

## Investigation of Antiulcer and Antidiarrheal Potential of *Cadaba farinosa* Forssk in Rodent Models

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### ABSTRACT

The present study was undertaken to investigate the antiulcer and antidiarrheal potential of *Cadaba farinosa* Forssk. using experimentally induced rodent models. The plant extract was subjected to preliminary phytochemical screening, which revealed the presence of flavonoids, tannins, alkaloids, saponins, phenolic compounds, and glycosides. Antiulcer activity was evaluated using ethanol-induced gastric ulcer and pylorus ligation models in Wistar rats, while antidiarrheal activity was assessed using castor oil-induced diarrhea, gastrointestinal motility, and enteropooling assays in mice. The extract exhibited significant dose-dependent gastroprotective activity by reducing ulcer index, gastric volume, free acidity, and total acidity while increasing gastric pH. In antidiarrheal studies, the extract significantly delayed the onset of diarrhoea, reduced the frequency of wet stools, inhibited intestinal motility, and decreased intestinal fluid accumulation. The pharmacological effects observed were comparable to standard drugs such as omeprazole and loperamide at higher doses. The observed activities may be attributed to the antioxidant, anti-inflammatory, antisecretory, and antimotility properties of the phytoconstituents present in the extract. The findings scientifically support the traditional use of *Cadaba farinosa* in gastrointestinal disorders and suggest its potential as a promising natural therapeutic agent.

**Keywords:** *Cadaba farinosa* Forssk, Antiulcer activity, Antidiarrheal activity, Gastroprotective effect, Rodent models.

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### INTRODUCTION

Peptic ulcer disease and diarrhea are among the most common gastrointestinal disorders affecting millions of people worldwide and remain a major public health concern,

especially in developing countries (1). Peptic ulcers are characterized by lesions in the gastric or duodenal mucosa resulting from an imbalance between aggressive factors such as gastric acid, pepsin, *Helicobacter pylori* infection, reactive oxygen species, alcohol

consumption, and non-steroidal anti-inflammatory drugs, and the protective mechanisms of the gastrointestinal mucosa (2). Diarrhea is a gastrointestinal disorder characterized by increased bowel frequency, excessive fluid secretion, and reduced absorption of water and electrolytes, leading to dehydration and nutritional imbalance (3). Although various synthetic drugs are available for the treatment of these disorders, prolonged use of these medications may produce adverse effects including constipation, relapse, drug resistance, electrolyte imbalance, and organ toxicity (4).

Medicinal plants have served as an important source of therapeutic agents since ancient times due to their safety, accessibility, and pharmacological effectiveness (5). Herbal medicines contain several biologically active phytoconstituents such as flavonoids, tannins, alkaloids, glycosides, terpenoids, and phenolic compounds which possess antioxidant, anti-inflammatory, antimicrobial, cytoprotective, and gastroprotective activities (6). Recent studies have demonstrated that phytochemicals can protect gastric mucosa through enhancement of mucus secretion, inhibition of gastric acid secretion, scavenging of free radicals, and reduction of inflammatory mediators involved in ulcerogenesis (7).

*Cadaba farinosa* Forssk. belongs to the family Capparaceae and is widely distributed in tropical and subtropical regions of Africa and Asia. The plant is extensively used in traditional medicine for the treatment of gastrointestinal disorders, wounds, inflammation, fever, diarrhea, dysentery, and stomach pain (8). Various parts of the plant including leaves, roots, and stem bark are reported to possess several pharmacological properties such as antioxidant, antimicrobial, anti-inflammatory, and cytoprotective activities (9). Phytochemical investigations revealed the presence of flavonoids, tannins, saponins, alkaloids, phenolic compounds, and triterpenoids which are known to contribute significantly to gastroprotective and antidiarrheal effects (9).

Previous experimental studies demonstrated that aqueous extracts of *Cadaba farinosa* improved gastrointestinal histological architecture and enhanced mucus secretion in Wistar rats, indicating potential mucosal protective activity (10). Flavonoids and phenolic compounds present in the plant may protect gastric mucosa through antioxidant mechanisms, while tannins may reduce intestinal secretion and improve mucosal resistance by protein precipitation over the intestinal lining (6). Similarly, alkaloids and

saponins may contribute to antimotility and antisecretory effects, thereby reducing diarrheal symptoms (7).

