International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 22s, 2025
https://www.theaspd.com/ijes.php

Hepatoprotective Effect of Aerial Parts of *Desmostachya bipinnata* Against CCl_4 Induced Liver Damage

Alok Bhatt[1], Praveen K. Ashok[1], Pallavi Ghildiyal[3], Anupama Singh[2*]
[1]School of Pharmaceutical Sciences, Jigyasa University (Formerly Himgiri Zee University), Dehradun, 248011, Uttarakhand, India. (alokbhatt03@gmail.com and praveen.kumar@jigyasauniversity.edu.in)
[2]Department of Pharmacognosy, School of Pharmaceutical Sciences and Technology, Sardar Bhagwan Singh University, Balawala, Dehradun, 248001, Uttarakhand, India. (anupama.cognosy@gmail.com)
[3]Department of Pharmacology, Uttaranchal Institute of Pharmaceutical Sciences, Uttaranchal University, Dehradun, 248007, Uttarakhand, India. (pallavighildiyal2@gmail.com)

Abstract
The human liver is exposed to high concentrations of toxicants and toxic metabolites during the process of metabolic detoxification, making it susceptible to injury. Liver damage is associated with cellular necrosis, an increase in tissue lipid peroxidation, and depletion of tissue GSH levels, among other factors. Herbs play a significant role in managing various liver disorders. In the present study, Desmostachya bipinnata aerial parts were subjected to soxhlet and cloud point extraction. Preliminary phytochemical identification was performed using chemical tests and TLC. Subsequently, the antioxidant and hepatoprotective potential of the extracts were assessed in a CCl_4-induced hepatotoxicity model. Regarding soxhlet extraction, the alcoholic extract exhibited the highest extractive yield (20.00 ± 0.68% w/w), with phenols and flavonoids present at 10.92% w/w and 20.81% w/w, respectively. Among the eight cloud point extracts (CPE), CPE 8 showed the best results (temperature: 60°C, time: 2 hours, and Triton X-100 concentration: 5%), with a maximum extraction yield of 11.2 ± 01.26% w/w, flavonoids at 13.44% w/w, and phenols at 43.63% w/w.
Phytochemical screening of the extracts revealed the presence of phenolics, flavonoids, alkaloids, and steroids in different extracts of D. bipinnata. In the CCl_4-induced hepatotoxicity model, based on tissue and serum analysis, the order of hepatoprotective effect was found to be alcohol extract < chloroform extract < C.P.E < standard marketed formulation. The difference in activity among these extracts in reducing hepatotoxicity is likely attributed to the nature and quantity of phytoconstituents present in each extract. Further studies are planned to isolate and characterize the active constituent responsible for this activity.
Keywords: *Hepatotoxicity, herbal, extraction, Soxhlet, liver, cloud point.*

INTRODUCTION

Carbon tetrachloride radicals can bind to both cellular lipids and proteins. Carbon tetrachloride-induced liver damage proceeds through a sequence of steps that contribute to different levels of damage. These steps include reductive dehalogenation, covalent binding of radicals, inhibition of protein synthesis (especially apolipoprotein synthesis), assemblage, packing, and release of VLDL and HDL, accumulation of fat, formation of CCl3-OO* radicals, lipid peroxidation, membrane damage, loss of Ca2+ sequestration, apoptosis, and fibrosis.[5,6] Radical formation and lipid peroxidation are the predominant cellular mechanisms involved in the development of fatty liver caused by CCl4 exposure, as well as excessive alcohol consumption. [19,36] Accumulation of fat is one consequence of CCl4-induced liver damage, which develops only in the presence of an intact cytochrome P450 oxygenase system.[5] The cytochrome P450 inducer metyrapone was used to enhance the bioactivation of CCl4 and formation of radicals, namely CCl3* and CHCl2*. This leads to the accumulation of triglycerides in hepatocytes. It is important to note that the liver is not the only target organ of CCl4; it also affects several other organs in the body, including the lungs, heart, testes, kidneys, and brain.

Silybum marianum, Coccinia grandis, Flacourtia indica, Wedelia calendulacea, Prostechea michuacana, Swertia chirata, Phyllanthus emblica, Desmostachya bipinnata, Picrorhiza kurroa, Azadirachta indica, Aegle marmelos, etc., have been used in many poly-herbal formulations meant for the treatment of liver diseases.[15] Several plant products are available in the market to protect the liver from damage. Cuscuta chinensis and Cuscuta reflexa contain alkaloids (cuscutamine, lupanine, agroclavine), glycosides (cuscutin, cuscutoside A&B, arbutin, odoroside H), sterols (gitoxigenin, campestrol,

stigmasterol, sitosterol), flavonoids (kaempferol, quercetin, hyperoside), etc., which have proven effective in CCl4 and N-acetyl-para-aminophenol-induced hepatotoxicity.[2]

Silymarin, a bioflavonoid from milk thistle, is effective in preventing CCl_4-induced liver injury.[18] Iridoid glycosides like picroside I and picroside II, isolated from extracts of *Picorrhiza kurroa* (Scrophulariaceae), have proven effects against liver intoxication in mice induced by CCl_4. Acubin and iridoid glycoside, isolated from both leaves and seeds of Plantoago asiatica, have shown potent liver-protecting activity.[13] The saponins of the gypsogenic series have been isolated from *Dianthus superbus* (Caryophyllaceae) and *Panax ginseng* (Arleaceae) for their potential role in elevated SGOT and SGPT levels in CCl_4-intoxicated rabbits.

