis* at different sub-lethal concentrations decreased in all selected sub-lethal concentrations (36) reported that effects of Cartap Hydrochloride and urea in freshwater fish *Oreochromis mossambicus*, glucose level appears also to be related at least in part to the detoxification mechanisms particularly in the hepatic tissue, which is the major site for metabolism in animals (37) reported that the glycogen depletion was observed when chlorophyrophos an organophosphate exposed to freshwater fish *Catla catla, Labeo rohita* and *Cirrhinus mrigala* (38) reported that carbohydrate metabolism is disturbed when *L. rohita* is exposed to fenvalerate; alterations in the blood glucose level indicate the variations in the carbohydrate metabolism of the fish under toxic stress (39) reported that decrease in the level of glycogen content was observed in all tissues with increase in exposure periods (38) reported that the glycogen depletion was observed, when chloropyriphos, an organophosphate exposed to freshwater fish *Catla catla, Labeo rohita, Cirrhinus mrigala* (27) reported that the effects of sub-lethal doses of Dimethoate and Malathion on growth parameters in *Oreochromis niloticus*, glycogen, protein and lipid in fish muscle gradually decreased with increased pesticidal concentrations, the effects of sub-

lethal doses of Dimethoate and Malathion in *Oreochromis niloticus* bio accumulated in the liver was higher than in gill or muscle.

4. CONCLUSION

In the present study, a comparison of lethal concentration values and behavior change indicated that Dimethoate was less toxic than other pesticides to fishes. The results of these studies may provide information to selection of acute toxicity to be considered in field Ecotoxicology and biomonitoring efforts designed to detect the bioavailability of Dimethoate.

Funding/ Support

Not mentioned any Funding/ Support by authors.

ACKNOWLEDGMENT

Not mentioned any ACKNOWLEDGMENT by authors.

AUTHORS CONTRIBUTION

This work was carried out in collaboration among all authors.

CONFLICT OF INTEREST

The authors declared no potential conflicts of interests with respect to the authorship and/or publication of this article.

