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Review Article

Gastric Acidity, Mucosal Integrity, and Ulceration: A Review of the Puzzles

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Abstract: Peptic Ulcer Disease, PUD, is a major public concern, globally, and affects every age, sex, race, and socioeconomic status. There is growing interest in phytomedicinal treatment of PUD. Patients' compliance to orthodox drugs management of the disease declines. The psycho-economic burden worsens. The ulcers mostly occur in the stomach and proximal duodenum, characterized by mucosal damage, predominantly caused by *Helicobacter pylori*, antiplatelet agents, nonsteroidal anti-inflammatory drugs (NSAIDs), alcohol consumption, and cigarette smoking. Symptoms of peptic ulcer disease include abdominal pain, vomiting, reflux symptoms, and loss of appetite and weight. The disease may lead to upper gastrointestinal haemorrhage and perforation or may progress to carcinoma. The stomach itself contains hydrochloric acid, but does it cause tissue injury, very frequently? What may be the best approach to the management of PUD and the complications? We have therefore attempted a review of history, available findings, recent advances, and challenges in gastrointestinal tract research.

Keywords: Gastric Mucosa, Barrier, Acidity, Ulcer.

1.0 INTRODUCTION

Among the viscera, the stomach is one of the earliest to have been described by priests, physicians, and anatomists, and to have been studied functionally by alchemists, chemists, and physiologists. The ancient EGYPTIANS recognized the gross anatomy and the infirmity of the stomach; at the time of burial, it was preserved separately in one of the four so-called ''canopic'' jars. HIPPOCRATES (460 – 370 BC) called digestion ''PEPSIS,'' likening it to cooking, and proposing that the heat of the stomach was responsible for the breakdown of food.

2.0 PHYSIOLOGY AND HISTORY

In 1547, Andreas Vesalius, in his *De Humani Corporis Fabrica*, provided anatomically correct descriptions of the human gastric structure and function as well as the intestines. In 1648, observations of animal digestion led J.B. van Helmont to postulate that different kinds of acids might play a role in digestion, calling them ferments. In the 1780s, Lazzaro Spallanzani published

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his *Dissertationi de Fisica Animale e Vegetale* where he described digestion as the chemical process of dissolution produced by vital organs. Spallanzani had experimental subjects, including himself, swallow enclosed receptacles (linen bags or perforated metal tubes). He observed that, over time, the contents disappeared from the receptacle and postulated the involvement of acid.

In 1823, Prout, Tiedemann, and Gmelin each independently identified the acid in the stomach as HYDROCHLORIC ACID. In 1833, International excitement and acclaim followed the publication, by the American army surgeon William Beaumont, of his Observations on the Gastric Juice and the Physiology of Digestion. In 1964, Davenport working on gastric mucosal injury by fatty and acetylsalicylic acids, demonstrated: "A mucosa through which acetic, propionic, butyric, or acetylsalicylic acid is diffusing develops increased fluxes of hydrogen, sodium, potassium, and chloride ions and the increases persist after the compounds are removed. The changes are chemical signs of injury, and desquamation and bleeding may accompany them".

Later on, still in 1964, Davenport *et al.,* on the functional significance of gastric mucosal barrier to sodium, showed that: "Normal gastric mucosa of man, dogs, cats, and rats offers a nearly complete barrier to the passage of sodium ions. The ready permeability to sodium of the mucous membranes in the rest of the alimentary canal suggests that the impermeability of the gastric mucosa to this ion has functional significance".

3.0 BUT, WHY DOES THE STOMACH NOT DIGEST ITSELF?

In I972, Davenport published the article, "*Why Does the Stomach not Digest Itself?*". Thirty-three years later, Wallace J L implicated prostaglandins and NSAIDs in the breakdown of gastric mucosal protection.

