Protective effect of alcoholic extract of *Entada pursaetha* DC. against CCl₄-induced hepatotoxicity in rats

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The alcoholic extract of stem of *E. pursaetha* (PSE, 30, 100, 300 mg/kg body weight, po for 7 days) showed hepatoprotective activity against CCl₄ (2 mL/kg body weight, ip)-induced hepatotoxicity. The extract exhibited a significant dose-dependent hepatoprotective effect comparable to standard drug silymarin, by preventing increase in serum levels of alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, total protein, and total bilirubin, lactate dehydrogenase; by lowering hepatic levels of malonaldehyde, nitrate-nitrite, myeloperoxidase activity; enhancing activities of antioxidant enzymes, superoxide dismutase, catalase and increasing reduced glutathione levels in liver, which suggests the antioxidant property of PSE. Histopathological studies also supported the above biochemical parameters. The results suggested that alcoholic extract of *E. pursaetha* possesses significant hepatoprotective activity in CCl₄-induced acute hepatotoxicity in rats and this is likely to be mediated through its antioxidant activities.

**Keywords:** Antioxidant, CCl₄, *Entada pursaetha*, Hepatoprotection Liver toxicity

Acute and chronic liver diseases constitute a global concern, but medical treatments for these diseases are often difficult to handle and have limited efficacy. Therefore, considerable efforts to obtain useful herbal medicines from medicinal plants for a wide variety of clinical conditions are currently underway. Developing therapeutically effective agents from natural products may reduce the risk of toxicity when the drug is used clinically.

Carbon tetrachloride (CCl₄) is widely used in animal models to induce acute liver injury.² It is generally believed that the toxicity of CCl₄ results from its reductive dehalogenation by the cytochrome P450 enzyme system into the highly reactive free radical trichloromethyl radical³. In addition, it has been shown that CCl₄-induced toxicity may stimulate endogenous reactive oxygen species (ROS) and reactive nitrogen species (RNS) that have also been suggested to play an important role in the pathogenesis of hepatotoxicity⁴.

*Entada pursaetha* DC. has been reported from Silhet (now Bangladesh), Manipur, the Andamans and Nicobar Islands and the Eastern and the Western Ghats in peninsular India⁵.

*Entada pursaetha* is a gigantic woody liana among legumes, which produces 90-150 cm long woody giant pods with 5-30 seeds. All parts of this species contain saponins and are thus used in the soap industry. The various parts of plant are used by the tribals as a broad spectrum compound. This species can be used as a narcotic or as a tonic, etc., or used in curing liver troubles, allaying body pains, in warding off cold, curing eye diseases, arthritis, and paralysis.⁶ The use of seed in hepatic complaints, paste applied over affected or inflamed swelling to reduce pain; leaf as anthelmintic antiemetic, in jaundice, antiseptic, infantile cold; stem as antiemetic and in diarrhoea and skin diseases has been reported by various tribal communities of India in ethnobotanical surveys⁷⁹.

Limited information on the use of stem in ethnomedicine coupled with report of hepatoprotective and antioxidant effects of the stem bark of *E. africana*¹⁰, use of stem by local population in painful conditions of joints and other gastrointestinal tract disorders from the areas where the plant material was collected prompted us to use stem in this study.

The present study has been carried out to explore the tribal’s knowledge and traditional uses of this species. This study is designed to investigate the scientific basis for their traditional usage in the treatment of liver diseases. As a part of our interest in
natural products and their pharmaceutical potential, the hepatoprotective effect of stem of *E. pursaetha* has been evaluated. In the present investigation, *in vivo* effects of ethanolic extract of *E. pursaetha* stem have been studied on acute hepatotoxicity induced by CCl₄. One of the objectives is to evaluate the effectiveness of *E. pursaetha* in normalization of biochemical parameters of acute liver injury as well as to investigate its antioxidant activity as one of the possible mechanisms of hepatoprotective activity.

**Materials and Methods**

**Collection of plant material**—The stems of *E. pursaetha* were obtained from the jungles of Bhawanipatna, District Kalahandi, Odisha (India). The plant specimen was botanically authenticated by Dr. B N Pandey, Department of Botany, Bareilly College, Bareilly, India. A voucher specimen has been maintained in the Division of Pharmacology and Toxicology, Indian Veterinary Research Institute, Bareilly, India, for ready reference.

