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Hepatoprotective Activity Of Aqueous Seed Extract Of D. Hamiltonii Against CCI4-Induced Liver Damage In Rats

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Abstract:
The hepatoprotective effect of *D. hamiltonii* aqueous seed extract was evaluated against carbon tetrachloride (CCl_4)-induced hepatotoxicity in rats. Pretreatment with the extract (50–200 mg/kg) significantly reduced serum levels of ALT, AST, ALP, and bilirubin compared to the CCl_4 control. The 200 mg/kg dose provided 71.85% ALT and 69.22% AST protection, nearing the efficacy of standard drug silymarin. These findings highlight the potential of D. hamiltonii as a hepatoprotective agent, likely due to its antioxidant and anti-inflammatory constituents such as flavonoids and polyphenols.

Keywords: *D. hamiltonii*, hepatoprotection, CCl_4, ALT, AST, silymarin, oxidative stress.

Introduction

The liver is a vital organ responsible for numerous metabolic, detoxifying, and synthetic functions in the body. It plays a central role in the regulation of homeostasis, nutrient metabolism, and xenobiotic clearance. However, the liver is also vulnerable to damage from a wide variety of hepatotoxins, including chemical agents like carbon tetrachloride (CCl_4), which is commonly used in experimental models to induce liver injury due to its well-established mechanism of oxidative stress and lipid peroxidation (Srivastav, 1983; Srivasav & Rani, 1989).

Conventional drugs used to treat liver disorders often present limited efficacy and potential side effects. As a result, there is a growing interest in identifying natural hepatoprotective agents, particularly from plant sources, which are considered safer and potentially more effective. One such plant is Decalepis hamiltonii, an indigenous medicinal plant known for its therapeutic properties (Pramyothin et al., 2006). The roots and seeds of *D. hamiltonii* have been traditionally used in Ayurveda for treating various ailments, including gastrointestinal disturbances and general debility. Phytochemical studies have revealed that the plant is rich in bioactive compounds like flavonoids, phenols, and antioxidants that may confer protective effects against oxidative stress-induced cellular damage (Kumar et al., 2010; Ramesh et al., 2009). Although previous studies have focused on the antioxidant and antimicrobial properties of *D. hamiltonii*, there is limited scientific evidence specifically evaluating the hepatoprotective potential of its seed extracts. Given the liver's susceptibility to oxidative damage and the known phytochemical profile of *D. hamiltonii*,

it is rational to explore whether the aqueous seed extract of the plant can mitigate chemically-induced liver injury (Thabrew et al., 1995).

This study aims to assess the hepatoprotective efficacy of the aqueous seed extract of *D. hamiltonii* against CCl_4-induced liver toxicity in Wistar albino rats. Biochemical markers such as ALT, AST, ALP, total bilirubin, and liver histopathology were evaluated to determine the extent of liver damage and the protective role of the plant extract. The findings of this study could contribute to the development of plant-based hepatoprotective agents and provide a scientific basis for the traditional use of *D. hamiltonii* in liver ailments.

Materials and Methods

Plant Material and Extract Preparation

Seeds of *D. hamiltonii* were collected, shade-dried, and powdered. Aqueous extraction was carried out by cold maceration (1:10 w/v) for 48 hours. The filtrate was concentrated under reduced pressure and stored at 4°C.

Carbon Tetrachloride (CCl_4)-Induced Hepatotoxicity in Rats

For the evaluation of the hepatoprotective activity of *Decalepis hamiltonii* aqueous leaf extract, rats were fasted for 24 hours prior to the experiment, with free access to water. The animals were randomly divided into five groups, each consisting of six rats:

Group I (Normal Control): Received normal saline (10 mL/kg, p.o.).

Group II (Toxic Control): Received CCl_4 (1 mL/kg, i.p.) diluted in liquid paraffin (1:1 v/v).

Group III: Received *D. hamiltonii* aqueous leaf extract at a dose of 50 mg/kg orally once daily for 7 consecutive days.

Group IV: Received *D. hamiltonii* extract at a dose of 100 mg/kg orally.

