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ORIGINAL ARTICLE

ATTENUATING PROPERTIES OF ATROPINE AGAINST THE CYPERMETHRIN TOXICITY IN THE OXIDATIVE STRESS IN THE FRESH WATER FISH *LABEO ROHITA* (HAMILTON)

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ABSTRACT

In the present investigation the effect of Cypermethrin on antioxidant enzyme activity in the fresh water fish *Labeo rohita* (Hamilton).The experimental fish were treated with cypermethrin the acute toxicity value was found to be 150µg/l and 1/5 as used for LC_{50} 30µg/l as sublethal concentration. Another group (III) of fish treated with cypermethrin in 120 hours, the group IV fish was exposed to atropine alone for 5 day. After the treatment fish was dissected out the organs like gill, liver and kidney were analysed enzymological parameters like Catalase (CAT) Superoxide dismutase (SOD), Lipid peroxidation (TBARS) level. Antioxidant enzymes are biomarkers used to indicating the cypermethrin toxicity. The SOD, CAT and LPO are increased during the cypermethrin exposure period (P>0.05). In the group III cypermethrin along with atropine exposure the antioxidant enzymes was recovered (P>0.05). Present study undertaken the toxic effect of cypermethrin on *Labeo rohita* fish and attenuating property of atropine.

Keywords: SOD, CAT, LPO and GPx, Cypermethrin, Atropine and *Labeo rohita*.

1.INTRODUCTION

Cypermethrin (416:30 C22 H19 Cl2 No3) is a synthetic pyrothroid insecticide that has been widely used over the past 30 year in India and other countries against pests, particularly Lepidoptera, cockroaches and termites. In animals, cypermethrin has been used as chemotherapeutic agent against ectoparasite infestations (Velisek *et al*., 2006). Cypermethrin can be found in trace amounts or at higher concentrations in soil and air. In mammals, cypermethrin can accumulate in body fat, skin, liver, kidneys, adrenal glands, ovaries, lung, blood, and heart the main target for cypermethrin is the central nervous system. (Wielgomas and Krechniak, 2007), globally, more than 520 tones of active ingredient of pyrethrorids are annually used in vector control programmers alone (Zaim and Jambulingam, 2004). Under the nrmal conditions, these antioxidants protect the cell and tissues from oxidative damage. The antioxidants in fish could be used as biomarkers of exposure to aquatic Fishes are sensitive to contamination of water and the pollutants may damage certain physiological and biochemical processes when they enter the organs of fishes (Tulasi *et al*., 1992). Under normal physiological conditions, ROS are rapidly eliminated in fish and other vertebrates by antioxidant enzymes including superoxidate dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) (Valavanidis *et al*., 2006; Zhang *et al*., 2009).

Stress can be counteracted by enzymatic and non enzymatic antioxidant system. Among enzymatic systems, the gluthione peroxides (GPx) belong to the first line of defense against peroxides, superoxide anion and hydrogen peroxide are assumes an important role in detoxifying lipid and hydrogen peroxide with the concomitant oxidation of glutathione (Almar *et al*., 1998). Hydroperoxides and H2O2 also are detoxified in the cytosolic and mitochondrial compartments by peroxidase (GPx), which uses reduced glutathione (GSH) as an electron doner and generates oxidized glutathione. Gluthathione reductase (GR) regenerates GSH in the glutathione redox cycle (Dorval and

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Hontela, 2003). GPx is found mainly in the cystosol and also in the matrix of mitochondria. Glutathione also shows similar distribution, some H2O2 produced peroxisomes is disposed by CAT, though some other H2O2 produced by mitochondria, endoplasmic reticulum or cytosolic enzyme such as SOD is eliminated by the peroxidase. Cypermethrin induced oxidative stress in fresh water mussels at various concentration has been reported (Koprucu *et al*., 2010) .The changes in activity of antioxidant enzymes such as SOD, CAT, GPx is considered as an effective method of denoting oxidative stress (Jin *et al*., 2011). In the present study, an attempt has been made to evaluate the attenuating properties of atropine of a against toxicity caused by pesticide cypermethrin.