**Extract preparation**—The stems collected from the fully mature plant were shade dried, powdered and then extracted with 85% ethanol under reflux. The ethanolic extract of stem was concentrated to a solid mass under reduced pressure and made free from any solvent. The alcoholic extract of *E. pursaetha* stem hereafter, referred as “PSE” was stored in air tight bottle at 4°C and used further in all the experimental studies. The yield of the extract (PSE) was 8.4% with reference to dry starting material. The extract was then suspended in 2% polysorbate 80 and used for different pharmacological studies.

**Phytochemical testing**—Preliminary phytochemical screening of the ethanolic extracts of *E. pursaetha* stem was done to test for the presence of the active chemical constituents such as alkaloids, glycosides, flavonoids, tannins, phenolic compounds, and saponins.

**Animals**—Colony bred male Wistar rats (180-220 g) were obtained from Laboratory Animal Resource Section of the Institute. The animals were given 7 days for acclimatization to the laboratory environment, prior to the start of the experiment. These animals were kept in polypropylene cages with chopped paddy straw as the bedding material in a temperature-controlled room (22±2 °C) with 30-70% RH. A 12:12 L:D cycle was followed. Animals were maintained on a balanced ration obtained from the Feed Technology Unit of the Institute. Fresh drinking water was offered to the animals daily *ad libitum*. All animal experiments were carried out according to CPCSEA guidelines, after getting the approval of the Institute’s Animal Ethics Committee.

**Drugs and chemicals**—Carbon tetrachloride (CCl₄) was purchased from Merck Chemicals Ltd. All other chemicals used were of analytical grade from Sigma-Aldrich, St. Louis, USA; SRL Chemicals, India and biochemical kits were from Span Diagnostics Ltd, Surat, India.

CCl₄-induced hepatotoxicity—CCl₄ was used to produce hepatotoxicity in rats. In this model, rats were divided into following 6 groups of 6 animals each: Gr. I, received normal saline orally, for 7 days and served as the naïve control; Gr. II, received 2% polysorbate 80 aqueous suspension by oral gavages for 7 days, as vehicle control; Gr. III, served as standard control and received silymarin by oral gavages at 50 mg/kg as suspension in 2% polysorbate 80 aqueous suspension for 7 days, Gr. IV, V and VI received PSE by oral gavages at 30, 100 and 300 mg/kg, respectively as suspension in 2% polysorbate 80 aqueous suspension, for 7 days. CCl₄ (2 mL/kg; 1:1 v/v in olive oil) was given ip for production of hepatotoxicity on 7th day, 3 h after the last drug administration to all groups of animals except group I (naïve control). On the 8th day, 24 h after CCl₄ administration, under light ether anesthesia; blood samples were collected by cardiac puncture. The blood was allowed to clot for 30 min at room temperature; serum was separated and stored at -20 °C for further biochemical estimations, separated and stored at -20 °C for further biochemical estimations. After collecting blood, the rats from each groups were sacrificed; liver was excised out rapidly, rinsed in normal saline, blot dried and weighed. Liver was then cut into two separate portions. While one portion of liver was processed for assessment of different biochemical parameters, other portion was quickly fixed in 10% normal buffer formalin for histopathological study.

From the portion of liver kept for biochemical assays, a 500 mg of liver was weighed and taken in 5 mL ice-cold PBS (pH 7.4). Another 200 mg of sample was weighed separately and taken in 2 mL of 0.02 M EDTA solution for reduced glutathione (GSH) estimation. The liver homogenate (10%) prepared under ice-cold condition was centrifuged for 10 min at 3000 rpm. The supernatant was stored at -70 °C until assayed for different oxidative stress-related biochemical parameters.
Serum biochemical assays—The serum was analyzed for aspartate aminotransferase (AST) and alanine aminotransferase (ALT), serum alkaline phosphatase (ALP), lactate dehydrogenase (LDH); total protein and total bilirubin by using kits (Span Diagnostics, Ahmedabad, India) as per manufacturer’s instructions.

