# Toxic Effects of Dimethoate Pesticide on and Biochemical Parameters in Freshwater Fish Arius dussumieri

**Dr. Dnyaneshwar S. Rathod**

Assistant professor

Department of Zoology and Fishery Science,

Rajarshi Shahu Mahavidyalaya, Latur (MS) India.

**Email of corresponding author**: dr_dsrathod@rediffmail.com

**ABSTRACT:**

The first objective of this investigation was to elucidate the biochemical alterations induced by the toxic effects of dimethoate pesticide under controlled laboratory conditions. Significant declines were observed in Biochemical parameters, including protein, glycogen, and cholesterol, of Arius dussumieri. Concurrently, an elevated trend was observed in blood glucose levels (31.05, 42.2, and 51.03 mg/dl), compared to the control group (27.51) mg/dl, with these increases corresponding to the concentration and duration of exposure. The findings of this study underscore the value of haematological and biochemical indicators as reliable measures to detect the impacts of chemical pollutants, such as pesticides, on organism health.

**KEY WORDS**: Dimethoate Pesticide, Biochemical Parameters, *Arius dussumieri*.

## INTRODUCTION:

The extensive use of pesticides to increase crop production and is constantly polluting water bodies as they enter surface and estuarine water through industrial effluents, run off or direct application. The effects of pesticide are manifold on living organisms including economically important fishes. An understanding of the effect of pesticide on fish would be more helpful for fish conservation and fisheries development.

Fish is one of the most important source of food. It is reported that more than 8.5 million of tons of fish are required annually to meet the present day demand of fish protein in the world. But fish habitat is affected by various pollutants. The effect of pesticides on the physiological and biochemical parameters have been known from the recent research. The biochemical characteristic and particularly the qualitative and quantitative occurrence of the major biochemical components viz protein, fats, carbohydrates etc. of the fish are prime importance as they determine nutritive value of the fish. Occurrence of polluted water bodies has exposed biota and particularly fish to an unlimited extent of damage. Pesticides alter body components to variable degree depending on the concentration of pollutants in water and they are unsuitable as food or a constant health hazard is posed knowingly or unknowingly if such fish is eaten by human population. Therefore, it becomes necessary to evaluate the nature and extent of alteration in biochemical component of fish, so that safely measures of ways to overcome the alter conditions in fish can be suggested.The purpose

of present investigation was to determine the effect of lethal and sublethal concentration of dimethoate (roger) on protein, glycogen and lipid content of test fish.

Materials and Methods:

The fish were collected from ManjraRiver, Latur district and brought to laboratory. These fishes were observed for any pathological symptoms and then placed in 0.1% potassium permagnate ($KMnO_4$) for two minutes so as to avoid any dermal infection. The fish were then washed with water and acclimatized to laboratory conditions for two weeks in glass aquaria with capacity of 20 litre. The physico-chemical parameters of water analysed by following standard method suggested by APHA, (1998), (1)

During acclimatization the fishes were provided with a diet consisting of live earthworms. Food supply was with drown 24 hours prior to experimentation. A commercial grade of pesticide, Dimethoate - 30% EC was used for bioassy test. A stock solution of the toxicant was prepared and few concentrations from stock solution were prepared as the dilution technique.

For experimentation, laboratory acclimatized fishes were divided into three groups of 10 fishes per aquarium. Group 'A' served as control was kept in tap water. Group 'B' and 'C' were exposed to lethal and sublethal i.e. ($LC_{50}$ of 96 hours, and $1/10^{th}$ of 96 hours $LC_{50}$) concentration of dimethoate solution.

In another set of experiment, similarly laboratory acclimatized fishes were divided into two groups of 10 fish per aquarium. Group 'A' served as control was kept in tap water. Group 'B' was exposed to sublethal concentration ($1/10^{th}$ of 96 hours $LC_{50}$) for two month and water was renewed every 24 hours in order to provide fresh oxygented water, to maintain the concentration of dimethoate and also to remove accumulated waste. In second set of experiment investigation were carried out 7, 15, 30, 45 and 60 day of exposure.

The fishes were sacrificed immediately at the end of exposure period and issue like liver, body muscle, gill and kidney were excised rapidly and processed for the biochemical estimations after homogenising the required media. The protein contain analysed by Biuret method (2)by using bovine serum albumin as standard. Carbohydrate estimation was done by Anthrone reagent (3) Lipid estimation was done by chloroform methanol method. Suggested by (4) in chloroform methanol extract. All values were expressed in mg 100 mg-1 and given in tabulated form.

