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PHYTOCHEMICAL INVESTIGATION AND HEPATOPROTECTIVE EFFECT OF *SCOPARIA DULCIS* AGAINST CARBON TETRACHLORIDE INDUCED LIVER DAMAGE IN RATS

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ABSTRACT: The objective of the present study is to carry out the photochemical investigation and hepatoprotective activity study of different extracts of *Scoparia dulcis*. *Scoparia dulcis* Linn. belongs to the family Scrophulariaceae and have ruminate medicinal properties. The different extracts of this plant were prepared by successive extraction with petroleum ether, chloroform and ethanol. These extracts (PEESD, CESD and EESD) were then taken for preliminary phytochemical screening using standard methods. The hepatoprotective activity of the different extracts of *Scoparia dulcis* was evaluated by using carbon tetrachloride induced liver damage in experimental rats. In hepatotoxicity induced animals, an oral dose of 300 mg/kg, of the petroleum ether, ethanol and chloroform extracts of *Scoparia dulcis* exhibited a compelling decrease in marker enzyme levels and increased levels of antioxidant enzymes. In the above dose the plant extracts produced a significant decrease in the Lipid peroxidase levels in the tested animals against Carbon tetrachloride induced liver toxicity. The chloroform extract was found rich in phytochemical constituents and had the highest hepatoprotective activity. From the results of our present study we concluded that, PEESD, CESD and EESD of *Scoparia dulcis* Linn. showed significant antioxidant defence mechanism and hepatoprotective activity. Result shows PEESD, CESD and EESD have significant antioxidant activity on comparison with standard silymarin. The hepatoprotective potential may be attributed to the presence of polyphenolic compound for their antioxidant properties.

INTRODUCTION: The liver is the main metabolic organ of the body and it also plays an important role for secretion and excretion which is repetitively exposed to various xenobiotics, surrounding toxicants and chemo remedial agents because of its vital position in the body. Liver disease is a global complication.[1 - 4]

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Typical drugs used in the treatment of liver diseases are sometimes insufficient and can causes serious adverse effects. So, it is necessary to find alternative drugs for the treatment of liver disease to replace currently used drugs of better efficacy and safety. Medicines derived from plant extract are to a greater extend utilized to treat a wide verity of disease, through relatively little idea about their mode of action is available. There is an increasing interest in the pharmacological screening of various plants for their therapeutic used in Indian traditional system of medicine.

The carbon tetrachloride (CCl_4) induced intoxication is widely used model for liver injury in rats.

Hepatotoxicity is linked with severe deterioration of cell protection mechanisms. The liver injury is defined mainly by the metabolism of CCl_4, which is cytochrome P - 450 dependant. Free radicals commence the process of lipid peroxidation, which causes the inhibition of the enzyme activity. [5, 6]

It is now generally confirmed that the hepatotoxicity of CCl_4 is the result of reductive dehalogination, which is catalyzed by P - 450, and which forms highly reactive trichloromethyl free radical. This easily interacts with molecular oxygen to form the trichloromethyl peroxy radical. Both trichloromethyl and its peroxy radicals are capable of binding to tissue proteins or lipids, or of releasing a hydrogen atom from an unsaturated lipid, initiating lipid peroxidation and liver damage and by doing so playing an important role in pathogenesis of diseases. [7]

Phytoconstituents such as flavonoids, terpenoids and steroids etc. have acquired great attention in recent years due to their varied pharmacological properties including antioxidant and hepatoprotective activity. [8, 9] There has been growing interest in the analysis of certain flavonoids, triterpinoids and steroids stimulate by heighten research into their probable benefits to human health.

Antioxidant activity is one of their main properties in this regard, which empower them to debilitate the development of tumor and inflammatory disease. Antioxidant plays an important role in hindering and tramping radicals, thus act as a safeguard to humans against infections and generative diseases. Realizing the fact, this research was carried out to evaluate the hepatoprotective activity of PEESD, CEESD and EESD.

Scoparia dulcis Linn. commonly known as Mithi Patti (Hindi) is distributed in the tropical region of India. It grows as a wasteland herb.

The traditional healers have developed its many promising traditional uses. Traditionally the leaves have been used for abortion, menstrual irregularities as female contraceptive. It also used against stomach aches, injuries, wounds, bronchitis, coughs, diarrhoea, eye infection, fever, and kidney failure and liver diseases. This has been used in case of infections such as gonorrhoea, skin infections and warts. [10 - 13]

MATERIALS AND METHODS:

Plant and Material: *Scoparia dulcis* whole plants were collected from Sheriguda village near to our institution and authenticated by Ms. Rupali, Associate Professor of Department of Pharmacognosy of our college, Hyderabad, India.

