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# Effect of Liv.52 on SH-Glutathione Levels in Carbon Tetrachloride-Induced Liver Damage

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# **INTRODUCTION**

The liver is the largest organ in the body performing multiple metabolic functions to maintain and promote health. Several enzymes present in the liver cells are responsible for the metabolic activities in the liver. Some of these enzymes can, by detoxification, offer protection against hepatotoxic agents. Glutathione is a major intracellular sulfhydryl tripeptide in the hepatocytes responsible for detoxification reactions<sup>1</sup>. Reduced Glutathione (GSH) preferentially conjugates with carbon tetrachloride intermediate and protects the liver against carbon tetrachloride induced liver damage<sup>2</sup>. It is also reported that liver damage caused by chemicals can be aggravated by deficient GSH<sup>3</sup>.

Liv.52, a herbal hepatoprotective preparation, has been found to be useful in many liver dysfunctions due to infections and hepatotoxic agents<sup>4-6</sup>. It was therefore decided to study the changes in serum GSH levels following carbon tetrachloride-induced liver damage and the influence of prior treatment with Liv.52.

# **MATERIALS AND METHODS**

Thirty male rats weighing 250 to 280 grams were divided into three groups of 10 rats each. Rats in Group I acted as controls and did not receive Liv.52 or carbon tetrachloride. Animals in Group II were given 1 gm/kg of Liv.52 in water once a day orally for 8 days prior to carbon tetrachloride administration. Rats in Group III received only water for 8 days.

On day 8, animals of Group II (Liv.52) and Group III (only water) received 0.7 ml/kg of carbon tetrachloride as a single injection intraperitoneally. Group I (control) animals received normal saline. Blood was collected on days 0, 8 and 10, ie. 2 days after carbon tetrachloride injection for estimation of serum SH-glutathione levels. Serum GSH was analyzed by the method of Beutler E. *et al.*<sup>7</sup>

## RESULTS

Table 1 shows the effect of carbon tetrachloride alone and with Liv.52 on serum glutathione levels.

There was no change in GSH levels in the control animals of Group I receiving only normal saline. There was significant reduction in GSH levels on day10 in animals of Group III (only water) due to

Table 1: Showing the mean serum GSH levels (mg/L)			
Day	Group I (Control)	Group II (1 gm/kg Liv.52)	Group III (Only water)
0	$316 \pm 36$	$318 \pm 24$	$304 \pm 41$
8	$324 \pm 21$	$358 \pm 31$	318 ± 29
10	$307 \pm 35$	$336 \pm 51*$	$214 \pm 16*$
* <i>p</i> <0.05			

carbon tetrachloride challenge. However, with prior treatment with Liv.52 in Group II animals, the effect of carbon tetrachloride injection was negated and there was no change in GSH levels on day 10 even after carbon tetrachloride challenge.

### DISCUSSION

On account of its metabolic functions the liver is more susceptible to injuries by toxic agents. Reduced glutathione present in hepatocytes can conjugate with toxic agents by virtue of its sulfhydryl group. However, a high dose of a toxic agent or metabolite can cause cell death and reduction in GSH levels. Rathore *et al.*, have reported a protective effect of Liv.52 on cell membranes. This effect could prevent cell lysis and reduction of GSH levels. This experiment suggests that Liv.52 can be used to protect the liver against hepatotoxic agents.

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