Asian Fisheries Science 12(1999):165-173
Asian Fisheries Society, Manila, Philippines
https://doi.org/10.33997/j.afs.1999.12.2.007

Influence of Pesticide Pyrethroid Deltamethrin Pollution on the Phospholipid Composition of Carp Erythrocyte Plasma Membrane

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Abstract

The effect of different concentrations of deltamethrin (0.5, 1.0, 2.0 and 4.0 ppm) on the carp erythrocyte plasma membrane was examined. Carp erythrocyte plasma membrane was exposed to deltamethrin for 48 and 96 hrs. Results showed that the deltamethrin pol-lutants significantly ($P \leq 0.01$) led to a decrease of phosphatidylethanolamine (PE), phosphoglyceride (PG) and phosphatidic acids (PA), with higher concentrations of deltamethrin leading to elimination of phosphatidic acids and cardiolipin (CL). Deltaméthrin also brought about a marked difference in the fatty acid patterns of the phospholipids in the erythrocyte plasma membrane.

Higher concentrations lead to increased levels of saturated fatty acids, primarily palm-itic (16:0) and stearic acid (18:0) as well as polyunsaturated fatty acids (PUFA), especially arachidonic acid (20:4 n-6). Of monounsaturated fatty acids (MUFA), palmitoleic acid (16:1 n-7) and oleic acid (18:1 n-9) did not increase significantly. At the same time the level of omega-3 polyunsaturated fatty acids [eicosapentaenoic (EPA, 20:5 n-3) and docosahexaenoic acid (DHA, 22:6 n-3)] significantly ($P \leq 0.01$) decreased, making the membrane more rigid and less permeable.

Introduction

Deltamethrin was synthesized in 1974 and first marketed in 1977. World consumption of deltamethrin was about 250 tons in 1987. Deltamethrin is mostly used on cotton and on crops such as coffee, maize, cereals, fruits, vegetables as well as on stored products. It is also used in animal health, in

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vector control and in public health. In Egypt it is widely used for plant protection and for the control of ectoparasites in farm animals (WHO 1990).

Deltamethrin [(s)-alpha-cyano-3-phenoxybenzyl (1R,3R)-cis-2,2-dimethyl-3-(2,2-dibromovinyl) cyclopropanecarboxylate] is a powerful pyrethroid insecticide which is highly toxic to aquatic organisms. Major routes of degradation or dissipation of parent deltamethrin in natural water are chemical and photochemical conversion to a mixture of inactive and active steroisomers, and hydrolysis with subsequent oxidation of products (Maguire 1992). Acute toxicity and bioconcentration capacity of deltamethrin was studied in the rainbow trout (*Oncorhynchus mykiss*) using filtered lake water, simulated (synthetic) water and filtered and unfiltered Aldrich humic acid (AHA) solutions. In the synthetic lake and filtered AHA solutions bioconcentration factors ranged from 43-320 (Muir et al. 1994).

In field experiments, the insecticide Glossinex-cocktail (endosulfan/deltamethrin) proved to be highly toxic to freshwater fish, affecting fatty acid concentrations in liver and acetylcholinesterase (AchE) activity in brain, muscle and liver of juvenile tilapia rendalli rendalli (Pisces Cichlidac) (Knauer et al. 1993). Ghosh and Banerjee (1992) found that catfish (*Heteropneustes fossilis*) exposed to lethal and prolonged sublethal concentrations of deltamethrin exhibited striking alteration in haemopoietic tissue smears and blood parameters. The in vivo effects of 2 μg/L of the insecticide deltamethrin on the activity and molecular form of acetyl cholinesterase of carp did not result in significant changes in AchE activity although it adversely affected the distribution of AchE molecular forms (Szegletes et al. 1995).

