Content available at: https://www.ipinnovative.com/open-access-journals

IP International Journal of Comprehensive and Advanced Pharmacology

Journal homepage: https://ijcap.in/

Review Article

A detailed exploration of screening techniques for the pharmacological evaluation of antiulcer agents

Arunkumar Vaghela[1]*, Mital Dulera[2]

[1]Dept. of Pharmacology, Shree Aryatej Institute of Pharmacy Morbi, Gujarat, India.
[2]HN Shukla Institute of Pharmaceutical Education and Research, Rajkot, Gujarat, India.

Abstract

Peptic ulcers are a common gastrointestinal issue that can develop due to various lifestyle and dietary habits. Factors such as unhealthy eating patterns, smoking, frequent alcohol intake, prolonged use of medications like NSAIDs, and a generally sedentary lifestyle are all known contributors. One of the earliest warning signs is a dull or sharp pain in the upper abdomen, which may point toward ulcer formation. This condition occurs when there is a disruption in the balance between the stomach's protective mechanisms and harmful (aggressive) factors, resulting in sores in the lining of the oesophagus, stomach, or duodenum. Individuals may also experience alarming symptoms such as vomiting that resembles coffee grounds, blood in the stool, or black, tar-like bowel movements. This discomfort often worsens after meals and, if left untreated, typically leads individuals to consult a gastroenterologist. Multiple experimental models have been developed to screen and evaluate the antiulcer potential of various drug candidates. The primary goal of this review is to explore and identify the most reliable and effective screening models for assessing antiulcer activity. To gather relevant information, a systematic literature search was performed using reputable scientific databases, including, Science Direct, and Pub Med. Keywords like *"anti-ulcer," "in-vitro models,"* and *"in-vivo models"* were used to filter the search results. Further filters were applied to ensure that only high-quality and relevant research articles were included in this review. Our review covered a range of studies, including both original research and review papers, focused on various experimental models used to evaluate the antiulcer effects of new pharmaceutical compounds.

Keywords: Helicobacter pylori, NSAIDs, Cushing's ulcer, Cyst amine, Histamine, Proton pump inhibitors.

Received: 04-08-2025; **Accepted:** 28-10-2025; **Available Online**: 19-11-2025

1. Introduction

Peptic ulcer is a pathological condition characterized by a localized erosion in the mucosal lining of the stomach or the proximal segment of the small intestine, most commonly the duodenum. This lesion arises when the delicate balance between aggressive factors, such as gastric acid and digestive enzymes, and protective mechanisms, such as mucus and bicarbonate secretion, is disrupted. As a result, the mucosal surface becomes vulnerable to injury, leading to ulceration.[1]

Clinically, peptic ulcers are often associated with epigastric pain described as burning or gnawing in nature. The discomfort typically intensifies when the stomach is empty and may temporarily subside following food intake. Additional symptoms may include bloating, early satiety, nausea, or, in severe cases, gastrointestinal bleeding evidenced by hematemesis or melena.[1]

The etiology of peptic ulcer disease is multifactorial. One of the primary contributing factors is the presence of certain bacterial infections that compromise mucosal integrity. Furthermore, chronic exposure to irritants—whether environmental, dietary, or behavioural—can exacerbate mucosal damage. Lifestyle factors such as smoking, excessive alcohol consumption, poor dietary habits, and psychological stress may not directly cause ulceration but are known to influence disease progression and symptom severity.[1]

*Corresponding author: Arunkumar Vaghela
Email: drxavaghela@gmail.com

https://doi.org/10.18231/j.ijcaap.12714.1761733274

If left untreated, peptic ulcers may lead to serious complications, including haemorrhage, perforation of the gastrointestinal wall, and gastric outlet obstruction. Therefore, timely diagnosis and appropriate therapeutic interventions are essential to prevent morbidity and promote mucosal healing. (**Figure 1**)

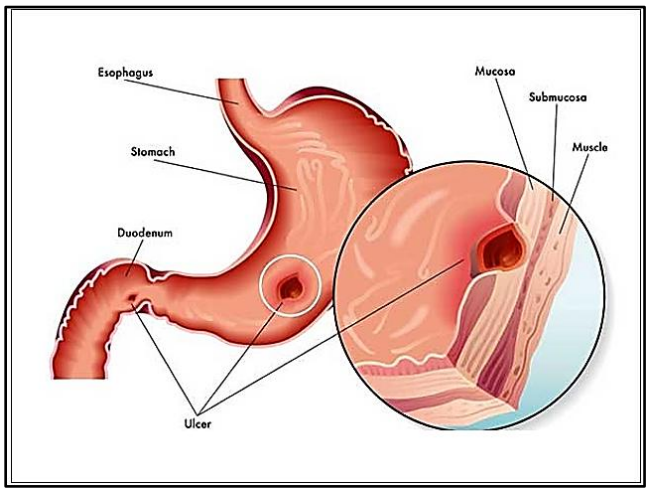

Figure 1: Ulcer in stomach

1.1. Sign and symptoms

Peptic ulcers can affect people in different ways, but one of the most common and noticeable symptoms is a burning or gnawing pain in the upper part of the stomach. This pain is often felt between meals or during the night when the stomach is empty. It may come and go, sometimes lasting for a few minutes, other times for several hours. Many people say the pain improves temporarily after eating something or drinking milk, only to return again later.[2]