Antihepatotoxic effects of flavnolignans and related constituents from *Silybum marianum* have been observed through CCl_4 and D-gal N-induced cytotoxicity studies in primary cultured rat hepatocytes (Hikino et al. 1984). The flavonoids in plants such as *Colinium goggyria, Anemone hepatica* (Ranumculaceae), *Convallaria majalis* (Liliaceae), and *Omonus arvenis* (Leguminosae) are proven hepatoprotectives.[13] The ethanolic extract of *Zinnia elegans* has shown hepatoprotective activity against CCl4-induced liver damage due to the presence of polyphenolic components in the plant, such as kaempferol 3-O-β-glucoside, kaempferol 3-O-β-xyloside-7-O-β-glucoside, quercetin 3-O-β-glucoside, apigenin 7-O-β-glucoside, apigenin 4'-O-β-glucoside, luteolin 7-O-β-glucosides.[9] Extracts of various samples of the crude drugs prepared from the rhizomes of *Atracylodes macrophala* and *Atracylodes lancae* (Compositae) exhibited anti-hepatotoxic activity. The major sesquiterpenoid active components atractylon, β-eudemol, and hinesol exhibited a significant liver-protecting effect.[7]

D. bipinnata is commonly known as sacrificial grass, kusha[14], drabh[27], dab.[26] It contains many flavonoids such as kaempferol, quercetin, quercetin-3-glucoside, trycin, trycin-7-glucoside, as well as coumarins such as scopoletin and umbelliferone, sugars, amino acids, and carbohydrates.[2] *D. bipinnata* oil contains camphene, β-eudesmol, eseroline, and calarene in significant amounts, while others like diphenyliodinium bromide, limenone, 2-cyclohexene-1-one, and 8-nitro-12-tridecanolide are present in smaller quantities.[15] The leaf paste is used to cure cuts and wounds[13], while the root is used in asthma, rheumatism[2], carbuncles, piles, cholera, dysuria[27], and as a diuretic, galactagogue, astringent, remedy for dysentery, leucorrhoea, and wounds.[14]

Through literature survey, we found only one study that estimates the hepatoprotective effect of the aqueous extract of roots of *D. bipinnata* using a paracetamol-induced hepatotoxicity model in rats.[24] The present study was carried out to examine the hepatoprotective activity of extracts prepared from the aerial parts of *D. bipinnata*. Hepatotoxicity in rats was induced with CCl_4, and the effect of the extracts in reversing it was assessed.

MATERIAL AND METHODOLOGY

Plant Material Collection and Authentication

The whole plants of *D. bipinnata* (L.) Stapf were collected in September 2013 from Chirawa, district Jhunjhunu, Rajasthan, India. The collected plant was authenticated by Dr. R. P. Pandey from the Botanical Survey of India, Jodhpur, India. A voucher specimen, JNU/PH/2010/Db D2, was deposited in the herbarium of Jodhpur National University, Jodhpur, India.

Extraction and Photochemical Screening

The plant material, i.e., aerial parts of *D. bipinnata*, was ground in an electric mixer-grinder and screened using a BSS standard sieve. The powder that passed through sieve no. 22, with an average aperture size of 710 μm, and was retained on sieve no. 44, with an average aperture size of 355 μm, was selected and used for extraction.

Soxhlet Extraction

The powdered drug was packed in a paper cylinder made from filter paper and placed in the body of a Soxhlet extractor. The solvent (alcohol and chloroform) was poured into the Soxhlet extractor and allowed to run for 3-4 cycles. After that, the apparatus fitted appropriately, and the drug was extracted. The obtained extracts were filtered through Whatman filter paper and concentrated and dried by evaporating the solvent on a water bath. The residual moisture in the extract was removed by drying in an oven, followed by keeping the extract in a desiccator.[23]

Cloud Point Extraction

Ultrasonic cloud point extraction was performed as per the illustration shown in Fig.1.[25] Three factors, viz. temperature, time and surfactant concentration, at two levels (Table No. 1) were investigated to optimize the extraction process.

Table No. 1. Factors used in cloud point extraction

Name	Factor	Lowest level	Highest Level
A	Temp	40°c	60 °C
B	Time	1 h	2 h
C	Surfactant	2% w/v	5%

