INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: https://www.ijpsjournal.com

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Research Article

Gastroprotective Activity of Ethanolic Leaf Extract of Biophytum Sensitivum (L.) DC. Against Indomethacin-Induced Gastric Ulcer in Male Wistar Rats: Effects on Gastric Secretion, Mucosal Defence, and Oxidative Stress

Reem Muayad Ahmed*, K. Eswar Kumar

Pharmacology Division, A.U. College of Pharmaceutical Sciences, Andhra University, Visakhapatnam – 530003, Andhra Pradesh, India.

ARTICLE INFO

Published: 25 Nov 2026
Keywords:
Biophytum sensitivum, gastroprotection, indomethacin, NSAID-induced gastric injury, mucosal defence, oxidative stress, Wistar rats
DOI:
10.5281/zenodo.24529248

ABSTRACT

Background: Indomethacin, a non-steroidal anti-inflammatory drug (NSAID), can compromise gastric mucosal defence and produce ulceration. Biophytum sensitivum (L.) DC. is traditionally used for gastrointestinal complaints and contains reported phenolic and flavonoid constituents. This study investigated the effects of an ethanolic leaf extract of B. sensitivum (EEBS) in a rat model of indomethacin-induced gastric injury. Methods: Thirty male Wistar rats were allocated to five groups (n = 6): normal control, ulcer control, omeprazole (20 mg/kg), EEBS (200 mg/kg), and EEBS (400 mg/kg). Treatments were administered orally for 14 days. Gastric injury was induced in Groups II–V using indomethacin (30 mg/kg, orally) on Day 14. Ulcer index, gastric volume, pH, free and total acidity, pepsin activity, mucin, gastric-juice protein, tissue nitrite, malondialdehyde (MDA), reduced glutathione (GSH), superoxide dismutase (SOD), catalase (CAT), and histological findings were assessed. Data were analysed using one-way ANOVA followed by Tukey's multiple-comparison test ($p < 0.05$). Results: The ulcer-control group showed marked gastric injury (ulcer index, 2.67 ± 0.52), increased gastric volume and acidity, reduced mucin and antioxidant status, and elevated MDA and nitrite. EEBS reduced the ulcer index to 1.67 ± 0.52 (200 mg/kg) and 1.17 ± 0.41 (400 mg/kg), corresponding to reported inhibition of 37.5% and 56.3%; omeprazole produced 87.5% inhibition. At 400 mg/kg, EEBS also improved gastric volume, pH, acidity, pepsin activity, mucin, protein, nitrite, MDA, GSH, SOD, and CAT relative to ulcer control. Histological descriptions indicated less mucosal damage in the high-dose group.

***Corresponding Author:** Reem Muayad Ahmed
Address: Pharmacology Division, A.U. College of Pharmaceutical Sciences, Andhra University, Visakhapatnam – 530003, Andhra Pradesh, India
Email ✉: reemalshik72@gmail.com

Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Conclusion: Under the conditions of this experiment, EEBS was associated with dose-related attenuation of indomethacin-induced gastric injury and improvement in secretory, mucosal-defence, and oxidative-stress endpoints. These findings warrant confirmation using a chemically characterized extract, quantitative histology, and mechanistic studies.

INTRODUCTION

Peptic ulceration develops when injurious luminal factors—including acid, pepsin, reactive oxygen species, and inflammatory mediators—overcome mucosal protective processes such as mucus and bicarbonate secretion, epithelial integrity, mucosal blood flow, prostaglandin signalling, and endogenous antioxidant activity [1,2]. NSAID exposure is an important cause of gastric mucosal injury and ulcer complications [1].

Indomethacin inhibits cyclooxygenase activity and reduces prostaglandin synthesis, weakening mucus and bicarbonate secretion and other mucosal protective functions. It can also contribute to oxidative injury and inflammatory responses, making it widely used to study experimental NSAID-associated gastric damage [2–5].