Measurement of liver biochemical parameters—Hepatic level of lipid peroxidation was evaluated in terms of malondialdehyde (MDA) formed by using thiobarbituric acid-reactive substances (TBARS)\textsuperscript{13}. Hepatic catalase\textsuperscript{14} and SOD activities\textsuperscript{15} were determined in liver homogenate. The reduced glutathione (GSH) level in liver tissue homogenate was determined by estimating free-SH groups, using 5-5' dithiobis 2-nitrobenzoic acid (DTNB) method\textsuperscript{16}. The hepatic myeloperoxidase (MPO) activity was determined by modification of O-dianisidine method\textsuperscript{17}. Nitrate and nitrite in the liver tissue were estimated following Sastry et al\textsuperscript{18}.

Histopathological evaluation of liver tissue—Small pieces of the liver tissues; fixed in 10% neutral buffer formalin (as above), were processed for embedding in paraffin. Sections (5-6 μm thick) were cut and stained with haematoxylin and eosin (H&E)\textsuperscript{19} and examined for histopathological changes under the microscope (BX41TF, Olympus Microsystems Corp. Japan). Images were captured using an Olympus DP12CCD camera at original magnification of 100 x (Olympus DP12 Microsystems Digital Imaging, Olympus, Japan). Histological evaluation was done in a blinded manner and quantitated by independent pathologist using a method by Hamza\textsuperscript{20}. The scoring was on a 0-3 scale as described below.

<table>
<thead>
<tr>
<th>Score</th>
<th>Inflammation</th>
<th>Necrosis</th>
<th>Sinusoidal dilation</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Absent</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>1</td>
<td>Present in one third of the lobules</td>
<td>Present in one third of the lobules</td>
<td>Present in one third of the lobules</td>
</tr>
<tr>
<td>2</td>
<td>Present in two third of the lobules</td>
<td>Present in two third of the lobules</td>
<td>Present in two third of the lobules</td>
</tr>
<tr>
<td>3</td>
<td>Present in all of the lobules</td>
<td>Present in all of the lobules</td>
<td>Present in all of the lobules</td>
</tr>
</tbody>
</table>

The histological score of individual rats represents the sum of the 3 sub scores of histological parameters.

Statistical analysis—Data were expressed as mean±SE. Statistical analysis of data was performed using GraphPad prism 4 and Microsoft Excel. Data were analyzed by ANOVA and means of various parameters were compared with Tukey’s multiple comparison post-hoc test. A value of \( P<0.05 \) was considered statistically significant.

Results

Effect on liver weight and relative liver weight—Increased liver weight (absolute; 12.27±0.32 g and relative 6.17±0.17 g/100 g body weight) were seen in the CCl\textsubscript{4}-treated group as compared to naïve control group (absolute; 8.43±0.20 g and relative 4.34±0.10 g/100 g body weight), whereas pre-treatment with the PSE 100 mg/kg (absolute; 10.38±0.25 g and relative 5.11±0.13 g/100 g body weight, PSE 300 mg/kg (absolute; 9.49±0.37 g and relative 4.72±0.19 g/100 g body weight) and standard drug silymarin (absolute; 9.12±0.21 g and relative 4.59±0.11 g/100 g body weight) showed significant reduction in liver weight, when compared with the CCl\textsubscript{4}-treated control group. However, PSE at 30 mg/kg did not reverse liver weight significantly (absolute; 11.56±0.44 g and relative 5.78±0.21 g/100 g body weight).

Effect on serum biochemical parameters—Administration of CCl\textsubscript{4}-induced a marked increase in serum biochemical parameters levels like ALT, AST, ALP, LDH, total bilirubin and marked decrease in total protein in vehicle control animals as compared to naïve control animals indicating liver damage (Table 1). Pre-treatment of rats with PSE prior to CCl\textsubscript{4} administration caused a significant reversal in the values of serum biochemical parameters towards normalization in a dose-dependent manner almost comparable to standard drug silymarin (50 mg/kg). The hepatoprotective effect of PSE at 300 mg/kg on serum biochemical parameters was fairly comparable to drug silymarin (50 mg/kg). However; PSE at 30 mg/kg did not reverse serum ALP level significantly (Table 1).