At the same time, the in vitro and in vivo effects of deltamethrin (DM) on acetylcholinesterase and some biochemical blood parameters, such as glutamicoxaloacetic transaminase (GOT), lactate dehydrogenase (LDH) and glucose in different tissues were examined in adult carp (*Cyprinus carpio L.*). The kinetic results showed that the inhibition of brain acetylcholinesterase was of mixed type, while the GOT and LDH activities increased 2.5-fold and 1.5-fold compared with those in the control carp. Also, the blood glucose level was 30% higher than that of the control fish (Balint et al. 1995). Pyrethroid deltamethrin may also interfere with the function of gamu aminobutyric acid (GABA) sub (A) receptors indirectly through an interaction with the voltage-dependent sodium channel in the brain of the trout and consequently disturb chloride influx, possibly through a voltage-dependent chloride channel (Eshleman and Murray 1991).

It has been demonstrated that the most significant change caused by deltamethrin is the prolonged elevation of the level of phosphorylation on a number of key synpatic proteins (Calcium-calmodulin dependent protein kinase and synapsin I) beyond the normal time of their recovery to the dephosphorylated state (Matsumura et al. 1989). Also demonstrating the immunosuppresive effect of deltamethrin are, Kowoiczyk et al. (1990) and Ashry and El-Ghareib (1997), who found that the insecticide suppressed both cell-mediated immunity and the antibody-forming ability of lymphocytes.

This study aimed to evaluate polar lipid metabolism changes in carp erythrocyte plasma membrane under the influence of deltamethrin.

Materials and Methods

A stock of carp fish (*Cyprinus carpio L.*) with body weight 200-250 g purchased from Alexandria Governorate Fish Farm, located 15 km west of Alexandria, and acclimated in the laboratory for one week under aquarium conditions were used in the experiment. Ten fishes were kept in an aquarium containing 100 L of O_2 saturated water. The temperature was maintained at 20°C ± 1°C. Deltamethrin [(s)-alpha-cyano-3-phenoxybenzyl (1R, 3R)-cis-2,2-dimethyl-3-(2,2-dibromovinyl) cyclopropanecarboxylate] used was of the purest chemical grade (98%) (Kafr El-Zayat Co. for Insecticides and Chemical Products. Egypt). Deltamethrin was dissolved in ethanol (1 mg/25 ml ethanol) and the stock solution was added to the water to achieve final concentrations of 0.5, 1.0, 2.0 and 4.0 ppm. The duration of treatments were 48 and 96 hrs, respectively. All experimental animals survived this exposure time. Six fishes from one concentrate were caught at each exposure time and killed by vertebral rupture. The blood of the fishes were rapidly collected and kept in ice for isolation of the erythrocyte plasma membrane.

Preparation of erythrocyte plasma membrane

Following Sorensen's (1983) method, blood was collected from each carp via the caudal vein in an Na-heparinized syringe and erythrocytes were collected by centrifugation and washed with 0.16 M NaCl. After washing, the cells were resuspended in 0.15 M NaCl, 3 mM $MgCl_2$ and 0.01 M Tris-HCl, pH 7.2 and homogenized in a virtis 45 homogenizer fitted with knives. Nuclei and unbroken cells were removed from the homogenate to low speed differential centrifugation (500-1200 g for 5 min). The broken plasma membrane was collected by centrifugation at 25000 g for 15 min, and the supernatant discarded. Then the membranes were washed several times with 0.1 M Choline chloride, 0.01 M Tris-HCl, pH 7.2. Finally, the washed membranes were resuspended in chloroform-methanol (2:1 v/v) for lipid extraction.

Lipid analysis

EXTRACTION OF LIPIDS

Lipid extraction was performed according to Folch et al. (1957) by homogenizing the erythrocyte plasma membrane in an all glass potter homogenizer in the presence of ice cold chloroform: methanol 2:1 containing 0.01% butylated hydroxytoluene (BHT). The homogenate was flushed with CO_2 and placed in a refrigerator overnight, then filtrated using fat filter paper and separated into two phases by adding 0.1 M KCl solution.

Phospholipids were separated by adsorption (solid-liquid) column chromatography (1.9 cm in diameter) using 15 gm silicic acid such as adsorbosi-CAB (100-140 mesh, Applied Science Lab.) Chloroform was used to remove neutral lipids and methanol to obtain the phospholipid fractions. The polar head group composition of the latter was determined according to Rouser et al. (1970).