Proton-pump inhibitors, including omeprazole, are established treatments for acid-related disorders. Their clinical benefits and potential risks have been discussed in the literature [6]. Because gastric injury reflects both aggressive luminal factors and impaired mucosal defence, agents that affect more than one protective pathway are of experimental interest.

Biophytum sensitivum (L.) DC. (Oxalidaceae), sometimes referred to as “Lajjalu,” is a touch-sensitive herb used in traditional medicine for several conditions, including gastrointestinal complaints. Published phytochemical reports describe flavonoids, biflavonoids, phenolic compounds, tannins, and polysaccharide constituents; extracts have also been investigated for biological activities [7,8]. Such reports provide a rationale for investigation but do not establish the composition of the extract used in this experiment.

Despite traditional use and reported bioactivities, the gastroprotective potential of B. sensitivum in NSAID-associated gastric injury requires further evaluation. The present study examined an ethanolic leaf extract at 200 and 400 mg/kg in male Wistar rats exposed to indomethacin, using macroscopic injury, gastric secretory and mucosal-defence measurements, oxidative-stress indices, and histological observations. Omeprazole was included as a reference treatment.

MATERIALS AND METHODS

2.1 Chemicals and plant extract

The ethanolic leaf extract of Biophytum sensitivum (EEBS) was obtained from Kshipra Biotech Private Limited, Indore, Madhya Pradesh, India. Indomethacin and omeprazole were pharmaceutical grade, and other reagents were analytical grade and obtained from Sri GhaMa Enterprises, India. The dried extract was triturated with cmc .

2.2 Experimental animals

Adult male Wistar rats (8 weeks old; 180–220 g; total n = 30) were obtained from Vyas Labs, Hyderabad, and housed in the CPCSEA-registered animal facility of A.U. College of Pharmaceutical Sciences. Housing conditions were 22–26 °C, 40–70% relative humidity, and a 12-h light/dark cycle, with standard pellet diet and water ad libitum. The study was reported to follow CPCSEA guidelines and to have IAEC approval (CPCSEA Reg. No. 516/01/A/CPCSEA.

2.3 Dose selection

Dose selection was based on a previously reported acute oral toxicity assessment of *Biophytum sensitivum* extract. The dose selected was 200 and 400 mg/kg .OECD Test Guideline 423 describes an acute oral toxicity testing procedure and is not, by itself, evidence of the toxicity profile of this extract.

2.4 Experimental design

Animals were allocated to five groups (six rats per group) and received oral treatment once daily for 14 days (Table 1). On Day 14, Groups II–V received indomethacin (30 mg/kg, orally) to induce gastric injury. Animals were monitored for behaviour, food and water intake, and signs of distress. The randomization procedure, blinding procedures, fasting status and duration , timing of the last treatment relative to indomethacin,

and exact timing of body-weight measurements are available from the study record .

Table 1: Experimental Groups and Treatment Schedule

Group	Treatment (p.o., days 1–14)	Ulcer induction (day 14)	n
I – Normal control	Distilled water (vehicle)	—	6
II – Ulcer control	Distilled water (vehicle)	Indomethacin 30 mg/kg, p.o.	6
III – Standard	Omeprazole 20 mg/kg	Indomethacin 30 mg/kg, p.o.	6
IV – EEBS low dose	EEBS 200 mg/kg	Indomethacin 30 mg/kg, p.o.	6
V – EEBS high dose	EEBS 400 mg/kg	Indomethacin 30 mg/kg, p.o.	6

2.5 Collection of gastric juice and tissue

At the end of the experiment, animals were euthanized according to the approved protocol. The stomachs were removed, gastric contents were collected into pre-weighed tubes, and samples were centrifuged at 3000 rpm for 10 min. Stomachs were opened along the greater curvature, rinsed with normal saline, and spread for lesion scoring .

