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INVESTIGATION OF IN-VITRO GASTROPROTECTIVE AND ANTIOXIDANTS EFFECTS OF MIMOSA PUDICA AND SESBENIA GRANDIFLORA

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Abstract

Gastric ulcer disease, a widespread condition driven by oxidative stress and acid imbalance, necessitates safer, multi-targeted therapies. This study investigated the gastroprotective potential of two traditional medicinal plants, *Mimosa pudica* (MPE) and *Sesbania grandiflora* (SGE), through in vitro assays. Hydro-methanolic extracts were screened for phytoconstituents and evaluated for antioxidant activity (DPPH, ABTS, FRAP, NO scavenging) and proton pump (H+,K+-ATPase) inhibition. SGE exhibited a higher yield and significantly greater total phenolic (152.7 mg GAE/g) and flavonoid (98.5 mg RE/g) content than MPE. Correspondingly, SGE demonstrated superior antioxidant potency across all assays (e.g., DPPH IC_{50}: 22.4 µg/mL) and more effectively inhibited H+, K+-ATPase (IC_{50}: 92.1 µg/mL) compared to MPE (IC_{50}: 185.4 µg/mL). The results validate the ethnomedicinal use of both plants and reveal a dual mechanism of action involving potent free radical scavenging and direct anti-secretory activity. The markedly stronger bioactivity of *S. grandiflora* is strongly correlated with its richer polyphenolic profile, positioning it as a promising source for developing standardized phytotherapeutics against gastric ulcers.

Introduction:

Gastric ulcer disease is an epidemic and disabling health problem worldwide, which is a serious violation of the gastrointestinal mucosa that may lead to serious complications including hemorrhage, perforation, and obstruction. The pathogenesis involves a complex multifactorial imbalance between harmful elements, including gastric acid, pepsin, Helicobacter pylori infection, and NSAIDs,[1] versus protective mucosal mechanisms encompassing mucus-bicarbonate production, prostaglandins, cellular renewal, and vascular perfusion. Oxidative stress represents a pivotal integrative mechanism underlying this tissue damage. Overproduction of reactive oxygen species including superoxide anions and hydroxyl radicals, combined with compromised endogenous antioxidant defenses[2] (such as glutathione, superoxide dismutase, and catalase), leads to lipid peroxidation, protein modification, DNA injury, and programmed cell death in

gastric epithelial tissue. This oxidative cascade is closely connected with the inflammatory pathways including the activation of the NF-kβ, and it is a popular endpoint in ethanol-induced ulcerogenesis triggered by stress, NSAIDs, and *H. pylori[3][4]*. Therefore, the therapeutic approaches of the present days are progressively aimed not at suppression of acid using proton pumping inhibitors (PPIs) but at the strengthening of the mucosal defense as well as alleviation of the oxidative damage; the long-term effects of the traditional medications, however, are rather inclined to adverse effects, drug interactions, recurrence on the withdrawal, and high costs, which makes the multi-targeted and less risky alternatives as much-needed[5].

Therapeutic flora, historically valued across various conventional medical frameworks, represents a valuable reservoir for novel gastric protective compounds[6][7]. The complex biochemical constituents of these plants encompassing flavonoids, tannins, alkaloids, saponins, and phenolic acids demonstrate cooperative interactions that target multiple ulcer-inducing mechanisms. Such bioactive molecules exhibit capabilities in reducing gastric acid production, enhancing protective mucus formation, scavenging reactive oxygen species, inhibiting Helicobacter pylori proliferation, and regulating inflammatory mediator expression[8]. The transformation of ethnopharmacological knowledge into scientifically substantiated therapeutic approaches requires rigorous experimental validation, representing a cornerstone of modern plant-based drug development that seeks to bridge ancestral medicinal practices with evidence-based clinical applications[9]. This paper examines two such medicinal plants; *Mimosa pudica* Linn. (the sensitive plant or touch-me-not) and *Sesbania grandiflora* (L.) Pers. (the agati or hummingbird tree) and tests their in vitro gastroprotective and antioxidant properties in order to scientifically justify their use in folk medicine. *Mimosa pudica* is a pan-tropical perennial herb or sub-shrub in the family Fabaceae which is botanically famous due to its fast thigmonastic (touch-sensitive) responses in leaf motions. In addition to this interest, it occupies an ancient status in the ethnomedicine of Asia, Africa and the Americas. Conventionally, different components of the plant particularly the root and the leaves have been used to treat a great number of conditions including injuries, inflammation, diarrhea, dysentery and most notably, gastro-intestinal disorders. In certain folk, ulcer is treated with decoctions or pastes and is a digestive aid. A pharmacological accumulated body supports this therapeutic reputation. Studies have established that *M. pudica* extracts have great anti-inflammatory, anti-microbial, analgesic, antidiarrheal and wound healing effects[10][11]. Essentially in gastroprotection, initial research has shown anti-ulcerogenic effects in rodent models and this is usually because of its high content of bioactive compounds[12][3]. The plant contains known sources of mimosine (an alkaloid), flavonoids (including quercetin and isoorientin), tannins and sterols. Flavonoids and tannins especially stand out due to their capacity to lower gastric acid secretion, develop a protective layer of proteinaceous nature on the mucosa, and possess a strong free radical scavenging effect. The antioxidant activity of M. pudica extracts has been verified in several models implying a direct process to counteract the oxidative stress which is central in the ulcer formation. Nevertheless, the precise in vitro mechanistic studies to establish the specific connection between its particularity of phytochemistry and specific gastroprotective effects, including proton pump inhibition, are progressively