Total phospholipids were transmethylated in the presence of 5% HCl in absolute methanol at 80°C in sealed vials for 2.5 hrs.

Gas liquid chromatography of fatty acid methyl esters

Methyl esters were separated using a Hewlett- Packard 5890 II gas chromatography, equipped with a capillary column coated with SP2330 of 0.25m thickness (0.25mm I.D. x 30mi CPS-Li Quadrex, New Haven, CT, USA). High-purity nitrogen was applied as carrier gas with a pressure of 230 KPa. Hydrogen was used at 100 KPa and 280 KPa. The dual column system was programmed from 160°C to 200°C to give partial separation of 18:3 n-3 and 20:1 n-9 at a flow rate of $1°C \cdot min^{-1}$. The detector temperature and injector temperature were 250°C and 230°C, respectively. The peaks were identified by means of primary standard mixture (e.g., for fatty acid methyl esters, laurate, myristate, palmitate and stearate in equal amounts, Applied Science Lab. N° K-108), by secondary standards (cod liver oil) and by plotting log relative elution temperature versus the number of carbon atoms (Schmit and Wynner 1966). The percentage composition was calculated as weight percentage (%w/w) using a Hewlett-Packard 3396A integrator. All peaks from myristic acid (14:0) to DHA (22:6 n-3) were included in the calculations.

Statistical analysis

Experimental results were subjected to one-way analysis of variance by Snedecor and Cochran (1982) t-test (SAS 1994). Results were presented as means ± S.E. A result of $P \leq 0.05$ was considered statistically significant.

Results

Effects of different deltamethrin concentrations on the polar head group

The results presented in Table 1 provide a quantitative phospholipids (PL) picture of carp erythrocyte plasma membrane in untreated (control) and treated animals with different doses of deltamethrin 0.5, 1.0, 2.0 and 4.0 ppm for short- and long-term exposure time 48 and 96 hrs, respectively. The phosphatidylcholine (PC) dominated the PL fraction (56.1 ± 7.0%) followed by phosphatidylethanolamine (PE) (21.2 ± 3.2%) and sphingomyline (SM) (7.7 ± 0.52%). There were smaller amounts of phosphatidylserine (PS) and phosphatidylinositol (PI), with traces of phosphatidic acid (PA), cardiolipin (CL) and lysophospholipids. The major differences between the control and polluted samples were the significant ($P \leq 0.01$) declines in PE, PS, PG, PA and CL. The amounts of marmoset erythrocyte PC were not altered by deltamethrin concentrations in the long-term, whereas, in the short-term, it significantly increased ($P \leq 0.05$) with an increase in deltamethrin concentrations.

The proportions of lysophosphatidylcholine (LPC) progressively increased from 0.94 ± 0.1% to 6.5 ± 0.7% in response to increases in deltamethrin concentrations.

Effects of the different deltamethrin concentrations on the phospholipid fatty acid compositions

The phospholipid (PL) fatty acid compositions of the control and polluted carp erythrocyte plasma membrane are listed in (Tables 2 and 3). Deltamethrin brought about marked differences in the fatty acid composition of the PL from the same tissue. The quantities of saturated fatty acids (SFA) increased considerably in fish given higher doses (2.0 and 4.0 ppm), especially in those exposed to deltamethrin, whereas, no significant changes were noted in lower doses (0.5 and 1.0 ppm). The quantities of monounsaturated fatty acids (MUFA) decreased in response to deltamethrin. Amounts of Σ n-3 polyunsaturated fatty acids (n-3 PUFA) also declined in response to deltamethrin. Despite the changes in the n-3 PUFA, eicosapentaenoic acid (EPA, 20:5 n-3) decreased from 8.7 ± 0.9 to 2.0 ± 0.2% in the short-term exposure time, but was eliminated in the long-term one. Docosahexaenoic acid (DHA, 22:5 n-6)

Table 1. Influence of different concentrations (ppm) of deltamethrin on the polar head composition of carp erythrocyte cytoplasmic membrane.