In addition to stomach pain, people with peptic ulcers might experience a feeling of bloating or fullness, even after eating a small amount of food. Some also report frequent burping or mild nausea. In some cases, there's a noticeable loss of appetite, which can lead to unintentional weight loss over time.[2]

When an ulcer becomes more serious, other warning signs can appear. These may include vomiting—sometimes with blood—or black, tar-like stools, which can indicate internal bleeding. Severe ulcers may even cause sharp, sudden stomach pain, which could be a sign that the ulcer has created a hole in the stomach or intestinal lining. This is a medical emergency and requires immediate attention.[2]

While not everyone with a peptic ulcer experiences strong symptoms, any ongoing or unusual stomach discomfort should be discussed with a healthcare provider. Early diagnosis and treatment can help prevent complications and promote healing.

2. Discussion

2.1. Etiology

Peptic ulcers usually develop when the natural balance between the stomach's protective lining and the strong digestive acids it produces is disturbed. The stomach and upper part of the small intestine are lined with a protective layer that helps prevent damage from acid. But when this barrier is weakened or breaks down, the acid can start to eat away at the tissue beneath, leading to the formation of an ulcer.[3]

One of the most common causes of this damage is a bacterial infection, specifically by a bacterium called Helicobacter pylori (often shortened to H. pylori). This spiral-shaped bacterium can survive in the harsh, acidic environment of the stomach by burrowing into the lining. Over time, it causes inflammation and weakens the stomach's natural defences, making it easier for acid to cause injury. Interestingly, many people carry H. pylori without ever developing symptoms, but in others, it can lead to serious issues like gastritis and peptic ulcers.[3]

Another significant factor in ulcer development is the long-term use of certain medications that can irritate the stomach lining. These drugs can reduce the stomach's ability to produce protective mucus, making the lining more vulnerable to acid damage—especially when taken frequently or at high doses.

In addition to H. pylori infection and medication use, certain lifestyle choices and health conditions may increase the risk of developing ulcers or make existing ones worse. Smoking, for example, not only raises the risk of developing an ulcer but also slows down the healing process. Heavy alcohol use can irritate and erode the stomach lining, further increasing acid damage. While emotional stress and irregular eating habits don't directly cause ulcers, they can aggravate symptoms or delay healing in people who already have them.[3]

In most cases, peptic ulcers are the result of more than one factor acting together. Understanding these causes—especially the role of H. pylori—is important for both treating the ulcer and preventing it from returning.

2.2. Pathophysiology

Peptic ulcer disease (PUD) develops when there's a disturbance in the delicate balance between the stomach's aggressive digestive elements—like acid and pepsin—and the protective systems that shield the stomach and duodenal lining. The two main culprits behind this condition are infection with Helicobacter pylori and the long-term use of certain pain-relieving medications, both of which can significantly weaken the stomach's natural defenses. H. pylori interferes with the protective mucus layer, releasing harmful substances and increasing acid exposure to the underlying tissue, while these medications reduce the

production of prostaglandins, which are vital for maintaining the stomach's protective lining. Other factors like smoking and alcohol further strain the mucosal barrier by reducing blood flow, decreasing protective secretions, and irritating the stomach lining directly. While stress was once thought to be a major cause, current research suggests it's more likely to worsen symptoms rather than directly cause ulcers. Some people may also be genetically more prone to developing ulcers, making them more sensitive to these risk factors. Ultimately, when the damaging forces outweigh the body's ability to protect and repair the stomach lining, an ulcer can form, leading to the symptoms and complications associated with PUD.[4] (**Figure 2**,**Figure 3**)

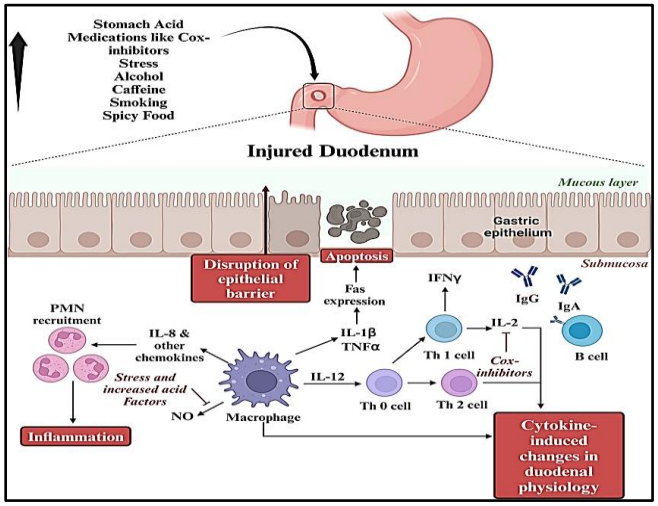

Figure 2: Explanation of pathophysiology of PUD5[5]