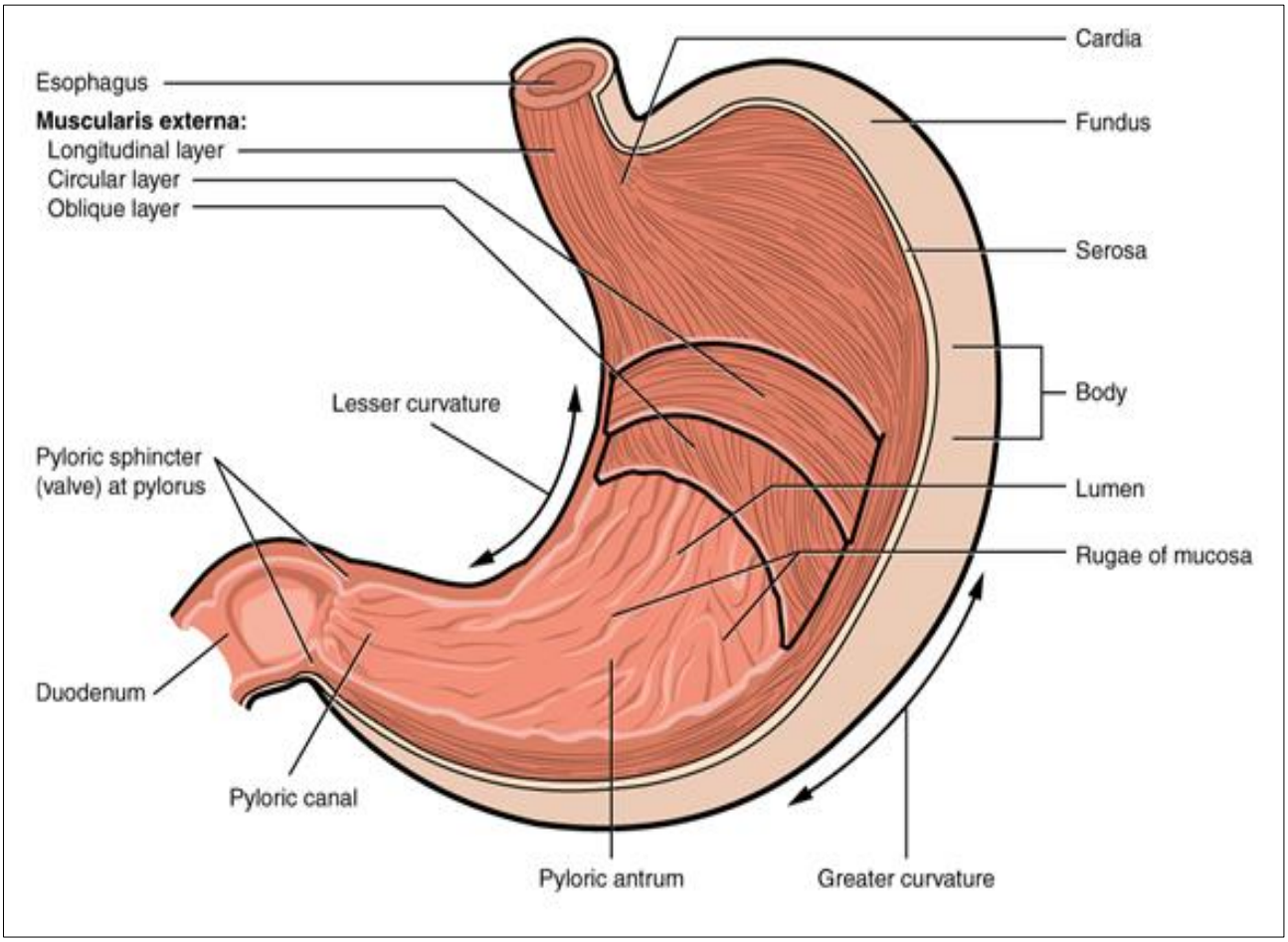

Fig. 1: Physiologic anatomy of the Stomach (Source: OpenStax, 2023).

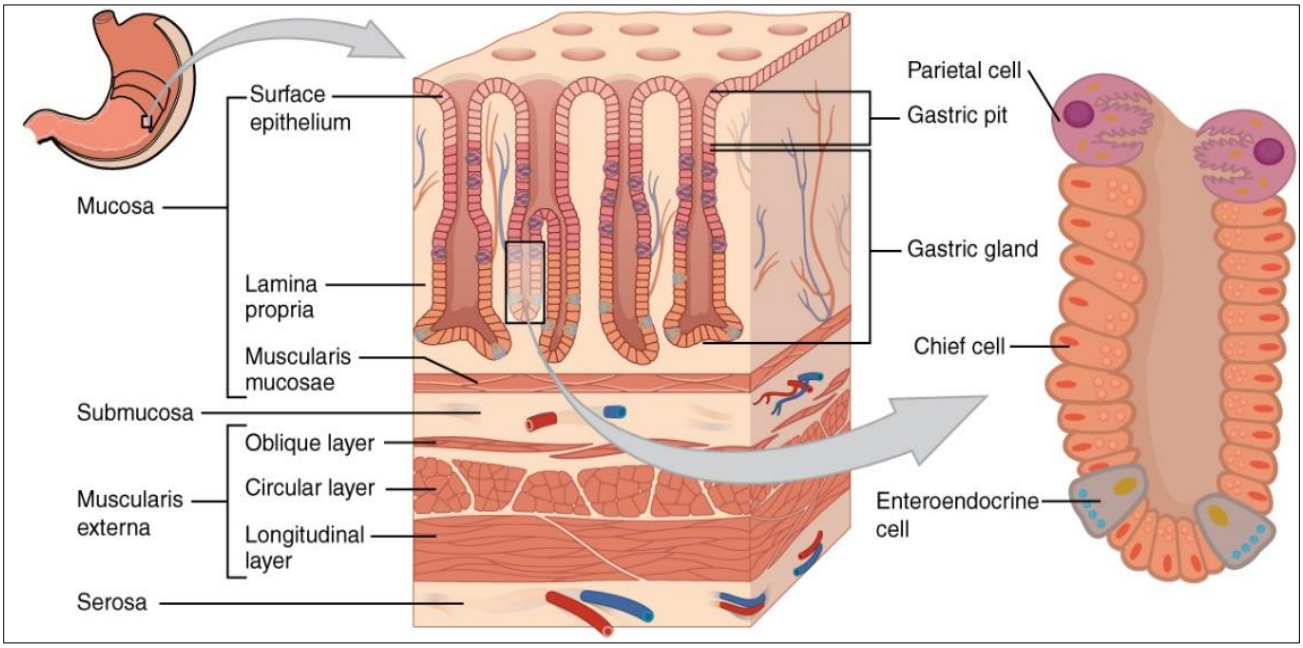

Fig. 2: Histology of the Stomach (Source: OpenStax, 2023).