	48 Hours					96 Hours				
	0.0	0.5	1.0	2.0	4.0	0.0	0.5	1.0	2.0	4.0
LPC	0.94 ± 0.1^{E}	2.06 ±0.15^{D}	3.0 ± 0.5^{C}	4.5 ± 0.4^{B}	6.55 ± 0.7^{A}	1.21 ± 0.1^{B}	2.0 ± 0.1^{A}	1.3 ± 0.1^{B}	2.5 ± 0.2^{A}	2.8 ± 0.2^{A}
SM	7.7 ±0.51^{C}	8.1 ±2.16^{C}	8.9 ± 1.7^{B}	10.3 ±2.12^{A}	12.5 ± 1.8^{A}	8.05 ±1.05^{B}	7.5 ± 1.2^{B}	8.9 ± 1.4^{B}	11.4 ±0.95^{A}	12.1 ± 1.3^{A}
PC	56.1 ± 7.0^{B}	55.3 ±5.35^{B}	58.8 ± 8.2^{B}	61.7 ± 7.4^{B}	66.1 ± 8.5^{A}	54.1 ± 8.2^{B}	56.0 ± 6.7^{B}	59.8 ± 5.4^{B}	65.4 ± 6.7^{A}	68.5 ± 6.9^{A}
PI	2.7 ±0.25^{C}	3.16 ±0.27^{B}	2.8 ±0.45^{C}	3.36 ± 0.1^{B}	5.6 ± 0.4^{A}	3.05 ±0.25^{C}	3.4 ±0.31^{C}	5.0 ±0.33^{B}	4.6 ± 0.4^{B}	8.2 ± 0.9^{A}
PS	6.12 ± 1.2^{A}	5.8 ±0.87^{A}	5.2 ±0.65^{A}	4.01 ± 0.5^{B}	2.17 ±0.36^{C}	7.18 ± 0.4^{A}	7.5 ±0.71^{A}	5.11 ± 0.5^{B}	5.0 ± 0.6^{B}	3.05 ±0.4^{C}
PE	21.16 ± 3.2^{A}	20.7 ± 2.4^{A}	18.2 ±3.08^{B}	15.0 ±4.59^{C}	6.3 ±2.81^{D}	21.0 ± 2.4^{A}	19.0 ± 1.8^{A}	16.12 ± 1.5^{B}	9.0 ± 1.3^{C}	5.45 ±1.1^{D}
PG	1.45 ± 0.2^{A}	1.3 ± 0.1^{A}	0.72 ± 0.1^{B}	0.04 ± 0.0^{C}	0.23 ± 0.1^{D}	0.8 ± 0.1^{A}	1.0 ± 0.1^{A}	0.8 ± 0.1^{A}	0.4 ± 0.1^{B}	0.4 ± 0.0^{B}
PA	1.4 ± 0.0^{A}	1.5 ± 0.1^{A}	0.93 ± 0.3^{B}	0.16 ± 0.0^{C}	0.36 ±0.15^{C}	1.9 ± 0.2^{A}	1.87 ± 0.2^{A}	1.4 ± 0.3^{B}	1.0 ±0.11^{B}	0.65 ± 0.1^{C}
CL	2.3 ±0.21^{A}	1.21 ± 0.3^{B}	1.1 ± 0.6^{B}	0.86 ± 0.4^{C}	0.3 ± 0.2^{D}	2.46 ± 0.3^{A}	1.5 ±0.21^{B}	1.05 ± 0.4^{B}	0.8 ±0.34^{B}	0.33 ± 0.5^{C}

The given values are the average of 6 fishes (±SD). Means within the same row with the same superscript are not significant at P≤0.05.

Table 2. Effects of different concentrations (ppm) of deltamethrin on the phospholipid fatty acid compositions in carp erythrocyte plasma membrane at exposure time of 48 hrs.

