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Review Article

Peptic Ulcer Disease and Screening Approaches: A Comprehensive Review

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ABSTRACT

Peptic ulcer disease (PUD) is a chronic gastrointestinal disorder caused by an imbalance between protective mucosal mechanisms and harmful factors such as gastric acid, pepsin, NSAIDs, alcohol, and Helicobacter pylori infection. Current therapies, including proton pump inhibitors, H₂ receptor antagonists, and prostaglandin analogs, are effective. Experimental assessment of anti-ulcer effects employs various in vivo models, including pylorus ligation, ethanol, histamine, NSAID, stress, and reserpine-induced ulcer models, alongside in vitro assays such as H⁺/K⁺-ATPase inhibition and titration-based neutralization tests. Every model provides additional insights into ulcer development and the effects of medication. Using in vivo and in vitro methods together may enhance the discovery of safer and more effective anti-ulcer medications, particularly from plant origins, offering appealing alternatives to conventional therapies.

INTRODUCTION

A peptic ulcer (PU) is defined as a specific lesion, which consists of a loss of continuity of the proximal gastric and/or duodenal mucosa.[35]

Ulcerations are primarily crater-shaped sores or lesions that may occur on the stomach, colon, or oral mucosa. Once an ulcer forms, the area may bleed or release disagreeable substances, creating a painful sore that is prone to infection [2]. Patients experiencing peptic ulcer disease may not exhibit any symptoms at all, or they might experience

heartburn, epigastric discomfort, anorexia, nausea, vomiting, and belching and bloating.

Stress, alcohol use, smoking, H. pylori infection, and drug and chemical ingestion are a few of the causes. Particularly, prolonged alcohol use, cigarette smoking, and long-term NSAID use are contributing factors to peptic ulcers. Free radicals are known to have a part in the pathophysiology of peptic ulcers caused by damage to the mucosa. [3] A gastric ulcer is an ulcer in the stomach, and a duodenal ulcer is an ulcer in the first segment of the intestine.

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Factors that enhance acid secretion include HCl, pepsin, refluxed bile, NSAIDs, alcohol, pancreatic proteolytic enzymes, ingested irritants, bacterial toxins, and physicochemical damage. Digestive factors include blood flow, mucus, bicarbonates, epithelium resolution, and the state of treatment. [1]. When defensive mechanisms and aggressive parameters are out of balance, mucosal integrity is disrupted, which eventually leads to the development of a peptic ulcer.

Antiulcer drugs aim to do more than only delay the disease's course. Additionally, it seeks to bring about remission as soon as possible and keep it going for a long time. Many traditional therapy techniques are being used to address PUD; however, it's important to keep in mind that each of these approaches has one or more unpleasant side effects. Currently known anti-ulcerative drugs aim to do more than just slow the progression of the condition. Additionally, it seeks to quickly establish and maintain a state of remission for a long time.

Table 1: The Anti-Ulcerative Effects of Diverse Plants in Preclinical Studies [2]

Medicinal Plants	Family	Phytoconstituents	Plant parts and solvents used	Animals used in preclinical studies	Reference
Annona muricata	Annonaceae	Acetogenins, Alkaloids, Flavonoids Tannins, Essential oil (trace)	Leaves; Maceration (Ethanol)	In vivo on male and female albino mice (<i>Mus musculus</i>),	Elizângela Beneval Bento et al 2018[11]
Arctium lappa	Asteraceae	Arctiin, Arctigenin, Inulin, Tannins, Polyacetylenes	Roots; Percolation (Ethanol 96%)	In vivo on female Wistar rats (<i>Rattus norvegicus</i>)	Luisa Mota da Silva et al 2018[12]
Artemisia absinthium	Asteraceae	Thujone, thujyl alcohol, cineole, Absinthin, anabsinthin, Flavonoids, Tannins, Organic acids	Aerial parts; Maceration (Water)	In vivo on female Wistar rats (<i>R. norvegicus</i>)	Thaise Boeing et al 2023[13]
Bryophyllum pinnatum	Crassulaceae	Bufoadienolides, quercetin, kaempferol, Triterpenes, Alkaloids, Tannins,	Leaves; Maceration (Methanol)	In vivo on Swiss mice (<i>M. musculus</i>)	Laure Brigitte Kouitchou Mabeku et al 2017 [14]
Casearia sylvestris	Salicaceae	Clerodane, diterpenes, Flavonoids, Tannins, Trace essential oil	Leaves; Infusion (Distilled Water)	In vivo on female Wistar rats (<i>R. norvegicus</i>)	Beatriz Monteiro Magalhães de Oliveira et al 2022 [15]
Cinnamomum glanduliferum	Lauraceae	Cinnamaldehyde, Eugenol, Linalool, Safrole, Camphor	Leaves; Hydro distillation (Water)	In vivo on male Wistar albino rats (<i>R. norvegicus</i>)	Azab SS et al 2017[16]
Phyllanthus niruri	Phyllanthaceae	Phyllanthin, hypophyllanthin, Quercetin, kaempferol, Phyllantine, Tannins, Saponins, Terpenoids,	Leaves; Maceration (Methanol)	In vivo on female Swiss albino rats (<i>R. norvegicus</i>)	Mostofa R et al 2017 [17]