Preparation of Extracts: The fresh plants were collected, cleaned, shade-dried and then powdered. Firstly the dried coarse powder of whole plant was subjected to successive extraction in a soxhlet apparatus using petroleum ether, ethanol and chloroform.

Preliminary Phytochemical Screening: Preliminary phytochemical screening was done for the extract as per standard methods.[14, 15]

Animals: Studied were carried out using Wistar albino rats (150 - 180 g) of both sex were used. They were obtained from the animal house of our institution animal house. The animals were grouped and housed in suitable laboratory animal cages (38 × 23 × 10 cm) with not more than six animals per cage and maintained under standard laboratory conditions of temp 25 ± 2 ℃ with dark and light cycle (14/10 h). They were allowed free access to standard dry pellet diet (Hindustan Lever, Kolkata, India) and water according to. The rats were acclimatized to laboratory conditions for 10 days before commencement of experiment. All procedures described were followed by the Animal Ethical Committee of our institution bearing IAEC approval no. 163/PO/Re/S/12/CPCSEA.

Drugs and Chemicals: Silymarin was purchased from Micro labs, India, 1-chloro-2,4-dinitrobenzene (CDNB), Bovine serum albumin (Sigma Chemical, USA), thiobarbituric acid, Nitro blue tetrazolium chloride (NBT, Loba Chemie, Mumbai, India), 5,5'-dinitro-bis-2-nitrobenzoic acid (DTNB), Carbon tetrachloride (SICCO Research laboratory, Mumbai). The solvents and reagents obtained were used as received.

Experimental Design:

Carbon tetrachloride – Induced Liver Damage in Rats: The animals were divided into 6 groups each containing 6 animals.

Group I: control – receive 1 ml of normal saline.

Group II: toxic control – received 30% carbon tetrachloride in liquid paraffin (1 ml/kg body weight, i.p.).

Group III: standard control – received the standard drug Silymarin (25 mg/kg p.o) once in a day and CCl_4 as maintained above.

Group IV: received PEESD 300 mg/kg p.o. with CCl_4 as maintained above.

Group V: received CESD 300 mg/kg p.o. with CCl_4 as maintained above.

Group VI: received EESD 300 mg/kg p.o. with CCl_4 as maintained above.

Treatment duration was 10 days and the dose of CCl_4 was administrated after every 72 hour. [16] Under the light anaesthetics ether animals were sacrificed 24 hour after the last injection. Blood was collected and allowed to clot and serum separated. The liver was dissected out and used for biochemical studies.

Histopathological Studies: One animal from each group was utilized for histopathological study. The livers were fixed in 10% formalin for 24 h.

Biochemical Studies: The blood was obtained from all animals from retro-orbital plexus by puncturing. The blood samples were allowed to clot for 45 minute at laboratory temperature. Serum was separated by centrifugation at 2500 rpm at 30 °C for 15 minute and utilized for the estimation of various biochemical parameters namely SGPT, SGOT [17], Serum bilirubin [18], and cholesterol [19] using the analytical kits from Span Diagnostics Ltd., India.

After collection of the blood samples the rats were sacrificed and their livers cut out, soak in ice cold normal saline followed by 0.15M Tris-HCl (pH 7.4) blotted dry and weighed.

Estimation of the Biochemical Parameters:

Estimation of Aspartate transaminase: Aspartate transaminase in both serum and liver was determined by the method of Reitman's and Frankel, 1957. 0.5ml of buffered substrate was incubated at 37 °C for 3 minutes and 0.1ml of serum was added, mixed well and incubated at 37 °C for 30 minutes. Then 0.5ml of 2, 4 dinitrophenyl hydrazine reagents was added, mixed well and kept at normal room temperature for 20 minutes and 0.5 ml of 4N working sodium hydroxide was added and allowed to stand at room temperature for 10 minutes. Blank and standards were also processed in a similar way and the absorbance was measured spectrophotometrically at 505 nm.