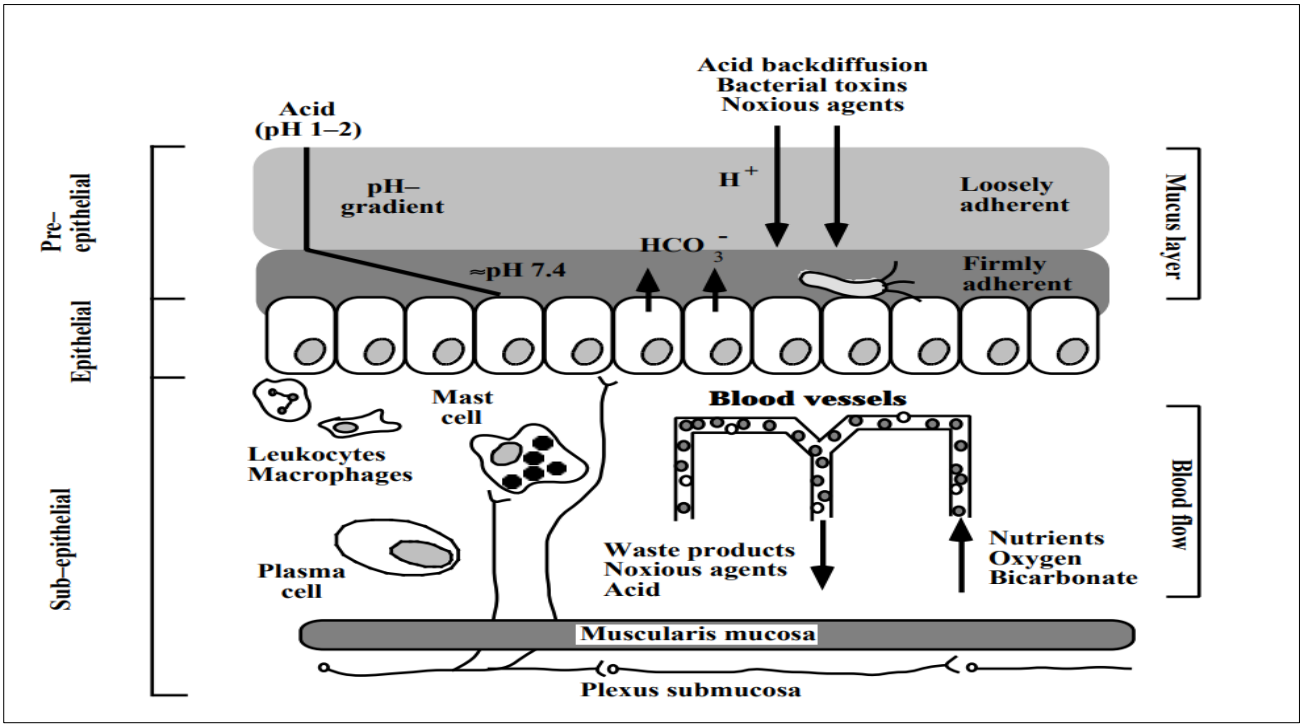

Fig. 3: Postulated Three Levels of Mucosal Defense (Source: Atuma, 2000).

Table 1: Gastric Mucosal Glandular Secretions

GASTRIC MUCOSAL GLANDULAR SECRETIONS

REGION	MAIN SECRETIONS	MINOR SECRETIONS	MAJOR GLANDULAR CELLS
PYLORIC ANTRUM	MUCUS GASTRIN	 PEPSINOGEN SOMATOSTATIN	MUCUOS CELLS G-CELLS MUCUOUS CELLS D CELLS
BODY	HYDOCHLORIC ACID INTRINSIC FACTOR PEPSINOGEN	 MUCUS HISTAMINE SEROTONIN	PARIETAL / OXYNTIC CELLS PARIETAL / OXYNTIC CELLS CHIEF/PEPTIC/ZYMOGENI C MUCUOUS CELLS ENTEROCHROMAFFIN CELLS ARGENTAFFIN CELLS
CARDIAC	MUCUS		MUCUOUS CELLS
FUNDUS	HYDOCHLORIC ACID INTRINSIC FACTOR PEPSINOGEN HISTAMINE		PARIETAL / OXYNTIC CELLS PARIETAL / OXYNTIC CELLS CHIEF/PEPTIC/ZYMOGEN ENTEROCHROMAFFIN

Table 2: Properties of Gastric Juice

PROPERTIES OF GASTRIC JUICE
Osim, 2002

1. pH : 0.8 – 2.5
2. Specific gravity: 1.002 – 1.004
3. Daily Secretion: 3000ml
4. Secretion Rate (fasting): 74ml/ hr
5. Secretion Rate (post -prandial): 100ml/ hr
6. Water Content: 99.5 %
7. Major Cations: H+, Na+, Mg2+,
8. Major Anions: Cl-, HPO4-, HCO3-
9. Enzymes: Pepsinogens, Carbonic Anhydrase, Rennin , Gastric Amylase & Lipase,
10. Hormone: Gastrin
11. Others: Mucus, Intrinsic Factor, Blood Group Substances

Ordinarily, gastric acidity is corrosive enough to dissolve a piece of metal. Gastric hydrochloric acid, HCL, with pH of about 0.8 – 2.5, kills bacteria, digests meats and some other particulate matter. The puzzle is, 'Why does the HCl not digest the stomach wall, always?' The answer is found in the integrity of the 'gastric mucosal barrier'.

4.0 INTEGRITY OF THE GASTRIC MUCOSAL BARRIER

The stomach wall histologically and molecularly is composed of the following properties: tight Junctions between the Cells, mucus, trefoil peptides, highly regenerative gastric mucosal cells, mucosal blood flow, prostaglandins and a layer of surface-active phospholipids in epithelium.

4.1 Presence of Tight Junctions (TJ) Between Cells Protects Gastric Epithelium

As a result, there is minimal leakage of Hydrogen ions, Sodium Ions, Interstitial fluid, through intact TJs.

