Indian Journal of Pharmacology 1992; 24: 163-165. **RESEARCH PAPER**

HEPATOPROTECTIVE ACTIVITY OF *OCIMUM SANCTUM* LEAF EXTRACT AGAINST PARACETAMOL INDUCED HEPATIC DAMAGE IN RATS

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Summary Effect of **Ocimum sanctum** leaf extract was studied on paracetamol induced hepatic damage in rats. **O.sanctum** was found to protect the rats from hepatotoxic action of paracetamol as evidenced by significant reduction in the elevated serum enzyme levels. Histopathological studies showed marked reduction in fatty degeneration in animals receiving **O.sanctum** along with paracetamol as compared to the control group. It is stipulated that the extract treated group was partially protected from hepatic cell damage caused by' paracetamol.

Key words **Ocimum sanctum** paracetamol hepatoprotective activity

Ocimum sanctum (Tulsi) is a medicinal plant commonly grown in India and considered sacred by many Indians. Different parts of this plant have been reported to exhibit several medicinal properties.[1,2] Pharmacological properties like anabolic, hypotensive, cardiac depressant, smooth muscle relaxant, antifertility and antistress activity of this plant have been reported by several workers.[3,4] *O.sanctum* have been reported to possess antihepatotoxicity[5,6] and two triterpenes from the leaves have been shown to possess hepatoprotective effect against CCl_4 induced damage in rats.[7] But systematic research on any possible effect of *O.sanctum* leaves on paracetamol induced hepatic damage seems to be scarce. The present investigation has been designed to study the effect of *O.sanctum* leaf extract on paracetamol induced hepatic damage in rats.

MATERIALS AND METHODS

The method for extraction of material was essentially the same as described by Bhargava et al.[5] The air dried powder of the leaves of *Osanctum* was extracted by percolation at room temperature with 70 per cent ethyl alcohol. The extract was concentrated under reduced pressure (bath temperature 50°C) and finally dried in a vacuum desiccator. The residue of *O.sanctum* (OSE) was dissolved in propylene glycol at a concentration of 100 mg/ml and was used in experiments.

Male albino rats of Wistar strain (5-6 weeks) weighing 100-l 50 g were randomly divided into three groups of 6 rats each. They were maintained on a standardised diet and water *ad* libitum For experimental purpose animals were kept fasting overnight but allowed free access to water. All the drugs were administered orally with the help of a feeding tube. OSE (200 mg/kg)[5] was administered daily for 7 days to one group of rats and paracetamol (2 g/kg)[8] was administered on the 5th day (after 5th administration of the OSE).[8] Forty eight hours after paracetamol administration the rats were sacrificed for serum enzyme and liver glutathione level analysis and histopathological study to determine the degree of hepatic damage. The normal group received no treatment, whereas the control (paracetamol treated) group received propylene glycol in place of OSE. Serum enzymes eg. aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline and acid phosphatase (ALP & ACP) which have been shown to be elevated by paracetamol' were used to evaluate the hepatoprotective activity. AST and ALT were measured by the method of Reitman et al[9] and ALP and ACP according to Wootton.[10] Liver glutathione (GSH) level was measured by the method of Woodward and Fry.[11] Histopathological studies of the liver were done using haematoxylin and eosin stain. Results were statistically analysed using Student's 't' test.

RESULTS

Table 1 shows that serum enzyme levels were significantly reduced and liver GSH level significantly higher in animals receiving paracetamol and OSE

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Figure 1. Histopathological studies showing fatty degeneration of liver in control (paracetamol treated) group.

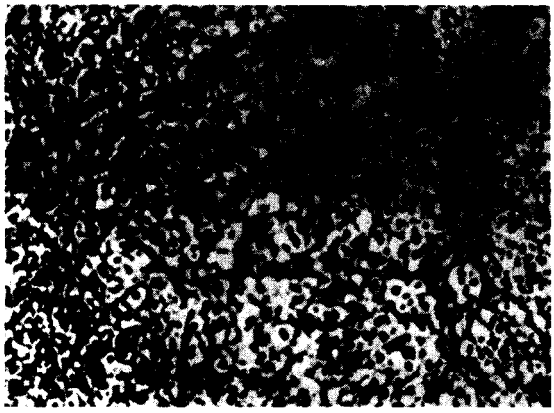

Figure 2. Histopathological studies showing mild fatty degeneration of liver in **O.sanctum** treated group.

than those given paracetamol alone. Histopathological studies of liver revealed that severe fatty degeneration of cells around the portal tract was observed in paracetamol treated group (Figure 1). But in OSE treated group mild fatty changes were observed (Figure 2).

Table 1, Alterations in the values of alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALT), acid phosphatase (ACP) and liver glutathione (GSH) following treatment with **Ocimum sanctum** leaf extract to rats intoxicated with paracetamol.

Parameters	A	B	C
ALT (Unit/dl)	25.24 ±2.48	45.00 ± 1.95	31.05"' ±1.92
AST (Unit/dl)	38.00 k2.52	67.76 ±4.00	45.00" ±2.94
ALP (K.A.U./dl)	48.16 ±2.00	87.00 ±4.02	**60.65"'** ±2.45
ACP (K.A.U./dl)	30.04 k2.85	55.10 ±2.05	39.82*** ± 1.87
Liver GSH (mg/g wet tissue)	1.42 ±0.16	0.55 ±0.18	1.07' ±0.14

Values are Mean±SE of six observations. A= Normal control; B = Paracetamol treated control; C = Paracetamol + OSE treated group.

*P < 0.05; **P < 0.01; ***P < 0.001 in comparison to control (paracetamol treated) group.

DISCUSSION

In living system liver is considered to be highly sensitive to toxic agents. The study of the aforesaid enzyme activity has been found to be of great value in the assessment of clinical and experimental liver damage.[12] In the present investigation it was observed that values of serum enzyme were significantly reduced (Table 1) in animals receiving OSE and paracetamol than those given paracetamol alone indicating that the degree of hepatic cell damage was of lesser magnitude in OSE treated group. The enzyme ALT is more abundant in liver cells than in any other cells in the body and is primarily used as a specific marker for hepatic damage. Thus an elevated serum ALT levels points to liver dysfunction. OSE has been found to reduce serum ALT levels significantly (P < 0.001; Table 1). Hepatic GSH depletion or even extra hepatic GSH depletion can provide a useful indication of the protective role of GSH against toxic foreign compounds. Thus GSH may be regarded as an endogenous protective agent for drugs, pesticides and other compounds. From our experiments it was observed that liver GSH level was much depleted in paracetamol treated animals than in those receiving a combination of paracetamol and OSE (Table 1). The attributivity of the observed alterations of serum enzyme levels to hepatic damage or health was confirmed by histopathological studies of the liver. Histopathological studies of liver in control (paracetamol treated) group showed severe fatty degeneration of cells around the portal tract (Figure 1) but the OSE treated group revealed only mild fatty changes (Figure 2). These observations point towards a hepatoprotective activity of OSE in this experimental model.

The results of this study are in corroboration with the earlier reports on the hepatoprotective activity of OSE against CCl_4 induced liver damage[6-8] and rationalise its use as a constituent of various herbal hepatoprotective formulations. Further extensive studies using some more models of experimental

hepatic damage may help in establishing a definite rationale for its therapeutic use as a hepatoprotective drug.

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