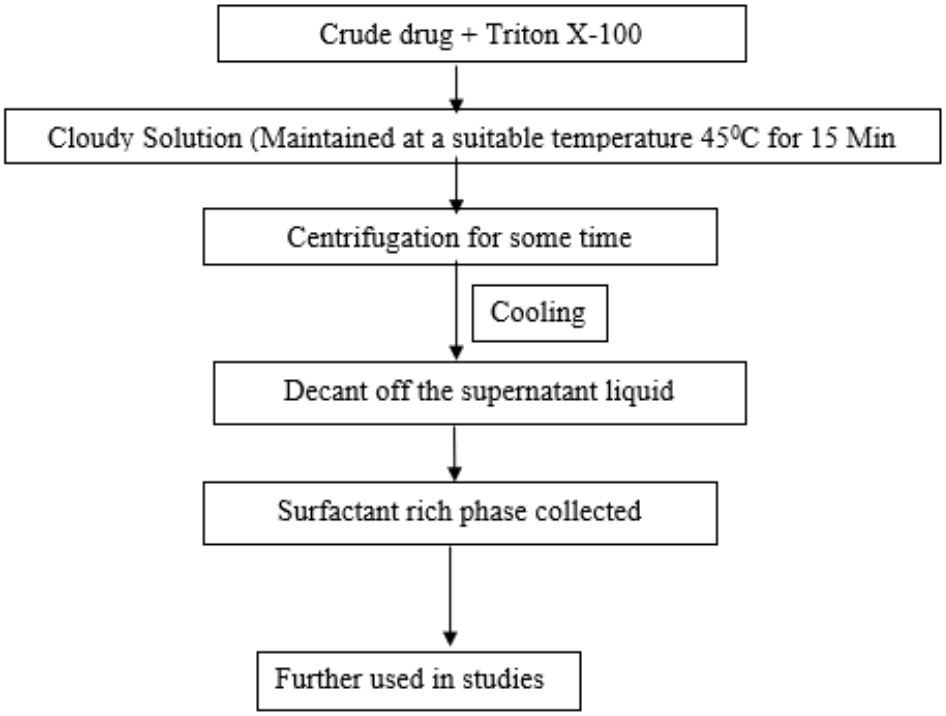


Fig. 1. Steps followed in cloud point extraction

The extracts of aerial parts of *D. bipinnata* were subjected to various qualitative chemical tests to determine the presence of various phytoconstituents like alkaloids, glycosides, carbohydrates, phenolics and tannins, fixed oils and fats, proteins, amino acids, flavonoids, saponins, etc. using reported methods.[23]

Carbon tetrachloride induced hepatotoxicity

Hepatotoxicity activity of chloroform and alcoholic extracts of aerial parts of *D. bipinnata* were evaluated using carbon tetrachloride induced hepatotoxicity in albino rats (n=5).[28]

Animals

Wistar albino rats of either sex weighing between 150-250 g were used for the study. Prior approval by institutional animal ethical committee, registration number 2650/SBS/2018, was obtained for conduct of experiments. Animals were kept in the departmental animal house at 25 ± 2°C and relative humidity 45-51.5% on a 12 h light/dark cycle.

Grouping and Treatment Protocol

Group I: Control group was treated with normal saline, i.e. 0.9% w/v of NaCl
Group II: Standard group received treatment of Himalaya Liv-52 at dose of 1 mg/kg
Group III: Positive control Carbon tetrachloride (1 mL/kg)
Group IV: Test group I was treated with alcoholic extract (500 mg/kg)
Group IV: Test group II received treatment of chloroform extract (500 mg/kg)
Group VI: Test group III received treatment of cloud point extract 8 extract (500 mg/kg)

Biochemical Estimation

Animals were fasted overnight, followed by treatments as mentioned above. Biochemical estimation was carried out 24 hours after the last dosing on the 8th and 15th day at the end of the treatment protocol. Serum parameters

including SGOT (serum glutamate oxaloacetate transaminases), SGPT (serum glutamate pyruvate transaminases), BIL (bilirubin), TP (total protein), and tissue parameters such as SOD (superoxide dismutase), GSH (reduced glutathione), LPO (lipid peroxidase), and CAT (catalase) were recorded. For serum parameters, blood was collected from the retro-orbital plexus under mild chloroform anesthesia, and serum was separated by centrifugation (C-24BL, Remi motor LTD. Mumbai) at 3000 rpm for 10 minutes. The resultant supernatant was used for biochemical estimation, and the analysis was performed using biochemical kits (ERBA).

All animals were fasted overnight. The estimation of oxidative stress markers was carried out 24 hours after the last dose on the 8th and 15th day at the end of the experimental protocol. The liver was quickly removed and washed with an ice-cool saline solution. The tissue was weighed and homogenized in tris buffer (pH 7.4). The homogenate was then centrifuged at 3000 rpm for 10 minutes, and the resultant cloudy supernatant liquid was used for estimation.

Reduced glutathione (GSH) was measured according to the method described by Glutathione peroxidase enzyme belongs to the peroxidase family, having a protective mechanism against oxidative damage. Glutathione contains a sulfhydryl group, i.e., 5,5 dithio bis 2-nitrobenzoic acid (DTNB).[22] A disulfide compound easily reacts with these sulfhydryl groups, resulting in the formation of a yellow-colored anion, which can be measured using a spectrophotometer at 412 nm.

Superoxide dismutase (SOD) was assayed by the method of Superoxide dismutase catalyzes the dismutation of superoxide into oxygen and hydrogen peroxide.[21] As superoxide has the ability to inhibit auto-oxidation of epinephrine to adenochrome at pH 10.2, this inhibition can be measured with a spectrophotometer at 480 nm.