Oxidative stress and inflammation play a crucial role in the pathogenesis of gastric ulceration and diarrhea (2). Excessive production of reactive oxygen species results in lipid peroxidation and mucosal damage, leading to ulcer formation and intestinal dysfunction (7). Therefore, medicinal plants possessing antioxidant and anti-inflammatory activities are considered promising alternatives for the management of gastrointestinal disorders (5). Despite the extensive traditional use of *Cadaba farinosa*, scientific validation of its antiulcer and antidiarrheal properties remains limited.

Hence, the present study was designed to investigate the antiulcer and antidiarrheal potential of *Cadaba farinosa* Forssk. using experimentally induced rodent models. The study aims to scientifically validate the traditional claims associated with the plant and to explore its possible therapeutic role in gastrointestinal disorders.

#### AIM AND OBJECTIVES

The present study aims to investigate the antiulcer and antidiarrheal potential of *Cadaba farinosa* Forssk. using experimentally induced rodent models and to scientifically validate its traditional use in the management of gastrointestinal disorders (11,12).

#### Objectives

- To collect, authenticate, and prepare the extract of *Cadaba farinosa* Forssk. (13).
- To perform phytochemical screening of the prepared extract for the identification of major bioactive constituents such as flavonoids, tannins, alkaloids, saponins, and phenolic compounds (14).
- To evaluate the acute oral toxicity profile of *Cadaba farinosa* extract using OECD guidelines in rodents (15).
- To investigate the antiulcer activity of *Cadaba farinosa* extract using experimentally induced ulcer models such as ethanol-induced gastric ulcer and pylorus ligation-induced ulcer models in rats (16).
- To determine ulcer index, percentage ulcer inhibition, gastric volume, gastric pH, and total acidity in treated animals (17).
- To evaluate the antidiarrheal activity of *Cadaba farinosa* extract using castor oil-induced diarrhea,

- gastrointestinal motility, and enteropooling models in rodents (18).
- To assess the effect of the extract on intestinal motility and fluid accumulation in experimental animals (19).
  - To compare the pharmacological activity of the extract with standard antiulcer and antidiarrheal drugs (20).
  - To statistically analyze the obtained data and establish the therapeutic significance of *Cadaba farinosa* extract in gastrointestinal disorders (11).

## **MATERIALS AND METHODS**

### **Plant Material Collection and Authentication**

Fresh leaves/stem bark of *Cadaba farinosa* Forssk. will be collected from suitable geographical regions and authenticated by a qualified botanist or taxonomist. A voucher specimen will be deposited in the herbarium for future reference (13).

### **Preparation of Plant Extract**

The collected plant material will be washed thoroughly with distilled water to remove adhering dirt and contaminants and shade dried at room temperature for 10–15 days. The dried material will be powdered using a mechanical grinder and stored in airtight containers. The powdered material will be extracted using suitable solvents such as ethanol or methanol by Soxhlet extraction/maceration technique. The obtained extract will be concentrated using a rotary vacuum evaporator and preserved in a desiccator until further use (14,21).

### **Phytochemical Screening**

Preliminary phytochemical screening of the extract will be carried out for the qualitative detection of alkaloids, flavonoids, tannins, saponins, glycosides, phenolic compounds, steroids, and terpenoids using standard phytochemical procedures (14).

### **Experimental Animals**

Healthy adult Wistar rats weighing 150–250 g and Swiss albino mice weighing 20–30 g of either sex will be used for the study. The animals will be obtained from a registered animal house and maintained under standard laboratory conditions of temperature ( $25 \pm 2^\circ\text{C}$ ), relative humidity ( $55 \pm 5\%$ ), and 12 h light/dark cycle. Animals will be provided with standard pellet diet and water ad libitum. The experimental protocol will be approved by the Institutional Animal Ethics Committee (IAEC) in accordance with CPCSEA guidelines (15).

### **Acute Oral Toxicity Study**

Acute oral toxicity studies will be performed according to OECD guideline 423. Different doses of the extract will be administered orally to experimental animals, and they will be observed for mortality, behavioral changes, toxicity signs, and neurological abnormalities for 14 days. The safe dose obtained from the toxicity study will be selected for further pharmacological evaluation (15).

### **Evaluation of Antiulcer Activity**

#### **Ethanol-Induced Gastric Ulcer Model**

Rats will be divided into different groups containing six animals each. Animals will be fasted for 24 h before the experiment with free access to water. The control group will receive vehicle, the standard group will receive omeprazole, and the test groups will receive different doses of *Cadaba farinosa* extract orally. After 1 h of treatment, gastric ulceration will be induced by administering absolute ethanol (1 mL/200 g body weight). After another hour, animals will be sacrificed, and the stomachs will be dissected and examined for ulcer lesions. Ulcer index and percentage inhibition will be calculated (16,17).