2.6 Ulcer index and percentage inhibition

The glandular mucosa was inspected under magnification and lesions were scored using the cited method [10]: 0, normal; 0.5, red coloration; 1, haemorrhagic streaks; 2, ulcers smaller than 3 mm; 3, ulcers 3–5 mm; and 4, perforated ulcers. The ulcer index was calculated as the mean lesion score per animal.

% Inhibition = [(UI*ulcer control* − UI*treated*) / UI*ulcer control*] × 100

2.7 Gastric secretory parameters

Gastric-fluid volume was recorded from the supernatant, and pH was measured using a calibrated digital pH meter. Free and total acidity were determined by titrating 1 mL gastric juice diluted with 9 mL distilled water against 0.01 N NaOH using Töpfer's reagent and phenolphthalein, respectively, and expressed as mEq/L [11]. Pepsin activity was assessed using the haemoglobin method, with the TCA-soluble product measured at 280 nm and expressed as µg tyrosine released/mL/min [12].

2.8 Mucosal defence parameters

Gastric wall mucus was quantified by Alcian-blue binding with absorbance measured at 605 nm [13]. Total protein in gastric juice was determined using the Lowry method at 750 nm [14]. Tissue nitrite was measured using Griess reagent at 540 nm [15].

2.9 Oxidative stress markers

The glandular stomach was homogenized (10% w/v) in ice-cold 0.1 M phosphate buffer (pH 7.4) and centrifuged at 10,000 rpm for 15 min at 4 °C. MDA was assessed using the thiobarbituric-acid-reactive method at 532 nm [16]; GSH with DTNB at 412 nm [17]; SOD by inhibition of adrenaline auto-oxidation at 480 nm [18]; and CAT by H_2O_2 decomposition at 240 nm [19]. .

2.10 Histopathology

Gastric tissues were fixed in 10% neutral-buffered formalin, processed using graded alcohol and xylene, embedded in paraffin, sectioned at 5 µm, and stained with haematoxylin and eosin. Sections were examined for epithelial integrity, erosion, oedema, necrosis, and inflammatory-cell infiltration .

2.11 Statistical analysis

Data are presented as mean ± SD (n = 6 per group). Group comparisons were performed using one-way ANOVA followed by Tukey's multiple-comparison test in GraphPad Prism 9.4.1, with $p < 0.05$ considered statistically significant. The analysis assumptions, exact p-values where requested, significance-symbol comparisons, and all summary statistics should be verified using animal-level data.

RESULTS

No mortality or overt toxicity occurred in any group during the 14-day treatment.

3.1 Body weight

Body weights were similar among groups at baseline and before ulcer induction. Following indomethacin administration, the ulcer-control group showed a lower reported body weight, while the EEBS- and omeprazole-treated groups showed less reduction (Table 2).

Table 2: Effect of EEBS on Body Weight (G)

Group	Day 0	Day 14 (pre-induction)	Day 14 (post-induction)
Normal control	165.83 ± 6.7	177.33 ± 6.0	179.17 ± 6.2
Ulcer control	165.17 ± 8.3	176.83 ± 9.5	158.67 ± 5.4$^{\#}$
Omeprazole 20 mg/kg	163.67 ± 5.7	177.33 ± 5.6	177.50 ± 5.0*
EEBS 200 mg/kg	166.50 ± 5.9	175.67 ± 5.0	168.83 ± 5.2*
EEBS 400 mg/kg	164.33 ± 8.5	175.00 ± 9.5	175.50 ± 4.7*

Values are mean ± SD (n = 6). $^{\#}p < 0.05$ vs normal control; $^{*}p < 0.05$ vs ulcer control (one-way ANOVA followed by Tukey's test).

3.2 Ulcer index and macroscopic appearance

Indomethacin was associated with haemorrhagic lesions in the glandular mucosa and a mean ulcer index of 2.67 ± 0.52, while the normal-control group had no observed lesions. EEBS treatment was associated with lower ulcer indices at both doses. The reported inhibition was 37.5% at 200 mg/kg and 56.3% at 400 mg/kg; omeprazole produced 87.5% inhibition (Table 3).

