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Research Article

Protective Action of Roots of *Abroma augusta* in Gastric Ulcer Prevention Induced by Ethanol in Rats

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ABSTRACT

Ulcers result from imbalanced factors that attack the stomach lining (pepsin, acid, NSAIDs) and those that protect it and free radical-associated damage. This research aims to understand how the roots of *Abroma augusta* can mitigate ethanol-induced gastric ulcers in rats. Omeprazole (20 mg/kg) and root extract (250 and 500 mg/kg*/p.o*) were administered for 21 days. Before ethanol administration (1-mL/200 g), the animals were fasted for 24 hours. Parameters like ulcer index, %inhibition of ulceration, plasma anti-oxidant levels and histopathology were assessed. The present study revealed that the 500 mg/kg root extract possesses a significant effect, which may be the presence of tannins, saponins, and alkaloids. The extract effectively countered ethanol-induced lesion formation and maintained plasma superoxide dismutase ($p < 0.01$), catalase ($p < 0.05$), and lipid peroxidase ($p < 0.01$) levels. At a dosage of 500 mg/kg, the root extract exhibited notable effectiveness in safeguarding the histological integrity of the gastric mucosa. This study has provided documentary evidence for the antiulcer property of *A. augusta* for its activity.

Introduction

Gastric ulcers remain a prevalent concern for numerous individuals globally in the 21st century, prompting some to label them as the modern-day "plague."[1] Ulcers result from imbalanced factors that attack the stomach lining (pepsin, acid, NSAIDs) and those that protect it, and free radical-associated damage. Disruption of the mucosal layer due to factors like increased acid secretion, hindered bicarbonate buffering, and mucosal damage leads to ulcers. Medication for gastric ulcers targets antagonizing aggressive factors or enhancing defensive factors. The ultimate objectives of managing gastric ulcers include symptom relief, ulcer healing, and prevention. However, no drug can fulfil all the objectives of therapy. Helicobacter pylori presence is linked to 70% of gastric ulcers.[2] Gastric ulcers is one of the most common problems nowadays. Excessive production of hydrochloric acid from the stomach's parietal cells, facilitated by a proton pump, defines the abnormal condition known as hyperchlorhydria. Several therapeutic approaches can help address gastric ulcers, including H2 blockers, cholinergic antagonists, proton pump inhibitors (PPI), and antacids. However, these medications can have adverse effects, including allergic reactions, arrhythmia, gynecomastia, etc.[3] Various aggressive factors like ethanol, stress, smoking, nutrient deficiency, and NSAIDs contribute to gastric ulcers by damaging the stomach's protective lining. The gastric mucosa counters this with the release of prostaglandin, bicarbonate, antioxidants, and nitric oxide. Nitric oxide aids ulcer healing by enhancing blood flow

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through vessel dilation, fostering gastric angiogenesis, promoting mucosal cell growth, and aiding granulation tissue formation at the ulcer base.[1]

Factors like NSAIDs, alcohol intake, and *Helicobacter pylori* infection frequently trigger the destruction of the stomach's mucosal lining, leading to gastric ulcers.[4] The development of stomach ulcers arises from an imbalance between factors that attack the stomach lining and those that protect it, particularly evident at the luminal surface of epithelial cells.[5] Alcohol harms the stomach through various mechanisms, including elevated gastric secretion, the liberation of pro-inflammatory cytokines, and oxidative stress.[6-8]

Ethanol (Etho) triggers various pathological processes linked to ulcer formation, causing damage to mucosal capillaries, congestion, heightened blood vessel permeability, and thrombosis in sub-epithelial microvasculature. These events lead to the liberation of pro-inflammatory cytokines, including TNF.[9,10] The factors outlined are crucial contributors to the escalated inflammatory reaction triggered by ethanol, the resultant oxidative stress-induced damage, tissue necrosis, apoptosis of gastric mucosal cells, and direct harm to the mucosal layer.[11] Although there exists a wide array of medications for treating gastric ulcers, including proton pump inhibitors, anti-histamines, and antacids, many of these drugs come with adverse side effects like gynecomastia and arrhythmias.[12,13] Due to several side effects, there is a growing interest in finding gastric ulcer medications that are both safe and effective, aiming for maximum benefits with minimal risks. Recent research has highlighted the potential effects of several naturally occurring plant chemicals in the treatment of stomach ulcers.[14]

