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# ALTERATIONS IN THE LIPID METABOLISM DURING DELTAMETHRIN INDUCED TOXICITY IN *CIPRINUS CARPIO*

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***Abstract:*** Present study is aimed at to investigate the effect of two different concentrations of (5ppm, 10ppm) deltamethrin in *Ciprinus carpio* different tissues i.e Liver, Heart, Kidney and Brain with reference to lipid metabolism. The fishes were randomly divided into 3 groups having 6 in each group: (1) Control (2) Deltamethrin-experimental toxic group (5ppm) (3) Deltamethrin-experimental toxic group (10 ppm). Deltamethrin- experimental groups increased the contents of Lipid peroxidation (LP), Super oxide dismutase (SOD) levels and decreased the levels of Phospholipids (PL), Total Cholesterol (TC) and Triglycerides (TG) in all the fish organs (Liver, Heart, Kidney and Brain). From the results it is presumed that the deltamethrin might be implicated to the either reduced synthesis / augmented degradation by lipoprotein lipase activity.

Key words: Deltamethrin, *Ciprinus carpio*, Lipids.

## 1. INTRODUCTION

Biological lipids are a chemically diverse group of compounds, the common and defining feature of which is their insolubility in water. The biological functions of the lipids are as diverse as their chemistry. Fats and oils are the principal stored forms of energy in many organisms. Phospholipids and sterols are major structural elements of biological membranes. Other lipids, although present in relatively small quantities, play crucial roles as enzyme cofactors, electron carriers, light absorbing pigments, hydrophobic anchors for proteins, "chaperones" to help membrane proteins fold, emulsifying agents in the digestive tract, hormones, and intracellular messengers (Nelson, D.L and M.M. Cox, 2005). The fats and oils used almost universally as stored forms of energy in living organisms are derivatives of fatty acids. The fatty acids are hydrocarbon derivatives, at about the same low oxidation state (that is, as highly reduced) as the hydrocarbons in fossil fuels. The cellular oxidation of fatty acids (to $CO_2$ and $H_2O$), like the controlled, rapid burning of fossil fuels in internal combustion engines, is highly exergonic. Lipids play a variety of cellular roles, some only recently recognized. They are the principal form of stored energy in most organisms and major constituents of cellular membranes. Specialized lipids serve as pigments (retinal, carotene), cofactors (vitamin K), detergents (bile salts), transporters (dolichols), hormones (vitamin D derivatives, sex hormones), extracellular and intracellular messengers (eicosanoids, phosphatidylinositol derivatives), and anchors for membrane proteins (covalently attached fatty acids, prenyl groups, and phosphatidylinositol). The ability to synthesize a variety of lipids is essential to all organisms. Lipids play an important role as source of energy for fish. Since, most insecticides are lipophilic compounds, they can easy pass through biological barriers which content lipids and accumulate in fat tissue. Lipids molecules are highly susceptible to oxidative reactions. Due to cell membrane lipid peroxidation of unsaturated fatty acids, short chain fatty acids with RCOOH, R-OOH, R-CHO, and R-OH bases are created which seriously affect the cellular membrane functions such as the activity of hormone receptors and neural mediators, ion transport channels and the activity of membrane enzymes and the transportation of specific molecules. On

the other hand, the formation of malondialdehyde (MAD) during peroxidation process of fatty acids having double bonds can create covalent bonds and polymerize cellular membrane components (Sureda *et al.*, 2006; Tejada *et al.*, 2007). In addition, accumulation of fatty acids in the cytosol increased peroxidation of fatty acids in peroxisomes and the endothelial reticulum, resulting in overproduction of ROS and further damage.