The method is based on the principle to estimate malondialdehyde (MDA), a product of lipid peroxidation.[35] One molecule of MDA reacts with two molecules of thiobarbituric acid (TBA) under mildly acidic conditions to form a pink-colored chromogen whose intensity can be measured with a spectrophotometer at 535 nm.

Catalase (CAT) was estimated according to the method of Catalase is a common enzyme that protects cells from oxidative damage.[1] It is found in all living organisms and catalyzes the decomposition of hydrogen peroxide into oxygen and water. In the UV-Visible spectrophotometer (200 - 400 nm), hydrogen peroxide shows an increase in absorption with decreasing wavelength. The decomposition of hydrogen peroxide can be followed with the decrease in absorbance at 240 nm. The difference in absorbance per unit time is a measure of catalase activity.

Histopathology of Liver

Harvested liver tissues were fixed in formalin (4% v/v) and sectioned perpendicularly. Tissue sections were stained with hematoxylin-eosin using standard histopathology protocol.[23]

Statistical analysis

All the data were represented as mean ± S.E.M., n=5, and analyzed by one-way ANOVA followed by Dunnett's t-test for the possible significant interrelation between the various groups.

RESULTS

Cloud point extraction and soxhlation were used to prepare various extracts from the aerial parts of *D. bipinnata*. In the soxhlation method, n-hexane, chloroform, and alcohol were used as solvents, while cloud point extraction employed triton X-100 as a surfactant. The maximum extractive yield of 20.00 ± 0.68% w/w was obtained in alcohol, followed by 4.44 ± 0.18% w/w in chloroform, and the least, 1.90 ± 0.57% in n-hexane.

The amount of phenolics and flavonoids present in the alcoholic extract was 10.92% w/w and 20.81% w/w, respectively. In the chloroform extract, phenolics and flavonoids were found to be 7.76% and 8.59% w/w, respectively. Among all eight extracts of cloud point extraction, CPE 8 (Temperature 60°C, time 2 h, and triton X-100 5% w/v) demonstrated the best results, with a maximum extraction yield of 11.2 ± 01.26% w/w, flavonoids at 13.44% w/w, and phenolics at 43.63% w/w.

During the phytochemical screening of extracts, phenolics, flavonoids, alkaloids, and steroids were found to be present in different extracts of *D. bipinnata*.

Hepatoprotective Activity

The serum parameters, such as SGOT, SGPT, BIL, and TP, were significantly higher in positive control rats compared to the standard drug and extracts of *D. bipinnata*. In the control group of rats, the estimated values of serum transaminases (SGOT and SGPT) were 16.40 ± 1.65 and 15.88 ± 1.62, respectively. However, after the

administration of a toxic dose of CCl4 (3 ml/kg), these values significantly increased to 46.80 ± 2.65 and 49.86 ± 3.52, respectively. However, pretreatment of animals with different extracts (alcoholic, chloroform, and C.P.E. 8) of *D. bipinnata* led to a reduction in serum SGOT and SGPT values. C.P.E. 8 showed a significant decrease in serum SGOT and SGPT values to 23.40 ± 0.67 and 27.50 ± 1.53, respectively, which were very close to the standard treatment.

Table No. 2: Serum analysis parameters after 14 days of treatment

Groups	**Serum biochemical parameters**			
	SGOT (U/L)	**SGPT (U/L)**	**BIL (mg/dl)**	**TP (g/dl)**
Control	16.40± 1.65	15.88± 1.62	0.38± 0.02	7.51± 0.19
Standard	25.87± 3.95***	22.34± 1.87***	0.62± 0.02***	8.70± 0.15***
Positive control	46.80± 2.65*	49.86± 3.52*	1.80± 0.15*	12.69± 0.35*
Test 1 (Alcohol)	45.98± 1.81*	49.66± 3.14*	0.94± 0.01***	11.27± 0.15*
Test 2 (chloroform)	35.97± 2.54**	35.16± 1.22**	1.06± 0.03**	10.54± 0.38**
Test 3 (C.P.E 8)	23.40± 0.67***	27.50± 1.53***	1.03± 0.25**	9.87± 0.12**

Note: The statistical significance of difference between means was calculated by Analysis of Variance (ANOVA), followed by Dunnett's test for multiple comparison and t-test followed by unpaired test. Values are expressed as Mean ± SEM. $^{*}P<0.05$, $^{**}P<0.01$, $^{***}P<0.001$, n=5 in each group.

Similarly, bilirubin and total protein levels were higher in the positive control group, i.e., 1.80 ± 0.15 and 12.69 ± 0.35, respectively. An increase in bilirubin concentration in the serum or tissue indicates obstruction in the excretion of bile, and thus, the elevated level of bilirubin observed in rats administered with CCl_4 alone (positive control) could be attributed to liver damage. However, the decrease in bilirubin levels in pretreated rats is indicative of the reversal of liver damage, which was observed in the pretreatment groups of different extracts (alcoholic, chloroform, and C.P.E. 8) of *D. bipinnata*. C.P.E. 8 significantly lowered the values of serum bilirubin and total protein to 1.03 ± 0.25 and 9.87 ± 0.12, respectively, which were close to the standard values.