2.MATERIALS AND METHODS

Experimental animal collection and maintenance

The fish *Labeo rohita* of size 14 to 16 cm and 50 to 70g weight were brought from a local fish farm in Pinnaloor, and Navarathna form. Fish collected and acclimatized at 28°C in the large sized aquarium for acclimatization in the laboratory condition for 15 days. During laboratory condition fishes were feed with artificial feed, water was renewed on every day to maintain water quality. The excess amount of feed and fecal matter was removed from the water and was provided the healthy environment before experimentation, to find out it's suitability for fish rearing. The LC_{50} concentration of Cypermethrin was noted at 120 hrs. Fishes were exposed in 4 groups.

Group-1 fish exposed to tap water
Group-2 fish exposed to cypermethrin
Group-3 fish exposed to cypermethrin Along with atropine
Group-4 Fish exposed to atropine alone

Experimental chemical

Experimental chemical Cypermethrin was purchased from (TATA Cypermethrin 50% WP) manufacture by Rallis India Limited, Mumbai.

Enzymatic assay

Superoxide dismutase (SOD) activity was determined by method of Kakkar *et al*., (1984), the in absorbance was recorded at 560nm. The activity of catalase (CAT) was determined by the method of Sinha, (1972) was recorded at Spectrophotometically read at 620 nm. Lipid peroxides in plasma and tissue were estimated by the method of Niehaus and Samuelson *et al*., (1968) which recorded at spectrophotometrically at 540 nm. Glutathione peroxidase (GPx) activity was assayed according to the method described by Rotruck *et al*., (1973) oxidation of NADPH was recorded specrophotometically read at 340 nm.

Statistically analyses

The data obtained in the present work were expressed as means ± SE, percentage changes and were statistically analyzed using student t-test (Milton and Tsokeg, 1983), to compare means of treated data against their control ones and the result were considered significant at ($P<0.05$) and ($P<0.01$) level.

3. RESULTS AND DISCUSSION

The effects of cypermethrin on formation in the different organs of *Labeo rohita*. During the past decade, pesticide - induced oxidative stress as a possible mechanism of toxicity has been focus on toxicological research (Sayeed *et al*., 2003). The activity of SOD observed in the tissue of gill, liver and kidney tissue of *Labeo rohita* during sub lethal concentration of cypermethrin for 24, 48, 72, 96 and 120 hours of exposure periods. The SOD activity significantly decreased in compared to control Group-1 in all tissue during the toxic exposure periods. The fish was exposed to group-3 the SOD content was recovered when compared to Group-2. While in the fish exposed to Group-4 when compared with Group-1 the slightly increased. The recorded SOD contents were statistically significant at 5% and 1% levels (Table -1). SOD is a link in the biological defense mechanism through disposition of endogenous cytotoxic O2, which are deleterious to structural proteins of plasma membrane. The decreased activity of SOD in erythrocyte of calves was observed by It is observed that the pesticides produce oxidative stress by inhibiting the activity of SOD, (Sathyanarayan, 2005).

Enzymatic degradation of O_2 to H_2O_2 is ensured by SOD. Monari *et al*. (2007) report that the extent of such a damage is dependent on the effectiveness of the antioxidant enzyme superoxide dismutase and changes in its activity have been proposed as a biomarker of pollutant-mediated oxidative stress. Considering SOD activity as an indirect measure of O_2 production, they also determine the positive relationship between tissue-specific SOD activity and index of superoxide production. Similarly, Qian *et al*. (2008) state that glufosinate exposure increases the activities of SOD, CAT and GPx is the first step in the removal of ROS. Therefore, increases in the activity of SOD in response to glufosinate suggest an increase in the production of O_2. CAT primarily occurs in peroxisomes and detoxifies H_2O_2 to O2 and water. GPx is the most important peroxidase for the detoxification of hydroperoxides (Lackner, 1998).