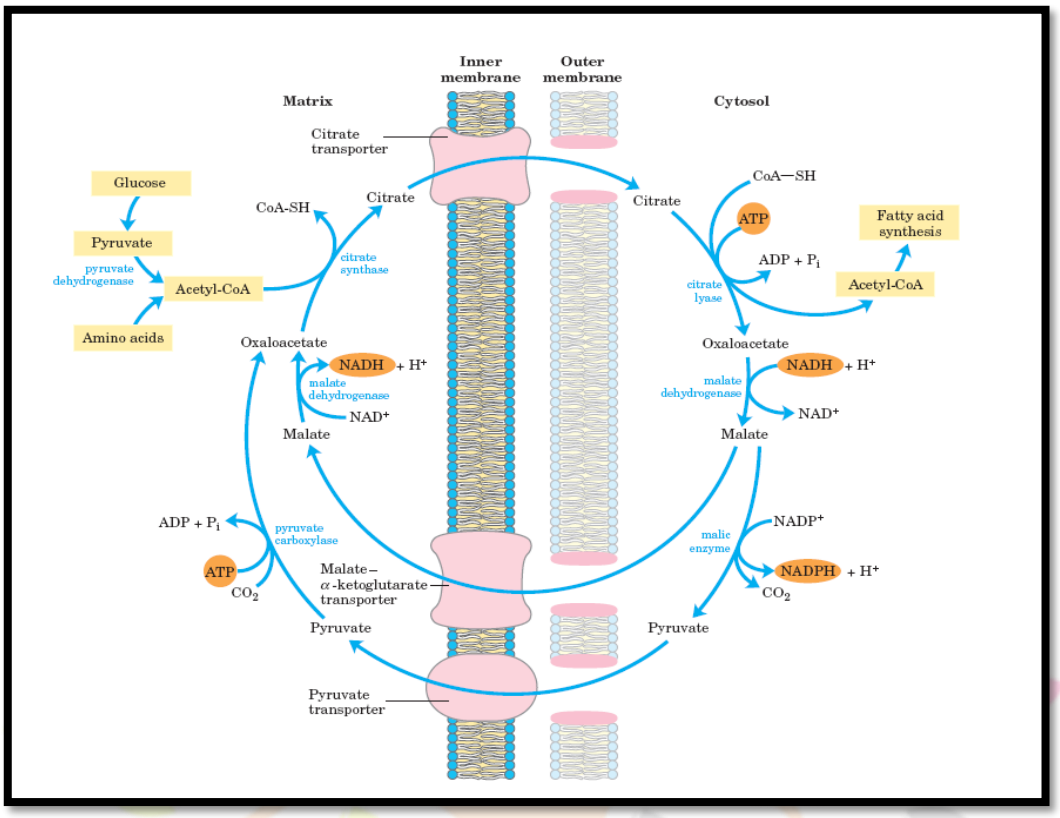



**FIGURE 1. Shuttle for transfer of acetyl groups from mitochondria to the cytosol.**

The mitochondrial outer membrane is freely permeable to all these compounds. Pyruvate derived from amino acid catabolism in the mitochondrial matrix, or from glucose by glycolysis in the cytosol, is converted to acetyl-CoA in the matrix. Acetyl groups pass out of the mitochondrion as citrate; in the cytosol they are delivered as acetyl-CoA for fatty acid synthesis. Oxaloacetate is reduced to malate, which returns to the mitochondrial matrix and is converted to oxaloacetate. An alternative fate for cytosolic malate is oxidation by malic enzyme to generate cytosolic NADPH; the pyruvate produced returns to the mitochondrial matrix. (Nelson, D.L and M.M. Cox 2005).

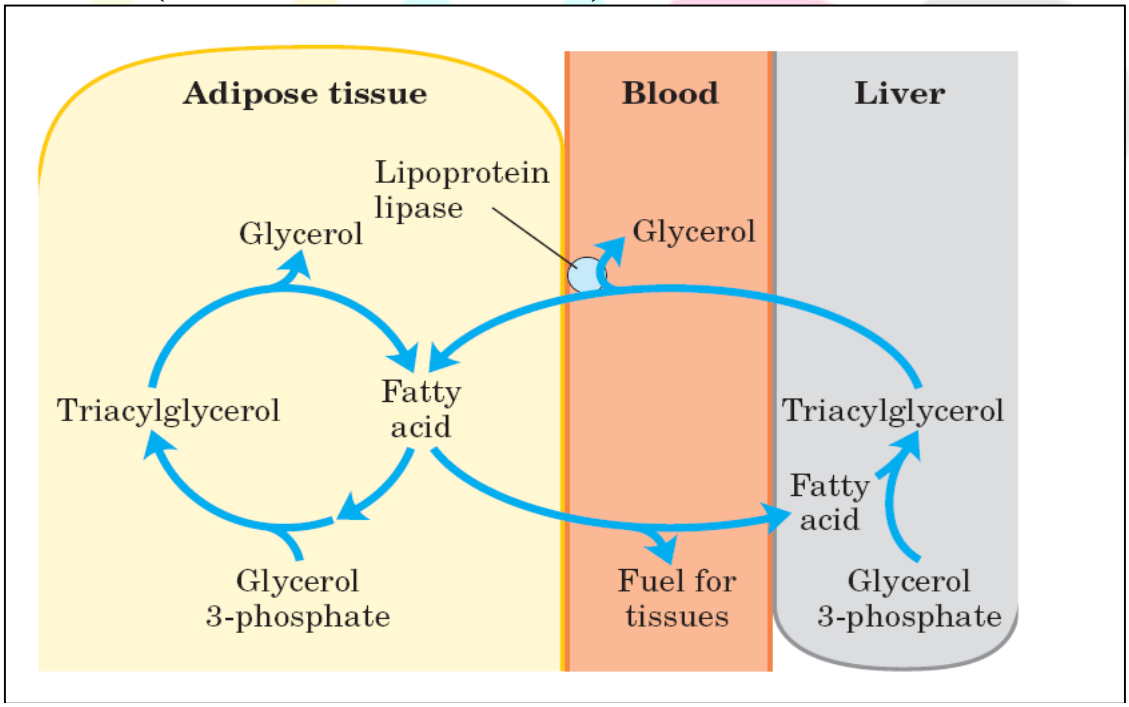



**FIGURE 2. The triacylglycerol cycle.**

Triacylglycerol molecules are broken down and resynthesized in a triacylglycerol cycle during starvation. Some of the fatty acids released by lipolysis of triacylglycerol in adipose tissue pass into the bloodstream, and the remainder are used for resynthesis of triacylglycerol. Some of the fatty acids released into the blood are used for energy (in muscle, for example), and some are taken up by the liver and used in triacylglycerol synthesis. The triacylglycerol formed in the liver is transported in the blood back to adipose tissue, where the fatty acid is released by extracellular lipoprotein lipase, taken up by adipocytes, and reesterified into triacylglycerol (Nelson, D.L and M.M. Cox, 2005).