Classification of anti-ulcer medicine

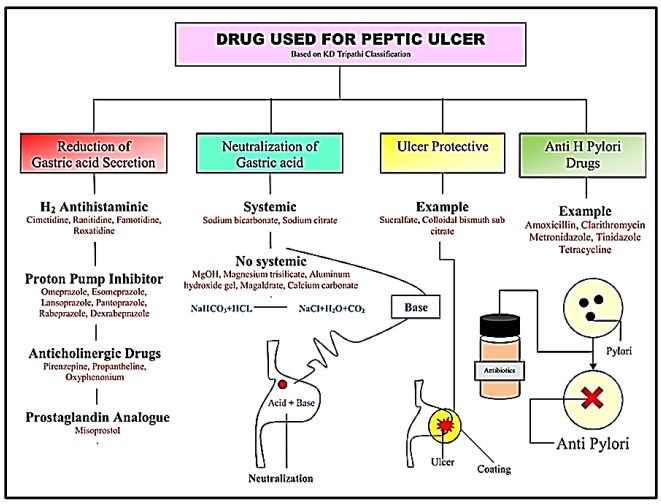

Figure 3: Drug used for peptic ulcer6[6]

2.3. Screening model

2.3.1. In vitro models for anti-ulcer screening

1. **Mucus secretion assay**: This model helps researchers evaluate whether a substance can stimulate the production of mucus from gastric cells. Since mucus acts as the stomach's first line of defense against acid and enzymes, its presence is essential for preventing ulcers.
   Process: Gastric epithelial cells (often cultured in lab dishes) are treated with the test compound. After a specific incubation period, the amount of mucus secreted is measured using dyes like alcian blue, which binds to mucus and allows quantification. An increase in mucus secretion suggests that the substance may offer protective benefits to the stomach lining.[7]
2. **Gastric cell viability assay (MTT or Trypan Blue Method)**
   This method focuses on the ability of a compound to protect stomach cells from damage caused by harmful agents such as alcohol, strong acid, or oxidative stress.
   Process: Gastric cells are exposed to a known damaging agent (like ethanol or hydrochloric acid), both with and without the test compound. Cell viability is then assessed using colorimetric assays like MTT (which turns purple in healthy cells) or by using trypan blue dye (which penetrates only dead cells). If more cells survive in the presence of the test compound, it suggests the substance has a protective or healing effect.[7]
3. **Urease inhibition assay**

Helicobacter pylori produces an enzyme called urease, which helps it survive in the acidic environment of the stomach by breaking down urea into ammonia, thus neutralizing acid. Inhibiting urease can reduce *H. pylori* survival and its ulcer-causing ability.
Process: Urease enzyme (from *H. pylori* or purified) is mixed with a urea solution and the test substance. If the compound inhibits urease activity, the amount of ammonia produced is reduced. This can be measured using color-change indicators. A lower ammonia level means stronger urease inhibition, indicating potential anti-ulcer action.[8]

4. **Anti-*H. pylori* activity (Disc Diffusion or MIC Assay)**
   Since *H. pylori* is one of the main causes of peptic ulcers, testing how a compound inhibits its growth is crucial.
   Process: A culture of *H. pylori* is spread on an agar plate, and the test substance is placed in wells or on paper discs on the surface. After incubation, the zone of bacterial growth inhibition is measured. A clear area around the disc suggests that the compound has antimicrobial effects against *H. pylori*. In the MIC (Minimum Inhibitory Concentration) method, different concentrations of the compound are tested in broth to find the lowest dose that stops bacterial growth.[8]
5. **Antioxidant assays (DPPH, ABTS, FRAP)**
   Although not ulcer-specific, antioxidants play a supportive role in ulcer healing, as oxidative stress contributes to mucosal damage.
   Process: In the DPPH method, for example, a purple solution of DPPH free radicals is mixed with the test compound. Antioxidants reduce the DPPH, causing the

solution to fade in color. The change in color is measured spectrophotometrically. A greater change means stronger antioxidant potential, which may support ulcer protection by reducing oxidative injury.[9]

6. **Prostaglandin E2 (PGE2) release assay**
   Prostaglandins are substances that help the stomach protect and repair its lining. NSAIDs often cause ulcers by reducing prostaglandin levels.
   Process: Gastric cells are treated with the test compound, and after incubation, the amount of PGE2 released is measured—often using ELISA kits. If the compound boosts prostaglandin release, it may help enhance mucosal defense and aid in healing or preventing ulcers.
7. **Mucin quantification assay**
   Mucin is a glycoprotein found in mucus, and its production is a direct marker of the stomach's ability to protect itself.