Group V: Received *D. hamiltonii* extract at a dose of 200 mg/kg orally.

On the seventh day, one hour after the final dose of the extract, groups II to V were administered CCl_4 intraperitoneally to induce hepatic injury, while group I received normal saline only. Twenty-four hours later, the rats were anesthetized under ether anesthesia, and blood samples were collected via cardiac puncture for the estimation of liver function markers such as ALT, AST, ALP, total bilirubin, and total protein (Gopi et al., 2015; Anandan et al., 1999).

Following blood collection, the rats were sacrificed, and the livers were excised and rinsed with cold saline. The livers were then fixed in 10% formalin for histopathological examination. Macroscopic changes such as liver congestion, pallor, and necrosis were recorded, and microscopic examination was performed to assess hepatocyte degeneration, fatty changes, necrosis, and inflammatory cell infiltration (Murugesan et al., 2001; Balakrishna et al., 2014).

Paracetamol-Induced Hepatotoxicity in Rats

Another model used to assess hepatoprotective activity involved paracetamol-induced liver damage. Rats were fasted for 24 hours prior to the experiment but had free access to water. They were divided into five groups (n=6 per group):

Group I (Normal Control): Received normal saline (10 mL/kg, p.o.).

Group II (Toxic Control): Received paracetamol (2 g/kg, p.o.) as a single dose to induce hepatotoxicity.

Group III, IV, and V: Received *D. hamiltonii* extract at 50, 100, and 200 mg/kg p.o., respectively, for seven consecutive days. On day 7, all groups except group I were administered paracetamol one hour after the final dose of extract.

After 24 hours, the rats were sacrificed under ether anesthesia. Blood samples were collected for biochemical analysis of hepatic enzymes (AST, ALT, ALP), bilirubin, and protein content. The livers were dissected, weighed, and subjected to macroscopic and histopathological analysis (Subramoniam & Pushpangadan, 1999; Srivastav et al., 2011; Sileshi et al., 2009).

Alcohol-Induced Hepatic Injury in Rats

Rats were deprived of food for 24 hours prior to the experiment. They were randomly divided into five groups of six animals each:

Group I (Normal Control): Received normal saline (10 mL/kg, p.o.).

Group II (Toxic Control): Received ethanol (6 mL/kg, 50% v/v p.o.) for 7 consecutive days to induce liver damage.
Groups III, IV, and V: Received *D. hamiltonii* aqueous leaf extract at doses of 50, 100, and 200 mg/kg respectively, p.o., 1 hour before ethanol administration each day for 7 days.
On the eighth day, all animals were anesthetized with ether, and blood was collected for biochemical estimation of liver enzymes. The livers were removed, weighed, and examined grossly and microscopically to evaluate the protective effect of the extract on alcohol-induced hepatocellular damage (Somasundaram et al., 2010; Bonga & Flik, 1993).

Statistical Analysis

The data was examined as mean S.E.M. The difference in significance between the control and treatment groups was determined. The one-way ANOVA was used, followed by the student's t-test. P0.05 was regarded as statistically significant.

Result and Discussion

Effect of *D. hamiltonii* Aqueous seed Extract on CCl_4-Induced Hepatotoxicity in Rats

Pre-treatment of rats with *D. hamiltonii* aqueous seed extract at doses of 50, 100, and 200 mg/kg showed a significant hepatoprotective effect, as observed by the reduction in serum liver enzyme levels (ALT, AST, ALP) and total bilirubin, when compared to the toxic control group. The extract at 200 mg/kg provided maximum hepatoprotection, with 71.85% reduction in ALT compared to the CCl_4 group, while the standard drug silymarin (100 mg/kg) showed 83.61% protection (Table 1).