Cholesterol is doubtless the most publicized lipid, notorious because of the strong correlation between high levels of cholesterol in the blood and the incidence of human cardiovascular diseases. Less well advertised is cholesterol's crucial role as a component of cellular membranes and as a precursor of steroid hormones and bile acids. Cholesterol is an essential molecule in many animals, including humans, but is not required in the mammalian diet—all cells can synthesize it from simple precursors. The structure of this 27-carbon compound suggests a complex biosynthetic pathway, but all of its carbon atoms are provided by single precursor acetate. The isoprene units that are the essential intermediates in the pathway from acetate to cholesterol are also precursors to many other natural lipids, and the mechanisms by which isoprene units are polymerized are similar in all these pathways. The normal regulation of cholesterol synthesis and its regulation in those with defects in cholesterol uptake or transport (Nelson, D.L and M.M. Cox 2005).

In the fish exposed to 0.1 mg/L diazinon, disorientation in the glomerular structure, dilation in the inter space of urinary tubular, cloudy swelling were observed. Histopathological damage in the kidney tissues of fish exposed to 0.2 mg/L diazinon were characterized by degeneration in the epithelial cells of renal tubule, necrosis in the hematopoietic tissue, degeneration of glomerulus, dilation of glomerular capillaries, appearance of vacuoles in cytoplasm epithelial cells of renal tubules with hypertrophied cells and narrowing of the tubular lumen. Increase of ROS production in the diazinon metabolism. process may play an important role in lipid peroxidation of components of cellular membrane resulting in reduced nephron number, glomerular lesions, and reduced glomerular filtration rate. Similar studies showed the toxic effect of insecticides on the histology of kidney of Atlantic salmon (Salmo salar), rainbow trout, O. mykiss, exposed to endosulfan, as pesticide and captan, as the fungicide, respectively (Glover *et al.,* 2007; Boran *et al.,* 2012).

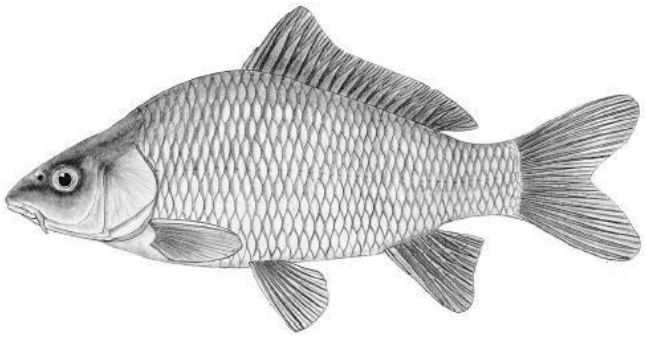
## 2.0. MATERIALS AND METHODS

### 2.1. Experimental animals

The tests were performed in a concrete holding tanks, glass aquaria, constant supply of water and good lighting system. The indoor tanks were filled with tap water and aerated for 3 days to help reduce the chlorine content. About 300 active test specimens ranging between 5 and 10 cm standard length were transported to the laboratory from a farm. The specimens were acclimatized to laboratory conditions for 7 days in the indoor holding tanks. The pH, dissolved oxygen concentration and temperature of water in the tanks were monitored.

Preliminary tests were conducted to provide guidance on range of concentration of pesticide to use in the bioassay. A stock solution of 25 mg/l was prepared from the original product concentration of 12.5 g/l. From the stock solution, the test solutions were prepared using distilled water. The specimens were not fed a day prior to and during toxicity tests to reduce faecal and excess food contaminating the test solution. The nominal test concentrations were 5ppm&10ppm with six replicates each. The results from the toxicity tests were analyzed, using a World Health Organisation (WHO). The concentrations used were converted by the programme to log dose and the number of dead fishes to mortality Probit values. A plot of these two parameters was made from which the $LC_{50}$ was estimated. The fishes were maintained according to the ethical guidelines.

**2.2 *Cyprinus carpio* (Common carp)**



| | |
|---|---|
| Kingdom: | Animalia |
| Phylum: | Chordata |
| Class: | Actinopterygii |
| Order: | Cypriniformes |
| Family: | Cyprinidae |
| Genus: | *Cyprinus* |
| Species: | *C. carpio* |

*Cyprinus carpio* (Common carp)

**2.3. Selection of Pesticide**

Deltamethrin, a synthetic pyrethroid pesticide, was selected for the present study. It was obtained as commercial grade chemical from Sigma chemicals, USA.

Deltamethrin

| **Properties** | |
|---|---|
| Molecular formula | $C_{22}H_{19}Br_2NO_3$ |
| Molar mass | 505.21 g $mol^{-1}$ |
| Density | 1.5 g $cm^{-3}$ |
| Melting point | 98 °C, 371 K, 208 °F |
| Boiling point | 300 °C, 573 K, 572 °F |

**2.4. Experimental design**

The fishes were divided into 3 groups, each consisted of 6 and used for studying the effects of different concentrations of deltamethrin.