Table 3: Effect of EEBS on Ulcer Index and Ulcer Inhibition

Group	Ulcer index	Ulcer inhibition (%)
Normal control	0.00 ± 0.00	—
Ulcer control	2.67 ± 0.52$^{\#}$	—
Omeprazole 20 mg/kg	0.33 ± 0.52*	87.50 ± 19.36
EEBS 200 mg/kg	1.67 ± 0.52*	37.50 ± 19.36
EEBS 400 mg/kg	1.17 ± 0.41*	56.25 ± 15.31

Values are mean ± SD (n = 6). $^{\#}p < 0.05$ vs normal control; $^{*}p < 0.05$ vs ulcer control (one-way ANOVA followed by Tukey's test).

3.3 Gastric secretory parameters

The ulcer-control group showed increased gastric volume and acidity and lower pH than the normal-control group (Table 4). EEBS treatment was associated with improvement in these parameters. At 400 mg/kg, gastric volume, total acidity, and pepsin activity were lower than in ulcer control. For several secretory endpoints, the response was less pronounced than with omeprazole.

Table 4: Effect of EEBS on Gastric Secretory Parameters

Group	Volume (mL)	pH	Free acidity (mEq/L)	Total acidity (mEq/L)	Pepsin (µg tyrosine/mL/min)
Normal control	1.49 ± 0.03	3.88 ± 0.12	11.25 ± 0.19	31.25 ± 0.19	46.00 ± 1.41
Ulcer control	4.97 ± 0.29$^{\#}$	1.70 ± 0.14$^{\#}$	21.63 ± 0.16$^{\#}$	75.05 ± 0.33$^{\#}$	86.00 ± 2.83$^{\#}$
Omeprazole 20 mg/kg	1.90 ± 0.04*	3.32 ± 0.12*	13.95 ± 0.18*	33.25 ± 0.19*	51.17 ± 1.72*
EEBS 200 mg/kg	3.43 ± 0.25*	2.70 ± 0.14*	15.52 ± 0.19*	44.65 ± 0.22*	70.00 ± 2.37*
EEBS 400 mg/kg	2.63 ± 0.19*	3.17 ± 0.15*	14.53 ± 0.15*	41.68 ± 0.15*	55.83 ± 1.17*

Values are mean ± SD (n = 6). $^{\#}p < 0.05$ vs normal control; $^{*}p < 0.05$ vs ulcer control (one-way ANOVA followed by Tukey's test).

3.4 Mucosal defence parameters

Compared with normal control, ulcer control showed lower mucin and gastric-juice total protein and higher

tissue nitrite (Table 5). EEBS, particularly at 400 mg/kg, was associated with higher mucin and protein values and lower nitrite than ulcer control. These findings are consistent with improved mucosal status but do not, by themselves, identify a specific molecular mechanism.

Table 5: Effect of EEBS on Mucin, Total Protein and Total Nitrite

Group	Mucin (µg/g wet tissue)	Total protein (mg/dL)	Total nitrite (µmol/g tissue)
Normal control	248.00 ± 5.09	6.08 ± 0.08	1.20 ± 0.07
Ulcer control	114.33 ± 3.01$^{\#}$	3.50 ± 0.07$^{\#}$	3.85 ± 0.19$^{\#}$
Omeprazole 20 mg/kg	234.17 ± 2.86*	6.03 ± 0.11*	1.50 ± 0.09*
EEBS 200 mg/kg	176.50 ± 4.93*	4.54 ± 0.07*	2.50 ± 0.07*
EEBS 400 mg/kg	210.17 ± 5.85*	5.44 ± 0.07*	1.72 ± 0.09*

Values are mean ± SD (n = 6). $^{\#}p < 0.05$ vs normal control; $^{*}p < 0.05$ vs ulcer control (one-way ANOVA followed by Tukey's test).