Effect on hepatic biochemical parameters—Reduced activities of enzymatic (CAT and SOD) and non-enzymic (GSH) antioxidants and enhanced activity of lipid peroxidation were seen in the CCl\textsubscript{4}-treated group. Standard drug silymarin and the PSE-treated groups showed dose-dependent rise in antioxidant levels with reduction in lipid peroxidation level when compared with the CCl\textsubscript{4}-treated control group. PSE at 100 and 300 mg/kg exhibited relatively higher protective action, which was comparable to silymarin (Table 2).

Increased hepatic myeloperoxidase activity was seen in CCl\textsubscript{4}-treated vehicle control group, compared...
to naïve control group, whereas treatment with PSE significantly reduced myeloperoxidase activity towards normalization almost comparable to silymarin indicating hepatoprotective effect of PSE exhibited through its inhibitory action on infiltration of neutrophils in hepatic parenchyma (Fig. 1).

Hepatic nitrosative stress, measured as nitrate-nitrite contents which are indicator of NO production in hepatic tissue was significantly increased in \( \text{CCl}_4 \)-treated groups, compared to naïve control groups. However, pre-treatment with PSE (100 and 300 mg/kg) significantly reduced hepatic nitrate-nitrite content towards normalization when compared to \( \text{CCl}_4 \)-treated group. These effects were comparable to standard drug silymarin (Table 2).

Liver histopathology—The histological observations basically supported the results obtained from other assays. A significant difference in histological score was observed between the naïve control group and \( \text{CCl}_4 \)-treated vehicle control group (Table 3). However, pre-treatment with PSE (100 and 300 mg/kg) and silymarin significantly reduced the histological score of liver damage (Fig. 2). The \( \text{CCl}_4 \)-intoxicated rats showed extensive changes in liver morphology including massive fatty changes, gross necrosis, broad infiltration of lymphocytes and Kuffer cells around the central vein, lymphomononuclear (LMN) cell (inflammatory cells like neutrophils) infiltration in hepatic parenchyma, sinusoidal dilatation and loss of cellular boundaries. These histological changes were ameliorated by PSE as well as by silymarin (Fig. 2).
Table 3—Effects of *E. pursaetha* stem extract (PSE) on liver histological score in CCl4-induced hepatotoxicity in rats [Values are mean±SE from 6 observations in each group].

<table>
<thead>
<tr>
<th>Groups</th>
<th>Dose (mg/kg)</th>
<th>Histological score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Naïve control</td>
<td>-</td>
<td>0.33±0.21</td>
</tr>
<tr>
<td>Vehicle control</td>
<td>-</td>
<td>5.83±0.70d</td>
</tr>
<tr>
<td>Silymarin</td>
<td>50</td>
<td>2.16±0.47c</td>
</tr>
<tr>
<td>PSE</td>
<td>30</td>
<td>4.66±0.33</td>
</tr>
<tr>
<td>PSE</td>
<td>100</td>
<td>3.33±0.70a</td>
</tr>
<tr>
<td>PSE</td>
<td>300</td>
<td>2.50±0.42c</td>
</tr>
</tbody>
</table>

P values: *<0.05; †<0.01 in comparison to vehicle control; ‡, §< 0.001 in comparison to †vehicle control, ‡naïve control, Tukey’s multiple comparison post hoc test.

Discussion

In the present study, the hepatoprotective effects of PSE in acute liver injury model were investigated. Effect of PSE was evaluated on acute hepatotoxicity induced by single injection of CCl4. In the vehicle-treated CCl4 group, ALT and AST levels increased dramatically compared with those in the control group, indicating severe hepatocellular damage. In contrast, treatment with PSE markedly attenuated the release of ALT and AST. Histological observations of liver samples strongly supported the release of aminotransferases by damaged hepatocytes as well as the protective effect of PSE. In a preliminary study, seed kernel extract of *E. pursaetha* has been reported.
to possess hepatoprotective activity\textsuperscript{21}. But to our knowledge no scientific and mechanistic study has been undertaken on the extract of stem of this plant for its hepatoprotective action. This led to our current investigation on the effects of PSE in hepatotoxicity. Here, the primary objective is to establish its efficacy in CCl\textsubscript{4}-induced hepatotoxicity in rats.