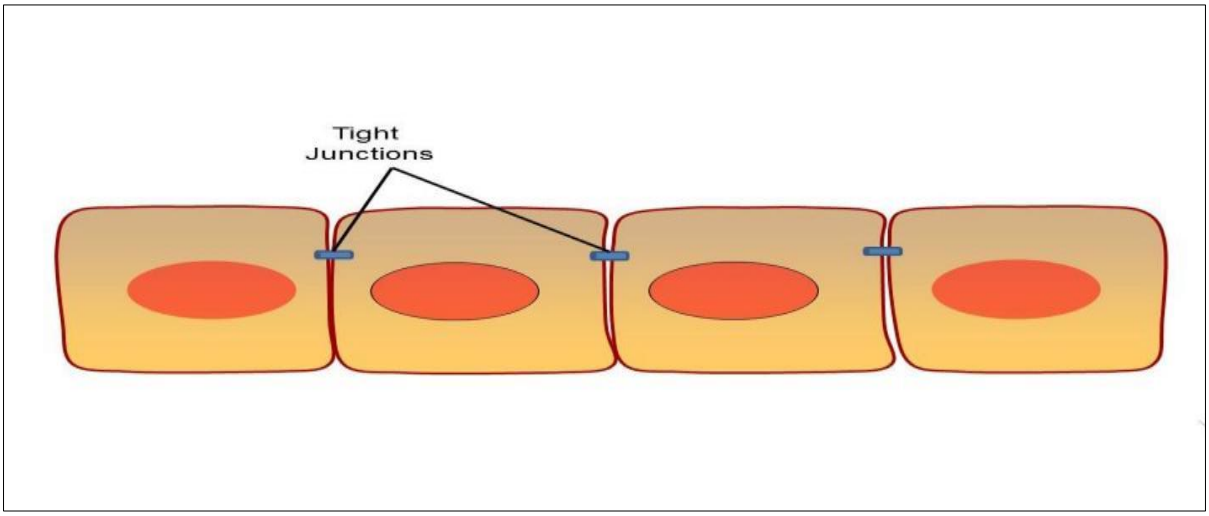

Fig. 3: Tight Junctions between adjacent cells in the stomach mucosa (Source: OpenStax, 2023)

4.2 Layer of Mucus is Gastroprotective

There is copious secretion of mucus by the mucus neck cells and surface mucus cells. The Mucus contains the glycoprotein MUCIN. Mucus cells also secrete HCO_3^- trapped in the mucus gel, creating a pH gradient with a pH of about 1-2 at the luminal side of the Gastric Mucosa, and a pH of about 7 at the surface of the epithelial cells.

4.3 Presence of Trefoil Peptides in Gastric Mucosa

Trefoil Factor Family, TFF, has a domain of 38 or 39 amino-acid residues that share a conserved motif, including three disulfide bonds that stabilize a well-defined three-loop-structure reminiscent of a trefoil. Secreted by mucus cells, the Trefoil Proteins are acid resistant protecting, maintaining, repairing the gastrointestinal tract. Therapeutic potential to treat and prevent a variety of gastrointestinal disorders associated with mucosal damage. Their mechanisms at the molecular level remain poorly understood.

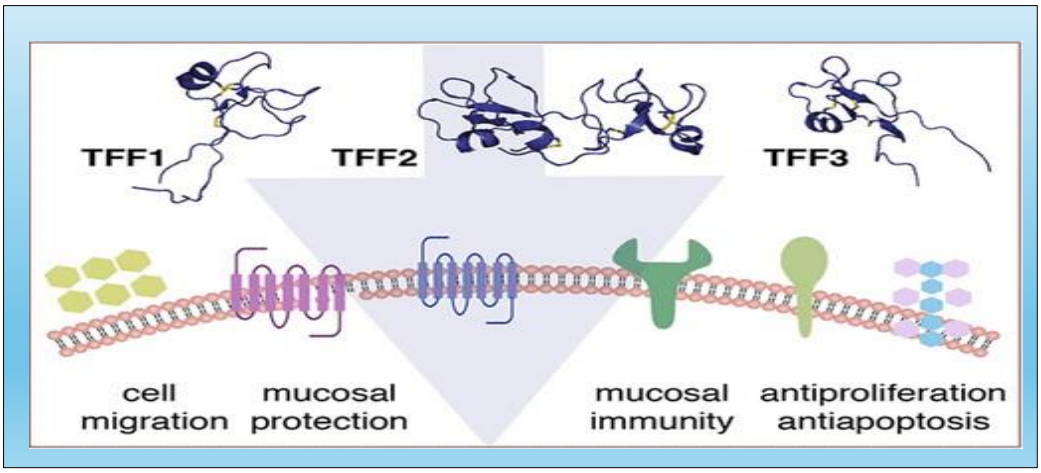

Fig. 4: Variety of Trefoil Proteins and their Biofunctions (Source: ACS Pharmacol, 2020)

4.4 The Presence of Highly Regenerative Cells in the Stomach

Gastric mucosal cells are highly regenerative. About 0.5 million cells/min are shed from the gastric surface lining. But this lining is completely renewed within 2-4 days. Thus, wound healing is greatly promoted, tissue repair is significantly enhanced and tissue injury does not easily degenerate to ulceration.

4.5 Gastric Mucosal Blood Flow is Adequate, Significantly

There is increased delivery of blood containing bicarbonate ions, $HC0_3^-$ to mucosa. This prevents local acidosis. The gastric arterioles penetrate the muscularis mucosae and the capillaries run in-between and parallel to the oxyntic glands to supply adequate amount of blood. This replenishes the supply of platelets, as well as factors for tissue growth, repair and healing.

4.6 Presence of Prostaglandins

The presence of certain prostaglandins confers a degree of cytoprotection. Found in a variety of cells in gastric mucosa, mast cells, macrophages, endothelial cells, prostaglandins are biosynthesized from arachidonic acid via the action of cyclo-oxygenase (COX) enzyme. Prostaglandins are known to cause decreased hydrochloric acid secretion, increased bicarbonate ion secretion and enhanced mucus production. They also regulate mucosal blood flow.

4.7 Presence of Phospholipids

Phospholipids are hydrophobic and acid resistant. Gastric epithelial cells contain a lipid bilayer that contains surface-active phospholipids. They confer a degree of hydrophobicity to the cell surface. For example, phosphatidylcholines demonstrate resistance to acidic attack.