Estimation of Alanine transaminase: Alanine transaminase in both serum and liver were determined by the manner of Reitman's and Frankel, 1957. 0.5 ml of buffered substrate was incubated at 37 °C for 3 minutes and 0.1 ml of serum was added, mixed well and incubated at 37°C for 60 minutes. Then 0.5 ml of 2, 4 dinitrophenyl hydrazine reagent was added, mixed well and kept at room temperature for 20 minutes and 0.5 ml of 4N working sodium hydroxide was added and kept at room temperature for 10 minutes. Blank and standards were also processed in a similar way and the absorbance was measured spectrophotometrically at 505 nm. The activity of SGPT was expressed as IU/L.

Estimation of Alkaline phosphastase: Alkaline phosphatase in both serum and liver were determined by the manner of King and Amstrong, 1934. 4 ml of substrate was a pipette out in tubes marked as test and control. They were placed in a boiling water bath at 37 °C for a few minutes. 0.2 ml of serum was added only to the test without removing the tubes from the water bath. Incubate for 15 minutes at 37 °C. At the end of 15 minutes added 1.8 ml of diluted phenol reagent to all tubes. Then 0.2 ml of serum was added to the tubes marked as a control. All the tubes were shaken well and centrifuged. 4 ml of supernatant was withdraw from each tube. Standards of various concentrations (0.2 - 1.0 ml) were taken and made up to 2.8ml with water. 1ml of sodium carbonate solution was added to all the tubes and the tubes were incubated in a water bath at 37 °C for 15 minutes. To a blank 2.8 ml of water was added to 1.2 ml of diluted phenol reagent. The activity of ALP was expressed in KA units.

Antioxidant Enzymes Activities:

Estimation of Catalase Activity: To measure the catalase the sample was homogenized in a pre chilled mortar and pestle with 0.067M phosphate buffer at 1 - 4 °C and centrifuged. The Sediment Stirred with cold phosphate buffer kept in the cold

with occasional shaking and then repeated the extraction once or twice. The extraction should not exceed than 24 hours and the combined supernatants were used for the assay.

Assay: Pipetted out 3.0 ml of H_2O_2 Phosphate buffer into a test tube. Mixed with 0.01 - 0.04 ml of sample with a glass rod. Noted the time Δt required for a decrease in absorbance from 0.45 to 0.4 at 240 nm. This value was used for the calculation. If Δt was greater than 60 seconds, then repeated the measurements with a more concentrated solution of the sample. Calculated the activity and expressed in units per mg protein. One enzyme unit was calculated as the amount of enzyme required to decrease the absorbance at 240 nm by 0.05 units.

Calculation: Calculated the concentration of H_2O_2 using the extinction coefficient 0.036 μ mole/ml. [20]

Determination of Lipid Peroxidase Activity:

Enzyme extract: To estimate the peroxidase activity measured one gram of the sample with 5ml (w/v) 0.1 phosphate buffer (pH 6.5) in a homogenizer. Centrifuge the homogenate at 300g for 15minutes. Use the supernatant as the enzyme source. All procedures were carried out in 0 - 50°C.

Procedure: Pipetted out 3ml of 0.05 M- Pyrogallol solution and 0.5 to 0.1ml of enzyme extract in a test tube. Adjust the spectrophotometer to read '0' at 400 nm. Add 0.5ml of 1% H_2O_2 in the cuvette. Record the change in the absorbance every 30 seconds up to 3 minutes. [21]

Assay of Superoxide Dismutase (SOD):

Procedure: The estimation of SOD activity was carried out by using 300µl of each reagent 50mM potassium phosphate buffer, 45µM Methionine, 5.3µM Riboflavin, 84µM NBT and 20mM potassium cyanide as incubation medium. To the test 300µl of sample was added and the final volume was made up to 3 ml with distilled water. The tubes were kept in a box lined with aluminium foil, at 25 ºC and equipped with 15W fluorescent lamps. Decreased NBT was measured spectrophotometrically at 600nm after exposure to light for 10 minutes. The maximum reaction was evaluated in the absence of enzymes. One unit of enzyme activity is defined as the enzyme reaction, which gives 50% inhibition of NBT reduction in one minute under the assay conditions and expressed as specific activity in units. [22]

Estimation of Reduced Glutathione:

Procedure: To estimate the reduced glutathione 1g of the sample was homogenized in 5% TCA to give 20% homogenate. The precipitated protein was centrifuged at 1000rpm for 10 minutes. The homogenate was collected and allowed to cool on ice and 0.1 ml of the supernatant was taken for the estimation. The volume of the aliquot was made up to 1ml with 0.2M Sodium phosphate buffer of pH 8.0. DTNB solution (0.6mM in 0.2M phosphate buffer - pH 8.0) was added to the tubes and the intensity of the yellow colour formed was read at 412nm in a spectrophotometer after 10 minutes. A standard curve of reduced glutathione was prepared using concentrations ranging from 2 to 10 nanomoles of reduced glutathione in 5% TCA. [23]

RESULTS: The Preliminary phytochemical analysis showed that the petroleum ether extract was rich in Alkaloids, Tannins- Phenolic Compounds, Proteins – Amino acids, Steroids & Sterols and Fat in. Alkaloids, Cardiac glycoside, Tannins-Phenolic Compounds, Flavones and Flavonoids and Steroids & Sterols were present in chloroform extract.

In ethanol extract Alkaloids, Carbohydrates, Glycosides (Cardiac glycoside), Tannins - Phenolic Compounds, Proteins –Amino acids, Flavones and Flavonoids, Saponins.

Hepatoprotective activity revealed that the all three extract (PEESD, CESD and EESD) of *Scoparia dulcis* provide protection against the toxic effect of CCl_4 on liver. In CCl_4 induced toxic hepatitis, toxicity begins with the changes in ER (endoplasmic reticulum), which result in the debt of metabolic enzymes located in intracellular structure. [24] The blood sample of CCl_4 treated group animals showed the drastic increase in the level of SGPT, SGOT, serum bilirubin, serum cholesterol and liver inflammation on compared to that of control group. The rapid elevation in the levels of serum aspirate transaminase indicates the extent of liver necrosis. [25]

Administration of the test extract showed recovery against the toxic effects of CCl_4 on comparison with the standard drug Silymarin.

TABLE: 1 EFFECT OF DIFFERENT EXTRACT OF *SCOPARIA DULCIS* ON CCl_4 INDUCED HEPATOTOXICITY IN RAT

Group	Biochemical Parameters						
	SGPT (IU/L)	SGOT (IU/L)	Billirubin (mg/dl)		Cholesterol (mg/dl)		Liver weight
			Total	Direct	Total	HDL	
Control	56.00 ±1.03	60.33 ± 1.58	0.394±0.03	0.232±0.04	115.38±0.39	9.61±0.39	8.55 ± 0.18
CCl_4	124.00±1.96	159.83 ± 0.49	6.729±0.54	1.215±0.03	176.28±1.54	1.68±0.20	10.55 ±0 .18
Silymarin	78.00 ±1.15	67.83 ± 1.04	0.794±0.04	0.910±0.02	121.47±2.29	23.63±0.26	9.42 ±0 .20
PEESD	88.5 ±0.76	139.66 ± 1.81	1.563±0.02	0.361±0.02	167.94±2.25	4.08 ±0.27	9.47 ± 0.24
CESD	81.33±1.55	105.00 ± 1.23	1.660±0.03	0.374±0.03	152.24±2.01	8.4 1±0.72	10.19 ± 0.04
EESDD	81.00±1.39	92.00 ± 1.41	1.240±0.03	0.368±0.26	138.14±1.62	5.92±0.36	10.12 ± 0.01