understudied and are one of the main justifications of the presented study[14][15]. Equally, *Sesbania grandiflora*, also of the Fabaceae, is an extensively used fast-growing tree in South and Southeast Asia not only as a source of fodder and wood, but also as a food medicine. Its huge and edible flowers and soft leaves are eaten as vegetables and are part of local cuisines, and different parts of the plant are used in Ayurveda, Siddha and other folk medicinal traditions. Historically, *S. grandiflora* has been used to treat a wide range of diseases: its flowers to treat headaches and night blindness, the leaves and the bark to treat fevers, infections and inflammatory diseases and the roots and the bark to treat gastritis and diarrhea and dyspepsia[16][17]. This is a direct gastrointestinal comfort application that is compatible with its reported pharmacological action that consists of antimicrobial, anti-inflammatory, hepatoprotective, and antidiabetic effects. Interestingly, scientific reports have also started to establish its gastroprotective activity in vivo with substantial decrease in ulcer indices in experimental models. The bioactivity of the plant is based on a wide phytochemical portfolio, comprising of alkaloids, flavonoids, saponins, tannins, triterpenoids, and distinct compounds such as isovanillic acid and linoleic acids. Notably, S. grandiflora is known to have a high antioxidant potential and researchers have attributed high radical scavenging and metal chelating properties of *S. grandiflori* to its phenolic and flavonoid compounds. Such a strong antioxidant profile is very pertinent in defence of the gastrointestinal mucosa against oxidative attack. In spite of these positive signs, a systematic, comparative in vitro study of its direct anti-ulcer mechanisms, including H^+ / K^+-ATPase inhibition, as well as a comprehensive evaluation of its antioxidant activity, is required to comprehensively know and standardize its therapeutic value[18]19]. In addition, a corresponding study using Mimosa pudica can be done to allow a comparative pharmacognostic analysis of which plant, or which group of compounds, could be of better multi-target activity regarding the prevention and treatment of ulcers. Thus, the proposed research is aimed at conducting a critical comparison of the in vitro gastroprotective and antioxidant activity of *Mimosa pudica* and *Sesbania grandiflora*.[20]21] The study will seek to offer a mechanistic basis of their traditional application by using a battery of assays such as proton pump inhibition and known antioxidant models (DPPH, ABTS, FRAP, NO scavenging), and screening of phytochemicals and quantification of total phenolics and flavonoids[22][23][24]. The results will not only hand in the validation of these ethnomedicinal resources, but also to the overall search of novel, safe and multi-functional phytotherapeutic agents against gastric ulcer disease a disease in which oxidative stress is a crucial and harmful factor[25].

Material & method:

Extraction technique:

To every plant, 100 g of dried leaf powder was macerated by mean of cold solvents of successively increasing polarity. The 500 mL of petroleum ether was initially used to extract the powder over 72 hours to take away chlorophyll as well as fatty substances. The marc was then air-dried after which the defatted marc was extracted in 500 mL of 70% aqueous methanol over 72 hours with occasional shaking. Whatman filter paper was used to filter the extracts and the solvent was evaporated at reduced pressure and 40o C using a rotary evaporator (Buchi, Switzerland). The resulting crude hydro-methanol extracts of Mimosa pudica (MPE) and Sesbania grandiflora (SGE) were lyophilized, dried to determine the

percentage of yield, and kept in at -20degC. All subsequent assays were replenished in distilled water or dimethyl sulfoxide (DMSO, less than 0.1 final concentration).