**RESULT AND DISCUSSION:**

Biochemical changes in muscle, gill, liver and kidney of fish exposed to sublethal ($1/10^{th}$ of $LC_{50}$ of 96 hour) and lethal concentration ($LC_{50}$ of 96 hours) of dimethoate are summarized in table. Overall depletion in glycogen, lipid and protein content was found in all the tissues i.e. muscle, gill, liver and kidney at 96 hours exposure and 60 days of exposure or chronic exposure period. The percent decrease of glycogen was maximum in muscle (16% and 30%) followed by Gill (9.4% and 17.6%) liver (8% and 14.3%) and kidney (5.5% and 12.3%) to sublethal and lethal concentration of dimethoate, for 96 hour exposure.

In long term expsoure (60 days of exposure) to sublethal concentration dimethoate, glycogen content was found to be gradually decreased in all the tissues.

The percent decrease of lipid was maximum in muscle (12% and 20%) followed by Gill (8.2% and 13%), Liver (5.6%) and Kidney (4.3% and 8.6%) to sublethal and lethal concentration of dimethoate, for 96 hour exposure.

In long term exposure (60 days of exposure) to sublethal concentration of dimethoate, lipid content was also found to be gradually decreased in all the tissues.

The percent decrease of protein was maximum in muscle (6.9% and 10.4%) followed by Gill (5.1% and 9.2%) Liver 2.8% and 5.9%) and Kidney (2.6% and 5.8%) to sublethal and lethal concentration of dimethoate for 96 hour exposure.

The long term exposure (60 day of exposure) to sublethal concentration of dimethoate, protein content was also found to be decreased. Effect of lethal and sublethal concentration of dimethoate on glycogen, lipid. Protein content of muscle, gill, liver and kidney of fish following 96 hours is epresented in Table No. 1, 2, 3.

**Table 1. Effect of lethal and sublethal concentration of dimethoate on glycogen content of muscle, gill, liver and kidney of fish following 96 hours exposure**

| Sr. No. | Tissues | Control Set Group 'A' | Sub lethal concentration Group 'B' (0.599 ppm) | Lethal concentration Group 'C' (5.99 ppm) |
|---|---|---|---|---|
| 1 | Muscle | 0.2421 ±0.013 | 0.2033 (16%)±0.011 | 0.1702 (30%)±0.014 |
| 2. | Gill | 0.1712 ±0.007 | 0.1550 (9.4%)±0.003 | 0.1410 (17.6%)±0.004 |
| 3 | Liver | 0.9242 ±0.023 | 0.8542 (8%)±0.026 | 0.7912 (14.3%)±0.028 |
| 4 | Kidney | 0.1619 ±0.005 | 0.1530 (5.5%)±0.002 | 0.1419 (12.3%)±0.006 |

All values expressed in mg/100mg$^{-1}$ wet weight of tissues.

2. Each value are the mean of three observation (±S.D.)
3. Brakets values indicates percent variation over control.
4. Values are significant * = $p < 0.05$; ** = $p < 0.01$, *** = $p < 0.001$

**Table: - 2:** Effect of lethal and sub lethal concentration of dimethoate on lipid content of liver, muscle, gill and kidney of *Arias dussumieri*following 96 exposure hours.

| Sr. No. | Tissues | Control Set Group 'A' | Sub lethal concentration Group 'B' (0.599 ppm) | Lethal concentration Group 'C' (5.99 ppm) |
|---|---|---|---|---|
| 1 | Muscle | 3.98 ±0.30 | 3.50 (12%) ±0.18 | 3.05 (30%) ±0.23 |
| 2. | Gill | 4.47 ±0.20 | 4.10(8.2%) ±0.10 | 3.88 (13%) ±0.12 |
| 3 | Liver | 7.89 ±0.26 | 7.45(5.6%) ±0.18 | 7.15 (9.4%) ±0.22 |
| 4 | Kidney | 3.45 ±0.17 | 3.30 (4.3%) ±0.09 | 3.15 (8.6%) ±0.15 |

All values expressed in mg/100mg$^{-1}$ wet weight tissues.
Each value are the mean of three observation (±S.D.)
Brakets values indicates percent variation over control.
Values are significant * = $p < 0.05$; ** = $p < 0.01$, *** = $p < 0.001$ NS = No significant.