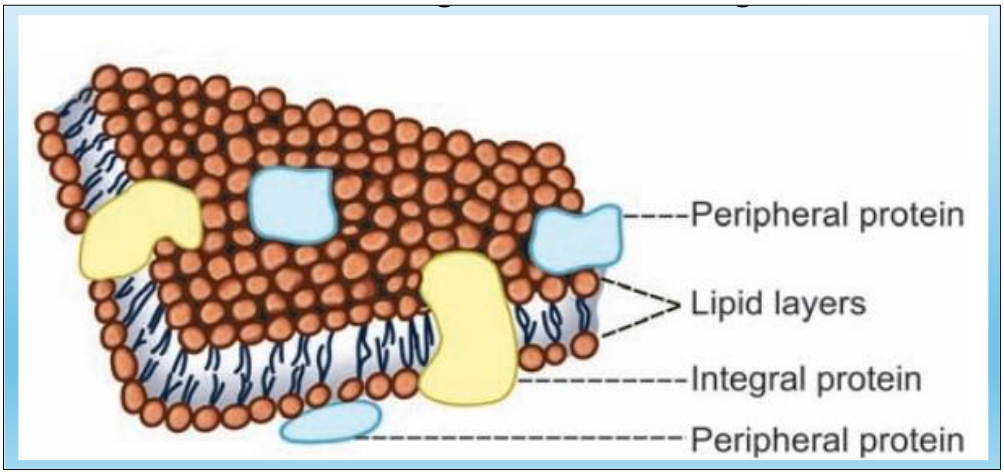

Fig. 5: Fluid Mosaic Modal of Epithelial Cell Membrane (Source: Sembulingam, 2012)

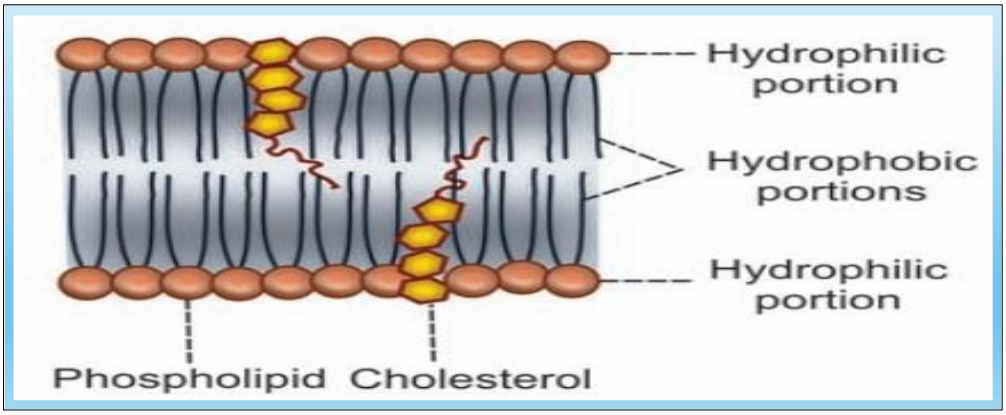

Fig. 6: Lipid Bilayer Model of the Gastric Epithelial Cell Membrane (Sembulingam, 2012)

5.0 AETIOLOGICAL FACTORS FOR THE BREAKDOWN OF GASTRIC MUCOSAL BARRIER

The gastric mucosal barrier, notwithstanding, the gastric mucosa barrier may be broken by many factors. These include, but not limited to, the following.

5.1 Aliphatic Compounds

These are unionized and fat-soluble moieties. They can easily diffuse through the cell membrane into the gastric mucosa. Examples are Butyric acid, Acetic acid, and Propionic acid.

5.2 Salicyclic Acid and Acetylsalicylic Acid in Acidic Solution

In acidic solution, salicylate and acetylsalicylate are unionized. They, therefore become fat-soluble, and diffuse *via* cell membrane into the gastric mucosa. They possess the propensity to kill the surface epithelial cells (cytotoxicity) and thus break the barrier. In neutral solution, however, they are ionized and so, absorbed slowly and may not cause damage. For example, in the presence of gastric hydrochloric acid, Aspirin (an NSAID) inhibits cyclo-oxygenase enzyme resulting in the decreased biosynthesis of Prostaglandins and consequent poor mucus production and release.

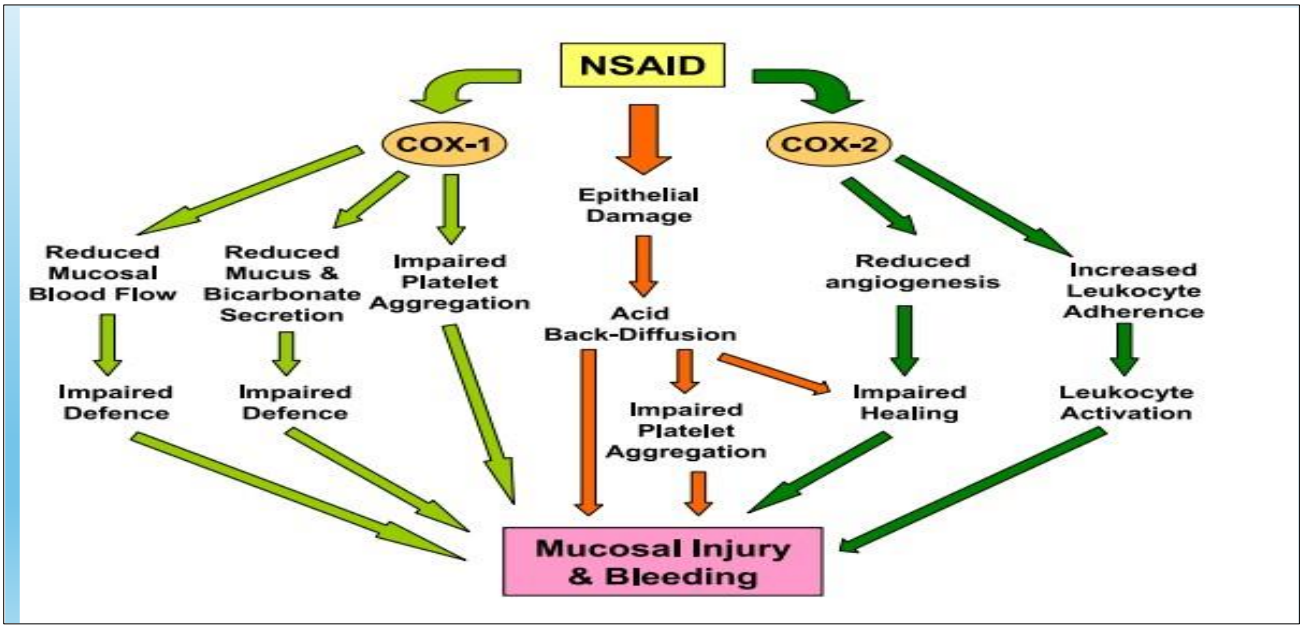

Fig. 7: Pathways of Mucosal Damage by Non-Steroidal Anti-Inflammatory Drugs, NSAIDs (Source: Wallace, 2008)