Group 1 - Control
Group 2 - 5ppm concentration
Group 3 - 10ppm concentration

**2.5. Isolation of Tissues**

The animals were sacrificed. Functionally different organs such as Liver, Heart, Kidney and Brain were separated and frozen in liquid nitrogen (-180$^0$C) and stored at -4$^0$C until further use. At the time of analyses the tissues were thawed and selected parameters were estimated by employing standard methods.

**2.6. Procurement of Chemicals**

All chemicals used in the present study were Analar grade (AR) and obtained from the following scientific companies: Sigma (St. Louis, MO, USA), Fisher (Pittsburg, PA, USA), Merck (Mumbai, India), Ranbaxy (New Delhi, India), Qualigens (Mumbai, India).

**2.7. Toxicity in aquatic fish for $LC_{50}$**

Fishes were reared and acclimatized to the laboratory conditions in the glass aquaria containing aerated tap water avoiding overcrowding. The water was renewed every day and the fishes were fed with poultry feed on alternate day. During the experimentation fishes were not fed. The physico-chemical characteristics of the test water such as temperature (22±2$^0$C), water hardness (420 mg/lit), pH (7±0.2) and Dissolved oxygen (4.27 ml/lit) were analyzed during the experiments according to standard methods suggested by APHA, (1989). The stock solution of pesticide, endosulfan (35%) was prepared by dissolving a known concentration of this pesticide into the distilled water and required concentrations were made from the stock solution. The acute toxicity as widely accepted for the period of 24, 48, 72 and 96 hrs. have been carried out in the present investigation and the observations were made on the percentage mortality of the fish. The $LC_{50}$ values were calculated by using regression equation method of probit analysis (Hubert and Schoch, 1984).

## 2.8. Lipid profiles and lipid peroxidation

### 2.8.1. Phospholipids

Phospholipids were estimated by the method of Zilversmidth and Davis (1950). The tissues were homogenized with 3 ml of 10% trichloro acetic acid (TCA) and centrifuged at 1500 rpm for 10 min. The supernatant was discarded and to the residue 1 ml of 60% perchloric acid (PCA) was added and digestion was continued on a heating-mantle to obtain a clear and colorless solution. Then the contents were cooled and 5 ml of distilled water was added to each tube.

To each sample, 1 ml of 4% (w/v) ammonium molybdate was added and mixed. Finally 1 ml of ANSA (1-amino 2-naphthalo 4-sulphonic acid) reagent was added and the contents were made up to 10 ml with distilled water. The time of addition of ANSA reagent was noted. The color developed was read at 660 nm against the blank within 6 min. Blank contained 0.8 ml of 60% PCA and 2 ml of phosphate standards (containing 0.01 mg phosphorous / ml). The blanks and standards were also diluted with distilled water to a volume of 10 ml. The color developed was read in the same manner as mentioned above. The results were finally expressed in mg of phospholipids / gram wet weight of tissue.

### 2.8.2.Total cholesterol (TC)

The total cholesterol content was estimated using Liebermann Burchard reaction as described by Natelson (1971). The tissue was homogenized in isopropanol. The contents were centrifuged at 1000g for 15 min. 0.5 ml of supernatant was taken and to it 4 ml of cholesterol reagent was added. Then the contents were heated at $90^0$C for 15 min. After cooling, the samples were read at 560nm in spectrophotometer against the reagent blank. The results were finally expressed in mg of total cholesterol/gram wet weight of the tissue.

### 2.8.3.Triglycerides (TG)

Triglycerides were estimated by the method of Natelson (1971) with slight modifications as given below. Triglycerides were assayed by hydrolyzing them to glycerol and the liberated glycerol was determined.

The tissue homogenates were prepared in 1N $H_2SO_4$ and to it 4 ml of chloroform was added. 0.5 ml of tissue homogenate was taken, to it 0.5 ml of 1N $H_2SO_4$ and 4 ml of chloroform were added. The contents were centrifuged at 1000 rpm for 15 min 0.5 ml of chloroform layer was taken and to it 0.4 ml of methanol and 0.1 ml of alkaline barium solution were added and the contents were heated for 30 min at $80^0$C, the total volume was made up to 1 ml with 2N $H_2SO_4$ and centrifuged for 10 min at 1000 rpm. 0.5 ml of this supernatant was taken and to it 0.1 ml of sodium periodate was added and shaken well for 1 min, 0.1 ml of sodium arsenate and 5 ml of chromotrophic acid reagent was added and heated for 30 min and cooled. The samples were read a 575 nm is Spectrophometer against the reagent blank. The results were finally expressed in mg of triglycerides/ gram wet weight of the tissue.