Phytochemistry and Phytochemical Screening:

Major bioactive classes were preliminary assessed through standard chemical tests of MPE and SGE to determine the presence of these classes in these extracts. The reagents of Mayer and Wagner were used to identify alkaloids. The presence of Flavonoids was determined using Shinoda test (magnesium turnings and concentrated HCl). Tannins were analyzed with the ferric chloride and gelatin solution. Froth test showed the presence of saponins with lead acetate and ferric chloride showing phenolics. The Salkowski and Libermann-Burchard tests were used to determine steroids and terpenoids, respectively.

***In vitro* gastroprotective assays:**

H+, K+-ATPase proton pump inhibition assay:

A spectrophotometric approach with minor adaptations was employed to evaluate the inhibitory impact on gastric H+, K+-ATPase activity using porcine gastric mucosal microsomal preparation (Sigma-Aldrich, USA). In brief, the reaction mixture (1 mL) contained 2 mM MgCl2, 10 mM KCl, 2 mM ATP, 50 µg of microsomal protein, and varying concentrations of plant extracts (50-400 µg/mL) or omeprazole (reference compound, 10-100 µM) in 40 mM Tris-HCl buffer (pH 7.4). The reaction was initiated by ATP addition and incubated for 30 minutes at 37°C. Termination was achieved by adding 1 mL of 10% trichloroacetic acid. The released inorganic phosphate (Pi) was quantified using the Fiske-Subbarow methodology, wherein the resulting blue coloration was measured at 660 nm. Control reactions (without inhibitor) and blanks (without enzyme) were conducted simultaneously. The concentration producing 50% inhibition of H+, K+-ATPase activity (IC50) was determined from the dose-response relationship.

***In Vitro* Antioxidant Assays:**

DPPH radical scavenging activity:

The 2,2-diphenyl-1-picrylhydrazyl (DPPH) radical represents a stable free radical employed for evaluating the antioxidant capacity of the plant extracts. Briefly, a 0.1 mM DPPH solution in methanol was formulated. Two milliliters of this DPPH solution were combined with varying concentrations of botanical extracts (10-100 µg/mL) or ascorbic acid (reference compound). The reaction mixture was subjected to vortex mixing and subsequently incubated under dark conditions at ambient temperature. Following a 30-minute incubation period, the absorbance was determined using UV-Vis spectrophotometry at 517 nm wavelength. Control samples (containing methanol and DPPH) and blank samples (comprising control plus individual extract concentrations) were prepared. The DPPH radical scavenging percentage was calculated, and the IC50 value (representing the concentration achieving 50% inhibition) was determined through linear regression analysis.

ABTS radical cation decolorization assay:

The ABTS+ radical scavenging capacity was assessed through the following methodology. The ABTS radical cation was generated by combining 7 µM ABTS stock solution with potassium persulfate at a final concentration of 2.45 mM, with the resulting mixture incubated in darkness at ambient temperature. Subsequently, the solution underwent dilution with ethanol to achieve an absorbance value of 0.70 (±0.02) at 734 nm. Plant extracts or Trolox standards (ranging from 0-100 µM) were then reacted with 2 mL of the diluted ABTS+ solution for a duration of up to 6 minutes. The decrease in absorbance at

734 nm was monitored and recorded. The antioxidant capacity was expressed as Trolox Equivalent Antioxidant Capacity (TEAC) in µmol Trolox equivalents per gram of dried extract.

Ferric reducing antioxidant power:

The reducing capacity was assessed using the FRAP assay. The FRAP solution was prepared fresh by combining 300 mM acetate buffer, 10 mM TPTZ solution (2,4,6-Tripyridyl-s-Triazine) in 40 mM HCl, and 20 mM $FeCl_3.6H_2O$ in equal ratios of 1:1:1. Plant extract (100 µL) or FeSO47H2O standard solutions (0-1000 µM) were added to 3 mL of the FRAP solution. The resulting mixture underwent incubation at 37°C, and the absorbance of the resulting blue-colored complex was determined at 593 nm. The reducing capacity was expressed as mM Fe (II) equivalents per gram of extract.