Process

Cultured gastric cells are exposed to the test compound, and mucin secretion is measured using dyes like alcian blue or periodic acid-Schiff reagent. Higher mucin levels suggest that the test compound strengthens the protective barrier against stomach acid and enzymes.[10]

8. **Gastric acid neutralization test (pH Titration Method)**
   Some anti-ulcer agents work by neutralizing excess stomach acid, making this a valuable screening method.
   Process: The test compound is added to a simulated gastric fluid (usually a solution with low pH), and the change in pH is monitored using a pH meter or indicator. If the pH increases significantly, it shows that the substance has acid-neutralizing properties, which could help relieve ulcer symptoms and prevent acid damage.
9. **In vitro wound healing assay (Scratch Assay)**
   This model simulates the healing process of damaged gastric tissue and assesses whether a substance promotes cell repair and regeneration.
10. **Process:** A single layer of gastric epithelial cells is grown in a dish, and a small "scratch" is made with a pipette tip to simulate an injury. The test compound is then added, and cell movement and coverage of the wound area are monitored over time under a microscope. Faster closure of the scratch area suggests enhanced healing potential.[10]

3. In Vivo Models for Anti-Ulcer Screening

3.1. Pylorus-Ligated (Shay Rat) Model

Principle

When the pylorus (the outlet of the stomach) is ligated, gastric contents including acid and pepsin accumulate because they can no longer pass into the small intestine. This leads to auto-digestion of the mucosa, creating ulcers. This model simulates hypersecretory conditions and impaired gastric emptying in humans.[11] (Fig 4)

Process

1. Animals (typically rats) are fasted for 18–24 hours with free access to water.
2. Under mild anesthesia (usually using ether or ketamine), a small abdominal incision is made.
3. The pyloric end of the stomach is carefully tied with silk thread, avoiding damage to nearby blood vessels.
4. The incision is sutured and the rat is allowed to recover in a controlled environment.
5. After 4–6 hours, animals are euthanized.
6. The stomach is dissected, contents are collected for volume, pH, and titratable acidity.
7. The mucosa is observed for ulceration, and ulcers are scored using a standard ulcer index.

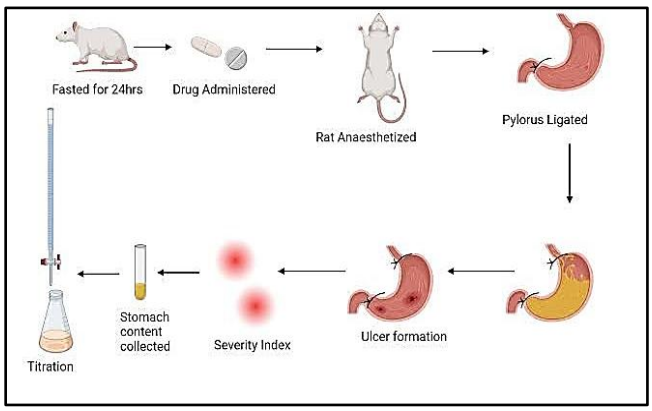

Figure 4: Pylorus-ligated (Shay Rat) Model[12]

3.2. Ethanol-induced ulcer model

Principle

Absolute ethanol penetrates the lipid layer of gastric epithelial cells, leading to necrosis, inflammation, and hemorrhagic lesions. It also generates oxygen-derived free radicals, further damaging tissue. This mimics the acute mucosal injury seen with excessive alcohol consumption or chemical exposure.[13] (**Figure 5**)

Process

1. Rats are fasted for 24 hours but have access to water to avoid dehydration.
2. The test compound is administered orally, usually 30–60 minutes before ethanol.
3. Ethanol (1 mL/200 g) is given orally using a gastric gavage.
4. After one hour, the animals are sacrificed.
5. The stomach is removed, opened along the greater curvature, rinsed with saline, and examined macroscopically.
6. Lesions are quantified using an ulcer scoring system (e.g., ulcer index = total area of ulcerated regions).