Abroma augusta, also known as ulatkambal in Hindi and devil's cotton in English, is a hairy shrub that grows wild in South Africa, tropical Asia, and Australia. *A. augusta* is one of the most effective plants against type –II diabetes mellitus. *A. augusta* has anti-inflammatory, wound healing, hypolipidemic, antimicrobial, and antioxidant properties. However, its ability to combat gynecological and urinary disorders remains one of its key physiological benefits. It possesses the ability to manage menstrual irregularities, minimize uterine leucorrhoea, and stimulate uterine contractions. This makes it valuable in treating conditions like amenorrhea, dysmenorrhea, and other menstrual disorders.[15]

While there is no existing research on the plant's anti-ulcer effects, this study aimed to establish the pharmacological foundation supporting its traditional use in treating gastric ulcers. Based on this, an attempt has been made to investigate the protective action of roots of *A. augusta* in gastric ulcer prevention induced by ethanol in rats with their phytoconstituents.

Materials And Methods

Plant Collection and Authentication

The collection site for roots of *A. augusta* was Amlachati Bhesojo Udyan, Shirshi, West Bengal, 721507. They were authenticated by Dr. Tapan Seal, scientist, Acharya Jagadish Chandra Bose Botanic Garden, Howrah, INDIA. Sample No. KCP/sample 01/M.Pharm/20022.

Preparation of Extract

The roots of *A. augusta* were thoroughly cleaned with fresh water. The roots were dried properly under shade for 20 days. Dried roots were coarsely powdered and utilized for methanolic extract. The soxhlet extractor technique carried out the methanolic extraction. Coarsely powdered roots were soxhlet with methanol for two cycles in 6 hours (Temperature-30°C). Following filtration through Whatman filter paper, the extract was accurately weighed and stored in sealed containers to facilitate upcoming phytochemical and pharmacological examinations.

Phytochemical Screening

Concentrated extracts were subjected to an initial screening process to detect phytoconstituents, including tannins, saponins, flavonoids, alkaloids, and terpenoids. The identification of these constituents was carried out using established methods outlined in standard tests.[16]

Treatment of Animals

The study employed female wistar albino rats (150–250 gm). These rats were sourced from Sri Venkateshwara Enterprises, Bangalore. Standard feed and water were available to the animals at all times, and they were allowed to acclimate for 15 days before the start of the study, following the ethical guidelines set by the CPCSEA.

Animal experiments were executed following the Experimental Protocols that the IAEC approved.

IAEC Registration number: KCP/IAEC/11/22-23/08/22/12/22.

Ethanol-induced gastric lesions method[1,17,18]

Each of the five groups in the study comprised six animals (N = 6). They were administered normal saline (NS) to the normal and disease control group, extract of roots of *A. augusta* (ERAA) to the low dose group and high dose group, and omeprazole (Ome) standard drug group for 21 days. Following that, the animals were kept fasting for 24 hours before being given ethanol (Etho) orally to induce gastric ulcers.

Below are the specific treatment groups and experimental protocol:

Group 1: The normal control group received only NS (2 mL/kg, *p.o.*).

Group II: The disease control group received NS (2 mL/kg, *p.o.*) + Etho (1-mL/200 g, *p.o.*).

Group III: The standard drug group received Ome

(20 mg/kg/*p.o.*) + Etho (1-mL/200 g, *p.o.*)
Group IV: The low dose test drug group received ERAA (250 mg/kg/*p.o.*) + Etho (1-mL/200 g, *p.o.*).
Group V: The high dose test drug group received ERAA (500 mg/kg/*p.o.*) + Etho (1-mL/200g, *p.o.*).
In this protocol, all animals were euthanized one hour after ethanol administration using a high dose of intraperitoneally administered phenobarbitone sodium. Following euthanasia, the stomachs were promptly extracted for parameter assessment.
Plasma LPO, CAT, and SOD were assayed by following the methods of Karuna R *et al*.[19] A surgical opening was made along the greater curvature of the stomach, washed with saline solution to clear away blood clots and gastric materials, and scrutinized with a 10X magnifier to gauge ulcer development.
Counting and scoring are conducted according to the method described by Abebaw M *et al*. to quantify the number and severity of ulcers.[18]
Gastric tissue samples were placed in 10% formalin for 24 hours to undergo fixation. Sections of stomach tissues were then histopathologically examined to explore the ulcerogenic or anti-ulcerogenic effects of *A. augusta*. These tissues underwent fixation in 10% formalin, processing with a tissue processor, embedding in paraffin blocks, and slicing into approximately 5 µm thick sections using a rotary microtome. H&E staining was applied to the sections following standard procedures. Microscopic analysis of the slides was carried out to identify pathomorphological changes such as edema, hemorrhage, congestion, and erosions, with severity assessed using an arbitrary scale.[20]