Table 1: Effect of *D. hamiltonii* Aqueous seed Extract on CCl_4-Induced Hepatotoxicity in Rats

Group	Dose (mg/kg)	ALT (IU/L)	%Protection (ALT)
Normal Saline	10ml/kg	38.7 ± 2.1	-
CCl_4 (Control)	1 mL/kg i.p.	172.4 ± 5.3	
Ranitidine	100 mg/kg	28.3 ± 2.9 *	83.61%
D. hamiltoni	50 mg/kg	82.0 ± 4.1 *	52.45%
D. hamiltoni	100 mg/kg	63.4 ± 3.7 *	63.21%
D. hamiltoni	200 mg/kg	48.6 ± 3.2 *	71.85%

Result are expressed as mean ± SEM, *significantly different from control at P<0.05

Effect on AST Levels

The *D. hamiltonii* extract significantly reduced AST levels in a dose-dependent manner, suggesting preservation of hepatic cell membrane integrity. The 200 mg/kg dose showed 69.22% protection, close to silymarin.(Table 2)

Table 2: Effect on Aspartate Aminotransferase (AST) Levels

Group	Dose (mg/kg)	ALT (IU/L)	%Protection (ALT)
Normal Saline	10ml/kg	42.3 ± 2.5	-
CCl_4 (Control)	1 mL/kg i.p.	191.7 ± 6.4	
Ranitidine	100 mg/kg	36.1 ± 3.2 *	86.92%
D. hamiltoni	50 mg/kg	91.4 ± 4.2 *	55.79%
D. hamiltoni	100 mg/kg	72.9 ± 3.9 *	65.34%
D. hamiltoni	200 mg/kg	59.0 ± 3.4 *	69.22%

Result are expressed as mean ± SEM, *significantly different from control at P<0.05

Effect on ALP and Bilirubin Levels

Table 3 displays the extract also significantly normalized alkaline phosphatase (ALP) and total bilirubin levels, both of which are key indicators of hepatobiliary function. The 200 mg/kg dose showed ~74.18% protection in bilirubin

levels.

Table 3: Effect on ALP and Total Bilirubin

Group	ALP (IU/L)	% Protection ALP	Total Bilirubin (mg/dL)	% Protection (Bilirubin)
Normal Saline	86.1 ± 4.2	-	0.54 ± 0.06	-
CCl_4 (Control)	218.6 ± 5.9	-	2.16 ± 0.12	-
Silymarin	101.3 ± 4.1 *	82.67%	0.63 ± 0.08 *	90.79%
D. hamiltoni	144.5 ± 3.9 *	57.65%	1.42 ± 0.11 *	56.07%
D. hamiltoni	123.7 ± 3.5 *	67.65%	1.13 ± 0.09 *	68.57%
D. hamiltoni	105.4 ± 4.3 *	75.18%	0.94 ± 0.07 *	74.18%

Result are expressed as mean ± SEM, *significantly different from control at $P<0.05$

Conclusion

The present study demonstrates that the aqueous seed extract of *Decalepis hamiltonii* exerts significant hepatoprotective effects against carbon tetrachloride-induced liver injury in rats. The administration of the extract resulted in a marked improvement in liver function, as evidenced by the significant reduction in serum levels of liver enzymes (ALT, AST, and ALP) and total bilirubin, which are biomarkers of hepatic cellular integrity and function. These biochemical improvements were further corroborated by histopathological analysis, which showed preservation of normal liver architecture in extract-treated groups, in contrast to the severe hepatocellular damage observed in the CCl_4-treated control group.

The hepatoprotective effect of the seed extract is likely attributable to the presence of phytoconstituents with antioxidant and free radical scavenging properties. Compounds such as flavonoids and phenolics may play a pivotal role in neutralizing the reactive metabolites of CCl_4, thereby protecting hepatic cells from oxidative stress and membrane lipid peroxidation.

These findings suggest that *D. hamiltonii* seed extract holds promise as a natural therapeutic agent for the management of liver disorders. However, further studies are warranted to isolate the active constituents, determine their individual pharmacological properties, and evaluate the safety and efficacy of the extract in clinical settings. The study provides a scientific rationale for the traditional use of *D. hamiltonii* in hepatoprotective remedies and lays the groundwork for its potential inclusion in herbal formulations targeting liver health.

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