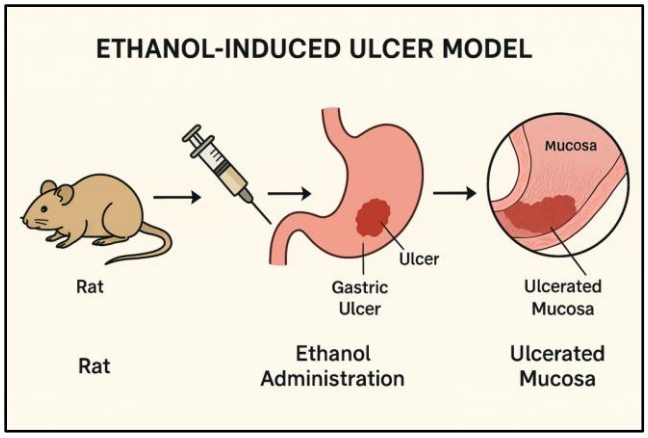

Figure 5: Ethanol-induced ulcer model

3.3.NSAID-induced ulcer model

Principle

NSAIDs like indomethacin inhibit cyclooxygenase (COX) enzymes, leading to a reduction in prostaglandins. These prostaglandins are essential for maintaining the mucosal defense by stimulating mucus and bicarbonate secretion, improving blood flow, and regulating cell turnover. Their inhibition compromises mucosal integrity, resulting in ulceration.[14] (**Figure 6**)

Process

1. Animals are fasted for 24 hours before the experiment.
2. The NSAID (e.g., indomethacin at 20–30 mg/kg) is administered orally or subcutaneously.
3. The test compound may be administered before or after the NSAID depending on whether protective or healing effects are being evaluated.
4. After 4–6 hours, rats are euthanized.
5. Stomachs are removed, opened, and inspected for ulceration and inflammation.
6. Ulcers are scored and sometimes histologically examined to confirm mucosal injury.

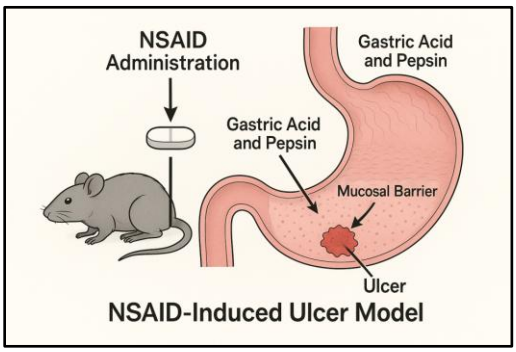

Figure 6: NSAIDS-induced ulcer model

4. Cold Restraint Stress-Induced Ulcer Model

4.1. Principle

Cold and physical restraint act as physiological stressors, triggering sympathetic nervous system activation and cortisol release. These responses reduce gastric mucosal blood flow, increase acid secretion, and weaken mucosal defenses, resulting in stress ulcers.[15] (**Figure 7**)

4.2. Process

1. Animals are fasted but hydrated.
2. They are placed in restraint devices such as metal cylinders or immobilized on boards.
3. They are then exposed to a cold environment (4°C) for 2–4 hours.
4. The test drug is given 30 minutes prior to stress exposure.
5. After the stress period, animals are euthanized.
6. The stomach is excised and examined for mucosal haemorrhages or ulcerative lesions.

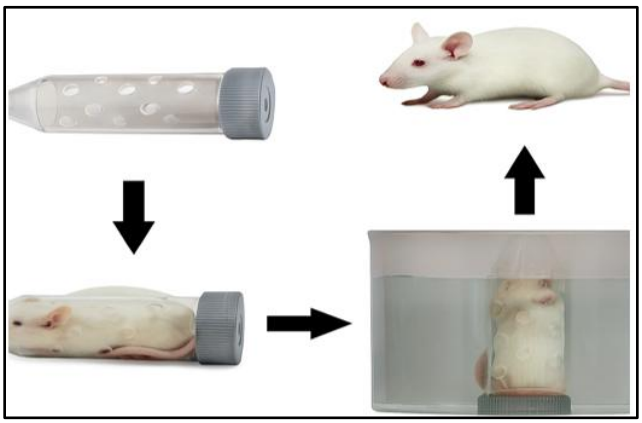

Figure 7: Cold restraint stress-induced ulcer model

4. Acetic Acid-Induced Chronic Ulcer Model

4.1. Principle

Topical application of acetic acid to the serosal surface causes localized necrosis and inflammation, mimicking the pathology of chronic gastric ulcers. This model allows long-term observation of the healing process and regenerative potential of test agents.[16]

Process

1. Under anesthesia, a midline laparotomy is performed to expose the stomach.
2. A glass ring is placed on the serosal surface and filled with 0.05 mL of glacial acetic acid for 60 seconds.
3. The acid is carefully removed and the area is rinsed with saline.
4. The abdominal wall is sutured in layers.
5. The test compound is administered daily (for 7–14 days).