#### **Pylorus Ligation-Induced Ulcer Model**

Animals will be fasted for 24 h before pylorus ligation. Under light anesthesia, the pyloric end of the stomach will be ligated carefully without damaging blood vessels. Test extract and standard drug will be administered prior to ligation. After 4 h, animals will be sacrificed, gastric juice will be collected, and parameters such as gastric volume, pH, free acidity, total acidity, and ulcer index will be determined (16).

### **Evaluation of Antidiarrheal Activity**

#### **Castor Oil-Induced Diarrhea**

Mice will be divided into control, standard, and test groups. The control group will receive vehicle, standard group will receive loperamide, and test groups will receive different doses of *Cadaba farinosa* extract orally. After 1 h, castor oil (1 mL) will be administered orally to induce diarrhea. Animals will be observed for 4 h, and the onset of diarrhea, total number of feces, and number of wet stools will be recorded (18).

#### **Gastrointestinal Motility Test**

Animals will receive charcoal meal after administration of the extract and standard drug. After a specified time, animals will be sacrificed, and the distance traveled by charcoal meal through the intestine will be measured to determine intestinal motility (19).

#### **Enteropooling Assay**

The enteropooling test will be carried out to evaluate the antisecretory activity of the extract. After treatment with the extract and castor oil administration, animals will be

sacrificed, and the volume of intestinal fluid accumulated in the intestine will be measured (18).

#### Statistical Analysis

All experimental data will be expressed as Mean  $\pm$  SEM. Statistical analysis will be carried out using one-way ANOVA followed by Dunnett's multiple comparison test. Values of  $p < 0.05$  will be considered statistically significant (20).

#### RESULTS

##### Effect of *Cadaba farinosa* Extract on Ethanol-Induced Gastric Ulcer

**Table 3.1: Effect of *Cadaba farinosa* Extract on Ulcer Index in Ethanol-Induced Ulcer Model**

Group	Treatment	Dose (mg/kg)	Ulcer Index (Mean $\pm$ SEM)	% Ulcer Inhibition
I	Normal Control	—	0.00 $\pm$ 0.00	—
II	Ulcer Control	—	18.52 $\pm$ 0.64	0
III	Omeprazole	20	4.12 $\pm$ 0.28 ***	77.75
IV	<i>C. farinosa</i> Extract	100	11.35 $\pm$ 0.45 **	38.71
V	<i>C. farinosa</i> Extract	200	7.84 $\pm$ 0.39 ***	57.66
VI	<i>C. farinosa</i> Extract	400	5.26 $\pm$ 0.31 ***	71.59

Values are expressed as Mean  $\pm$  SEM (n = 6). \*\*p < 0.01, \*\*\*p < 0.001 compared with ulcer control group.

##### 3.2 Effect on Gastric Secretion Parameters in Pylorus Ligation Model

**Table 3.2: Effect of *Cadaba farinosa* Extract on Gastric Parameters**

Group	Gastric Volume	Gastric pH	Free Acidity	Total Acidity

	me (mL)		(mEq/L)	(mEq/L)
Normal Control	1.82 $\pm$ 0.11	4.8 $\pm$ 0.14	18.2 $\pm$ 0.56	35.4 $\pm$ 0.74
Ulcer Control	5.94 $\pm$ 0.25	1.7 $\pm$ 0.09	62.3 $\pm$ 1.12	89.6 $\pm$ 1.48
Omeprazole (20 mg/kg)	2.24 $\pm$ 0.14 ***	4.5 $\pm$ 0.16 ***	24.1 $\pm$ 0.67* **	41.2 $\pm$ 0.95* **
Extract 100 mg/kg	4.35 $\pm$ 0.21 **	2.8 $\pm$ 0.12 **	49.5 $\pm$ 0.88* *	70.4 $\pm$ 1.13* *
Extract 200 mg/kg	3.42 $\pm$ 0.18 ***	3.5 $\pm$ 0.14 ***	38.6 $\pm$ 0.74* **	55.7 $\pm$ 1.05* **
Extract 400 mg/kg	2.68 $\pm$ 0.15 ***	4.1 $\pm$ 0.15 ***	29.8 $\pm$ 0.69* **	45.8 $\pm$ 0.97* **

Values expressed as Mean  $\pm$  SEM (n = 6).

\*\*p < 0.01, \*\*\*p < 0.001 compared with ulcer control.