### 2.8.4.Lipid peroxidation (LP)

This assay was used to determine MDA levels as described by Ohkawa *et al.,* (1979). The tissue was homogenized (5% w/v) in 50 mM phosphate buffer (pH 7.0) containing 0.1 mM EDTA. The homogenates were centrifuged at 10,000 rpm for 10 min at $4^0$C in ice cold centrifuge. The separated supernatant part was used for the estimation. 200 µl of the tissue extract was added to 50 µl of 8.1% sodium dodecyl` sulphate (SDS), vortexed and incubated for 10 min at room temperature. 375 µl of twenty percent acetic acid (pH 3.5) and 375 µl of thiobarbituric acid (0.6%) were added and placed in a boiling water-bath for 60 min. The samples were allowed to cool at room temperature. A mixture of 12:5 ml of butanol: phyridine (15:1) was added, vortexed and centrifuged at 1000 rpm for 5 min. The colored layer (500 µl) was measured at 532 nm using 1, 1, 3, 3-tetraethoxypropane as a standard. The values were expressed in µ moles of malondialdehyde formed / gram wet weight of the tissue.

### 2.8.5.Xanthine Oxidase (XOD)

Xanthine oxidase activity was assayed by the dye reduction method of Srikanthan and Krishnamurthy, (1955). The assay mixture contained 100 µ moles of sodium phosphate buffer (pH 7.4), 50 µ moles of xanthine, 0.1 µ moles of NAD, 0.4 µ moles of INT and the enzyme source. The reaction was initiated by the addition of 20 mg of enzyme source and incubated at $37^0$C for 30 min. The reaction was stopped by the addition of 5 ml glacial acetic acid and the formazan formed was extracted into 5 ml of toluene and read at 495 nm against toluene blank. The activity was expressed in µ moles of formazan formed / mg protein / hour.

### 2.8.6. Statistical treatment of data

All assays were carried out with six separate replicates from each group. The mean, standard error (SE) and Analysis of Variance (ANOVA) were done using SPSS statistical software for different parameters. Difference between control and experimental assays was considered as significant at $\rho<0.05$.

## 3. RESULTS AND DISCUSSION

Lipid peroxidation is a complex process generating reactive radicals, which is enriched with polyunsaturated fatty acids (PUFAs), are more prone to free radical mediated lipid peroxidation. Lipid peroxidation of cell membranes causes a loss of the fluid properties of the membrane as well as increase in membrane permeability (Packer 1984; Pradhan *et al.,* 1990). Lipid peroxidation refers to the oxidative degradation of lipids. It is the process whereby free radicals "steal" electrons from the lipids in cell membranes, resulting in cell damage.

Phospholipids are complex lipids containing in addition to fatty acids, glycerol or other ethanol, a phosphoric acid residue, protein containing base and other substituent's. A major function of these phospholipids is to serve as structural components of membranes of the cell surface and sub cellular organelles. It also serves as donors of arachidonic acid for the synthesis of prostaglandins, thromboxanes. leukotrienes and related compounds (Devlin, 1986).

Phospholipids participate in the formation of lipoprotein complexes, which are thought to constitute the matrix of cell walls and membranes, the myelin sheath and of such structures as mitochondria and microsomes. Phospholipids play an important role in the blood coagulation process (Chatterjea and Ranashinde, 1993). These have an important role in the activities of the membrane bound enzyme complex and were also found to be sensitive to lipophilic inhibitor toxic chemicals (Cutkomp and Koch, 1981). It was also suggested that phospholipids are essential for the activities of Na+, K+ and Mg2+ ATPases and also for the activity of several dehydrogenases and other enzymes concerned with oxidative phosphorylation (Gregoriadis *et al.,* 1977). It has also been reported that the decrease in the activity of above mentioned enzymes might be due to the loss of phospholipids (Nadamuni Chetty, 1991). Since the alterations in the phospholipid content has significant impact on the membrane architecture, an attempt is made in the present investigation to study the alterations in phospholipids content after the exposure of different concentrations of deltamethrin on fish *Cyprinus carpio*.

The triglycerides are esters of alcohol, glycerol and fatty acids and play an important role in the metabolism as energy sources and transporters of dietary fat. They contain more than twice as much energy (9 K Cals/g) as carbohydrates and proteins. The triglycerides are hydrolyzed to produce fatty acids and glycerol in order to meet the energy demands. Since deregulated lipid metabolism may be of particular importance for tissues in fish Liver, Heart, Kidney and Brain an attempt was made in the present investigation to study the alterations in triglyceride content in different tissues of fish after exposure of different concentrations of deltamethrin.

Cholesterol is an amphipathic lipid, synthesized in many tissues from acetyl Co-A, occur in all tissues either as free cholesterol or as cholesterol ester. It forms a precursor for all steroids in the body such as corticosteroids, sex hormones, bile acids and vitamin-D (Nelson and Cox, 2001).

Xanthine oxidase (XOD) is an essential enzyme that converts hypoxanthine to xanthine, subsequent to uric acid. This enzyme contains FAD, molybdenum and Iron are exclusively found in liver, intestine and little amount in other tissues of animals (Sathyanarayana, 2005) also stated XOD played a vital role in transformation of toxic ammonia into nontoxic uric acid. Xanthine oxidase produces hydrogen peroxide which is very dangerous to the animal, and then it converts into HO and $O_2$. Further, the uric acid may act as an antioxidant and free radical scavenger protects the cells from oxidative damage (Sheehan *et al.,* 2001; Guskov *et al.,* 2002).