Fatty acids %	48 Hours				
	0.0	0.5	1.0	2.0	4.0
14:0	6.1 ± 0.5	5.0 ± 0.6	5.1 ± 0.6	6.7 ± 0.2	6.5 ± 0.7
16:0	23.8 ± 2.1	26.2 ± 2.5	27.4 ± 3.1	26.8 ± 2.7	20.8 ± 2.1
18:0	5.0 ± 0.3	6.6 ± 0.5	6.5 ± 0.5	8.3 ± 0.7	8.8 ± 1.0
T.SFA	33.3 ± 3.1^{A}	35.8 ± 2.7^{A}	38.6 ± 3.21^{A}	41.8 ± 3.8^{A}	46.1 ± 3.4^{A}
16:1 n-7	3.6 ± 0.2	3.9 ± 0.4	3.8 ± 0.3	3.02 ± 0.1	3.6 ± 0.2
18:1 n-9	12.4 ± 1.1	10.9 ± 0.9	10.7 ± 1.0	8.8 ± 0.6	8.9 ± 1.0
T.MUFA	16.0 ± 1.7^{A}	14.8 ± 1.6^{A}	14.5 ± 1.3^{A}	11.8 ± 1.2^{B}	12.5 ± 1.3^{B}
18:2 n-6	2.5 ± 0.2	2.1 ± 0.3	2.4 ± 0.1	2.5 ± 0.22	4.5 ± 0.3
20:2 n-6	1.6 ± 0.1	2.3 ± 0.2	1.9 ± 0.06	3.4 ± 0.2	2.2 ± 0.2
20:3 n-6	1.8 ± 0.1	1.7 ± 0.1	2.8 ± 0.2	3.1 ± 0.1	4.4 ± 0.3
20:4 n-6	7.4 ± 0.6	7.5 ± 0.7	7.8 ± 0.9	10.0 ± 1.8	10.7 ± 1.4
22:4 n-6	2.4 ± 0.1	2.5 ± 0.2	2.1 ± 0.4	3.6 ± 0.3	2.7 ± 0.3
22:5 n-6	2.3 ± 0.21	2.7 ± 0.5	2.8 ± 0.6	3.7 ± 0.4	2.8 ± 0.5
T. n-6	17.5 ± 1.3A	18.8 ± 2.01A	19.8 ± 2.3A	26.1 ± 3.3B	27.8 ± 1.4B
18:3 n-3	2.2 ± 0.1	2.1 ± 0.1	1.7 ± 0.2	1.5 ± 0.1	—
20:5 n-3	8.7 ± 0.9	8.3 ± 0.9	6.9 ± 0.7	4.0 ± 0.7	2.04 ± 0.2
22:5 n-3	2.1 ± 0.2	2.0 ± 0.1	2.7 ± 0.2	1.6 ± 0.2	1.8 ± 0.1
22:6 n-3	17.6 ± 2.1	17.3 ± 1.9	15.6 ± 1.6	11.7 ± 1.3	10.4 ± 1.5
T. n-3	30.6 ± 2.4^{A}	29.7 ± 2.1^{A}	26.9 ± 2.8^{A}	18.8 ± 1.9^{B}	14.2 ± 1.3^{C}
Polyene index A	0.92 ± 0.04^{A}	0.83 ± 0.05^{B}	0.70 ± 0.08^{C}	0.45 ± 0.05^{D}	0.31 ± 0.03^{E}
Polyene index B	2.0 ± 0.3^{A}	1.8 ± 0.2^{A}	1.6 ± 0.2^{B}	1.4 ± 0.1^{B}	1.2 ± 0.1^{C}

The given values are the average of 6 fishes (±SD). Means within the same column with the same superscript are not significant at P≤0.05).
Polyene index A = S% 18:4 + 20:5 + 22:6 per 14:0 + 16:0 + 18:0.
Polyene index B = S% Unsaturated FA per 14:0 + 16:0 + 18:0,
Calculated according to Ke et al.,(1975).