Oxidative stress is a result of an increase in reactive oxygen species or an impairment of antioxidant enzymes. Several studies provide evidence that antioxidant may be used as biomarkers of exposure to environmental pollutants (Di Giulio *et al*., 1993; Hasspieter *et al*. 1994; Regoli and Pricipate, 1995). Similar report that various pesticides can induce oxidative stress in different tissues of mouse (Bagchi *et al*., 1995; Hassoun and Stohs, 1996; Bachowski *et al*., 1998) could also be taken into account. The activity of catalases (CAT) is observed in the tissue of gill, liver and kidney tissue of *Labeo rohita* during sublethal concentration of cypermethrin for 24, 48, 72, 96 and 120 hours of exposure periods. The catalases activity significantly decreased when compared to group-1 in all atropine group-3 the CAT content being recovered when compared to Group-2 while in the fish exposed to group-4 when compared to their control group-1 The slightly increased of (CAT) in the fish tissue (CAT) statistically significant at level of 5% and 1% (Table - 2).

Table 1. Variations of superoxide dismutase (SOD) (U/min/mg protein) activity in the freshwater fish *Labeo rohita* exposed to cypermethrin and atropine exposed for 120 hours

Tissues	Groups	Hours of exposure				
		24	48	72	96	120
Gill	Group-I Control	17.825 ± 0.437	17.879 ± 0.631	17.944 ± 0.520	17.980 ±0.539	17.975 ±0.65
	Group-II CYP	15.905* ± 0.554	14.668* ± 0.733	13.906** ± 0.546	13.125** ± 0.845	12.550** ± 0.651
	% COC	% -10.82	% -18.00	% -22.50	% -27.00	% -30.18
	Group-III CYP+Atropine	16.445^{NS} ± 0.432	16.220^{NS} ± 0.354	15.903* ± 0.470	15.127* ±0.661	14.900* ± 0.587
	% COC	% -7.79	% -9.33	% -11.37	% -15.87	% -17.11
	% COT	% +3.39	% +10.58	% +14.36	% +15.25	% +18.72
	Group-IV Atropine	17.843^{NS} ± 0.634	17.915^{NS} ± 0.535	18.079^{NS} ± 0.470	18.175^{NS} ± 0.461	18.220^{NS} ± 0.506
	% COC	% +0.10	% +0.20	% +0.70	% +1.14	% +1.36
Liver	Group-I Control	29.711 ± 0.470	29.754 ± 0.654	29.796 ± 0.844	29.811 ± 0.634	29.805 ± 0.780
	Group-II CYP	27.643* ± 0.378	26.256** ± 0.563	25.340** ± 0.414	24.136** ± 0.841	23.456** ± 0.786
	% COC	% -6.96	% -11.76	% -14.95	% -19.04	% -21.30
	Group-III CYP+ Atropine	27.979* ± 0.366	27.380* ± 0.461	27.056* ± 0.523	26.753* ± 0.480	26.221* ± 0.618
	% COC	% -5.83	% -7.98	% -9.19	% -10.26	% -12.02
	% COT	% +1.21	% +4.28	% +6.77	% +10.84	% +11.79
	Group-IV *Atropine*	29.786^{NS} ± 0.655	29.884^{NS} ± 0.714	29.997^{NS} ± 0.718	30.085^{NS} ± 0.625	30.179^{NS} ± 0.886
	% COC	% +0.25	% +0.44	% +0.67	% +0.92	% +1.25
Kidney	Group-I Control	16.201 ± 0.480	16.236 ± 0.561	16.255 ± 0.446	16.272 ± 0.406	16.277 ± 0.576
	Group-II CYP	14.626^{NS} ± 0.472	13.831* ± 0.550	12.717** ± 0.343	11.880** ± 0.606	11.008** ± 0.419
	% COC	% -9.84	% -14.25	% -21.83	% -27.11	% -32.37
	Group-III CYP+ Atropine	15.189^{NS} ± 0.567	14.914^{NS} ± 0.583	14.426* ± 0.446	14.195* ± 0.380	14.051* ± 0.396
	% COC	% -6.25	% -8.20	% -11.31	% -12.76	% -13.67
	% COT	% +3.98	% +7.68	% +13.45	% +19.69	% +27.64
	Group-IV Atropine	16.220^{NS} ± 0.472	16.266^{NS} ± 0.514	16.320^{NS} ± 0.660	16.377^{NS} ± 0.380	16.418^{NS} ± 0.406
	% COC	% +0.12	% +0.18	% +0.40	% +0.64	% +0.87

Values are mean ± S.E-Mean of six individual observations; and student t-test. Significant at *P<0.05; Significant at ** P<0.01 levels. (+,-) denotes decreased and increased. % COC (change over control); % COT (change over treated).