6. On completion, the stomach is examined for healing, and histopathological analysis is performed.

5. Hydrochloric Acid–Ethanol-Induced Ulcer Model

Principle
The combination of HCl and ethanol potentiates mucosal damage. HCl disrupts the mucus barrier, and ethanol extracts lipids and increases oxidative stress. This synergistic effect causes intense hemorrhagic lesions.[17]

Process

1. Rats are fasted for 24 hours with water ad libitum.
2. The test compound is administered orally.
3. After 1 hour, a mixture of 0.3 M HCl and 60% ethanol (1:1 ratio) is administered.
4. One hour later, animals are sacrificed.
5. The stomach is removed, washed, and examined under magnification for lesion severity and ulcer area.

6. Water-Immersion Stress Model

Principle
Submersion in cold water while under restraint combines physical and thermal stress. The resulting vasoconstriction and increased acid secretion mimic real-world stress-related ulcer conditions in humans.[17]

Process

1. Animals are placed in cylindrical restrainers and immersed vertically in cold water (20–25°C) up to the neck.
2. The immersion lasts for 4–6 hours.
3. The test drug is given orally 30 minutes prior.
4. Post-exposure, the animals are euthanized and stomachs examined for ulcers.
5. Both ulcer index and biochemical markers (e.g., MDA for oxidative stress) may be assessed.

7. Cysteamine-Induced Duodenal Ulcer Model

Principle
Cysteamine administration causes increased gastric acid and pepsin output, and induces oxidative stress and inflammation in the duodenum. It produces chronic, deep ulcers suitable for healing studies.[17]

Process

1. Rats are fasted and given two oral doses of cysteamine hydrochloride (400 mg/kg), 4 hours apart.
2. The test substance may be given before or after induction.
3. After 24 hours, animals are sacrificed and the duodenum is opened longitudinally.
4. The number and severity of ulcers are recorded, and tissue may be collected for histology and biochemical assays.

8. Helicobacter pylori-Infected Model

Principle
Infection with H. pylori leads to persistent inflammation, mucosal damage, and increased gastrin secretion. This mimics human peptic ulcer disease and is ideal for testing both antimicrobial and mucosal healing activities.[17]

Process

1. Animals (mice or gerbils) are pre-treated with an antacid to increase gastric pH.
2. They are orally infected with a live culture of *H. pylori* for 3–5 consecutive days.
3. After infection is established (confirmed by biopsy or stool antigen test), treatment with the test compound begins.
4. After 1–3 weeks, animals are euthanized.
5. Stomachs are assessed for ulcers, bacterial load (via urease test or PCR), and histological changes.

9. Histamine or Serotonin-Induced Ulcer Model

Principle
Histamine acts on H2 receptors and serotonin on 5-HT receptors to stimulate gastric acid secretion. These models help evaluate the antisecretory effects of test compounds.[17]

Process

1. Animals are fasted for 18–24 hours.
2. Histamine (5 mg/kg) or serotonin (1 mg/kg) is administered subcutaneously.
3. The test compound is given prior to stimulant injection.
4. After 4–6 hours, stomachs are collected, contents are analyzed for acidity, and ulceration is assessed.

9.1. Ulcer scoring and ulcer index

Ulcer scoring remains a fundamental method for evaluating the extent of gastric damage. Ulcers are observed macroscopically, and lesions are scored based on severity—ranging from no lesions (score 0) to perforated ulcers (score 3 or more). The Ulcer Index (UI) quantifies this damage using the following formula:[18]

Ulcer Index (UI) = (UN + US + UP) × 10^{-1}
Where:
UN = Average number of ulcers per animal
US = Average severity score
UP = Percentage of animals with ulcers
This index offers a consolidated numeric representation of ulcer severity for statistical comparison.

9.2. Measurement of gastric volume and pH

After experimental treatment and sacrifice, gastric contents are collected and measured. *Gastric volume* (in mL) indicates the amount of secreted fluid, and *pH* is measured using a digital pH meter. A lower volume and a higher pH suggest that the test drug reduces acid secretion, which is beneficial in managing peptic ulcers. This method is often coupled with acidity measurements for a complete profile.[18]

9.3. Estimation of total and free acidity

To determine the level of acid secretion, gastric juice is titrated with 0.01 N NaOH using Topfer's reagent and phenolphthalein. The formula used is:

Acidity (mEq/L) = Volume of NaOH (mL) × Normality of NaOH × 100 / Volume of gastric juice (mL) This gives the total acidity. Free acidity is determined when only Topfer's endpoint is considered. A significant decrease in acidity levels in test groups suggests anti-secretory potential.[18]

9.4. Evaluation of mucus production

The stomach's mucus layer is gently scraped and weighed or stained using dyes like alcian blue to estimate mucosal protection. The mucus adherent weight (mg) or dye-binding capacity provides a quantifiable index of mucus content. A higher mucus level in treated animals implies better cytoprotection and mucosal defense.