Results expressed as mean ± SEM from six observations

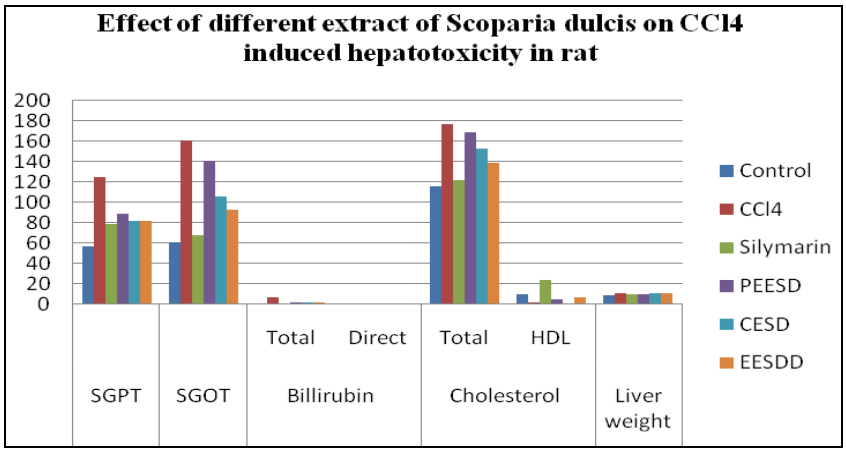

FIG. 1: EFFECT OF DIFFERENT EXTRACT OF *SCOPARIA DULCIS* ON CCl_4 INDUCED HEPATOTOXICITY IN RAT

TABLE: 2 THE INFLUENCE OF ORAL ADMINISTRATION OF *SCOPARIA DULCIS* EXTRACT TO CARBAN TETRACHLORIDE TREATED ANIMALS ON ANTIOXIDANT ENZYMES ACTIVITIES

Sr. No.	Group (n=5)	SOD (unit/mg tissue)	GSH (mmol/mg tissue)	LPO (nmol MDA/mg tissue)	CAT (unit/mg tissue)
1	Control	11.52±.025	4.48±0.1	1.54±0.03	13.85 ±0.25
2	CCl_4	3.01±0.05	0.3±0.01	6.26±0.25	3.96± 0.07
3	Silymarin	10.7±0.25	4.25±0.11	1.89±006	13.12 ±0.19
4	PEESD	3.91±0.38	0.95±0.16	5.88±0.121	3.81±0.64
5	CESD	3.86±0.423	0.98±0.21	6.12±0.222	4.12±0.48
6	EESDD	7.12±0.341	2.27±0.25	3.34±0.33	9.85±0.37

Values are Mean ± SEM, n=6, when compared with Toxic control

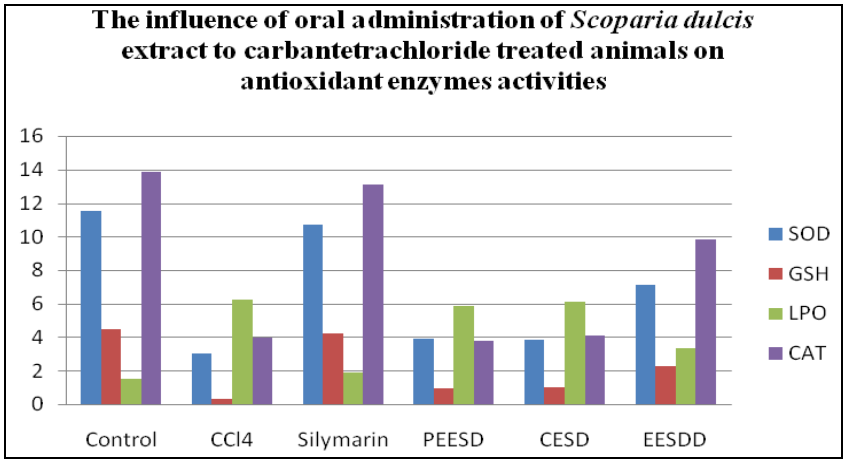

FIG. 2 THE INFLUENCE OF ORAL ADMINISTRATION OF *SCOPARIA DULCIS* EXTRACT TO CARBAN TETRACHLORIDE TREATED ANIMALS ON ANTIOXIDANT ENZYMES ACTIVITIES

DISCUSSION: In the assessment of liver damage by CCl_4 hepatotoxin the determination of the enzyme levels such as SGPT and SGOT is widely used. Necrosis releases the enzyme into circulation; therefore it can be measured in serum. Increase levels of SGPT indicate liver damage, cardiac infraction and muscle injury. SGPT catalyzes the conversion of alanine to pyruvate and glutamate, and is released in a same way. Therefore SGPT is more specific to the liver and is thus a better parameter for detecting liver injury. [26] Our results using the model of CCl_4 induced hepatotoxicity in the rats demonstrated that the test extract caused significant inhibition of SGPT and SGOT levels. Serum bilirubin and cholesterol levels are related to the function of hepatic cell. Our results using the model of CCl_4 induced the hepatotoxicity in rats demonstrated that our test extract (PEESD, CESD and EESD) caused significant inhibition of bilirubin levels.

CONCLUSION: The results of this study demonstrate that PEESD, CESD and EESD have a potent hepatoprotective action upon carbon tetrachloride induced hepatic illness in rats. Our results show that the hepatoprotective effects of our test sample may be due to the antioxidant and free radical scavenging properties contributed by the presence of phytoconstituents like phenolic compounds and flavonoids.

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CONFLICT OF INTEREST: Nil

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