**Table:-3:** Effect of lethal and sub lethal concentration of dimethoate on Protein content of liver, muscle, gill and kidney of *Arias dussumieri*following 96 exposure hours.

| Sr. No. | Tissues | Control Set Group 'A' | Sub lethal concentration Group 'B' (0.599 ppm) | Lethal concentration Group 'C' (5.99 ppm) |
|---|---|---|---|---|
| 1 | Muscle | 19.02 ±1.02 | 17.70 (6.9%) ±0.38 | 17.05 (10.4%) ±0.28 |
| 2. | Gill | 11.07 ±0.90 | 10.50(5.1%) ±0.28 | 10.05(9.2%) ±0.25 |
| 3 | Liver | 17.21 ±0.35 | 16.55 (2.8%) ±0.22 | 16.19 (5.9%) ±0.12 |
| 4 | Kidney | 10.89 ±0.18 | 10.60 ±0.15 | (2.6%) ±0.10 |

All values expressed in mg/100mg$^{-1}$ wet weight tissues.
Each value are the mean of three observation (±S.D.)
Brakets values indicates percent variation over control.
Values are significant * = $p < 0.05$; ** = $p < 0.01$, *** = $p < 0.001$ NS = No significant.

Effect of long term exposure of sublethal concentration of dimethoate on glycogen, lipid and protein content of muscle, gill, liver and kidney of fish is represented in Table No. 1, 2, 3.

Maruthi*et al*.(5) worked on effect of sugar mill effluent on oxygen consumption of freshwater fish *Channa punctatus*, observed that decrease in glycogen, total protein, lipid with increasing concentration of distillery effluent indicate a decrease in energy supply metabolism through oxidative pathways which ultimately lead to less growth in the fish, *Channa punctatus*.

Research carried out on endosulfan induced metabolic alteration in freshwater fish, *Catlacatla*observed that the decrease in the protein content under the pesticidal stress.(6)

Research carried out oneffect of cypermethrin on protein metabolism of the fish, *Labeo rohita*, observed that the total protein level decreased in all the tissues tested whereas the free amino acid levels were increased.(7)

studies on impact of hildan on biochemical constituents in the freshwater mussel, *Lamellidenscorrianus*and reported that significant decrease in total protein and total free amino acid content in foot were observed after 24, 48, 72 and 96 hours. The total protein of hepatopancreasshowed no significant decrease after 24 hours. In gills significant decrease in total protein content.(8)

Suggestedresearch of depletion of protein may be due to utilization of protein for the production of energy to mitigate the pesticide stress and to prevent from fatique due to effect of pesticide.(9)

Studied on impact of hildan on total protein content of freshwater crab *Barytecphusaguerini*and reported that the total protein content in muscles, hepatopancreas and gill of crab were found to decrease in all sublethal concentration of hildan and at all exposure periods.(10)

Biochemical changes induced by pesticide phosalone in *Cyprinus carpio* (Linn) and reported that the subacute period of exposure a significant depletion in protein contents were observed in different tissues. He reported that it may be due to the more utilization of protein to meet out the energy demand when the fish was under stress condition.(11)

Carbohydrate is used as the principle and immediate energy precursor in fish under stress conditions. According to Rate and Rao (1991) the decreased level of lipid might be due to the utilization of lipid to meet the additional energy required under stress.(12)

studied on the alterations in the total protein during exposure to Nuvan toxicity on catfish *Clariasbatrachus*and reported that the alteration in various metabolites appear to the tissue specific and time dependent which can be attributed the absorption distribution and elimination patent of Nuvan in the tissue(13). Narayana Swamy(14) reported that the changes and decrease in protein level might be due to inhibition or induction of metabolizing enzymes by administration of toxicants.

The decrease in the level of glycogen content was observed in all tissue with increase in exposure periods. In sub lethal concentration also, decrease in glycogen reserves was observed during the initial period, which further slowly regressed to normal. The carbohydrate metabolism is disturbed when *L.rohita*is exposed to fenvalerate. Alternation in the blood glucose level indicate the variations in the carbohydrate metabolism of the fish under toxic stress.(15)

Toxicity and effect of fenvalerate on fish *Ctenopharyngodon idella* and reported that the decreasing trend of glycogen and protein under pesticidal stress.(16)

Enzyme related to sex difference in mice with muscular dystrophy and reported that the decreased glycogen synthesis due inhibition of glycogen synthetase which mediate glycogen synthesis.(17)

Exposure of fish to sublethal and lethal concentration of dimethoate showed veriation in level of biochemical components of muscle, gill, liver and kidney.

The amount of glycogen content of muscle, liver, gills and kidney was decreased on 96 hours exposure to sublethal concentration and lethal concentration of dimethoate. In chronic exposure to dimethoate glycogen level decreased continuously in all the tissue of fish in throughout the expsoure period.