5.3 Infection with a Specific Bacterium: *Helicobacter Pylori*

In 1875 this specific bacterium was isolated as a spiral bacterium, in the margins of gastric ulcers (Atuma 2000). In 1982, it was originally named as *Campylobacter pyloridis* (Warren & Marshall, 1983) Subsequently, it was renamed as H*elicobacter pylori* (Goodwin *et al.*, 1989). H. pylori is Gram negative bacterium and colonizes the gastric epithelium. It is a major risk factor for gastritis, gastric ulcer, and gastric cancer. 50% of the population is positive; 10% of this manifest the disease. *Others are symptomatic. The b*acterium is commonly acquired during childhood and follows the individual as a chronic infection throughout life (Atuma, 2000).

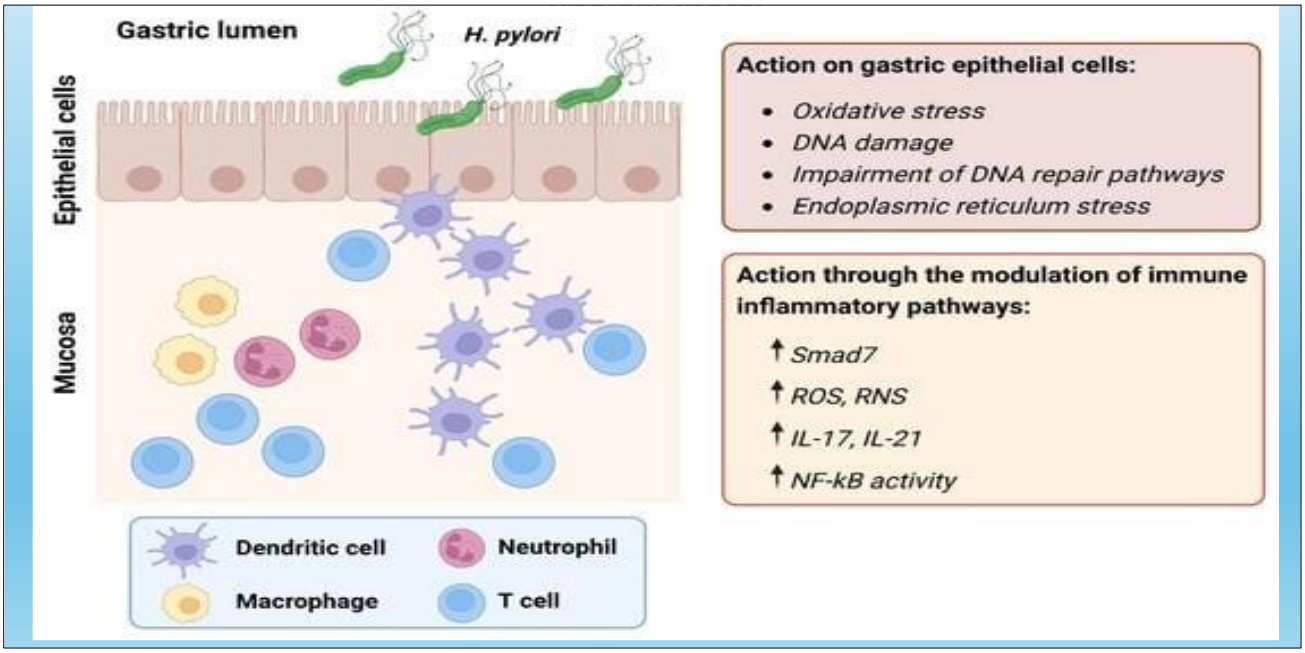

Fig. 8: *H. pylori* Direct and Indirect Actions on the Stomach Mucosa (IJMS. 2023)