Statistical Analysis

Six rats per group (n = 6) are used to determine the mean ± SEM for each set of results. To conduct statistical analysis, Graph Pad Prism (version 10) was used. Using one-way ANOVA and Tukey's multiple comparisons test group disparities were analyzed by comparing each group to the disease control group. The $p < 0.05$ was used to indicate statistical significance.

Result and Discussion

Phytochemical Screening

The phytochemical study, it has evaluated the presence of tannins, alkaloids, saponins, and terpenoids in the absence of flavonoid root extract of *A. augusta* (Table 1).

Pharmacological Studies

Effect of test drug and standard drug on plasma antioxidants enzyme

The use of ERAA at doses of 250 and 500 mg/kg, alongside the standard drug, effectively reversed the ethanol-induced reduction in superoxide dismutase (SOD) and catalase (CAT) activities, while simultaneously inhibiting the increase in lipid peroxidase (LPO) activity in the stomach of the studied animals. Particularly noteworthy is the superior efficacy of the 500 mg/kg dose of ERAA compared to the 250 mg/kg dose. (Table 2).
The protected normal catalase activity in plasma evidenced the antioxidant effects of the ERAA (500 mg/kg). ERAA (250 mg/kg) and ome (20 mg/kg) gave a similar type of effect. ERAA (500 mg/kg) protected normal SOD level in plasma (Fig. 1). The enzyme CAT is pivotal in the antioxidant defense system, responsible for converting hydrogen peroxide (H_2O_2) into oxygen and water (Fig. 2). The High dose of ERAA also exhibited protection against LPO (Fig. 3). It was shown that ERAA could effectively

Table 1: Results of preliminary phytochemical screening of *A. augusta*

S. No	*Constituents*	*Observation*
1	Alkaloids	+
2	Flavonoids	-
3	Terpenoids	+
4	Saponins	+
5	Tannins	+

(-) indicates the absence of compound (+) indicates the presence of compound

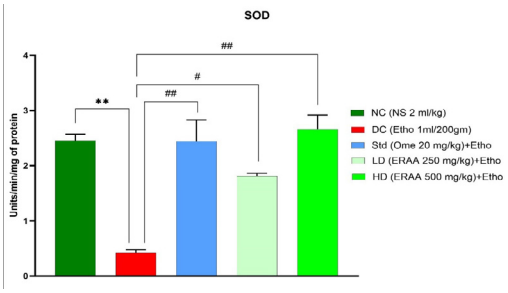

Fig. 1: This figure shows effect of methanolic extract of *A. augusta* on SOD enzyme. Values are expressed as Mean ± SEM (n = 6), $p^{**} < 0.01$ compared with normal control, $p^{\#\#} < 0.05$, $p^{\#\#} < 0.01$ compared with Disease control. NC, Normal Control, DC, Disease Control, Std, Standard Control, LD, Low Dose, HD, High dose, NS, normal saline, etho, ethanol, ome, omeprazole, ERAA, extract of roots of *A. augusta* (One way ANOVA followed by Tukey's multiple comparison test)

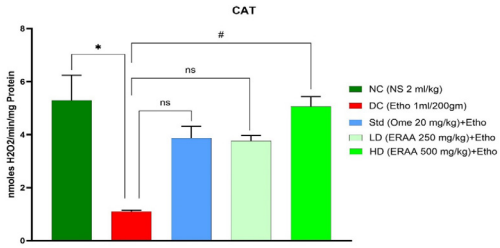

Fig. 2: This figure shows effect of Methanolic extract of *A. augusta* on CAT enzyme. Values are expressed as Mean ± SEM (n = 6), $p^{*} < 0.05$ compared with Normal control, $p\# < 0.05$ compared with Disease control. NC, Normal Control, DC, Disease Control, Std, Standard Control, LD, Low Dose, HD, High dose, NS, normal saline, Etho, ethanol, ome, Omeprazole, ERAA, extract of roots of *A. augusta* (One way ANOVA followed by Tukey's multiple comparisons test)