Lipids level also decreased in all the tissues exposed to lethal and sublethal concentration of dimethoate for period of 96 hours. In chronic exposure to dimethoate lipid level decrerasedcontinuasly in all the tissues in throughout the exposure period.

Protein levels decreased in all the tissues exposed to lethal and sublethal concentration of dimethoate for period of 96 hours. In chronic exposure to dimethoate protein level decreased continuously in all the tissues in throughout the exposure period.

**CONCLUSION:**

In the present investigation, it has been found that decrease in biochemical components of all tissues. It has been also found that among these biochemical components, glycogen was maximum decreased than lipid and protein. Decrease in biochemical component of tissues might be due glycogenolysis, proteolysis, and lipolysis to meet energy demand of fish under pesticide stress.

**Reference:**


1. APHA. (1998) Standard Methods for the Examination of Water and Wastewater. American Public Health Association, 20th edition, Washington. D.C.
2. GORNALL AG, BARDAWILL CJ, DAVID MM. Determination of serum proteins by means of the biuret reaction. J Biol Chem. 1949 Feb;177(2):751-66. PMID: 18110453.
3. SEIFTER S, DAYTON S, et al. The estimation of glycogen with the anthrone reagent. Arch Biochem. 1950 Jan;25(1):191-200. PMID: 15401229.
4. BREIL C, ABERT VIAN M, ZEMB T, KUNZ W, CHEMAT F. "BLIGH AND DYER" AND FOLCH Methods for Solid-Liquid-Liquid Extraction of Lipids from Microorganisms. Comprehension of Solvatation Mechanisms and towards Substitution with Alternative Solvents. Int J Mol Sci. 2017 Mar 27;18(4):708.
5. MARUTHI, M. N., NAVANEETHAN, S., COLVIN, J. AND HILLOCKS, R. J. (2004B): Bionomics, morphometric measurements and molecular characterisation of a cassava Bemisia afer (Priesner & Hosny) population. International Journal of Tropical Insect Science (In press).
6. SHIVKUMAR (2005), Bioaccumulation of heavy metals in fish species from the Meiliang Bay, Taihu Lake, China, Toxicology Reports,Volume 5, 2018, Pages 288-295
7. KHAN, R.R. & BHASKER DEV (1982) *Toxicology data sheets on chemicals: dimethoate,* Lucknow, India, Lucknow Industrial Toxicology Research Centre (No. 6).

8. KHERA, K.S., WHALEN, C., TRIVETT, G., & ANGERS (1979) Terato-genicity studies on pesticidal formulations of dimethoate,diuron, and lindane in rats. *Bull. environ. Contam. Toxicol.*, **22**: 522-529.

9. KISS, Z. & FAZEKAS, T. (1983) Organophosphates and torsade de pointes ventricular tachycardia. *J. R. Soc. Med.*, **76**: 984-985.
10. KNAAK, J.B. (1980) Minimizing occupational exposure to pesticides. Techniques for establishing safe levels of foliar residues. *Residue Rev.*, **75**: 81-96.

11. KNAAK, J.B., SCHLOCKER, P., ACKERMAN, C.R., & SEIBER, J.N. (1980) Reentry research: Establishment of safe pesticide levels on foliage. *Bull. environ. Contam. Toxicol.*, **24**: 796-804.
12. KÖPPEL, C., FORYCKI, Z., & IBE, K. (1986) Hemoperfusion in severe dimethoate poisoning. *Intensive Care Med.*, **12**(2): 110- 112.
13. KRUEGER, H.R., O'BRIEN, R.D., & DAUTERMAN, W.C. (1960) Relationship between metabolism and differential toxicity in insects and mice of dimethoate, parathion, and acethion. *J. econ. Entomol.*, **53**(1): 25-31.
14. KUNDIEV, YU.I. (1979) [ *Absorption of pesticides through the skin and prophylaxis of poisonings,*] Kiev, Zdorovye Publishers, 200 pp (in Russian).
15. LARRIPA, I., MATOS, E., & DE SALUM, S.B. (1979) [Cytogenetic study of human population accidentally exposed to an organophos- phate pesticide.] *Medicina (Buenos Aires)*, **39**(6): 773-774 (in Spanish).
16. LARRIPA, I., MATOS, E., DE VINUESA, M.L., & DE SALUM, S.B. (1983) Sister chromatid exchanges in a human population accidentally exposed to an organophosphorus pesticide. *Rev. Braz. Genet.*, **4**: 719-727.
17. LEBLANC, F.N., BENSON, B.E., & GILG, A.D. (1986) A severe organophosphate poisoning requiring the use of an atropine drip. *Clin. Toxicol.*, **24**(1): 69-76.