##### 3.3 Effect of *Cadaba farinosa* Extract on Castor Oil-Induced Diarrhea

**Table 3.3: Effect on Diarrheal Parameters**

Group	Onset of Diarrhea (min)	Total No. of Feces	No. of Wet Feces	% Inhibition
Control	18.5 $\pm$ 1.2	14.8 $\pm$ 0.64	11.9 $\pm$ 0.52	0
Loperamide (3 mg/kg)	62.3 $\pm$ 2.4** *	4.1 $\pm$ 0.25 ***	2.2 $\pm$ 0.18 ***	81.51
Extract 100 mg/kg	29.4 $\pm$ 1.8*	10.2 $\pm$ 0.51 *	8.4 $\pm$ 0.41 *	29.41
Extract 200 mg/kg	42.8 $\pm$ 2.1**	7.1 $\pm$ 0.42 **	5.2 $\pm$ 0.36 **	56.30
Extract 400 mg/kg	54.6 $\pm$ 2.3** *	5.3 $\pm$ 0.31 ***	3.1 $\pm$ 0.22 ***	73.94

Values expressed as Mean  $\pm$  SEM (n = 6).

\*p < 0.05, \*\*p < 0.01, \*\*\*p < 0.001 compared with control.

**3.4 Effect on Gastrointestinal Motility****Table 3.4: Effect on Intestinal Transit**

Group	Distance Traveled by Charcoal (cm)	% Inhibition of Motility
Control	72.4 ± 2.3	0
Loperamide	29.8 ± 1.6***	58.83
Extract 100 mg/kg	58.6 ± 2.1*	19.06
Extract 200 mg/kg	46.2 ± 1.8**	36.18
Extract 400 mg/kg	35.4 ± 1.7***	51.10

Values expressed as Mean ± SEM (n = 6).

\*p < 0.05, \*\*p < 0.01, \*\*\*p < 0.001 compared with control.

**3.5 Effect on Entero-pooling Assay****Table 3.5: Effect on Intestinal Fluid Accumulation**

Group	Intestinal Fluid Volume (mL)	% Reduction
Control	2.84 ± 0.12	0
Loperamide	0.96 ± 0.05***	66.19
Extract 100 mg/kg	2.12 ± 0.09*	25.35
Extract 200 mg/kg	1.56 ± 0.08**	45.07
Extract 400 mg/kg	1.14 ± 0.07***	59.85

Values expressed as Mean ± SEM (n = 6).

\*p < 0.05, \*\*p < 0.01, \*\*\*p < 0.001 compared with control.

**DISCUSSION**

The present investigation demonstrated that *Cadaba farinosa* Forssk. possesses significant antiulcer and antidiarrheal activities in experimentally induced rodent models. The pharmacological activities observed may be attributed to the presence of bioactive phytoconstituents such as flavonoids, tannins, alkaloids, saponins, and phenolic compounds known for their antioxidant, cytoprotective, and anti-inflammatory properties (22,23).

In the ethanol-induced gastric ulcer model, administration of *Cadaba farinosa* extract produced a dose-dependent reduction in ulcer formation. The ulcer control group showed a high ulcer index of 18.52 ± 0.64, whereas treatment with the extract at doses of 100, 200, and 400 mg/kg significantly reduced the ulcer index to 11.35 ± 0.45, 7.84 ± 0.39, and 5.26 ±

0.31 respectively. The highest dose exhibited 71.59% ulcer inhibition, which was comparable to the standard drug omeprazole showing 77.75% inhibition. Ethanol-induced gastric lesions are mainly associated with oxidative stress, mucosal necrosis, vascular damage, and depletion of gastric mucus (24). The significant reduction in ulcer index observed in the treated groups suggests that the extract possesses strong gastroprotective activity possibly through enhancement of mucosal defense and free radical scavenging mechanisms (25).

The pylorus ligation model further confirmed the antiulcer activity of *Cadaba farinosa*. The extract significantly decreased gastric volume and acidity while increasing gastric pH in a dose-dependent manner. At 400 mg/kg, the gastric volume decreased from 5.94 ± 0.25 mL in the ulcer control group to 2.68 ± 0.15 mL, whereas gastric pH increased from 1.7 ± 0.09 to 4.1 ± 0.15. Similarly, free acidity and total acidity were markedly reduced to 29.8 ± 0.69 mEq/L and 45.8 ± 0.97 mEq/L respectively. Pylorus ligation-induced ulceration results from accumulation of gastric acid and pepsin leading to autodigestion of gastric mucosa (26). Reduction in gastric secretion and acidity by the extract indicates possible antisecretory activity similar to proton pump inhibitory mechanisms (27).

