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GLUTATHIONE PEROXIDASE ACTIVITY IN THE LIVER OF RATS TREATED WITH CCL_4 AFTER EXPERIMENTAL HYPO AND HYPERTHYROIDISM

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ABSTRACT
Glutathione peroxidase is the general name of an enzyme family with peroxidase activity whose important biological role is to protect the organism from oxidative destruction.In our study glutathione declined in the liver of rats after carbon tetrachloride treatment. Afterthyroidic manipulations, glutathione content in the liver declined further.
Key words: Glutathione peroxidase, carbon tetrachloride, L-thyroxine, thyroid, liver.

INTRODUCTION
Very little is known about link between non-enzymatically generated free radical and cyclooxygenase (COX) catalysed reactive pathways. Indeed, induced oxidative injury and subsequent inflammatory response have been demonstrated following CCL_4 administration to rats (Basu, 1999). It was postulated that cyclooxygenase dependent inflammatory response through PGF2α formation in CCl_4- induced hepatotoxicity may be a secondary effect to oxidative injury and might have a conceivable link between inflammatory response and oxidative injury involving both non-enzymatic and enzymatic oxidation of arachidonic acid (Chaudhary and Rana, 2012).

MATERIALS AND METHODS
For the proposed investigations, the rats were divided into 6 groups, each containing 10 rats. The body weight of rats was recorded each day and the food intake was regularly monitored. A record of the change in body weight was maintained. Experimental protocol followed in this study is described in table 1.

Table 1: Experimental protocol employed in present study

Group	Treatment	Dose administered/kg body weight	Vehicle/route	Duration of treatment
A	Control	0.2 ml/kg body weight	Olive oil only	Each alternate day for 30 days
B	Carbon tetrachloride	0.2 ml/kg body weight of 2% carbon tetrachloride	Olive oil/intramuscular	Each alternate day for 30 days
C	6-N-propyl-2-thiouracil (PTU)	6-N-propyl-2-thiouracil (2.5 µg/100 gm body weight)	Distilled water/intramuscular	Twice a week for 30 days
D	L-thyroxine	L-thyroxine (25-30 µg/100 gm body weight)	Distilled water/intramuscular	On each 4th day for 3 weeks
E	PTU + carbon tetrachloride	After 2.5 µg PTU/100 gm body weight for 30 days (twice a week). Intramuscular 0.2 ml/kg body weight of 2% carbon tetrachloride.	Distilled water/olive oil/intramuscular	30 days + 30 days
F	L-thyroxine + carbon tetrachloride	After L-thyroxine (25 - 30 µg/100 gm body weight) for 3 weeks. Intramuscular 0.2 ml/kg body weight of 2% carbon tetrachloride	Distilled water/olive oil/intramuscular	3 weeks + 30 days

After required days of treatment, the rats were starved for 24 hours and then sacrificed by decapitation in the morning hours.

Glutathione peroxidase activity in the liver homogenate was assayed with a coupled enzyme system (Wendel, 1980) in which glutathione-di-sulphide reduction was coupled to NADPH oxidation by glutathione reductase. The oxidation of NADPH was followed spectrometrically at 340 nm on a spectrophotometer.

OBSERVATIONS

The enzyme glutathione peroxidase activity play a very important role in scavenging the free radicals. As such its activity increased in the liver of carbon tetrachloride treated rats. It further increased in the liver of hyperthyroidic and carbon tetrachloride treated rats as well as in hypothyroidic and carbon tetrachloride treated rats (Table 2).

Table 2: Glutathione peroxidase activity in the liver of rats treated with carbon tetrachloride after experimental hypo and hyperthyroidism.

Group	Treatment	Glutathione peroxidase (n moles NADPH/ mg protein/minute)
A	Control	12.48± 0.2856
B	Carbon tetrachloride	18.090±1.0336
C	Hypothyroidic	13.5300± 0.7925
D	Hypothyroidic + carbon tetrachloride	24.640± 0.5149
E	Hyperthyroidic	40.800± 0.5423
F	Hyperthyroidic + carbon tetrachloride	20.5400± 0.8276

Results are expressed as mean ± SE (n=5)

DISCUSSION

Glutathione-peroxidase was discovered by Mills (1957) as an enzyme, protecting haemoglobin from oxidative destruction by hydrogen peroxide. Later on, the enzyme was rediscovered as a "contraction factor" of mitochondria, i.e. as a compound preventing loss of contractibility of mitochondria under special conditions. Further it is found that glutathione-peroxidase not only catalyzes the reduction of hydrogen peroxide but also of organic hydroperoxides including those derived from unsaturated lipids. Present results on glutathione-peroxidase help in drawing the following conclusions:

Glutathione-peroxidase, which helps glutathione in the formation of stable alcohols from lethal lipid peroxides, declined after CCl4 treatment and was elevated only in unilaterally parathyroidectomized and carbon tetrachloride treated rats. The tendency of the mitochondria to swell and to form malondialdehyde is inversely correlated with their content of glutathione-peroxidase (Clouet and Michel, 1969). However, present results justify a low malondialdehyde and a low glutathione-peroxidase relationship.

RESULT

Glutathione declined in the liver after carbon tetrachloride treatment. After thyroidic manipulations, glutathione content in the liver declined further.

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