Nitric oxide scavenging activity:

Various concentrations of botanical extracts or ascorbic acid reference standard were mixed with sodium nitroprusside (10 mM) in phosphate buffer solution (PBS, pH 7.4). Following a 150-minute incubation period at 25°C under illuminated conditions, Griess reagent (containing 1% sulfanilamide and 0.1% N-(1-naphthyl)ethylenediamine dihydrochloride dissolved in 2% H3PO4, 1.5 mL) was incorporated into the reaction mixture. The absorbance of the resulting chromophore was determined spectrophotometrically at 546 nm. The relative percentage inhibition of nitrite production, representing NO scavenging activity, was computed in comparison to the control sample.

Quantification of Bioactive Compounds:

Total Phenolic Content (TPC):

TPC determination was conducted using the Folin-Ciocalteu colorimetric technique. Each extract sample (0.5 mL) was combined with 0.5 mL of Folin-Ciocalteu reagent. Following a 5-minute interval, 2 mL of 7.5% (W/V) sodium carbonate solution was incorporated. The mixture underwent incubation under dark conditions at ambient temperature for 60 minutes, after which absorbance measurements were taken at 765 nm wavelength. A calibration curve was established using gallic acid standards ranging from 0-100 µg/mL. Results were expressed as milligrams of Gallic Acid Equivalent (GAE) per gram of dried extract.

Total Flavonoid Content (TFC):

The aluminum chloride colorimetric technique was employed for TFC quantification. Plant extract (1 mL) was mixed with distilled water (4 mL) and 5% NaNO2 solution (0.3 mL). Following a 5-minute interval, 10% AlCl3 (0.3 mL) was added, succeeded by the addition of 1 M NaOH (2 mL) after 6 minutes. The final mixture was adjusted to 10 mL using distilled water. Following vortex mixing, absorbance measurements were obtained at 510 nm. Quantification was achieved through calibration against a rutin standard curve ranging from 0-100 ug/mL, with TFC expressed as milligrams of Rutin Equivalent (RE) per gram of dried extract.

Statistical Evaluation: Each experiment was performed three times (n=3). Results are expressed as mean ± standard deviation (SD). Statistical evaluation was conducted using GraphPad Prism (version X.X). Comparative analysis utilized one-way ANOVA with Tukey's post-hoc testing. IC_{50} determination was accomplished through non-linear regression analysis. Correlation assessment employed Pearson's correlation coefficient.

Result & Discussion

Phytochemical Screening and Extract Yield:

Mimosa pudica (MPE) and Sesbania grandiflora (SGE) hydro-methanolic extracts had a hydro-methanolic yield of

14.2% and 18.7% (w/w), respectively. The preliminary qualitative analysis showed that significant phytoconstituents exist (Table 1).

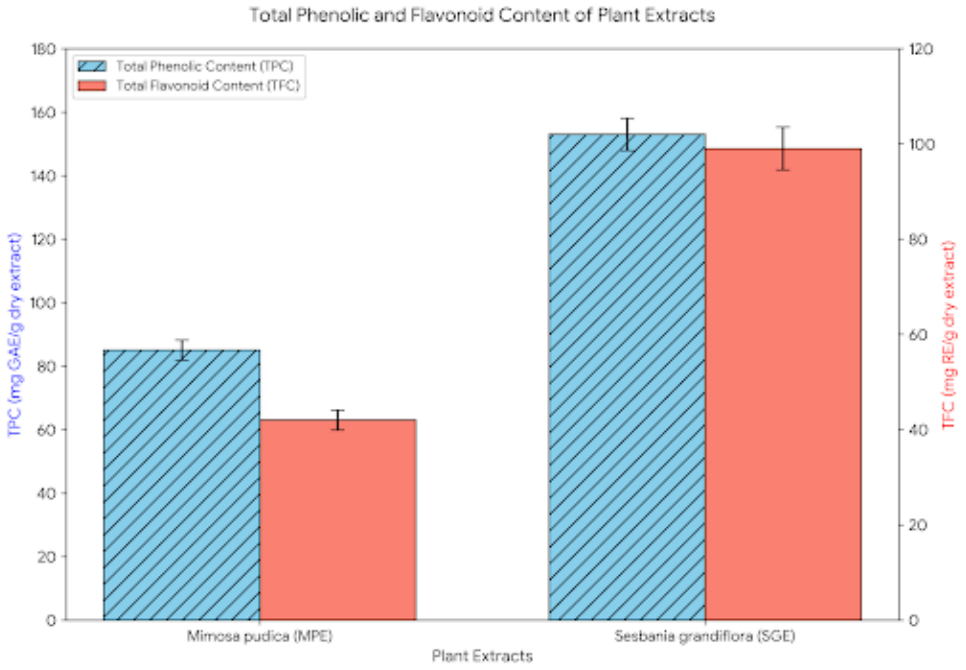
Table 1: Preliminary Phytochemical Screening of MPE and SGE:

Phytoconstituent	Test/Reagent	*Mimosa pudica* (MPE)	*Sesbania grandiflora* (SGE)
Alkaloids	Mayer's & Wagner's	+	++
Flavonoids	Shinoda Test	+++++	++++
Tannins	$FeCl_3$ & Gelatin	+	++
Saponins	Froth Test	+	+
Phenolics	$FeCl_3$ & Lead Acetate	+++	++++
Steroids	Salkowski Test	+	+
Terpenoids	Libermann-Burchard	+	+

Key: + = Present, ++ = Moderately present, +++ = Abundantly present)

***In Vitro* Antioxidant Activity:**

TPC and TFC was much better in SGE than in MPE (Fig. 1A). In line with this, SGE exhibited a better antioxidant activity in all the assays, with significantly lower values of IC50 in DPPH and NO scavenging experiments and higher TEAC and FRAP values (Table 2, Fig. 1B).



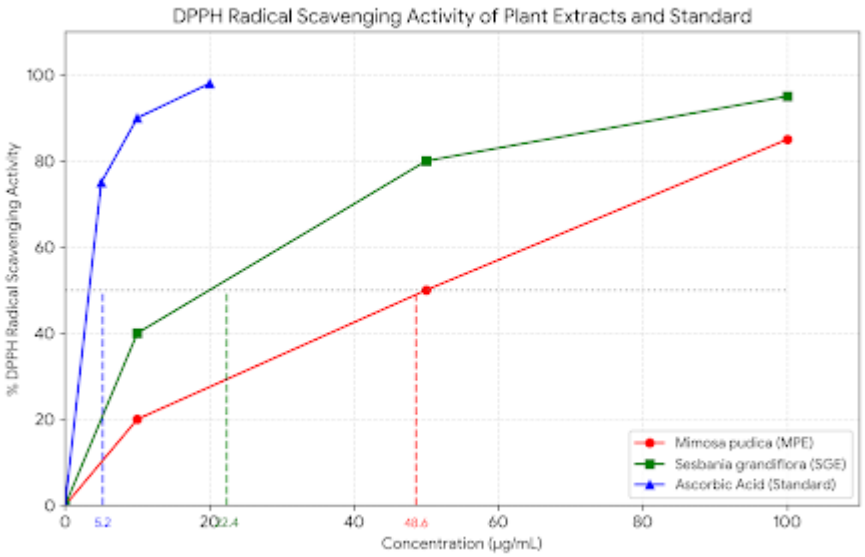


Fig 1. (A)Show comparing TPC (GAE) and TFC (RE) of MPE and SGE. (B showing dose-dependent DPPH radical scavenging activity of MPE, SGE, and Ascorbic Acid (Std).

Table 2: Antioxidant Potential and Bioactive Content of MPE and SGE:

Parameter	***Mimosa pudica* (MPE)**	***Sesbania grandiflora* (SGE)**	**Standard**
TPC (mg GAE/g extract)	85.3 ± 3.2	152.7 ± 5.1*	-
TFC (mg RE/g extract)	42.1 ± 1.8	98.5 ± 4.3*	-
DPPH Scavenging (IC_{50}, µg/mL)	48.6 ± 1.5	22.4 ± 0.9*	Ascorbic Acid: 5.2 ± 0.3
ABTS Scavenging (TEAC, µmol/g)	325 ± 12	580 ± 18*	-
FRAP (mM Fe(II)/g)	1.8 ± 0.1	3.5 ± 0.2*	-
NO Scavenging (IC_{50}, µg/mL)	105.3 ± 4.8	55.7 ± 2.1*	Ascorbic Acid: 28.4 ± 1.3

***In vitro* gastroprotective activity: H+, K+-ATPase inhibition:**

Both plant extracts significantly inhibited proton pump activity in a dose-dependent manner (Fig. 2). SGE again showed more potent activity, with an IC_{50} value closer to the standard drug omeprazole.