In the tissue parameters, the levels of antioxidant enzymes SOD, GSH, and CAT were decreased, but the level of LPO significantly increased in positive control rats. However, in different pretreatment groups with standard, alcoholic, chloroform, and CPE8 extracts, the levels of antioxidant enzymes SOD, GSH, and CAT were increased compared to the control and positive control groups.

Table No. 3. Tissue analysis parameters after 14 days of treatment

	Tissue parameters (IU/l)			
	SOD	**GSH**	**LPO**	**CAT**
Control	94.25± 1.57	18.48± 1.59	60.73± 2.73	47.35± 1.52
Positive control	61.43± 3.55	11.63± 1.45	161.30± 9.37	38.88± 2.71
Standard	78.28± 3.20**	15.50± 1.45**	94.12± 4.46***	46.38± 1.45***
Test 1 (Alcohol)	58.58± 1.46	17.73± 1.18***	109.60± 2.31**	37.81± 2.24*
Test 2 (chloroform)	62.08± 1.57*	18.16± 1.69***	111.20± 1.29**	36.75± 2.27*
Test 3 (C.P.E 8)	69.69± 1.40**	16.00± 1.21**	114.40± 4.25**	41.86± 1.36**
Note: LPO (Lipid peroxide), SOD (Superoxide dismutase), CAT(Catalase), and GSH (Glutathione)				

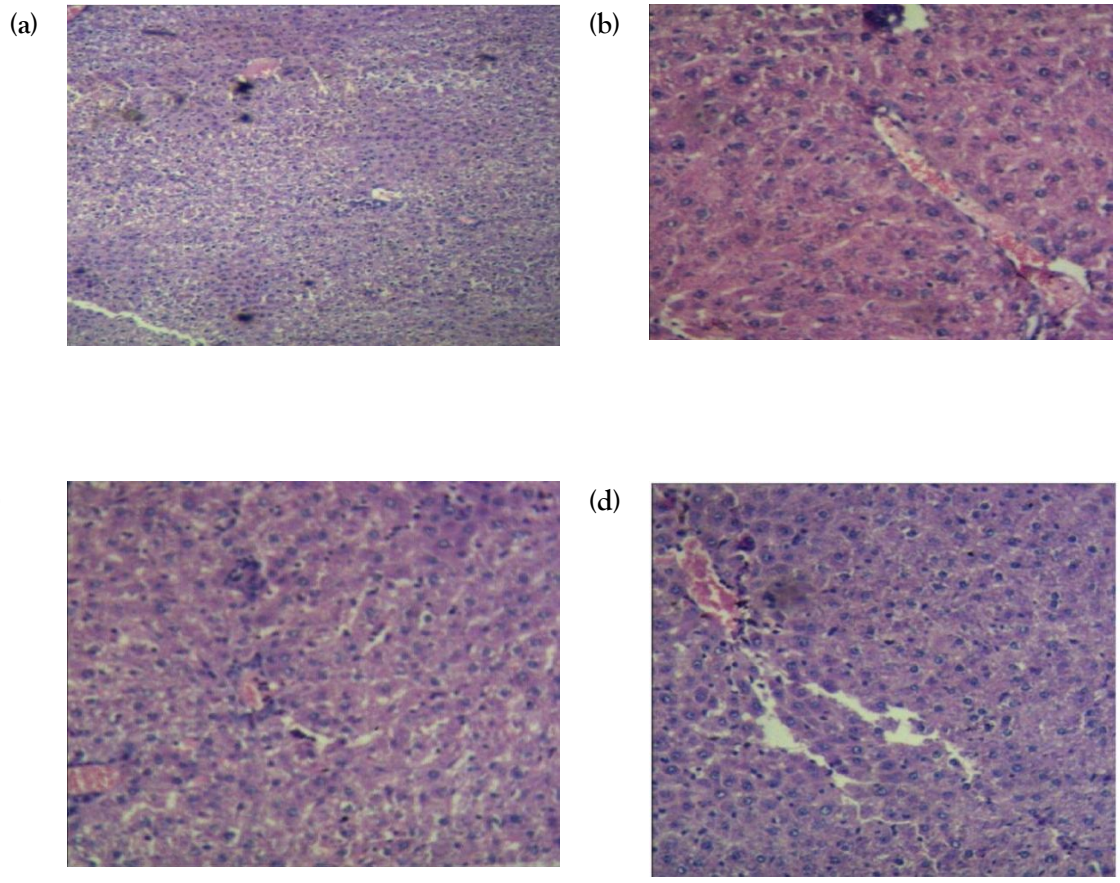
Note: The statistical significance of difference between means was calculated by Analysis of Variance (ANOVA), followed by Dunnett's test for multiple comparison and t-test followed by unpaired test. Values are expressed as Mean ± SEM. $^{*}P<0.05$, $^{**}P<0.01$, $^{***}P<0.001$, n=5 in each group.