Pathogenesis of ulcer [6]:**Figure no: I****Treatment strategies for peptic ulcer include [1]****Table No: II**

Class	Subclass	Drugs
Gastric acid secretion inhibitors	Proton pump inhibitors	1. Omeprazole 2. Lansoprazole 3. Pantoprazole 4. Rabeprazole
	H2 Receptor antagonist	1. Cimetidine 2. Ranitidine 3. Famotidine 4. Roxatidine
	Prostaglandin analogues	1. Misoprostol 2. Enprostil 3. Rioprostil
	Anticholinergics	1. Pirenzepine 2. Propanthelin 3. Oxyphenonium
Gastric acid neutralizers (Antacids)	Systemic	1. Sodium bicarbonate 2. Sodium citrate
	Non systemic	1. Aluminium hydroxide 2. Magnesium hydroxide
Ulcer protective		1. Sucralfate 2. Colloidal bismuth subcitrate
Anti-H.Pylori		1. Amoxicillin 2. Clarithromycin 3. Metronidazole 4. Tinidazole 5. Tetracycline

Antiulcer screening methods include –

In vivo screening methods -

Pylorus ligated induced ulcer model (Shay rat method)

One of the oldest animal models for gastric ulcers [5]. The tying off of the pyloric region of the stomach results in the build-up of gastric acid within the stomach, leading to the formation of ulcers. [4] The autodigestion of the gastric mucosa causes the breakdown of the gastric mucosal barrier, resulting in ulcer formation, making the "Shay-rat" a useful method for assessing a drug molecule's anti-ulcer properties [6].

In this approach, Albino rats weighing 150-200 g are kept in separate cages and deprived of food for 24-36 hours before undergoing pyloric ligation. With light ether anaesthesia, a small midline incision is made in the abdomen just below the xiphoid process. The pyloric section of the stomach is tied off, ensuring no harm is done to its blood supply. The stomach is carefully replaced, and the abdominal wall is sutured closed with interrupted stitches. The medications are given subcutaneously right after pyloric ligation. The animals undergo a 48-hour fast during the postoperative phase and are euthanized 19 hours following the surgery. Stomachs are removed, and the contents are emptied into tubes. Then, the contents are analysed for pH, free acidity, and total acidity. The stomachs are subsequently incised along the greater curvature, and the inner surface is inspected for ulceration. The ulcer index is determined in this manner:

$$\text{Ulcer Index} = 10 / X,$$

where X = Total mucosal area / Total ulcerated area. [20], [21]

Ethanol-induced gastric ulcer model

Ethanol causes ulcer lesions by exposing the gastric mucosa to the hydrolytic and photolytic actions of HCl and pepsin [4]. These lesions are characterised by sub-epithelial haemorrhages, cellular exfoliation, inflammatory cell infiltration, and mucosal oedema [6].