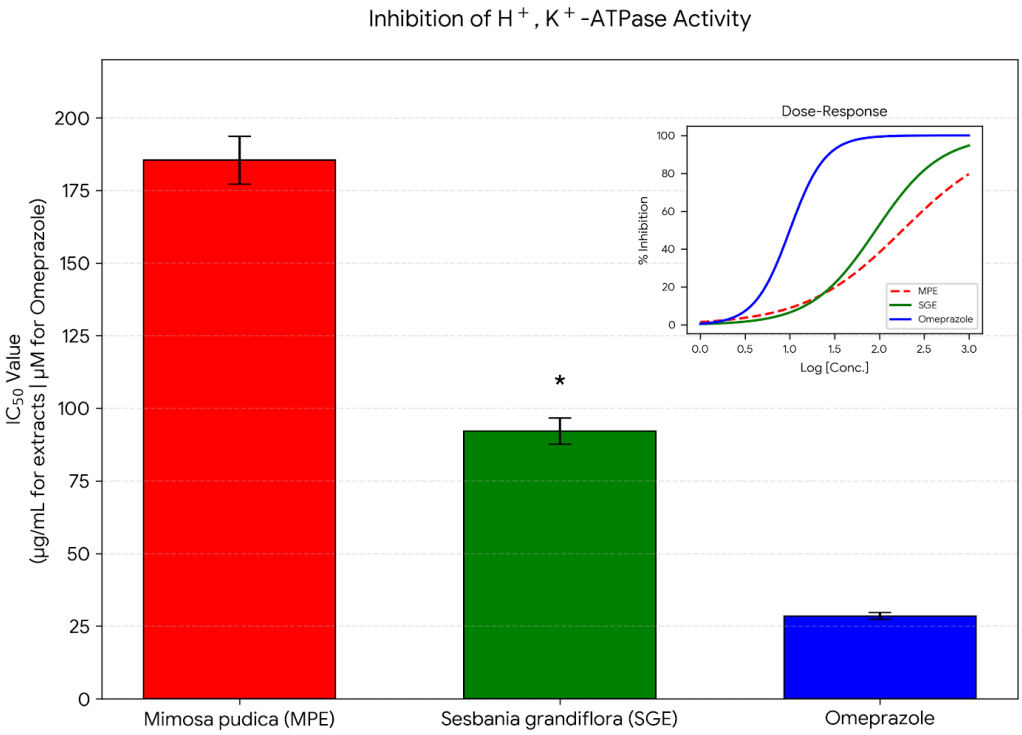


Fig 2. Comparing the IC_{50} values for H+, K+-ATPase inhibition by MPE, SGE, and Omeprazole.

Table 3: H+, K+-ATPase Inhibitory Activity

Sample	**IC_{50} Value**
Mimosa pudica (MPE)	185.4 ± 8.7 µg/mL
Sesbania grandiflora (SGE)	92.1 ± 4.2 µg/mL*
Omeprazole (Standard)	28.5 ± 1.3 µM

Discussion:

The current research gives a coherent scientific support of the traditional ethnomedicinal use of M. pudica and S. grandiflora in the management of gastrointestinal diseases, which explains a complex dual mechanism of action and involves both potency antioxidant defense and direct anti-secretory action. The multi-targeted method is especially useful in the environment of gastric ulcer pathology where both oxidative stress and acid hypersecretion are tightly connected and, in most cases, form a vicious cycle of mucosal damage. These results prove that these botanical extracts are not palliative, and they participate in certain pharmacological processes related to cytoprotection and acid modulation. This high bioactivity of S. grandiflora extract (SGE), which is significantly better demonstrated in all assays, can be directly explained by the fact that the yield of extraction of the extract is great enough, and the concentration of the phenolic and flavonoid compounds is significantly higher, as evaluated by TPC and TFC.