**3.1. Results :**

The changes in the Lipid peroxidation content in different organs of fish after exposure of different concentrations of deltamethrin were represented in tables 5.1-5.4

The elevation in the Lipid peroxidation content can be represented as:

LP: Liver>Heart>Kidney>Brain

The changes in the Phospholipids content in different organs of fish after of exposure of different concentrations of deltamethrin represented in tables 5.1-5.4

The of depletion in the Phospholipids content after of exposure of different concentrations of deltamethrin can be represented as:

PL: Liver>Heart>Kidney>Brain

The changes in the Total cholesterol content in different tissues of fish after exposure of different concentrations of deltamethrin were represented in tables 5.1-5.4

The percent change of depletion in the Total cholesterol content can be represented as:

TC: Liver>Heart>Kidney>Brain

The changes in the triglycerides content in different tissues of fish were represented in tables 5.1-5.4 The depletion in the triglycerides content can be represented as:

TG: Liver>Heart>Kidney>Brain

The changes in the XOD levels in different organs of fish after exposure of different concentrations of deltamethrin were represented in tables 5.1-5.4

The elevation in the XOD levels can be represented as:

XOD : Liver>Heart>Kidney>Brain

**3.2. Discussion:**

Deltamethrin, GABA antagonist, induced oxidative stress as indicated by a significant rise in the MDA levels, an indicator of lipid peroxidation in fish. The disorders in lipid organization of biological membranes result in alterations in the activity of a number of membrane bound enzymes (Ohkawa *et al.*, 1979). Oxygen free radical reacts with polyunsaturated fatty acid (PUFA) in the cell membrane and new free radical (Peroxyradical) are formed. In the presence of oxygen, the peroxyl radical initiates chain reaction called lipid peroxidation. Lipid peroxidation products are constantly involved in some of the pathophysiological effects associated with oxidative stress in cell and tissues. Unlike reactive free radicals, aldehydes can produce lipid peroxidation products, which are rather long lived and can therefore diffuse from the site of their origin, reaching and attacking intracellular and extra cellular targets (Esterbauer *et al.*, 1991). They disrupt various important structural and protective functions associated with bio-membranes in various in vivo pathologic events and are implicated as a result of this oxidation (Barclay. 1993). Although generation of low concentrations of ROS plays a significant role in signal transduction cascades triggered by growth factors, cytokinines, hormones etc. high production of ROS impose a number of direct and indirect consequences on signaling pathways resulting in the induction of apoptosis or neuro-necrosis. Since lipoperoxidation of neural membrane is a harmful process, altering membrane fluidity, protein structure and cell signaling pathways. Free radicals can peroxidise polyenoid acids and lead to the formation of malondialdehyde (MDA) and also participates in the oxidation of proteins, which causes enzymatic inactivation (Dean *et al.*, 1991). Free radicals inactivate enzymes, break DNA and initiate chain reactions that peroxidise the lipids. Hence, the present investigation is taken up to determine the alterations in lipid profiles in different organs of fish after exposure of different concentrations of deltamethrin.

Membrane phospholipids have a dual role as structural building blocks of cell membranes and as precursor molecules involved in signal transduction such as the lipid second messengers diacyl glycerol, phosphatidic acid, lysophosphatidic acid and arachidonic acid (Hodgkin *et al.*, 1998). The decreased PL content may serve as a metabolic alarm to the animal in the sense that the membrane integrity is lost. The phospholipid content was decreased in the present study in all the tissues of fish which might be implicated to the enzymatic hydrolysis of membrane phospholipids by phospholipases leading to loss of membrane integrity. From the observation of present study coupled with the above reports, it can be speculated that both the inhibition of phospholipid synthesis and activation of phospholipases have been involved in the reduced levels of phospholipids in different tissues of fish after exposure of different concentrations of deltamethrin.

From the results it is clear that deltamethrin might be implicated to the either reduced synthesis / augmented degradation by lipoprotein lipase activity. The results are also in congruence with the previous reports where substantial loses of lipids including cholesterol and occurrence of peroxidative damage during arsenic induced oxidative damage (Haider and Najar, 2008) and during exposure to environmental pollutants and heavy metals (Haider and Hasan, 1984; Haider *et al.,* 1981; Pande *et al.,* 1989).

The decreased levels of triglycerides in different organs of fish might be due to enhanced lipolysis through lipase activity. Hence, decreased triglyceride content observed in the present study might be implicated to the activation of phospholipases and lipases due to excitotoxin-induced calcium flux resulting in accumulation of free fatty acids, diacylglycerols, eicosonoids and lipid peroxides.

Naveed and Janaiah (2011) reported that the reduction in XOD activity in liver of fish, Channa punctatus exposed to triazophos leads to increase in cellular damage and may be due to nonavailability of Iron to the fish during toxic period. Free radicals are formed during the normal metabolic processes (Yoshika *et al*., 1990), in addition to being generated by exposure to toxic agents (Halliwell and Gutteridge 1985) and several other disease states (Kellog and Fridovich 1977; Lambert and Bondy 1989). It has been well established that uric acid is the most abundant antioxidant and a powerful free radical scavenger (Waring, 2002) and particularly effective in quenching hydroxyl, superoxide and peroxynitrite radicals and may serve a protective physiological role by preventing lipid peroxidation (Squadrito *et al.,* 2000) and increased uric acid concentrations during oxidative stress might be considered as a compensatory mechanism that confers protection against increased free radical injury (Nieto *et al.,* 2000) that occurs after exposure of deltamethrin observed in the present study.