Table 2.Variations of catalase (CAT) (U/min/mg protein) activity in the freshwater fish *Labeo rohita* exposed to cypermethrin and atropine exposed for 120 hours

Tissues	Groups	Hours of exposure				
		24	48	72	96	120
Gill	Group-I Control	2.067 ± 0.038	2.077 ± 0.031	2.089 ± 0.026	2.095 ± 0.038	2.099 ± 0.043
	Group-II CYP	1.836** ± 0.023	1.680** ± 0.028	1.526** ± 0.035	1.501** ± 0.034	1.454** ± 0.020
	% COC	% -11.13	% -19.04	% -26.91	% -28.32	% -30.73
	Group-III CYP+Atropine	1.916* ± 0.026	1.885** ± 0.023	1.817** ± 0.039	1.788** ± 0.025	1.759 ± 0.034
	% COC	% -7.31	% -9.64	% -12.98	% -15.90	% -16.67
	% COT	% +4.30	% +11.61	% +19.07	% +18.45	% +20.29
	Group-IV Atropine	2.076^{NS} ± 0.033	2.088^{NS} ± 0.023	2.112^{NS} ± 0.029	2.120^{NS} ± 0.045	2.139^{NS} ± 0.024
	% COC	% +0.48	% +0.63	% +1.15	% +1.24	% +1.90
Liver	Group-I Control	4.630 ± 0.028	4.655 ± 0.021	4.679 ± 0.038	4.685 ± 0.034	4.691 ± 0.041
	Group-II CYP	3.996** ± 0.029	3.647** ± 0.039	3.449** ± 0.024	3.299** ± 0.033	3.091** ± 0.044
	% COC	% -13.80	% -21.67	% -26.31	% -29.60	% -34.32
	Group-III CYP+ Atropine	4.395** ± 0.029	4.186** ± 0.039	4.070** ± 0.042	3.974** ± 0.033	3.909** ± 0.024
	% COC	% -5.07	% -10.75	% -13.01	% -15.18	% -16.67
	% COT	% +10.01	% +14.81	% +18.04	% +20.50	% +26.87
	Group-IV *Atropine*	4.652^{NS} ± 0.039	4.686^{NS} ± 0.028	4.715^{NS} ± 0.039	4.726^{NS} ± 0.029	4.736^{NS} ± 0.051
	% COC	% +0.45	% +0.69	% +0.80	% +0.85	% +0.96
Kidney	Group-I Control	1.918 ± 0.016	1.936 ± 0.024	1.943 ± 0.025	1.950 ± 0.014	1.956 ± 0.027
	Group-II CYP	1.780* ± 0.036	1.591** ± 0.020	1.475** ± 0.031	1.395** ± 0.028	1.312** ± 0.018
	% COC	% -7.19	% -17.82	% -24.09	% -28.46	% -32.92
	Group-III CYP+ Atropine	1.834* ± 0.031	1.785** ± 0.029	1.719** ± 0.035	1.687** ± 0.036	1.635 ± 0.022
	% COC	% -4.90	% -8.32	% -11.58	% -13.49	% -16.92
	% COT	% +2.47	% +11.56	% +16.47	% +20.93	% +23.86
	Group-IV Atropine	1.929^{NS} ± 0.020	1.952^{NS} ± 0.047	1.965^{NS} ± 0.037	1.977^{NS} ± 0.026	1.986^{NS} ± 0.035
	% COC	% +0.57	% +0.83	% +1.13	% +1.38	% +1.53

Values are mean ± S.E-Mean of six individual observations; and student t-test. Significant at *P<0.05; Significant at ** P<0.01 levels. (+,-) denotes decreased and increased. % COC (change over control); % COT (change over treated).