Plant-based antioxidant therapy is grounded on the phenolics and flavonoids; the established capacity to donate hydrogen atoms, chelate transition metals, and quench a wide range of free radicals, such as the stable nitrogen-centered DPPH and the previously-formed cation radical ABTS+, or even the pro-inflammatory mediator nitric oxide, was evident. The robust statistical evidence demonstrating a substantial inverse relationship between TPC/TFC concentrations and IC50 values in antioxidant evaluations (including DPPH radical neutralization and NO scavenging activities) confirms that these phenolic constituents serve as primary determinants of the demonstrated free radical elimination capacity and reductive potential. This pronounced antioxidant efficacy represents an essential characteristic for direct gastric protection rather than a secondary phenomenon, enabling the inactivation of reactive oxygen intermediates (ROS), encompassing hydroxyl and superoxide radical species, which play direct roles in triggering membrane lipid oxidation in gastric epithelial tissues, enhancing inflammatory cascades via NF-kB pathway stimulation, and inducing programmed cell death in gastric epithelial structures.

In addition to protective effects on the mucosa against oxidative attack, the dose-dependent, potent (again, significantly greater by SGE) inhibition of the H +, K + -ATPase by both extracts, indicates a direct anti-ulcer effect pharmacologically similar to synthetic proton pump inhibitors (PPIs) such as omeprazole. This process can be logically explained by the presence of the phytoconstituents. Flavonoids and tannins which are active in plants and especially in SGE are known to react with sulfhydryl (-SH) groups which are essential in the conformational activity of the proton pump and may act as reversible inhibitors. In addition, tannin is capable of producing proteinaceous complex, which provides a protective coating on gastric mucosa, and thus, provides a physical barrier to acid and pepsin. The hypothesis that these polyphenols are the active principles is supported by the positive relationship existing between the richness of the extract in phenolic compounds and its proton pump inhibitory activity. Such a pharmacological effect combined with the one that inhibits the main cause of acid secretion and the one that strengthens the mucosal lining against oxidative and inflammatory harm is a holistic approach to treatment. It is the opposite of the traditional mono-therapeutic methods that may be beneficial in the treatment of ulcers in which several pathogenic agents interact, including those caused by NSAIDs or prolonged stress. Although the hydro-methanolic extract of M. pudica (MPE) was more moderate in its activity, which perhaps can be attributed to its lower phytochemical density, it is important not to overlook its bio-efficacy. Its antioxidant property and its having a significant but lower proton pump inhibition confirm the traditional use. The appearance of unique constituents such as the alkaloid mimosine, and other secondary metabolites, may lead to a different pharmacological profile that may include pathways not well studied by the current in vitro-based design, including a change in the production of inflammatory cytokines (e.g., TNF-a, IL-6) or an increase in endogenous production of prostaglandins. This is an indicator that MPE can work via a synergistic combination of actions, and the complexity of plant-based therapeutics is a reality. Finally, these results put S. grandifloras, especially, in a better place as a very promising and potentially powerful source of bioactive compounds to build into the subsequent drug development. Its activity is probably not independent of a single molecule but a product of

synergistic or additive interactions between its diverse phytochemical ensemble a unique benefit of standardized botanical extracts. This encouraging in vitro data is the basis upon which a logical advancement towards more complex experimental models should be carried out. The bioassay-guided fractionation and isolation of the lead inhibitory compounds of SGE followed by validation in established in vivo ulcer models (e.g., ethanol-induced, pylorus ligation, H. pylori-infected models) should be given priority when further studies are conducted on the subject. At the same time, full toxicological profiling and pharmacokinetic analysis is required to determine safety and bioavailability. This orderly line of research may eventually result in the creation of a standardised, evidence-based phytomedicine, which is a possible alternative or supplement to the existing synthetic medicines in managing gastric ulcers particularly given the known complications and recurrence rate of using PPI over an extended period.

Conclusion:

This study scientifically validates the traditional use of *Mimosa pudica* and *Sesbania grandiflora* in managing gastric disorders, demonstrating their efficacy through dual mechanisms: potent antioxidant activity and direct inhibition of the gastric proton pump. The significantly stronger activity of *S. grandiflora* extract is attributed to its abundant phenolic and flavonoid constituents, which effectively scavenge free radicals and suppress acid secretion. These findings bridge ethnopharmacological knowledge with evidence-based science, highlighting these plants, particularly *S. grandiflora*, as rich sources of multi-target bioactive compounds. The results warrant further research, including bioassay-guided isolation of active principles, in vivo validation, and toxicological studies, to develop these extracts into safe, effective, and standardized adjuncts or alternatives to conventional anti-ulcer therapies.

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