Male Wistar rats (200–300 g) are used for this experiment. Animals are fasted for 18 hours with access to water. The test drug is given to animals orally. Administer the test drug or vehicle 30 minutes before ethanol. Absolute ethanol (95%–99%) is administered at a dose of 1mL/200g body weight to each animal, and after 1 hour, the animals are sacrificed by euthanasia with CO₂, and their stomachs are dissected out. The stomach is opened along the greater curvature, washed with warm water, and examined for ulcer scoring (tissue damage score 0 (least) – 3 (most)). [28, 29]

$$\text{HR} = \text{CA} - \text{B}(\text{mmHg/h})$$

Where: HR = Healing Rate Equation, A = tensile strength (mmHg) at time-point C after puncture, B = tensile strength 30 min after puncture (avg. 143 mmHg), C = duration of experiment (h) [30, 31, 32, 33, 34]

Histamine-induced gastric ulcer model

Both increased stomach acid output and the vasospastic action of histamine influence the experimental induction of gastric ulcers in a number of animals [7]. Histamine's strong acid-stimulating and vasodilating properties cause increased vascular permeability, which is the mechanism by which it causes gastric ulcers. The histamine-induced ulcer model is based on these pharmacological actions of histamine, which makes it suitable for assessing the anti-secretory effects of possible ulcer-fighting medications and substances that act as H₂-receptor antagonists. [34]



The experiment utilises male guinea pigs. Animals undergo a fasting period of 36 hours. A dosage of 50 mg of histamine acid sulphate is administered intraperitoneally. To avoid histamine toxicity, 5 mg of promethazine hydrochloride is administered intraperitoneally 15 minutes prior to and 15 minutes following the histamine injection. The test medication is given 30-45 minutes following the histamine injection. After 4 hours, the animals are killed, and the stomach is extracted and analysed. The assessment of ulcers is determined by the severity of the ulcers.

Assess ulcer severity

- 0 - No abnormalities
- 1 - Oedema of the mucosa
- 2 - 1-5 tiny lesions (1-2 mm in diameter)
- 3 - > 5 small to moderate (3-4 mm in diameter) lesions
- 4 - ≥ 2 moderate lesions or 1 large (> 4 mm in size) lesion
- 5 - Perforated wounds [27]

NSAIDs Induced Gastric Ulcer Model

NSAIDs lead to ulcers by blocking prostaglandin production via the inhibition of the cyclooxygenase enzyme in the COX pathway. Prostaglandins have a protective function by promoting the release of bicarbonates and mucus, preserving blood circulation, and controlling the regeneration and repair of mucus cells. A decrease in prostaglandin production may lead to inflammation and weaken the protective mucosal barrier in the stomach, potentially resulting in ulcer development.

In this approach, animals are subjected to fasting for 24 to 36 hours. An NSAID (either aspirin or indomethacin) is administered orally with an appropriate vehicle (water or 1% carboxymethylcellulose). The test drug is

administered to animals one hour later. After four hours, the animals are euthanised, the stomach is removed, and the severity of the ulcer is evaluated. Amount of NSAIDs used to induce ulcers: Aspirin – 150 mg/kg of body weight, and Indomethacin – 40 to 100 milligrams for each kilogram of body weight. The maximum diameters of the lesions are measured and summed to give a total lesion score (in mm) for each animal, and the average score for each group is calculated. [18], [19]

Stress Ulcer

This form of ulcer is frequently observed in severely ill patients in the intensive care unit and occasionally leads to gastrointestinal bleeding, which is associated with significantly higher morbidity and mortality, even with intensive acid suppression therapy. [6] Stress ulcers are especially linked to heightened gastric acid secretion, decreased blood circulation to the stomach lining, and harm to the mucosal barrier of the gastrointestinal tract. The ways in which immobilization stress leads to stress ulcers are intricate and involve interactions among the nervous, endocrine, and immune systems. [22]

In this approach, 10 female Wistar rats, each weighing between 150 and 170 g, are randomly chosen per dose of the test medication and for control groups. Animals are denied food and water for 24 hours before the start of the experiment. Following oral or subcutaneous administration of the test compound/placebo, the animals are mildly anesthetized using appropriate anaesthesia such as ether. They are suspended horizontally in darkness at 20°C for 24 hours and ultimately euthanized using CO₂ anaesthesia. The stomach is excised and secured onto a cork plate, and the count and intensity of ulcers are recorded with a stereomicroscope using these scores: 0 = no ulcer, 1 = superficial ulcers, 2 = deep ulcers, 3 = perforation



An ulcer index (UI) is calculated:

$$UI = UN + US + UP \times 10^{-1}$$

- UN = average number of ulcers per animal
- US = average severity score
- UP = percentage of animals with ulcers [34].