Histopathology Studies on Rats' Liver

Histopathological studies provided supportive evidence for the biochemical analysis. The histology of the normal control group [Fig. 2(a)] shows reparative changes in a few hepatocytes, with no evidence of cirrhosis and toxicity. As for the treatment groups [Fig. 2(e) and 2(f)], they exhibit similar results, except for the alcoholic group [Fig. 2(d)], where the absence of glycogen degeneration was observed. Among all treatment groups, CPE 8 was found to be more effective in tissue, serum, and histopathological studies.

The difference in the activity of these extracts in reducing hepatotoxicity is likely due to variations in the active constituents present in them. Phytoconstituents such as alkaloids, monoterpenoids, diterpenoids, triterpenoids, steroids, lignans, glycosides, phenolics, flavonoids, and coumarin compounds have been reported to possess hepatoprotective activity and are found in different plant parts.[30,31] Agents possessing antioxidant[1], free radical scavenger[29], and anti-lipid per oxidant activities can effectively manage reactive species-mediated hepatotoxicity upon administration.[20]

Various phytoconstituents have been identified from *D. bipinnata* and reported in the literature, including coumarins (scopoletine and umbelliferone), carbohydrates, sugars, proteins, amino acids, alkaloids, tannins, phenolics, xanthenes (2,6-dihydroxy-7Zmethoxy-3H-xanthen-3-one)[32] steroids (stigmasterol, β-sitosterol, daucosterol, etc.)[32], flavonoids (kaempferol, quercetin, quercetin-3-glucoside, trycin, and trycin-7-glucoside)[4], triterpenoids, and essential oils obtained from the aerial parts of D. bipinnata consist of camphene, isobornyl acetate, tricyclene, caryophyllene diepoxide, eudesmol, eseroline, and calarene as the main components of the oil.[8,10,11,15]



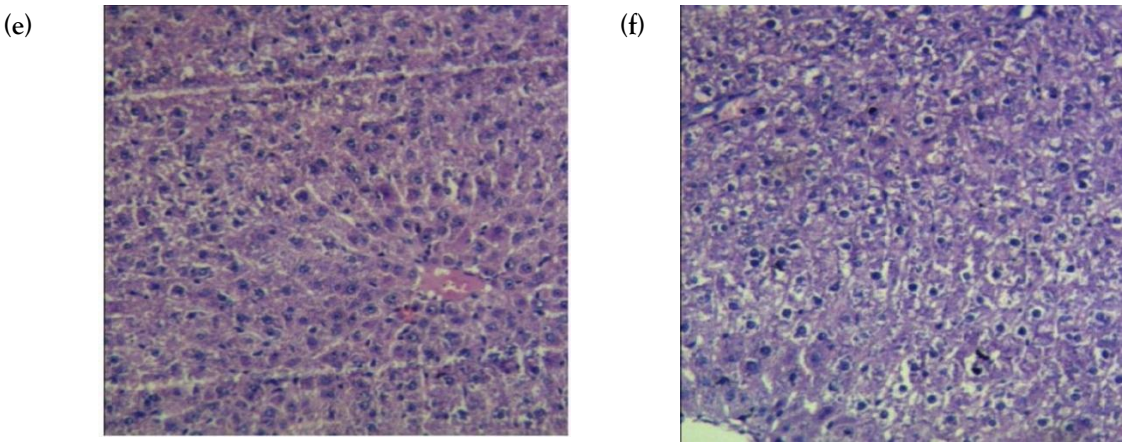


Fig. No. 2. Histopathology of liver in CCl_4 induced model of hepatotoxicity. (a) Control group (b) standard group (c) positive control group (d) alcoholic extract group (e) chloroform extract group (f) CPE 8 extract group

DISCUSSION

In the preliminary phytochemical screening of extracts, phenolics, flavonoids, alkaloids, and steroids were found in different extracts of *D. bipinnata*. In hepatoprotective activity, CPE 8 was found to be more effective among all treatment groups in tissue, serum, and histopathological studies. The results of our studies validate the traditional use of the plant as a hepatoprotective agent. Detailed studies, including phytochemical analysis, isolation of phytoconstituents, mechanisms, formulation, etc., are warranted for exploitation of this plant for developing herbal medicinal product.

ACKNOWLEDGEMENT

We extend our gratitude to Dr. R. P. Pandey from the Botanical Survey of India, Jodhpur, India, for the identificatioand taxonomic confirmation of collected plants.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

REFERENCES

1. Aebi, Hugo. 1984. "[13] Catalase in Vitro." In , 121–26. doi:10.1016/S0076-6879(84)05016-3.

2. Ahmad, Ateeque, Sudeep Tandon, Tran Dang Xuan, and Zulfa Nooreen. 2017. "A Review on Phytoconstituents and Biological Activities of Cuscuta Species." *Biomedicine & Pharmacotherapy* 92 (August): 772–95. doi:10.1016/j.biopha.2017.05.124.

3. Attri, S, S V Rana, K Vaiphei, C P Sodhi, R Katyal, R C Goel, C K Nain, and K Singh. 2000. "Isoniazid – and Rifampicin–Induced Oxidative Hepatic Injury – Protection by N–Acetylcysteine." *Human & Experimental Toxicology* 19 (9): 517–22. doi:10.1191/096032700674230830.