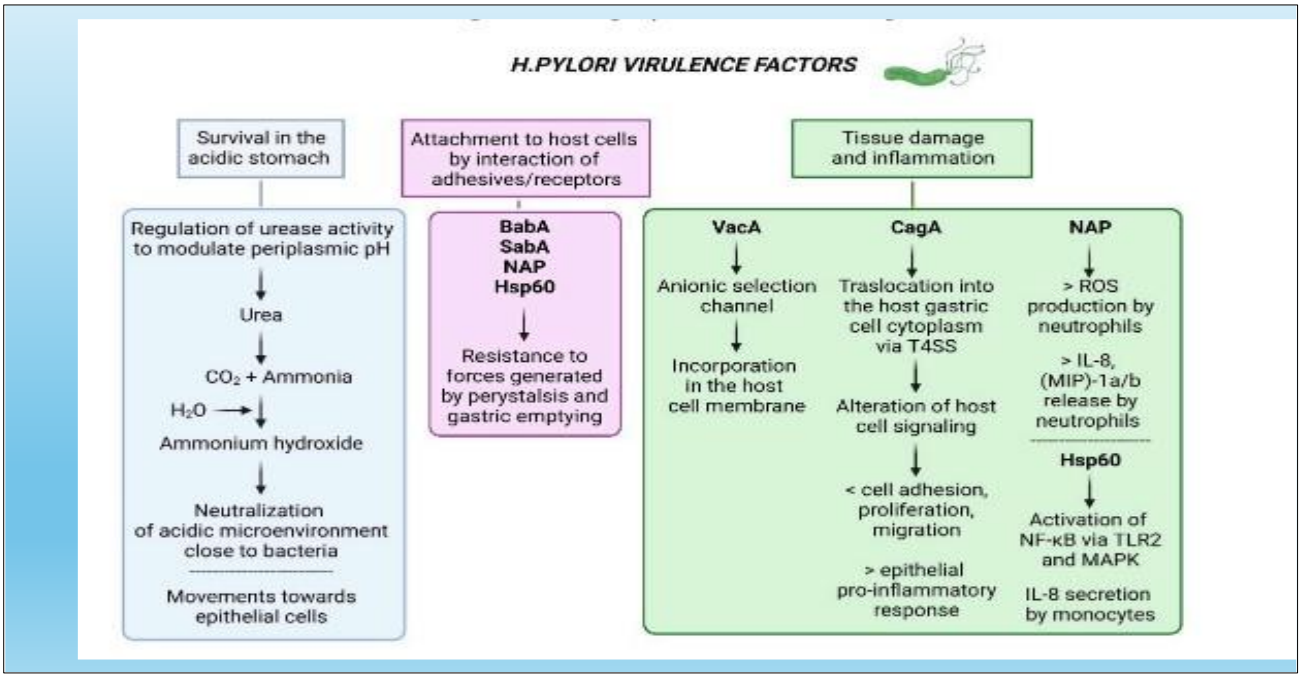

Fig. 9: *H. pylori* Virulence Factors (IJMS, 2023)

5.4 COMPOUNDS WITH DETERGENT ACTIVITY

Natural Detergents – e.g. Bile acids or bile salts and lysolecithin (from lecithin *via* hydrolysis by *phospholipase A*) break mucosal barrier by emulsifying fats and rendering them water-soluble. Regurgitated Bile salts provoke gastric ulcer by detergent action.

Synthetic Detergents – e.g. Omo, Klin, Ariel act like Bile acids and lysolecithin. Sodium Lauryl Ether Sulphate, SLES, a common component of commercial detergents, has high cleansing, surfactant, properties. They dissolve lipids of cell membranes resulting in *gastric ulceration.*

5.5 Alcohol, Caffeine and Tobacco

Consumption of substances like alcohol, caffeine and nicotine has been implicated in the breakdown of gastric mucosa. In the presence of gastric acidity, ethanol is absorbed rapidly. Aspirin, in the presence of both ethanol and gastric acid is very dangerous. Alcohol, caffeine and nicotine are known to dissolve the lipid content of cell membrane leading to gastric ulceration.

5.6 Ischaemia

Ischemia in the presence of gastric acid and bile acids is very damaging to the stomach wall.

5.7 Prolonged Excess Secretion of Acid

There is increase in both gastric secretion and gastric hydrochloric acid output in cases of gastrinomas, as in Zollinger Ellison Syndrome. In these circumstances, tumours of gastrin secreting cells are replete. Incidence of gastric ulceration is high.

5.8 Some Other Factors That Risk Breaking the Gastric Mucosal Barrier

The integrity of the gastric mucosal barrier is greatly challenged by traumatic accident, naturally weak Gastric Mucosa Barriers, hydrogen ions, pepsin, severe illness, nervous stress.

6.0 WHAT IS THE EFFECT OF ACID ON NORMAL AND BROKEN MUCOSAL BARRIER?

Hydrogen ions, H+, from Hydrochloric acid cause no harm if the barrier is intact. When the barrier is broken, the H+ ions penetrate the underlying cells and stimulate them to release HISTAMINE which in turn stimulates the secretion of more and more acid. This causes the blood vessels in the mucosa to dilate and become more permeable. Fluid leak out also, causing OEDEMA. H+ ions also attack the blood vessels. Concurrent CHOLINERGIC stimulation raises blood pressure. HAEMORRHAGE may set in. Nerve cells in the Gastric wall, stimulated by acid, cause stomach wall to contract, leading to PAIN.

7.0 WHAT ARE GASTRIC ULCERS?

Gastric ulcer is a break in the mucosa of the stomach lining. It has a BASE, MARGINS and WALLS. The break penetrates through the muscularis mucosa and extends more than 5 mm in diameter. It altered defense mechanisms of the stomach.

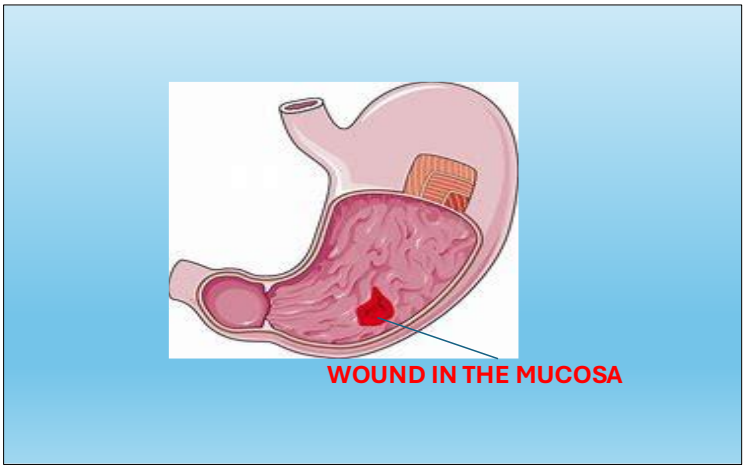

Fig. 10: Wound in the Gastric Mucosa, Stomach Ulceration (OpenStax, 2023)

7.1 Changes in the Gastric Mucosa

Inflammation leads to erosion which subsequently results in desquamation or ulceration.

7.2 Tendency to Transform to Gastric Carcinoma

Gastritis transforms to gastric ulcer. Ulcer may advance to gastric cancer.