Reserpine-Induced Duodenal Ulcer

Reserpine is derived from the root of *Rauwolfia serpentina*, utilized for managing hypertension; however, it can lead to side effects such as gastric mucosal lesions (GMLs), sexual dysfunction, and depression due to excessive dosage and extended use. Reserpine causes gastric lesions by lowering sympathetic tone and raising cholinergic tone, leading to excessive secretion of gastric acid [6].

In this approach, rats deprived of food for 36 hours receive reserpine mixed in 10% Tween 80 (5–8 mg/kg, i.p.) as stated in [23]. Despite the model's dependence on acid, hypermotility appears to be more crucial than hypersecretion for causing gastric mucosal lesions. Typically, the substances or plant extracts under assessment are given to the test animals no less than 30 minutes prior to the administration of reserpine. The test animals are subsequently euthanized 24 hours later [7].

In vitro screening methods -

Preparation of H⁺/K⁺ - ATPase enzyme

The fresh Goat stomach was used to prepare the H⁺/K⁺ - ATPase enzyme sample. The stomach of a goat was dissected open, washed, and the mucosa of the gastric fundus was removed, and the inner layer was scraped off to obtain parietal cells. Next, 10% Triton X-100 was added to a homogenate of parietal cells in 16 mM Tris buffer (pH 7.4), and the mixture was centrifuged at 5000xg for 10 minutes. The activity of the enzyme H⁺/K⁺-ATPase inhibitor was measured by isolating the

supernatant. Bovine serum albumin was utilised as a reference reagent to calculate the protein concentration in the supernatant. The activity of H⁺/K⁺ ATPase was measured in parietal cell extract. [9][10]

Using a Titration Method of Fortran's Model for the Determination of Neutralizing Capacity

A 250 ml beaker is used, and 90 ml of the prepared solution, along with the reference drug, is placed in separate beakers at 37°C. Continuous stirring is performed using a magnetic stirrer at 30 rpm to simulate stomach movements or the stomach environment. The prepared sample and standard reference are titrated individually with artificial gastric juice until the endpoint of pH 3 is reached. The volume of artificial gastric juice consumed is quantified as a reference drug. [24, 25, 26]

Neutralization Effects of Prepared Preparation on Artificial Gastric Acid

In this approach, we prepare laboratory acid, and the pH is modified to match stomach conditions, particularly between pH 1.2 and 2.5. This is a significantly acidic state where the mucosal layer starts to sustain damage. If this pH is not managed promptly, it leads to the development of gastric ulcers. The plant extract or formulated drug and reference drug are individually mixed with artificial gastric juice at a pH of 1.2 - 2.5, and subsequently, this combination is analysed for its neutralizing effect on artificial gastric juice using the titration method. During titration, surplus acid is neutralized, allowing us to find the precise quantity of the prepared solution that balances the artificial gastric juice. To prepare artificial gastric juice, dissolve NaCl (2 grams) and pepsin (3.2 mg) in distilled water (500 ml). Subsequently, hydrochloric acid (7.0 ml) is introduced, and an adequate quantity of water is added to raise the volume [24, 25].



CONCLUSION

In conclusion, a variety of anti-ulcer examination methods have been established worldwide, and we have a large number of experimental models accessible for assessing anti-ulcer therapies. Every model has a unique set of advantages and disadvantages. This paper's main goal is to give a summary of the antiulcer models that are most commonly used by researchers in their gastrointestinal protection studies. Analysing antiulcer models, we discovered that the outlined models are frequently employed in accordance with treatment demands, and the timing and outcomes of the study impact the model selection. Although *in-vivo* models are used more frequently compared to *in-vitro* models, our research indicates that combining the two could lead to better results and help find new pharmacological agents with better antiulcer capabilities.

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