Phytochemical screening of the plant in present study revealed the presence of triterpenes, saponins, tannins, flavanoids and alkaloids. Earlier phytochemical studies of \textit{E. pursaetha} have described several natural products structurally related to aminoacid glycosides and triterpenoids\textsuperscript{22}. Many triterpenoids and their glycosides have been shown to possess anti-inflammatory activity\textsuperscript{23}, and some triterpene saponins from the family Leguminosae have been reported to be hepatoprotective\textsuperscript{24,25}.

Many hepatotoxicants including CCl\textsubscript{4}, nitrosamines, and polycyclic aromatic hydrocarbons require metabolic activation, particularly by liver CYP enzymes to form reactive toxic metabolites, which in turn cause liver injury in experimental animals and humans. The trichloromethyl radical formed during the metabolism of CCl\textsubscript{4} is capable of binding to lipids, and this binding initiates lipid peroxidation and liver damage\textsuperscript{26}.

Exposure to CCl\textsubscript{4} increases liver weight and relative liver weight in rats as compared to naïve control rats. This is due to the inflammation of the hepatocytes produced by oxidative and nitrosative stress implicated by the exposure of CCl\textsubscript{4} to which the weight of the liver increases. The decrease in weight of liver towards normalcy after pre-treatment with PSE might be because of its anti-inflammatory action on hepatocytes.

The hepatic activity of MPO, an index of tissue neutrophil infiltration\textsuperscript{27,28}, in CCl\textsubscript{4}-intoxicated rats is increased at an early stage of liver injury and further increased at a progressed stage of the injury, as reported previously\textsuperscript{29}. PSE administered before CCl\textsubscript{4}-induced liver injury reduced the increase in hepatic MPO in a dose-dependent manner. Thus, PSE administered orally to rats intoxicated once with CCl\textsubscript{4} can prevent increased hepatic neutrophil infiltration in the liver tissue.

Marked increase in serum levels of LDH, AST, ALT and ALP indicates a severe damage to tissue membranes during CCl\textsubscript{4}-induced liver injury\textsuperscript{30}. A significant increase in the activities of ALT, AST, ALP and LDH in serum on exposure to CCl\textsubscript{4} revealed considerable hepatocellular injury. Since these enzymes are cytoplasmic in nature, during liver injury, these enzymes enter into the circulatory system due to necrosis or altered permeability of membrane. AST is found in the liver, cardiac muscles, skeletal muscles, pancreas, lungs, kidney, brain, etc., whereas ALT concentration is highest in the liver and therefore, it appears to be a more sensitive test to hepatocellular damage than AST\textsuperscript{31}. Administration of PSE attenuated the increased levels of these biomarkers produced by CCl\textsubscript{4} and caused a subsequent recovery towards normalization almost like that of silymarin treatment. This is an indication of the stabilization of plasma membranes as well as repair of hepatic tissue damage caused by CCl\textsubscript{4}. This effect is in agreement with the commonly accepted view that serum levels of transaminases return to normal with healing of hepatic parenchyma and the regeneration of hepatocytes\textsuperscript{32}. The present results of stem extract are substantiated by previous work on aqueous and ethanolic extracts of \textit{E. pursaetha} seed kernel\textsuperscript{21}. Histological observations of liver samples strongly supported the release of aminotransferases by damaged hepatocytes as well as the protective effect of PSE. CCl\textsubscript{4} caused various histological changes in the liver, including cell necrosis, fatty metamorphosis in the adjacent hepatocytes, ballooning degeneration, cell inflammation, and infiltration of lymphocytes and Kupffer cells. These alterations were significantly attenuated by the PSE so that treated livers showed only minimal hepatocellular necrosis and inflammatory cell infiltration and mild portal inflammation. These results suggest that PSE has potential clinical applications for treating liver disorders.

Hyperbiliurinaemia indicates the severity of liver cell necrosis and its increased levels in serum with CCl\textsubscript{4} exposure in the present study is in agreement with previous studies\textsuperscript{32,33}. Administration of PSE attenuated the increased levels of bilirubin in serum, produced by CCl\textsubscript{4} and caused a subsequent recovery towards normalization almost like that of silymarin. Since PSE prevented the effects of CCl\textsubscript{4} on bilirubin levels in serum, it is reasonable to suggest that PSE limited the severity of liver injury which is confirmed by histological changes in CCl\textsubscript{4}-induced liver damage.