Table 3. Variations of lipid peroxidation (LPO) (nmole/mg protein) activity in the freshwater fish *Labeo rohita* exposed to cypermethrin and atropine for 120 hours

Tissues	Groups	Hours of exposure				
		24	48	72	96	120
Gill	Group-I Control	1.716 ± 0.020	1.726 ± 0.017	1.728 ± 0.031	1.746 ± 0.024	1.735 ± 0.032
	Group-II CYP	1.971** ± 0.020	2.085** ± 0.026	2.215** ± 0.039	2.323** ± 0.034	2.440** ± 0.024
	% COC	% +14.93	% +21.01	% +28.11	% +33.81	% +40.80
	Group-III CYP+Atropine	1.894** ± 0.018	1.969** ± 0.010	1.975** ± 0.022	2.047** ± 0.023	2.068** ± 0.028
	% COC	% +9.85	% +13.70	% +14.81	% +17.91	% +19.33
	% COT	% -4.41	% -6.04	% -10.38	% -11.88	% -15.24
	Group-IV Atropine	1.717^{NS} ± 0.013	1.725^{NS} ± 0.022	1.732^{NS} ± 0.009	1.746^{NS} ± 0.018	1.750^{NS} ± 0.008
	% COC	% +0.2	% +0.12	% +0.17	% +0.58	% +0.98
Liver	Group-I Control	1.005 ± 0.023	1.012 ± 0.023	1.016 ± 0.038	1.022 ± 0.018	1.020 ± 0.034
	Group-II CYP	1.148** ± 0.024	1.290** ± 0.037	1.375** ± 0.027	1.443** ± 0.048	1.527** ± 0.029
	% COC	% +14.23	% +27.47	% +35.33	% +41.19	% +49.70
	Group-III CYP+ Atropine	1.122* ± 0.035	1.145** ± 0.021	1.169* ± 0.030	1.188** ± 0.021	1.254* ± 0.035
	% COC	% +10.65	% +13.14	% +15.06	% +16.14	% +18.04
	% COT	% -3.13	% -11.24	% -14.98	% -17.74	% -21.15
	Group-IV *Atropine*	1.008^{NS} ± 0.023	1.017^{NS} ± 0.011	1.022^{NS} ± 0.022	1.035^{NS} ± 0.044	1.040^{NS} ± 0.035
	% COC	% +0.29	% +0.49	% +0.59	% +1.27	% +1.96
Kidney	Group-I Control	1.076 ± 0.023	1.085 ± 0.016	1.091 ± 0.039	1.098 ± 0.027	1.105 ± 0.032
	Group-II CYP	1.188* ± 0.018	1.281** ± 0.031	1.399** ± 0.029	1.461** ± 0.039	1.581** ± 0.045
	% COC	% +9.48	% +17.14	% +28.14	% +33.06	% +42.17
	Group-III CYP+ Atropine	1.165^{NS} ± 0.027	1.198* ± 0.030	1.229* ± 0.027	1.234* ± 0.028	1.264* ± 0.034
	% COC	% +7.34	% +10.32	% +11.73	% +12.39	% +14.30
	% COT	% -1.95	% -5.82	% -12.80	% -15.54	% -19.60
	Group-IV Atropine	1.079^{NS} ± 0.026	1.089^{NS} ± 0.033	1.097^{NS} ± 0.029	1.109^{NS} ± 0.030	1.119^{NS} ± 0.021
	% COC	% +0.28	% +0.37	% +0.46	% +1.00	% +1.27

Values are mean ± S.E-Mean of six individual observations; and student t-test. Significant at *P<0.05; Significant at ** P<0.01 levels. (+,-) denotes decreased and increased. % COC (change over control); % COT (change over treated).