4. Awaad, Amani, Nawal Mohamed, Derek Maitland, and Gamal Soliman. 2008. "Anti-Ulcerogenic Activity of Extract and Some Isolated Flavonoids from Desmostachya Bipinnata (L.) Stapf." *Records of Natural Products* 2: 76–82.

5. Boll, Meinrad, Lutz W. D. Weber, Eberhard Becker, and Andreas Stampfl. 2001. "Pathogenesis of Carbon Tetrachloride-Induced Hepatocyte Injury Bioactivation of CCl 4 by Cytochrome P450 and Effects on Lipid Homeostasis." *Zeitschrift Für Naturforschung* C 56 (1–2): 111–21. doi:10.1515/znc-2001-1-218.

6. Clawson, Gary A. 1989. "Mechanisms of Carbon Tetrachloride Hepatotoxicity." *Pathology and Immunopathology Research* 8 (2): 104–12. doi:10.1159/000157141.

7. Doreswamy, R, and Darshan Sharma. 1995. "Plant Drugs for Liver Disorders Management." *Indian Drugs* 32 (4). Indian Drug Manufacturers' Association: 139–54.

8. Golla, Upendarrao, Praveen Kumar Gajam, and Solomon Sunder Bhimathati. 2014. "Evaluation of Diuretic and Laxative Activity of Hydro-Alcoholic Extract of Desmostachya Bipinnata (L.) Stapf in Rats." *Journal of Integrative Medicine* 12 (4): 372–78. doi:10.1016/S2095-4964(14)60029-7.

9. Gomaa, Alshymaa, Mamdouh Samy, Samar Desoukey, and Mohamed Kamel. 2018. "A Comprehensive Review of Phytoconstituents and Biological Activities of Genus Zinnia." *Journal of Advanced Biomedical and Pharmaceutical Sciences* 2 (1): 29–37. doi:10.21608/jabps.2018.5599.1024.

10. Hegde, Medha, Kuruba Lakshman, K Girija, Ashok Kumar Bs, and V Lakshmiprasanna. 2010. "Assessment of Antidiarrhoeal

Activity of Desmostachya Bipinnata L. (Poaceae) Root Extracts." *Boletin Latinoamericano y Del Caribe de Plantas Medicinales y Aromaticas* 9: 312–18.

11. Hifnawy, M S, H H Ammar, S k. Kenawy, M E Zaki, A K Yossef, and A Awaad S. 1999. "Phytochemical and Biological Studies on Alkaloidal Content of Some Allergy Producing Plants Growing in Egypt." *Bulletin of the Faculty of Pharmacy (Cairo University)* 37 (2): 107–17.

12. Hikino, Hiroshi, Yoshinobu Kiso, Hildebert Wagner, and Manfred Fiebig. 1984. "Antihepatotoxic Actions of Flavonolignans from Silybum Marianum Fruits." *Planta Medica* 50 (03): 248–50. doi:10.1055/s-2007-969690.

13. Katewa, S.S., and Anita Jain. 2006. *Traditional Folk Herbal Medicines*. Apex Publishing House. https://www.bagchee.com/books?author=33163.

14. Khare, C.P. 2007. "Launaea Pinnatifida Cass." In *Indian Medicinal Plants*, 1–1. New York, NY: Springer New York. doi:10.1007/978-0-387-70638-2_887.

15. Kumar, K Ashok, Sharvanee Sharvanee, Jitendra Patel, and Ram Kumar Choudhary. 2010. "Chemical Composition and Antimicrobial Activity of the Essential Oil of Desmostachya Bipinnata Linn." *International Journal of Phytomedicine* 2: 436–39.

16. Kumar, Vinod, Rajeev Kumar, Sanjay Yadav, Satyawan Singh, and Surendra Nath Pandeya. 2010. "Evaluation of Analgesic & Anti-Inflammatory Activity of Hydro-Alcoholic Extract of Desmostachya Bipinnata (L.) Stapf Root on Experimental Animals." *International Journal of Pharmaceutical Sciences and Drug Research* 2 (3): 213–15.

17. https://ijpsdr.com/index.php/ijpsdr/article/view/126.

18. Lettéron, Philippe, Gilles Labbe, Claude Degott, Alain Berson, Bernard Fromenty, Marcel Delaforge, Dominique Larrey, and Dominique Pessayre. 1990. "Mechanism for the Protective Effects of Silymarin against Carbon Tetrachloride-Induced Lipid Peroxidation and Hepatotoxicity in Mice." *Biochemical Pharmacology* 39 (12): 2027–34. doi:10.1016/0006-2952(90)90625-U.

19. Lieber, Charles S. 2000. "Alcoholic Liver Disease: New Insights in Pathogenesis Lead to New Treatments." *Journal of Hepatology* 32 (January): 113–28. doi:10.1016/S0168-8278(00)80420-1.

20. Lim, Hwa-Kyung, Hack-Seang Kim, Hong-Serck Choi, Seikwan Oh, Choon-Gon Jang, Jongwon Choi, Seung-Hwan Kim, and Myung-Jei Chang. 2000. "Effects of Acetylbergenin against D -Galactosamine-Induced Hepatotoxicity in Rats." *Pharmacological Research* 42 (5): 471–74. doi:10.1006/phrs.2000.0730.