3.5 Oxidative stress markers

Ulcer control showed higher MDA and lower GSH, SOD, and CAT than normal control (Table 6). EEBS treatment was associated with improvement in all four measures, generally greater at 400 mg/kg. This indicates an association with improved tissue oxidative-stress status; direct antioxidant mechanisms in vivo were not established by these measurements alone.

Table 6: Effect of EEBS on Gastric Oxidative Stress Markers

Group	MDA (nmol/mg tissue)	GSH (µM/g tissue)	SOD (U/mg protein)	CAT (U/mg protein)
Normal control	1.84 ± 0.07	8.17 ± 0.09	12.61 ± 0.24	45.56 ± 0.89
Ulcer control	4.98 ± 0.09$^{\#}$	4.10 ± 0.09$^{\#}$	6.55 ± 0.22$^{\#}$	20.43 ± 0.59$^{\#}$
Omeprazole 20 mg/kg	2.19 ± 0.05*	7.48 ± 0.09*	11.46 ± 0.27*	41.78 ± 0.84*
EEBS 200 mg/kg	3.29 ± 0.05*	6.04 ± 0.08*	8.79 ± 0.32*	30.12 ± 0.28*
EEBS 400 mg/kg	2.53 ± 0.05*	7.09 ± 0.07*	10.47 ± 0.27*	38.45 ± 0.77*

Values are mean ± SD (n = 6). $^{\#}p < 0.05$ vs normal control; $^{*}p < 0.05$ vs ulcer control (one-way ANOVA followed by Tukey's test).

3.6 Histopathology

Normal-control sections were described as showing intact surface epithelium and regularly arranged gastric pits and glands (Figure 1). Ulcer-control sections showed mucosal erosion, disrupted glands, and inflammatory infiltration. Omeprazole-treated sections showed near-normal mucosa with mild infiltration. EEBS 200 mg/kg was described as partial protection, whereas EEBS 400 mg/kg showed less epithelial damage and inflammation. Support these observations with original images and, preferably, blinded quantitative scoring.

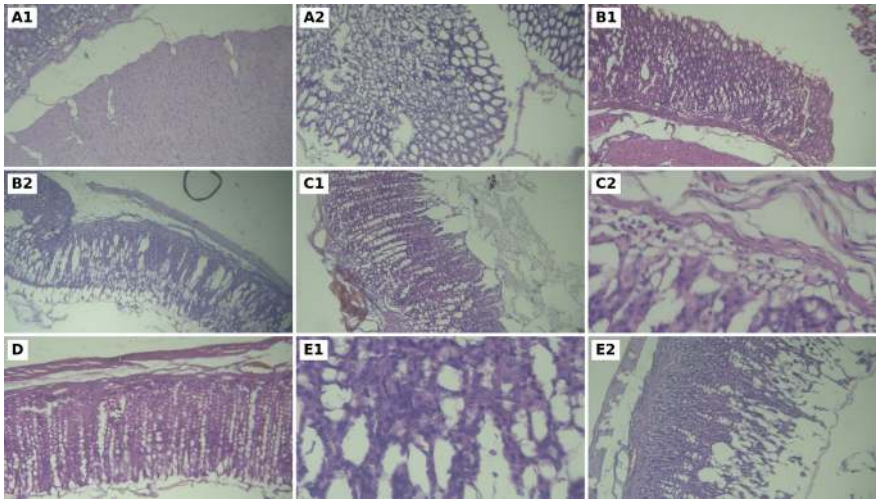

Figure 1. Histopathological changes in rat gastric tissue (H&E staining). (A) Normal control: normal gastric mucosa with normal crypts, lamina propria and muscularis mucosae (400×). (B) Ulcer control (indomethacin): mucosal erosion with chronic inflammatory infiltrate in the lamina propria (200×). (C) Omeprazole 20 mg/kg: normal gastric mucosa with mild inflammatory-cell infiltration (400×). (D) EEBS 200 mg/kg: mild protective response (200×). (E) EEBS 400 mg/kg: very little epithelial destruction, almost similar to the omeprazole group (200×/400×). Panels with the same letter (A1, A2, etc.) are separate images from the same group.