Table 4. Variations of glutathione peroxidase (GPx) (µg/min/protein) activity in the freshwater fish *Labeo rohita* exposed to cypermethrin and atropine for 120 hours

Tissues	Groups	Hours of exposure				
		24	48	72	96	120
Gill	Group-I Control	4.139 ± 0.046	4.146 ± 0.035	4.159 ± 0.054	4.152 ± 0.066	4.149 ± 0.039
	Group-II CYP	3.798** ± 0.066	3.640** ± 0.058	3.425** ± 0.043	3.276** ± 0.025	3.053** ± 0.039
	% COC	% -8.22	% -12.16	% -17.45	% -21.10	% -26.40
	Group-III CYP+Atropine	3.957* ± 0.027	3.896** ± 0.038	3.805** ± 0.033	3.754** ± 0.025	3.703** ± 0.048
	% COC	% -4.40	% -6.01	% -8.31	% -9.61	% -10.63
	% COT	% +4.16	% +7.00	% +11.06	% +14.56	% +21.42
	Group-IV Atropine	4.157^{NS} ± 0.054	4.179^{NS} ± 0.061	4.182^{NS} ± 0.048	4.192^{NS} ± 0.039	4.20^{NS} ± 0.054
	% COC	%+0.43	% +0.60	% +0.77	% +0.93	% +1.30
Liver	Group-I Control	4.580 ± 0.035	4.586 ± 0.044	4.593 ± 0.038	4.598 ± 0.056	4.595 ± 0.059
	Group-II CYP	4.123** ± 0.030	3.902** ± 0.028	3.679** ± 0.044	3.442** ± 0.023	3.218** ± 0.054
	% COC	% -9.42	% -14.91	% -19.90	% -25.14	% -29.97
	Group-III CYP+ Atropine	4.265** ± 0.031	4.174** ± 0.042	4.109** ± 0.030	4.077** ± 0.029	4.009** ± 0.047
	% COC	% -6.88	% -8.98	% -10.54	% -11.33	% -12.75
	% COT	% +3.44	% +6.97	% +11.69	% +18.45	% +24.58
	Group-IV *Atropine*	4.593^{NS} ± 0.026	4.608^{NS} ± 0.047	4.623^{NS} ± 0.056	4.635^{NS} ± 0.033	4.646^{NS} ± 0.069
	% COC	% +0.28	% +0.48	% +0.65	% +0.80	% +1.11
Kidney	Group-I Control	3.115 ± 0.026	3.121 ± 0.031	3.128 ± 0.049	3.135 ± 0.054	3.132 ± 0.037
	Group-II CYP	2.892** ± 0.025	2.717** ± 0.030	2.549** ± 0.028	2.439** ± 0.034	2.281** ± 0.038
	% COC	% -7.19	% -12.98	% -18.54	% -22.23	% -27.21
	Group-III CYP+ Atropine	2.988* ± 0.023	2.877** ± 0.021	2.805** ± 0.040	2.769** ± 0.038	2.696** ± 0.012
	% COC	% -4.08	% -7.82	% -10.33	% -11.74	% -13.92
	% COT	% +3.35	% +5.93	% +10.09	% +13.58	% +18.24
	Group-IV Atropine	3.136^{NS} ± 0.024	3.147^{NS} ± 0.031	3.169^{NS} ± 0.029	3.173^{NS} ± 0.043	3.186^{NS} ± 0.040
	% COC	% +0.64	% +0.81	% +0.99	% +1.21	% +1.69

Values are mean ± S.E-Mean of six individual observations; and student t-test. Significant at *P<0.05; Significant at ** P<0.01 levels. (+,-) denotes decreased and increased. % COC (change over control); % COT (change over treated).

Catalase plays an important role in protection of cell from the hydrogen peroxide toxicity. Gaetani *et al*., (1994) have reported that catalases consist of four protein sub units containing heam group and it acts as antioxidant enzyme. Mostly these catalases are found in peroxisomes. The activity of catalases reduced under toxicity of pesticides because pesticides inhibit the catalases activities in C. punctuatus (Xu, 1997). Catalase in mammalian cells. It is a tetramer hermin enzyme located in sub cellular organelles such as peroxisomes of the liver and kidney (Liedias *et al*., 1998). Deltamethrin exposure also caused significant decreases in CAT activities in liver, kidney and gill tissues of Channa punctatus (Sayeed *et al*., 2003). This decline in CAT activity could be due to the excess production of O_2 as indicated by Bainy *et al*., (1996).