Table 2: Effect of methanolic extract of *A. augusta* on antioxidant enzymes

Groups	*SOD (Units/min/mg of protein)*	*CAT (nmoles H_2O_2/min/mg Protein)*	*LPO (nmoles of MDA/g protein)*
Normal control (NS 2 mL/kg, *p.o*)	2.45 ± 0.11	5.29 ± 0.94	0.27 ± 0.03
Disease control (Etho 1 mL/ 200 g, *p.o.*)	0.42 ± 0.05**	1.10 ± 0.05*	1.06 ± 0.02**
Standard control (Ome 20 mg/ kg, p.o.)	2.44 ± 0.39##	3.86 ± 0.45	0.55 ± 0.08##
Low dose (ERAA 250 mg/kg p.o)	1.81 ± 0.04#	3.76 ± 0.20	0.80 ± 0.08**
High dose (ERAA 500 mg/kg p.o)	2.65 ± 0.26##	5.05 ± 0.37#	0.54 ± 0.04##

Values are expressed as Mean ± SEM (n = 6), *p*< 0.05, p**< 0.01* compared with Normal control, *p# < 0.05, p## < 0.01* compared with Disease control. NS, normal saline (2 mL/kg, *p.o*) etho, ethanol, ome, omeprazole, ERAA, extract of roots of *A. augusta* (One way ANOVA followed by Tukey's multiple comparisons test).

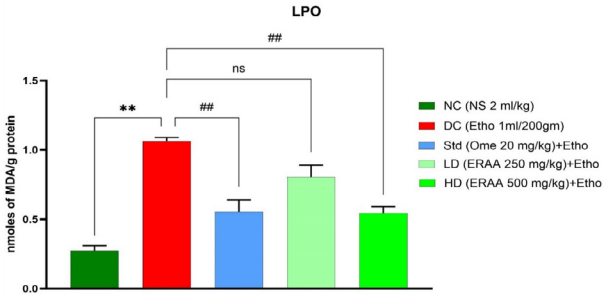

Fig. 3: This figure shows effect of Methanolic extract of A. augusta on LPO enzyme. Values are expressed as Mean ± SEM (n = 6), *p*** < 0.01 compared with Normal control, *p*# < 0.05, *p*## < 0.01 compared with Disease control. Normal Control (NC), Disease Control (DC), Std, Standard Control, Low Dose (LD), High dose (HD), normal saline (NS), Etho, Ethanol, Ome, Omeprazole, ERAA, Extract of roots of *A. augusta* (One way ANOVA followed by Tukey's multiple comparison test)

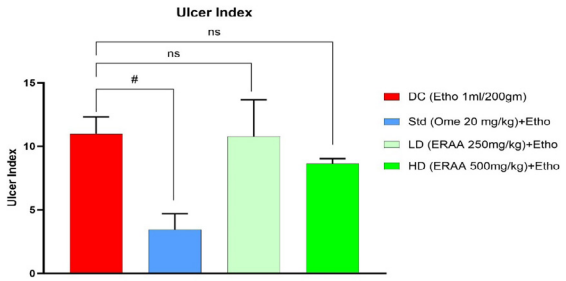

Fig. 4: Effect of Methanolic extract of A. augusta on Ulcer Index. Values are expressed as Mean ± SEM (n = 6), *p*# < 0.5 compared with Disease control group. Normal Control (NC), Disease Control (DC), Std, Standard Control, Low Dose (LD), High dose (HD), normal saline (NS), Etho, Ethanol, Ome, Omeprazole, ERAA, Extract of roots of *A. augusta* (One way ANOVA followed by Tukey's multiple comparisons test).

defend cell membranes against damage inflicted by reactive species (RS). Ethanol's aggressive effects on gastric cells, including damage and cell death, are linked to the abnormal rise in RS.[1]