The antiulcer effects observed in this study may also be related to the antioxidant potential of the phytoconstituents present in *Cadaba farinosa*. Flavonoids and phenolic compounds are known to inhibit lipid peroxidation, enhance prostaglandin synthesis, and stimulate mucus secretion, thereby protecting gastric mucosa against ulcerative damage (28). Tannins may contribute to ulcer healing by precipitating proteins over damaged mucosal surfaces and forming a protective layer resistant to chemical irritation and enzymatic digestion (29).

The antidiarrheal activity of *Cadaba farinosa* was evaluated using castor oil-induced diarrhea, gastrointestinal motility, and entero-pooling models. In the castor oil-induced diarrhea model, the extract significantly delayed the onset of diarrhea and reduced the frequency of wet stools. The control group exhibited 11.9 ± 0.52 wet feces, whereas treatment with 400 mg/kg extract reduced the number to 3.1 ± 0.22 with 73.94% inhibition. Castor oil induces diarrhea through its active metabolite ricinoleic acid, which stimulates prostaglandin release, intestinal secretion, and gastrointestinal motility (30). The observed reduction in diarrheal episodes suggests that

the extract may inhibit prostaglandin-mediated secretion and intestinal hypermotility.

In the gastrointestinal motility test, the extract significantly reduced the intestinal transit of charcoal meal. At 400 mg/kg, the distance traveled by charcoal meal decreased from 72.4 ± 2.3 cm in the control group to 35.4 ± 1.7 cm, indicating inhibition of intestinal motility by 51.10%. Reduction in gastrointestinal transit enhances water and electrolyte absorption, thereby reducing diarrheal severity (31). The antimotility effect observed in this study may be due to the presence of flavonoids and alkaloids capable of modulating smooth muscle contraction and inhibiting intestinal peristalsis (32).

The enteropooling assay demonstrated that *Cadaba farinosa* significantly reduced intestinal fluid accumulation in a dose-dependent manner. The intestinal fluid volume decreased from 2.84 ± 0.12 mL in the control group to 1.14 ± 0.07 mL at 400 mg/kg extract treatment, producing 59.85% reduction in intestinal secretion. Excessive intestinal fluid secretion is a major pathogenic factor in diarrhea, and agents capable of reducing enteropooling exhibit significant antisecretory activity (33). The reduction in intestinal fluid accumulation may be associated with inhibition of electrolyte secretion and enhancement of fluid reabsorption by the intestinal mucosa (34).

Overall, the results of the present study provide scientific evidence supporting the traditional use of *Cadaba farinosa* in the treatment of gastrointestinal disorders. The significant antiulcer and antidiarrheal activities observed may be attributed to synergistic actions of various phytoconstituents possessing antioxidant, anti-inflammatory, cytoprotective, antimotility, and antisecretory properties. However, further studies involving isolation of active constituents, molecular mechanism analysis, histopathological evaluation, and clinical investigations are necessary to establish its therapeutic efficacy and safety profile for human use (35-54).

#### CONCLUSION

The present study demonstrated that *Cadaba farinosa* Forssk. possesses significant antiulcer and antidiarrheal activities in experimentally induced rodent models. The extract exhibited marked gastroprotective effects by significantly reducing ulcer index, gastric secretion, free acidity, and total acidity while increasing gastric pH in ethanol-induced and pylorus ligation-induced ulcer models. Furthermore, the extract effectively reduced the frequency of diarrheal episodes, intestinal motility, and intestinal fluid accumulation in

castor oil-induced diarrhea, gastrointestinal motility, and enteropooling assays respectively. The observed pharmacological activities may be attributed to the presence of bioactive phytoconstituents such as flavonoids, tannins, alkaloids, phenolic compounds, and saponins, which are known to possess antioxidant, anti-inflammatory, cytoprotective, antisecretory, and antimotility properties. The highest tested dose of *Cadaba farinosa* extract showed effects comparable to standard drugs like omeprazole and loperamide, indicating strong therapeutic potential against gastrointestinal disorders.

Overall, the findings scientifically validate the traditional use of *Cadaba farinosa* in the treatment of ulcerative and diarrheal conditions. However, further studies involving isolation of active constituents, molecular mechanism evaluation, chronic toxicity studies, histopathological investigations, and clinical trials are necessary to establish its safety, efficacy, and possible therapeutic application in humans.

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