9.5. Histopathological assessment

Stomach tissues are preserved in 10% formalin, embedded in paraffin, sectioned, and stained with Hematoxylin and Eosin (H&E). Microscopic analysis helps evaluate epithelial erosion, inflammatory infiltration, edema, and ulcer depth. Although no formula is used, a semi-quantitative scoring system is often applied to rate histological damage, aiding in statistical evaluation.[19]

9.6. Oxidative stress and antioxidant markers

Markers such as malondialdehyde (MDA) indicate oxidative damage, while superoxide dismutase (SOD), glutathione (GSH), and catalase reflect antioxidant defenses.

For MDA (via TBARS method):

MDA (nmol/mg protein) = (Absorbance × Molar extinction coefficient^{-1} × dilution factor)
Increased antioxidant levels and reduced MDA in test groups demonstrate the protective role of antioxidants against ulcer-induced oxidative stress.19[19]

9.7. Microbial assessment for Helicobacter pylori

When studying H. pylori-infected models, gastric tissues are cultured or analyzed using PCR or urease tests. Bacterial reduction is expressed as:

% Reduction = [(CFU control – CFU treated) / CFU control] × 100
where CFU = colony-forming units. A significant reduction reflects the antibacterial efficacy of the test agent, aligning with histological improvements.[19]

9.8. Calculation of healing or protection rate

The protection or healing percentage gives a straightforward measure of the treatment effect:

% Protection = [(Ulcer Index control – Ulcer Index treated) / Ulcer Index control] × 100[19]

10. Conclusion

Based on the analysis of the collected literature, it was observed that certain experimental models offer significant advantages in antiulcer drug testing. Moreover, combining both in-vitro and in-vivo screening methods may provide a more accurate and comprehensive assessment of a drug's antiulcer potential, enhancing the reliability of research outcomes in this field.

11. Source of Funding

None.

12. Conflict of Interest

None.

References

1. Mishra AP, Bajpai A, Chandra S. A Comprehensive Review on the Screening Models for the Pharmacological Assessment of Antiulcer Drugs. *Curr Clin Pharmacol.* 2019;14(3):175-196. https://doi: https://doi.org/10.2174/1574884714666190312143846
2. Kajihara Y, Shimoyama T, Mizuki I. Evaluation of the effects of a proton pump inhibitor on Helicobacter pylori stool antigen testing. Helicobacter. 2023;28(3):e12961. https://doi:10.1111/hel.12961.
3. Hojgaard L, Nielsen AM, Rune SJ. Peptic Ulcer pathophysiology: Acid bicarbonate, and mucosal function. *Scandinavian J Gastroenterol*, 1996; 31(1): 10-15.
4. Snowdex FM. Emerging and re-emerging: "Historical and perspective". Immunological Reviews, 2008; 225: 9-26.
5. Isenberg JI, Flemstrom G. Physiology and pathophysiology of gastro duodenal bicarbonate secretion. *Diges Diseases.* 1991;23:27-33.
6. Agurav M, Rimpi A, Shaba P. Study Different Experiment Models Used In Peptic Ulcer. *Int J Rec Adv Pharma Res.* 2015;5(2):31-37.
7. Shirisha B, Subash V. Animal Models in Experimental Gastric Ulcer Screening -A Review. *Int Res J Pharm*. 2012; 3(6): 57-77.
8. Graham DY, Rakel RE, Fendrick AM. Recognizing peptic ulcer disease: keys to clinical and laboratory diagnosis. Postgraduate *Med J.* 1999; 105(3):113-33.
9. Harsh Mohan. Text book of pathology. New Delhi: Jaypee publishers; 2009. 549-555 p.
10. Mahajan N, Sanghai D. Evaluation of anti-uler potential of leaves of Jasminumgrandiflorum L. International