## 4.0. SUMMARY AND CONCLUSION

Lipid profiles are important components of the biochemical environment in the fish. They serve several functions in the biological systems such as structural components of the membranes, storage and transport forms of metabolic fuel, protective coating on the surface concerned in cell recognition, species specifity and tissue immunity. Lipid peroxidation is one of the most widely accepted assays for oxidative damage.

Free radicals are formed during the normal metabolic processes in addition to being generated by exposure to toxic agents. Deltamethrin, GABA antagonist, induced oxidative stress as indicated by a significant rise in the MDA levels, an indicator of lipid peroxidation in fish. The disorders in lipid organization of biological membranes result in alterations in the activity of a number of membrane bound enzymes. Free radicals can peroxidise polyenoid acids and lead to the formation of malondialdehyde (MDA) and also participates in the oxidation of proteins, which causes enzymatic inactivation.

The survival of an organism may depend upon its ability to overcome the toxic effects of reactive oxygen species (ROS) because oxidation of thiol groups during oxidative stress can inactivate enzymes and hydroxyl radicals are known to attack cellular proteins, lipids and nucleic acids.

Phospholipids are complex lipids containing in addition to fatty acids, glycerol or other ethanol, a phosphoric acid residue, protein containing base and other substituent's. Phospholipids play an important role in the blood coagulation process. These have an important role in the activities of the membrane bound enzyme complex and were also found to be sensitive to lipophilic inhibitor toxic chemicals. Phospholipids content has significant impact on the membrane architecture, an attempt is made in the present investigation to study the alterations in phospholipid content during Deltamethrin toxic exposure.

Cholesterol is an amphipathic lipid, synthesized in many tissues from acetyl Co-A, occur in all tissues either as free cholesterol or as cholesterol ester. It forms a precursor for all steroids in the body such as corticosteroids, sex hormones, bile acids and vitamin-D. Substantial loses of lipids including cholesterol and occurrence of peroxidative damage during deltamethrin oxidative damage.

The triglycerides are esters of alcohol, glycerol and fatty acids and play an important role in the metabolism as energy sources and transporters of dietary fat. They contain more than twice as much energy (9 K Cals/g) as carbohydrates and proteins. The triglycerides are hydrolyzed to produce fatty acids and glycerol in order to meet the energy demands. The triglycerides are hydrolyzed to produce fatty acids and glycerol in order to meet the energy demands. Since deregulated lipid metabolism may be of particular importance for tissues in fish Liver, Heart, Kidney and Brain an attempt was made in the present investigation to study the alterations in triglyceride content in different tissues of fish after exposure of different concentrations of deltamethrin. The decreased levels of triglycerides in different organs of fish might be due to enhanced lipolysis through lipase activity. Hence, decreased triglyceride content observed in the present study might be implicated to the activation of phospholipases and lipases due to excitotoxin-

induced calcium flux resulting in accumulation of free fatty acids, diacylglycerols, eicosonoids and lipid peroxides.

Xanthine oxidase (XOD) is an essential enzyme that converts hypoxanthine to xanthine, subsequent to uric acid. This enzyme contains FAD, molybdenum and Iron are exclusively found in liver, intestine and little amount in other tissues of animals. XOD played a vital role in transformation of toxic ammonia into nontoxic uric acid. Xanthine oxidase produces hydrogen peroxide which is very dangerous to the animal, and then it converts into HO and $O_2$. Further, the uric acid may act as an antioxidant and free radical scavenger protects the cells from oxidative damage. Increased XOD levels during oxidative stress might be considered as a compensatory mechanism that confers protection against increased free radical injury that occurs after exposure of deltamethrin observed in the present study.

The present study reveals that the Deltamethrin toxic/adverse effects in target and non-target areas of the different organs of fresh water fish *Ciprinus carpio*. However, further in depth studies are required to understand the physiological mechanism and adverse effects of Deltamethrin in fish *Ciprinus carpio*.

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**Table-5.1**
Alterations in the Lipid metabolism in **Liver** of fish *Cyprinus carpio* after insecticide deltamethrin exposure.

Values are expressed as - LP : µ moles of malondialdehyde formed / gram wet wt of the tissue. PL: mg of phospholipids/g wet wt of the tissue. TC: mg of total cholesterol / g wet wt of the tissue. TG: mg of triglycerides / g wet wt of the tissue. XOD: µ moles of formazan formed / mg of protein / hour.

| Liver | Control | 5ppm | 10ppm |
|---|---|---|---|
| LP | 34.012 | 41.181* | 47.686* |
| | ±0.867 | ±0.131 | ±0.331 |
| | | (21.26) | (39.2) |
| PL | 40.824 | 33.809* | 26.324* |
| | ±0.401 | ±0.668 | ±0.313 |
| | | (-19) | (-35.9) |
| TC | 24.693 | 19.216* | 13.923* |
| | ±0.226 | ±0.216 | ±0.015 |
| | | (-21.44) | (-43.34) |
| TG | 34.962 | 30.269 | 26.139* |
| | ±0.356 | ±0.862 | ±0.804 |
| | | (-7.73) | (-26.74) |
| XOD | 23.519 | 30.174* | 37.555* |
| | ±0.213 | ±0.386 | ±0.496 |
| | | (28.96) | (63.07) |

All the values are mean, ±SE of six individual observations.
Values in '( )'parentheses are % change over control.
*Values are significant at $P < 0.05$ in Scheffe test.