Table 3. Effects of different concentrations (ppm) of deltamethrin on the phospholipid fatty acid compositions in carp erythrocyte plasma membrane at exposure time of 96 hrs.

Fatty acids %	48 Hours				
	0.0	0.5	1.0	2.0	4.0
14:0	5.8 ± 0.6	5.6 ± 0.7	5.5 ± 0.51	7.5 ± 0.65	7.9 ± 0.8
16:0	23.8 ± 2.1	26.3 ± 2.2	26.6 ± 2.8	30.8 ± 2.4	31.7 ± 4.2
18:0	5.7 ± 0.8	6.7 ± 0.7	6.6 ± 0.41	8.0 ± 0.8	10.4 ± 1.1
T.SFA	35.3 ± 2.8^{A}	39.6 ± 2.1^{A}	38.7 ± 3.2^{A}	46.3 ± 4.1^{B}	50.0 ± 4.7^{B}
16:1 n-7	3.54 ± 0.4	4.1 ± 0.3	4.0 ± 0.4	3.7 ± 0.21	2.5 ± 0.20
18:1 n-9	12.0 ± 1.1	12.9 ± 2.3	11.84 ± 1.3	10.9 ± 1.1	6.1 ± 0.7
T.MUFA	15.5 ± 1.3^{A}	17.0 ± 1.6^{A}	15.8 ± 1.5^{A}	14.6 ± 1.2^{A}	8.6 ± 0.8^{B}
18:2 n-6	2.4 ± 0.2	2.5 ± 0.3	2.5 ± 0.16	2.9 ± 0.3	4.7 ± 0.3
20:2 n-6	1.4 ± 0.1	3.3 ± 0.2	1.2 ± 0.07	2.7 ± 0.2	3.7 ± 0.22
20:3 n-6	1.9 ± 0.05	3.7 ± 0.1	5.8 ± 0.15	5.17 ± 0.2	6.5 ± 0.2
20:4 n-6	9.9 ± 1.2	10.5 ± 1.0	14.5 ± 1.3	14.8 ± 2.1	16.6 ± 2.4
22:4 n-6	2.7 ± 0.4	1.2 ± 0.08	1.5 ± 0.1	1.1 ± 0.7	0.8 ± 0.05
22:5 n-6	2.9 ± 0.4	3.6 ± 0.1	2.2 ± 0.3	2.03 ± 0.2	2.80 ± 0.3
T. n-6	18.5 ± 1.2^{A}	20.6 ± 1.4^{A}	26.2 ± 2.2^{B}	27.6 ± 3.1^{B}	33.3 ± 2.8^{C}
18:3 n-3	2.1 ± 0.1	0.92 ± 0.1	0.18 ± 0.0	-	-
20:5 n-3	7.2 ± 0.5	,5.5 ± 0.5	4.1 ± 0.4	2.0 ± 0.1	-
22:5 n-3	1.3 ± 0.2	1.4 ± 0.1	1.1 ± 0.2	0.4 ± 0.1	-
22:6 n-3	16.8 ± 1.7	16.2 ± 1.5	12.0 ± 0.24	8.9 ± 0.7	6.5 ± 0.4
T. n-3	27.4 ± 2.0^{A}	22.02 ± 2.4^{B}	18.38 ± 1.6^{C}	11.3 ± 0.4^{D}	6.5 ± 0.21^{E}
Polyene index A	0.78 ± 0.09^{A}	0.56 ± 0.5^{B}	0.47 ± 0.03^{C}	0.24 ± 0.01^{D}	0.13 ± 0.00^{E}
Polyene index B	1.8 ± 0.1^{A}	1.5 ± 0.1^{B}	1.6 ± 0.07^{B}	1.2 ± 0.01^{C}	1.0 ± 0.02^{D}

The given values are the average of 6 fishes (±SD). Means within the same column with the same superscript are not significant at P≤0.05).
Polyene index A = S% 18:4 + 20:5 + 22:6 per 14:0 + 16:0 + 18:0.
Polyene index B = S% Unsaturated FA per 14:0 + 16:0 + 18:0.
Calculated according to Ke et al.,(1975).