This study investigated the protective action of roots of *A. augusta* in gastric ulcer prevention induced by ethanol in rats, compared to ome, a drug whose ulcer-healing properties comprehensively studies have been carried out, and to an ulcer control group. Both the ERAA (500 mg/kg) and ome (20 mg/kg) were observed to offer protection to the gastric mucosa. However, the protective effect of ome (20 mg/kg) was more pronounced than that of the high-dose group. In comparison to the ulcer control group, both ome and ERAA (500 mg/kg) were found to have protective effects. This suggests that ERAA produced a dose-dependent anti-ulcer effect. ERAA at a dose of 500 mg/kg exhibited significant protection of the gastric mucosa against Etho challenge, evident from reduced ulcer index values compared to the normal control group (Fig. 4). This underscores its potent cytoprotective effect and significant inhibition of gastric ulcer formation. In addition, the %inhibition of ulceration of ome and ERAA 500 mg/kg was 68.67 and 21.31, respectively (Fig. 5). The ERAA is capable of dose-dependent inhibiting of lesion formation induced by etho (Fig. 6).

The section studied shows a normal esophagus lined by keratinizing squamous epithelium and a stomach lined by normal mucosa. No ulceration or inflammation is seen in the normal control group animal (Fig. 7a). Etho administration at a dosage of 1-mL/200 g consistently caused microscopic damage in comparison to the control group animals. In the disease control group animal shows normal oesophageal mucosa. Gastric mucosa shows necrosis of the upper half to 2/3 of the mucosal thickness in the ulcerated areas along with hemorrhage and no significant inflammation. Adjacent mucosa shows mild laminal inflammation (Fig. 7b). Moreover, the animals that received omeprazole and ERAA at 20 and 500 mg/kg were able to substantially prevent the damage induced by ethanol (Fig. 7c and 7d). Animal of the standard group shows normal oesophagus. The stomach shows laminal infiltration by scattered neutrophils at places (Fig. 7c) and animals of the high-dose group show normal esophagus

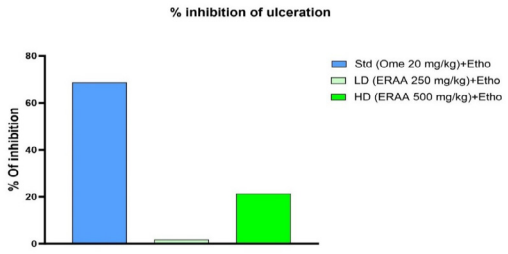

Fig. 5: Bar chart of %inhibition of ulceration in ethanol induced

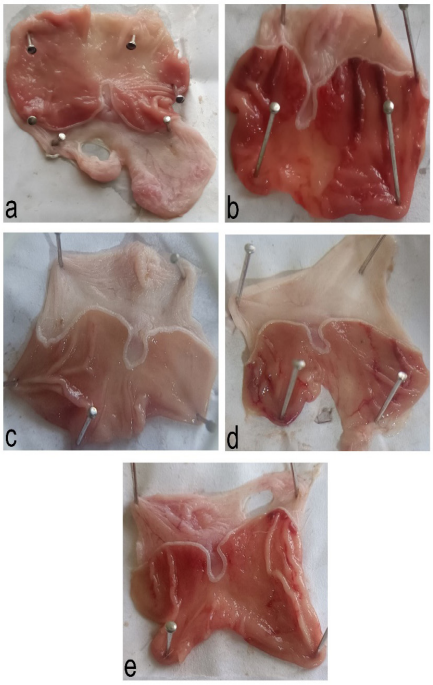

Fig. 6: Effect of methanolic extract of *A. augusta* on Gross appearances of stomach. Stomach of Normal Control group (a), Stomach of Disease control group Gastric lesions induced by Etho (1-mL/200 g) (b), Stomach of Standard group. Absence of gastric lesions in Ome (20 mg/kg) (c), Stomach of Low dose group. Fraction inhibition in gastric lesions at 250 mg/kg of ERAA (d), Stomach of High dose group Inhibition in gastric lesions at 500 mg/kg of ERAA (e). All drugs were administrated by oral way.