DISCUSSION

In this experiment, 14-day oral pretreatment with EEBS was associated with attenuation of indomethacin-induced gastric injury. The 400 mg/kg dose produced a lower ulcer index and improvements across several secretory, mucosal-defence, and oxidative-stress measurements compared with ulcer control. With six animals per group and two extract doses, findings should be considered preliminary and limited to this model.

Indomethacin-induced gastric injury is linked to cyclooxygenase inhibition and reduced prostaglandin-dependent mucosal protection, alongside inflammatory and oxidative processes [2–5]. The ulcer-control findings—including higher ulcer index, increased gastric-fluid volume and acidity, lower pH, and altered mucosal and oxidative-stress measurements—were consistent with gastric injury. Weight changes may reflect stress or reduced intake, but additional measurements would be needed to interpret them.

Antisecretory findings. EEBS was associated with lower gastric volume, free and total acidity, and pepsin activity, together with higher gastric pH. These findings are consistent with reduced gastric secretory burden. Proton-pump inhibition or histamine modulation was not directly measured and should be presented only as a hypothesis for future investigation.

Mucosal-defence findings. EEBS was associated with higher mucin and gastric-juice protein and lower tissue nitrite relative to ulcer control. These findings are compatible with improved mucosal condition.

Oxidative-stress findings. Indomethacin was associated with higher MDA and lower GSH, SOD, and CAT, while EEBS improved these measures. This supports an association with improved tissue oxidative-stress status.

Histological observations. Qualitative descriptions suggest less mucosal injury in the high-dose EEBS group than in ulcer control.

Comparison with omeprazole. Omeprazole produced greater ulcer inhibition than either EEBS dose in the reported results. EEBS nonetheless improved several mucosal and oxidative-stress endpoints. Claims of a distinct multi-target mechanism should remain provisional because active constituents and pathway-specific target engagement were not assessed.

Limitations. The extract was commercially obtained and not chemically fingerprinted or standardized; botanical authentication and batch characterization should be documented. Only two doses, one injury

model, and six animals per group were studied. Prostaglandin E_2, myeloperoxidase, cytokines, mucosal blood flow, and pathway-specific targets were not measured. Histology was qualitative, and original images and blinding/scoring details are needed. Statistical outputs and ulcer-inhibition calculations must be checked against animal-level data. These limitations restrict mechanistic interpretation and generalization. Future studies should include extract fingerprinting, dose-ranging, blinded histological scoring, and targeted prostaglandin/inflammatory assays.

CONCLUSION

In this rat model, oral EEBS at 200 and 400 mg/kg was associated with dose-related attenuation of indomethacin-induced gastric injury. Treatment improved the ulcer index and several secretory, mucosal-defence, and oxidative-stress endpoints, with the 400 mg/kg dose generally showing greater improvement than 200 mg/kg. These preliminary findings do not establish clinical efficacy, safety, or specific molecular mechanisms.

Declarations

Ethics approval: The study is reported to have been approved by the Institutional Animal Ethics Committee, A.U. College of Pharmaceutical Sciences, Andhra University (CPCSEA Reg. No. 516/01/A/CPCSEA).

Author contributions:

Reem Muayad Ahmed performed the experiments, analysed the data and drafted the manuscript. K.Eswar Kumar conceived and supervised the study and revised the manuscript. Both authors approved the final version.

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HOW TO CITE: Reem Muayad Ahmed, K. Eswar Kumar, Gastroprotective Activity of Ethanolic Leaf Extract of Biophytum Sensitivum (L.) DC. Against Indomethacin-Induced Gastric Ulcer in Male Wistar Rats: Effects on Gastric Secretion, Mucosal Defence, and Oxidative Stress, Int. J. of Pharm. Sci., 2026, Vol 4, Issue 11, 4776-4783. https://doi.org/10.5281/zenodo.24529248