11. Hojgaard L, Nielsen AM, Rune SJ. Peptic ulcer pathophysiology: Acid bicarbonate, and mucosal function. *Scand J Gastroenterol*. 1996; 31(1):10-15.
12. Snowdex FM. Emerging and re-emerging: "Historical and perspective". *Immunol Rev.* 2008; 225: 9-26.
13. Isenberg JI, Flemstrom G. Physiology and pathophysiology of gastro duodenal bicarbonate secretion. *Diges Dis*. 1991;23: 27-33.
14. Agurav M, Rimpi A, Shaba P. Study Different Experiment Models Used In Peptic Ulcer. *Int J Recent Adva Pharma Res.* 2015; 5(2): 31-37.
15. Shirisha B, Subash V. Animal Models in Experimental Gastric Ulcer Screening -A Review. *Int Res J Pharm*. 2012;3(6): 57-77.
16. Graham DY, Rakel RE, Fendrick AM. Recognizing peptic ulcer disease: keys to clinical and laboratory diagnosis. *Postgraduate Med J*. 1999; 105(3):113-33.
17. Mohan H. Text book of pathology. New Delhi: Jaypee publishers; 2009. 549-555 p.
18. Mahajan N, Sanghai D. Evaluation of anti-uler potential of leaves of Jasminumgrandiflorum L. International Hojgaard L, Nielsen AM, Rune SJ. Peptic ulcer pathophysiology: Acid bicarbonate, and mucosal function. Scandinavian Journal
19. Hojgaard L, Nielsen AM, Rune SJ. Peptic ulcer pathophysiology: Acid bicarbonate, and mucosal function. Scandinavian Journal
20. Hojgaard L, Nielsen AM, Rune SJ. Peptic ulcer pathophysiology: Acid bicarbonate, and mucosal function. Scandinavian Journal
21. Snowdex FM. Emerging and reemerging: "Historical and perspective". Immunological Reviews, 2008; 225: 9- 26.
22. Comprehensive insights on treatment modalities with conventional and herbal drugs for the treatment of duodenal ulcers - Scientific Figure on ResearchGate. Available from: https://www.researchgate.net/figure/Explains-the-pathophysiology-of-duodenal-ulcers-The-diagram-depicts-the-various-stages_fig2_381191769. 2025
23. Isenberg JI, Flemstrom G. Physiology and pathophysiology of gastro duodenal bicarbonate secretion. *Diges Dis.* 1991;23:27-33. https://doi:10.1111/j.1365-2036.1994.tb00319.x
24. Agurav M, Rimpi A, Shaba P. Study Different Experiment Models Used In Peptic Ulcer. *Int J Recent Adv Pharma Res*. 2015;5(2): 31-7.
25. Shirisha B, Subash V. Animal Models in Experimental Gastric Ulcer Screening -A Review. *Int Res J Pharm*. 2012;3(6):57-77. https://doi:10.1111/j.1365-2036.1994.tb00319.x
26. Graham DY, Rakel RE, Fendrick AM. Recognizing peptic ulcer disease: keys to clinical and laboratory diagnosis. *Postgrad Med J*. 1999; 105(3):113-33. https://doi:10.3810/pgm.1999.03.594.
27. Ludovico B, Eric S, Annemarthe G, Van DV, Rino R, Antonello C. Helicobacter Pylori cytotoxinassociated gene A subverts the apoptosis-stimating protein of p53ASPP 2tumor suppressor pathway of the host. *Proce Nat Acad Sci USA*. 2011;108(22):9238–43. https://doi:10.1073/pnas.1106200108
28. https://www.google.com/url?sa=i&url=https%3A%2F%2Fwww.myschoolexams.com.ng%2F2025%2F04%2F13%2Fthe-study-of-anti-ulcer-activity-of-a-drug%2F&psig=AOvVaw3nNemZS4q-1QKkz00JC_3y&ust=1753344667590000&source=images&cd=vfe&opi=89978449&ved=0CBUQjRxqFwoTCMDj-ITE0o4DFQAAAAAdAAAAABAE
29. Martin JB. Not all Helicobacter pylori strains are created equal; should all be liminated. *The Lancet*. 1997;349:1020- 22. https://doi:10.1016/S0140-6736(96)09133-7.
30. Fan XG, Kelleher D, Fan XJ, Xia HX, Keeling PW. Helicobacter pylori increases proliferation of gastric epithelial cells. *Gut*. 1996;38(1):19-22. https://doi.org/10.1136/gut.38.1.19.
31. Arige SD, Lakshmana Rao A. A Review on Pharmacological Screening of Anti-Ulcer Agents. *Int J Med Lab Res*. 2017:2(3):32-42.
32. Chey WD, Wong BC. American College of Gastroenterology guideline on the anagement of Helicobacter pyloriinfection. *Am J Gast.* 2007;102(8):1808-15. https://doi:10.1111/j.1572-0241.2007.01393.x
33. Ford AC, Delaney BC, Forman D, Moayyedi P. Eradication therapy for peptic ulcer disease in Helicobacter pylori positive patients. *Cochrane Databa Syst Rev*. 2006;19(2):38-40. https://doi:10.1002/14651858.CD003840.pub5.
34. Szabo S, Vincze A, Sandor Z, Jadus M, Gombos Z, Pedram A. Vascular approach to gastroduodenal ulceration. New stud-ies with endothelins and VEGF. *Digestive Dis Sci.* 1998;43(9):40-5
35. Talley NJ, Vakil N. American gastroenterological association technical review on the evaluation of dyspepsia. Gastroenterology. 2005;129(5):1756-80. https://doi:10.1053/j.gastro.2005.09.020.

Cite this article: Vaghela A, Dulera M. A detailed exploration of screening techniques for the pharmacological evaluation of antiulcer agents. *IP Int J Compr Adv Pharmacol*. 2025;10(3):144-151.