The levels of total protein in serum were also decreased in CCl\textsubscript{4}-induced hepatotoxicity because the metabolic machinery of hepatocytes and mitochondrial chain are affected by lipid peroxides and free radicals produced by CCl\textsubscript{4}\textsuperscript{35}. The decreased
levels of total protein in serum by CCl₄ were significantly elevated by pretreatment with PSE and the improvement is comparable with standard drug silymarin. Attainment of near normalcy in protein content of PSE-treated rats further suggested antihapatotoxic effects of PSE.

In recent years, there is increasing interest in free radicals. CCl₄ is known to generate ROS and free radicals like H₂O₂, hydroxyl radical (OH•), nitric oxide, •O₂⁻ in living systems. Excess levels of ROS and RNS can attack biological molecules such as DNA, protein and phospholipids, which leads to lipid peroxidation, nitration of tyrosine and depletion of the antioxidant enzymes (SOD, CAT and GPx) that further results in oxidative stress. SOD and CAT enzymes are important scavengers of superoxide ion and hydrogen peroxide, respectively. SOD catalyzes dismutation of superoxide radical anion (•O₂⁻) to H₂O₂ and O₂. GPx and CAT are the major enzymes that remove H₂O₂ generated by SOD in cytosol and mitochondria by oxidizing GSH (reduced glutathione) to GSSG (oxidized glutathione). The decrease in the activity of SOD in the liver of CCl₄-treated rats may be due to the increased lipid peroxidation or inactivation of the enzyme by cross linking with malonaldehyde. This will cause an increased accumulation of superoxide radicals, which could further stimulate lipid peroxidation. In the present study, PSE prevented the rise of MDA and also prevented the decrease in hepatic catalase and SOD in CCl₄-intoxicated rats, indicating an inhibitory activity of the extract on oxidative stress.

GSH, a major known protein thiol and nonenzymatic antioxidant in living organisms plays a central role in coordinating the body’s antioxidant defense process. Excessive peroxidation causes increased GSH consumption, which in turn results in enhanced lipid peroxidation. Studies suggest that there exists an inverse relationship between peroxidative decomposition of membrane polyunsaturated fatty acid and GSH level. It can be hypothesized that, administration of PSE promoted the conversion of GSSG (oxidized glutathione) into GSH by the reactivation of hepatic glutathione reductase enzyme in CCl₄-treated rats. The availability of sufficient amount of GSH thus increased the detoxification of active metabolites of CCl₄. The restoration of GSH levels after the administration of PSE to CCl₄-treated rats account for the protective efficacy of the extract. Hence, it is possible that the mechanism of hepatoprotection of PSE may be due to its antioxidant action.

NO is a short-lived free radical with multiple cell- and organ-specific functions. NO reacts with O₂ and H₂O in the extracellular milieu to form the stable oxidized products nitrate and nitrite, a marker of NO synthesis. Overproduction of RNS is called nitrosative stress. The significance of NO to a large extent stems from the fact that O²⁻ is a starting compound in two intoxication pathways. One pathway leads to formation of H₂O₂ and whereas the other one produces peroxynitrite (ONOO⁻) a potent oxidizing agent that can cause DNA fragmentation and lipid peroxidation. In the present study it was observed that, CCl₄ exposure increased nitrosative stress in rat liver, which was measured as liver nitrate-nitrite concentration. This is in agreement with previous work done on nitrosative injury in CCl₄-induced hepatotoxicity. Administration of PSE attenuated the increased levels of nitrate-nitrite, produced by CCl₄.

Conclusion

PSE has a significant hepatoprotective effect on liver injury induced by CCl₄. Therefore the possible hepatoprotective effect of PSE on the CCl₄-induced liver injuries may be due to: (i) preventing the process of lipid peroxidation, (ii) attenuation of oxidative and nitrosative stress (iii) preventing the inflammatory response by attenuation of the recruitment of neutrophils and (iv) stabilization of cellular membranes. This study provides evidence that PSE could be used to prevent hepatocellular damage and revealed that E. purusaetha is a promising candidate herb for the development of a phytomedicine against liver ailments.

References