**Table-5.2**
Alterations in the Lipid metabolism in **Heart** of fish *Cyprinus carpio* after insecticide deltamethrin exposure.
Values are expressed in - LP : µ moles of malondialdehyde formed / gram wet wt of the tissue. PL: mg of phospholipids/g wet wt of the tissue. TC: mg of total cholesterol / g wet wt of the tissue. TG: mg of triglycerides / g wet wt of the tissue. XOD: µ moles of formazan formed / mg of protein / hour.

| Heart | Control | 5ppm | 10ppm |
|---|---|---|---|
| LP | 30.124 | 38.773* | 44.224* |
| | ±0.084 | ±0.182 | ±0.25 |
| | | (28.02) | (47.02) |
| PL | 35.415 | 27.859* | 22.785* |
| | ±0.265 | ±0.373 | ±0.309 |
| | | (-20.89) | (-36.19) |
| TC | 22.351 | 15.978* | 11.447* |
| | ±0.237 | ±0.212 | ±0.074 |
| | | (-28.6) | (-49.4) |
| TG | 34.567 | 28.262* | 24.989* |
| | ±0.366 | ±0.426 | ±0.45 |
| | | (-16.86) | (-28.2) |
| XOD | 19.226 | 26.025* | 32.963* |
| | ±0.176 | ±0.248 | ±0.142 |
| | | (35.66) | (72.38) |

All the values are mean, ±SE of six individual observations.
Values in '( )'parentheses are % change over control.
*Values are significant at P < 0.05 in Scheffe test.

**Table-5.3**
Alterations in the Lipid metabolism in **Kidney** of fish *Cyprinus carpio* after insecticide deltamethrin exposure.

Values are expressed in - LP : µ moles of malondialdehyde formed / gram wet wt of the tissue. PL: mg of phospholipids/g wet wt of the tissue. TC: mg of total cholesterol / g wet wt of the tissue. TG: mg of triglycerides / g wet wt of the tissue. XOD: µ moles of formazan formed / mg of protein / hour.

| Kidney | Control | 5ppm | 10ppm |
|---|---|---|---|
| LP | 23.445 | 31.198* | 39.217* |
| | ±0.135 | ±0.328 | ±0.971 |
| | | (33.28) | (66.82) |
| PL | 29.193 | 23.326* | 19.147* |
| | ±0.402 | ±0.447 | ±0.647 |
| | | (-20.89) | (-36.91) |
| TC | 18.291 | 14.872* | 10.717* |
| | ±0.702 | ±0.576 | ±0.850 |
| | | (-18.1) | (-41.55) |
| TG | 32.498 | 31.072 | 23.067* |
| | ±0.329 | ±0.593 | ±0.275 |
| | | (-8.1) | (-41.55) |
| XOD | 18.177 | 24.751* | 30.928* |
| | ±0.343 | ±0.439 | ±0.726 |
| | | (37.23) | (70.71) |

All the values are mean, ±SE of six individual observations.
Values in '( )'parentheses are % change over control.
*Values are significant at P < 0.05 in Scheffe test.

**Table-5.4**
Alterations in the Lipid metabolism in **Brain** of fish *Cyprinus carpio* after insecticide deltamethrin exposure.

Values are expressed in - LP : µ moles of malondialdehyde formed / gram wet wt of the tissue. PL: mg of phospholipids/g wet wt of the tissue. TC: mg of total cholesterol / g wet wt of the tissue. TG: mg of triglycerides / g wet wt of the tissue. XOD: µ moles of formazan formed / mg of protein / hour.

| Brain | Control | 5ppm | 10ppm |
|---|---|---|---|
| LP | 16.228 | 24.274* | 33.222* |
| | ±0.408 | ±0.639 | ±0.108 |
| | | (49.33) | (104.64) |
| PL | 22.157 | 17.604* | 12.849* |
| | ±0.411 | ±0.543 | ±0.072 |
| | | (-20.89) | (-36.18) |
| TC | 16.504 | 12.676* | 9.828* |
| | ±0.126 | ±0.161 | ±0.835 |
| | | (-22.6) | (-40.56) |
| TG | 31.992 | 26.291* | 20.347* |
| | ±0.410 | ±0.266 | ±0.773 |
| | | (-17.15) | (-35.94) |
| XOD | 12.76 | 17.444* | 27.522* |
| | ±0.370 | ±0.339 | ±0.290 |
| | | (35.32) | (112.84) |

All the values are mean, ±SE of six individual observations.
Values in '( )'parentheses are % change over control.
*Values are significant at P < 0.05 in Scheffe test.