REFERENCES

1. Kumar R, Nagpure N, Kushwaha B, Srivastava SK, Lakra W. Investigation of the genotoxicity of malathion to freshwater teleost fish Channa punctatus (Bloch) using the micronucleus test and comet assay. Archives of environmental contamination and toxicology. 2010;58(1):123-30.
2. Leiss JK, Savitz DA. Home pesticide use and childhood cancer: a case-control study. American Journal of Public Health. 1995;85(2):249-52.
3. Khan M. Pesticides in aquatic environments: Springer Science & Business Media; 2013.
4. Adedeji O, Okocha R. Overview of pesticide toxicity in fish. Advances in Environmental Biology. 2012;6(8):2344-51.
5. APHA A. WEF (American Public Health Association, American Water Works Association, and Water Environment Federation). 1998. Standard methods for the examination of water and wastewater.20.
6. OECD O. Guidance Document on Acute Oral Toxicity Testing. OECD, Paris, France; 2000.
7. Finney D. Radioligand assay. Biometrics. 1976:721-40.
8. Kemp A, Van Heijningen AJK. A colorimetric micro-method for the determination of glycogen in tissues. Biochemical Journal. 1954;56(4):646.
9. Lowry OH, Rosebrough NJ, Farr AL, Randall RJ. Protein measurement with the Folin phenol reagent. J biol Chem. 1951;193(1):265-75.
10. Mushigeri S, David M. Fenvalerate induced changes in the Ach and associated AchE activity in different tissues of fish Cirrhinus mrigala (Hamilton) under lethal and sub-lethal exposure period. Environmental toxicology and pharmacology. 2005;20(1):65-72.
11. Marigoudar SR, Ahmed RN, David M. Cypermethrin induced respiratory and behavioural responses of the freshwater teleost, Labeo rohita (Hamilton). Veterinarski Arhiv. 2009;79(6):583-90.
12. Prashanth M, Sayeswara H, Patil HR. Impact of copper cyanide on behavioral changes and oxygen consumption in Indian major carp Catla catla (Hamilton). Electronic Journal of Environmental, Agricultural and Food Chemistry. 2010;9(9):1433-42.
13. Eyer P. The role of oximes in the management of organophosphorus pesticide poisoning. Toxicological reviews. 2003;22(3):165-90.
14. Eggen RI, Behra R, Burkhardt-Holm P, Escher BI, Schweigert N. Peer reviewed: Challenges in ecotoxicology. Environmental Science & Technology. 2004;38(3):58A-64A.
15. Gilliom RJ. Pesticides in US streams and groundwater. Environmental science & technology. 2007;41(10):3408-14.
16. Mathivanan R. Effects of sublethal concentration of quinolphos on selected respiratory and biochemical parameters in the fresh water fish Oreochromis mossambicus. Journal of Ecotoxicology & Environmental Monitoring. 2004;14(1):57-64.
17. Begum G. Carbofuran insecticide induced biochemical alterations in liver and muscle tissues of the fish Clarias batrachus (linn) and recovery response. Aquatic toxicology. 2004;66(1):83-92.
18. Rao JV. Toxic effects of novel organophosphorus insecticide (RPR-V) on certain biochemical parameters of euryhaline fish, Oreochromis mossambicus. Pesticide Biochemistry and Physiology. 2006;86(2):78-84.
19. Agrahari S, Gopal K. RETRACTED: Fluctuations of certain biochemical constituents and markers enzymes as a consequence of monocrotophos toxicity in the edible freshwater fish, Channa punctatus. Pesticide biochemistry and physiology. 2009;94(1):5-9.
20. Singh D, Yadav R, Singh S, Sing A. Botanicals as pesticides and their future perspectives in India. Frontiers in Environmental Research, Emma B Davis, ed. 2006:95-108.
21. Tripathi P, Singh A. Toxic effects of dimethoate and carbaryl pesticides on protein metabolism of the freshwater snail Lymnaea acuminata. Bulletin of environmental contamination and toxicology. 2003;70(1):0146-52.
22. Sahib IK, Prasada Rao KS, Sambasiva Rao KR, Ramana Rao KV. Sublethal toxicity of malathion on the proteases and free amino acid composition in the liver of the teleost, Tilapia mossambica (Peters). Toxicology letters. 1984 Jan;20(1):59-62. PubMed PMID: 6364457. Epub 1984/01/01. eng.
23. Gill TS, Pande J, Tewari H. Sublethal effects of an organophosphorus insecticide on certain metabolite levels in a freshwater fish, Puntius conchonius Hamilton. Pesticide Biochemistry and Physiology. 1990;36(3):290-9.
24. Tilak K. Toxicity of Carbaryl and 1-naphthol to the fresh water fish, Channa punctata: Ph. D. Thesis, Andhra University, Visakhapatnam, Andhra Pradesh, India; 1979.
25. Sarma K, Pal A, Sahu N, Mukherjee S, Baruah K. Biochemical and histological changes in the brain tissue of spotted murrel, Channa punctatus (Bloch), exposed to endosulfan. Fish physiology and biochemistry. 2010;36(3):597-603.
26. Borah S, Yadav R. Biochemical and haematological responses to starvation in an air breathing fresh water teleost Heteropneustes fossilis (Bloch). Indian Journal of Fisheries. 1996;43(3):307-11.
27. Mastan S, Ramayya P. Biochemical profile of Chana gachua (Ham.) exposed to sub-Lethal doses of dichlorovos (DDVP). Internat J Toxicol. 2010;8(1):27-32.
28. Ghosh D, Bhattacharya S, Mazumder S. Perturbations in the catfish immune responses by arsenic: organ and cell specific effects. Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology. 2006;143(4):455-63.
29. Murthy KS, Kiran B, Venkateshwarlu M. A review on toxicity of pesticides in fish. Int J Open Sci Res. 2013;1:15-36.
30. Khare A, Singh S, Shrivastava K. Malathion induced biochemical changes in the kidney of fresh water fish Clarias batrachus. Journal of Ecotoxicology & Environmental Monitoring. 2000;10(1):11-4.
31. Khare A, Singh S. Impact of Malathion on protein content in the freshwater fish Clarias batrachus. Journal of Ecotoxicology & Environmental Monitoring. 2002;12(2):129-32.
32. Tripathi P, Srivastava V, Singh A. Toxic effects of dimethoate (organophosphate) on metabolism and enzyme system of freshwater teleost fish Channa punctatus. Asian fisheries science. 2003;16(3/4):349-60.
33. Yeragi S, Rana A, Koli V. Effect of pesticides on protein metabolism of mud skipper Boleophthalmus dussumieri. Journal of Ecotoxicology & Environmental Monitoring. 2003;13(3):211-4.
34. Tilak K, Satyavardhan K, Thathaji P. Biochemical changes induced by fenvalerate in the freshwater fish Channa punctatus. Journal of Ecotoxicology & Environmental Monitoring. 2003;13(4):261-70.
35. Shaikh N, Yeragi S. Effect of Rogor 30 E(organo phosphate) on muscle protein in the freshwater fish Lepidocephalecthyes thermalis. Journal of Ecotoxicology & Environmental Monitoring. 2004;14(3):233-5.
36. Palanivelu V, Vijayavel K, Ezhilarasibalasubramanian S, Balasubramanian M. Combined effects of cartap hydrochloride and urea on biochemical constituents of fresh water fish Oreochromis mossambicus. Journal of Ecotoxicology & Environmental Monitoring. 2004;14(1):23-9.
37. Tilak K, Veeraiah K, Rao DK. Biochemical changes induced by chlorpyrifos, an organophosphate compound in sublethal concentrations to the freshwater fish Catla catla, Labeo rohita and Cirrhinus mrigala. Journal of environmental biology/Academy of Environmental Biology, India. 2005;26(2 Suppl):341-7.
38. David M, Kumar R, Mushigeri S, Kuri R. Blood glucose and glycogen levels as indicators of stress in the freshwater fish, Labeo rohita under fenvalerate intoxication. Journal of Ecotoxicology & Environmental Monitoring. 2005;15(1):1-5.
39. Sweilum MA. Effect of sublethal toxicity of some pesticides on growth parameters, haematological properties and total production of Nile tilapia (Oreochromis niloticus L.) and water quality of ponds. Aquaculture Research. 2006;37(11):1079-89.