The activity of GPx observed in the tissue of Gill, liver and kidney tissue of *Labeo rohita* during sublethal concentration of cypermethrin 120 hours of exposure periods. The GPx activity significantly decreases in compared to control in all tissue during the toxic exposure periods, the fish is exposed to Group-3 the GPx content is recovered when compared to group-2 while in the fish exposed to group-4 when compared with group-1 the observed values are significant at the level of 5% and 1% (Table - 3).

The decreased activity of GPx in gills and muscle observed in the present study could be related to the O2 production (Bagnasco *et al*., 1991) or to the direct action of pesticides on the enzyme synthesis (Bainy *et al*., 1993).

Fatima *et al*., (2000) report a low activity of GPx in different fish tissues after exposure to paper mill effluent, indicating an inefficiency of these organs in neutralizing the peroxide impacts. A similar decrease in GPx activity in rat liver is reported after 90 days of treatment with lindane, an organochlorine pesticide (Yarsan *et al*., 1999). GPx inhibition is reported after combined treatment with the pesticides 2,4-D and azinphosmethyl in the brain of carp, C. carpio (Oruc *et al*., 2004) and in the liver of Nile tilapia, Oreochromis niloticus (Oruc and Uner, 2000).

A reduced GPx activity could indicate that its antioxidant capacity is surpassed by the amount of hydroperoxide products of lipid peroxidation (Remacle *et al*., 1992). The GST activity is involved in xenobiotic detoxification and excretion of xenobiotics and their metabolites, including MP (Jokanovic, 2001). It plays an important role in protecting tissues from oxidative stress (Fournier *et al*., 1992 and Banerjee *et al*., 1999).

Toxicant induced stress at a biochemical level is based on the production of free radicals. Oxidative stress caused by the toxicants in biological system may be involved in a variety of disease states and toxic reaction, thereby contributing indirectly to injury (Sonia *et al*., 2004).

The activity of lipid peroxidation LPO is observed in the tissue of gill, liver and kidney tissue of *Labeo rohita* during sublethal concentration of cypermethrin for 24, 48, 72, 96 and 120 hours of exposure periods. The LPO activity significantly increased compared to control group-1 in all tissue during the exposure period the cypermethrin along with atropine Group-3 the LPO content being recovered when compared to group-2 while in the control group-1. The increased of LPO level. Statistically significant at 5% and 1% level. (Table - 4).

The increase in lipid peroxidation observed in perchloroethylene (PER) administered animals might be a consequence of higher levels of superoxide radicals which are produced in significant amounts in response to PER exposure or inhibition of free radicals scavenging metalloenzymes that play a key role in the defence against ROS by transforming superoxide anions into hydrogen peroxide (Yim *et al*., 1993). Antioxidant enzyme, such as superoxide dismutase (SOD) was changed in the liver of red and white muscle of fish with cadmium exposure (Almeida *et al*., 2002).

Lipid peroxidation may be due to the oxidation of molecular oxygen to produce super oxide radicals. This reaction is also the source of H2O2, and O2 produced highly reaction hydroxyl radical with haber weiss reaction. The hydroxyl radical with haber weiss reaction. The hydroxyl radical can indicate lipid peroxidation, which is a free radical chain leading to less of membrane structure and function (Ray *et al*., 1991).

4.CONCLUSION

In conclusion, the present study pesticides toxicity in cypermethrin, enzymological parameters to observe in selected tissue in fish, the antioxidant enzyme of SOD, CAT and GPx level increased and LPO level decreased the control of atropine can prevent or slow down the oxidative damage induced in fish *labeo rohita*. The effects of treated fish by treatment with attenuating properties of atropine , further studies to identify the active antidote in the atropine and determine their structure and mechanism of action control of atropine.

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