decreased from 17.6 ± 2.1 to 10.4 ± 1.5% and from 16.8 ± 1.7 to 6.5 ± 0.4% in the short- and long-term exposure time, respectively. At the same time, the proportions of Σ n-6 PUFA significantly ($P \leq 0.01$) increased under the higher doses (2.0 and 4.0 ppm). Despite these changes, the acylchain indexes (polyene index A and B) had significantly higher values in control fish than in polluted ones. Differences due to the different deltamethrin concentrations were more significant in the case of polyene index A compared to polyene index B.

Discussion

Measurements of quantitative changes in fatty acid desaturase and other enzymes relating mainly to the PL metabolism may provide a further explanation of the mechanism behind the molecular action of deltamethrin.

The major metabolic reactions were oxidation, cleavage and conversion of the cyano portion to thiocyanate (Miyamoto 1981). The resultant carboxylic can contribute to the depletion of dissolved oxygen in polluted waters (Phipps 1981), action explained by the formation of free radicals (O_2) which oxidize various cellular substrates, especially unsaturated fatty acid, (Rady et al. 1993) and enhance aging of the cell membrane. The lack of unsaturated fatty acids and the blocking of glycerolipid formation result in the inhibition of cell membrane formation as well as necrosis and cell death (Rady and Korshom 1995).

It is evident from the results obtained here that the polar lipid composition, with particular emphasis on PC, is in general agreement with that of other freshwater fish analyzed to date (Henderson and Tocher 1987; Olsen et al. 1990). The high levels of PC in the blood lipid is consistent with high-density and low-density lipoproteins being major lipoproteins in the blood of *Cyprinus carpio* as in rainbow trout (Fremont et al. 1981) and Tilapia nilotica (Olsen et al. 1990). Also, decreasing PE and plasmalogen species as a result of increased deltamethrin concentrations enhance oxidative stress and aging of the cell membrane (Morand et al. 1988).

The results of the present study are consistent with the well established observation that fatty acid composition of polar lipids in carp erythrocyte plasma membrane is influenced by the toxic action of deltamethrin. This is particularly true for the long-chain SFA and MUFA. The higher proportions of SFA and MUFA in the PL of polluted fish in comparison with control fish, suggest that long-term exposure to higher deltamethrin (2.0 and 4.0 ppm) concentrations leads to decreased desaturase activity (Barabas et al. 1983), therby inhibiting the conversion of linoleic (18:2 n-6) and linolenic (18:3 n-3) acids to longer-chain PUFA. Furthermore, the low levels of n-3 PUFA in the erythrocyte plasma membrane of polluted fish in comparison with those in control fish are in keeping with the formation of oxidizable fatty acids (Rady and Korshom 1995).

It is well known that the 22:6 n-3 in fish tissues is responsible for the biophysical properties of membrane, modulation of lipid protein interactions and membrane bound enzymes as well as the precursor of functionally important lipooxygenase products (Neuringer et al. 1988; Bazan 1990). The decrease of

this fatty acid and the double bond index/saturation of fatty acid, which is taken as indicative of membrane fluidity, led to changes in allosteric transitions and could indicate that the condensing effect of the SFA of the membrane increases the strength of interactions between the allosteric effector sites (Bloj et al. 1973). Meanwhile, effects of low doses become mild at long exposure and even disappeared.

These measurements proved the conception that, in aquatic animals (fish), the toxicity of deltamethrin, which is related to intercellular O_2 generation affects the membrane lipids directly, changing their quantitative values, presumably by the direct action of deltamethrin on the enzymes influencing fatty acid metabolism.

Acknowledgments

We are indebted to Dr. Abdel-Aziz Nour and Dr. Nadia Saber for their kind contribution to the acclimation experiment. We also thank Prof. Dr. Farkas Tibor for the gas liquid chromatography analysis. This work was supported by the Fisheries Research Institute in Alexandria.

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Manuscript received 06 January 1998; Accepted 09 August 1998.