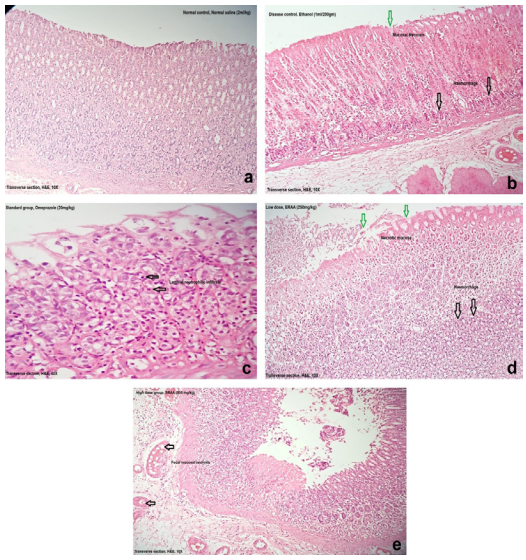

Fig. 7: Effect of Methanolic extract of *A. augusta* on histopathological evaluation of stomach. The stomach of the Normal Control group (a), the Stomach of the Disease control group with mucosal necrosis and haemorrhage induced by Etho (1 mL/200 g) (b), Stomach of the Standard group. Mild laminal neutrophilic infiltrate in Ome (20 mg/kg) (c), Stomach of Low dose group. focal mucosal necrosis at 250 mg/kg of ERAA (d), Stomach of High dose group necrotic mucosa, and haemorrhage at 500 mg/kg of ERAA (e). All drugs were administrated by oral way.

and gastric mucosa with one small ulcer showing superficial necrosis of the mucosa and infiltration by neutrophils. The rest of the mucosa is unremarkable except for congested vessels at places (Fig. 7e). However animal in low dose group shows mild inhibition of ulcers. The section shows necrotic gastric mucosa below the gastro-oesophageal junction, involving the upper 1/2 to 2/3 of the mucosal thickness, along with hemorrhage. No significant inflammation was noted (Fig. 7d).

Gastric ulcers arise from damage to the protective gastric mucosal barrier due to factors like excess acid, reduced mucus, or blood flow imbalance. Prostaglandins shield the duodenum by bolstering mucosal resistance and lowering aggressive factors. Ethanol-induced ulcers, often used for research, trigger radical release, leading to mucosal harm, particularly in the stomach's glandular part. The presence of oxygen-derived free radicals is linked to the occurrence of acute and chronic ulcers, and removing them promotes the healing process. Despite available drugs with adverse effects, attention has shifted to natural sources of antiulcer agents. Medicinal plants, rooted in traditional knowledge, show promise in managing gastric ulcers, tapping into the potential of nature for novel treatments.

Many diseases, such as diabetes, gastric disorders, inflammation, and cancer, are believed to be caused by oxidative stress. This has led to a focus on researching natural antioxidants sourced from plants. Plants have developed robust defense mechanisms against environmental oxidative stress, leading to the evolution of diverse biomolecules. These plant antioxidants, honed over millions of years, exemplify perfect functional optimization.

Tannin,[21] triterpenoids,[22] alkaloids,[23] and saponin[24] are reported for their anti-ulcer activity. Significant elevation in epidermal growth factor levels and accelerated ulcer healing were observed with the administration of total alkaloids. The mechanism underlying their gastroprotective effects involved the regulation of serotonin and noradrenaline levels.[23] Studies have indicated that triterpenoids exert their gastroprotective effects primarily through the activation of mucous membrane secretion.[22] The saponin group demonstrated significant improvement in alcohol-induced gastric mucosal injury by reducing edema and cell necrosis.[24] The presence of all the mentioned phytoconstituents in *A. augusta* suggests that they may be responsible for its anti-ulcer activity.

The examination of *A. augusta* extract confirmed the existence of tannins, alkaloids, and triterpenoids. Any of these compounds may contribute to the antioxidant and anti-ulcer characteristics of *A. augusta*.

Lastly, ERAA (500 mg/kg) gave a significant gastro-protective effect compared to the disease control group but the significant level was not more in the ome group. ERAA has a dose-dependent effect so we need to do further study with a higher dose level of ERAA.

Conclusion

Based on the results of this study, it can be concluded that the methanolic extract from *A. augusta* roots, administered at a dose of 500 mg/kg, demonstrated significant anti-ulcer properties in animal models. Furthermore, its favorable antioxidant activities in different assays provided additional evidence of its effectiveness in protecting against gastric ulcers induced by etho.

Through this study, it was evident that the methanolic root extract of *A. augusta* at 500 mg/kg exhibited significant antiulcer and antioxidant properties. The presence of alkaloids, saponins, tannins, and terpenoids in the extract likely contributed to these effects.

These results emphasize the need for additional research to delve into the mechanisms underlying the anti-ulcer and antioxidant effects of high-dose ERAA at the molecular level. Such insights could lead to more effective therapeutic approaches for associated disorders.

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