21. Misra, H P, and I Fridovich. 1972. "The Role of Superoxide Anion in the Autoxidation of Epinephrine and a Simple Assay for Superoxide Dismutase." *The Journal of Biological Chemistry* 247 (10). United States: 3170–75.

22. Moron, M, J Depierre, and B Mannervik. 1979. "Levels of Glutathione, Glutathione Reductase and Glutathione S-Transferase Activities in Rat Lung and Liver." *Biochimica et Biophysica Acta (BBA) - General Subjects* 582 (1): 67–78. doi:10.1016/0304-4165(79)90289-7.

23. Mukherjee, Pulok. 2002. "Quality Control of Herbal Drugs: An Approach to Evaluation of Botanicals." *Business Horizons*.

24. Nair, Gouri, and Subeesh Viswam. 2014. "Protective Effect of Desmostachya Bippinata Against Paracetamol Induced Hepatotoxicity in Rats." *Universal Journal of Pharmacy* 3.

25. Paleologos, Evangelos K., Dimosthenis L. Giokas, and Miltiades I. Karayannis. 2005. "Micelle-Mediated Separation and Cloud-Point Extraction." *TrAC Trends in Analytical Chemistry* 24 (5): 426–36. doi:10.1016/j.trac.2005.01.013.

26. Parveen, B. Upadhyay, Shikha Roy, and Ashwani Kumar. 2007. "Traditional Uses of Medicinal Plants among the Rural Communities of Churu District in the Thar Desert, India." *Journal of Ethnopharmacology* 113 (3): 387–99. doi:10.1016/j.jep.2007.06.010.

27. Qureshi, Rahmatullah, Ghulamraza Bhatti, And Rabia, and Rabia Memon. 2010. "Ethnomedicinal Uses of Herbs from Northern Part of NARA Desert, Pakistan." *Pakistan Journal of Botany* 42 (April): 839–51.

28. Ranawat, Lalitsingh, Jigar Bhatt, and Jagruti Patel. 2010. "Hepatoprotective Activity of Ethanolic Extracts of Bark of Zanthoxylum Armatum DC in CCl4 Induced Hepatic Damage in Rats." *Journal of Ethnopharmacology* 127 (3): 777–80. doi:10.1016/j.jep.2009.10.019.

29. Sadanobu, Shinro, Masaki Watanabe, Chika Nakamura, and Masakatsu Tezuka. 1999. "In Vitro Tests of 1,3-Dithia-2-Thioxo-Cyclopent-4-Ene to Evaluate the Mechanisms of Its Hepatoprotective Action." *The Journal of Toxicological Sciences* 24 (5): 375–81. doi:10.2131/jts.24.5_375.

30. Sharma, Bhawna, and Sharma Kumar. 2008. "Hepatoprotective Activity of Some Indigenous Plants." *International J. Pharm. Tech. Res* 1.

31. Sheikh, R A, D Babu, Nimmagadda Venkat Rao, P R Irene, Sachin More, and A Turaskar. 2012. "Hepatoprotective Activity of Alcoholic and Aqueous Extracts of Bark of Bassia Latifolia Roxb. Against Paracetamol Induce Hepatotoxicity in Rats." *Der Pharmacia Lettre* 4: 1272–84.

32. Shrestha, Sabina, Ha-Na Lyu, Ji-Hae Park, Dae-Young Lee, Jin Gyeong Cho, En-ji Cui, In-Sik Chung, and Nam-In Baek. 2011. "Sterols from the Leafy Culms of Desmostachya Bipinnata." *Chemistry of Natural Compounds* 47 (5): 852–53.

33. https://www.academia.edu/1387981/Sterols_from_the_leafy_culms_of_Desmostachya_bipinnata.

34. Shrestha, Sabina, Ji-Hae Park, Dae-Young Lee, Jin-Gyeong Cho, En-ji Cui, In-Sik Chung, Byoung-Mog Kwon, Moon-Hee Cho, Tae-Sook Jeong, and Nam-In Baek. 2011. "A New Xanthene from Desmostachya Bipinnata (L.) Stapf Inhibits Signal Transducer and Activator of Transcription 3 (STAT3) and Low-Density Lipoprotein-Oxidation." *Journal of the Korean Society for Applied Biological Chemistry* 54 (2): 308–11. doi:10.3839/jksabc.2011.049.

35. Slater, T. F., and B. C. Sawyer. 1971. "The Stimulatory Effects of Carbon Tetrachloride and Other Halogenoalkanes on Peroxidative Reactions in Rat Liver Fractions in Vitro . General Features of the Systems Used." *Biochemical Journal* 123 (5): 805–14. doi:10.1042/bj1230805.

36. Tribble, Diane L., Tak Yee Aw, and Dean P. Jones. 1987. "The Pathophysiological Significance of Lipid Peroxidation in Oxidative Cell Injury." *Hepatology* 7 (2): 377–86. doi:10.1002/hep.1840070227.Valan, M, A John, De Britto, and Ramaswamy Venkataraman. 2010. "Phytoconstituents with Hepatoprotective Activity." *Int J Chem Sci* 8.