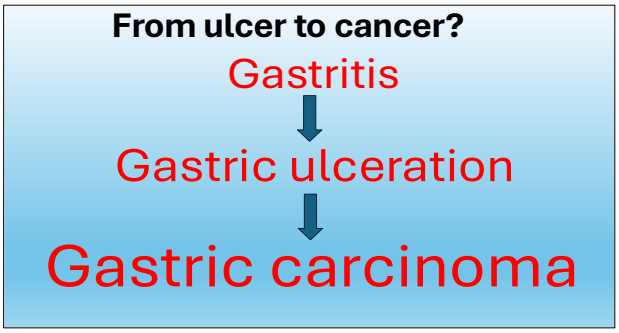

Fig. 11: Progression from gastritis to Cancer

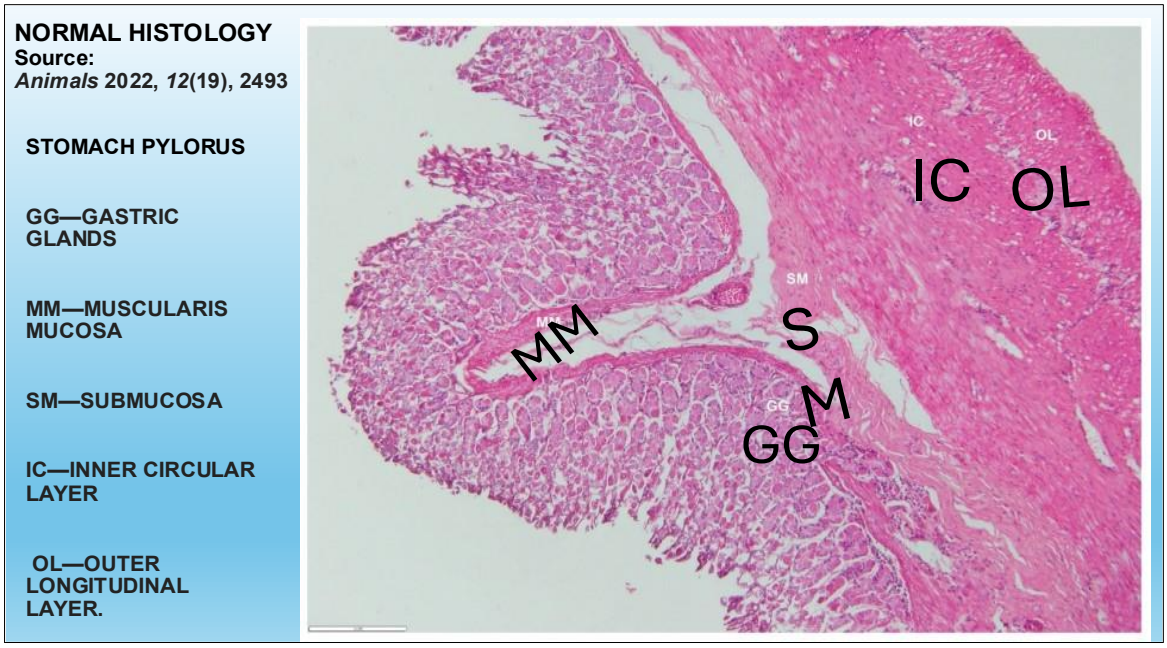

Fig. 12: Micrograph showing histological features of the stomach pylorus (Source: *Animals* 2022)

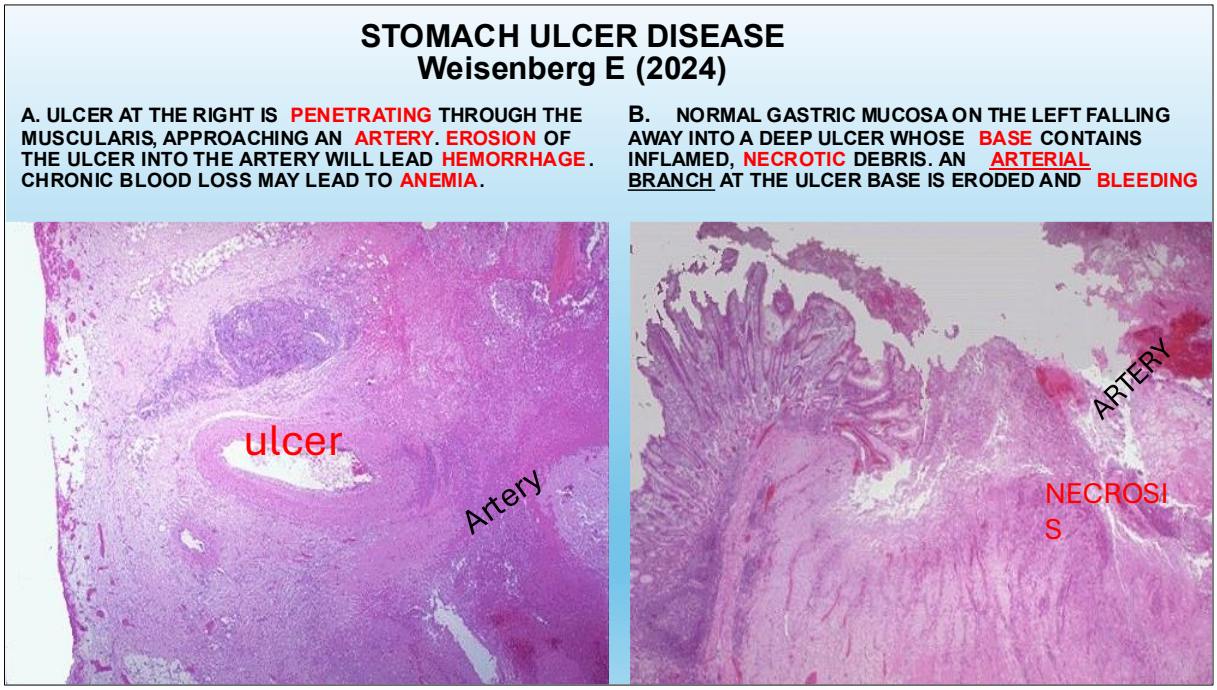

Fig. 13: Peptic Ulcer Disease, A, versus Normal Stomach Mucosa (Source: Weisenberg, 2024)

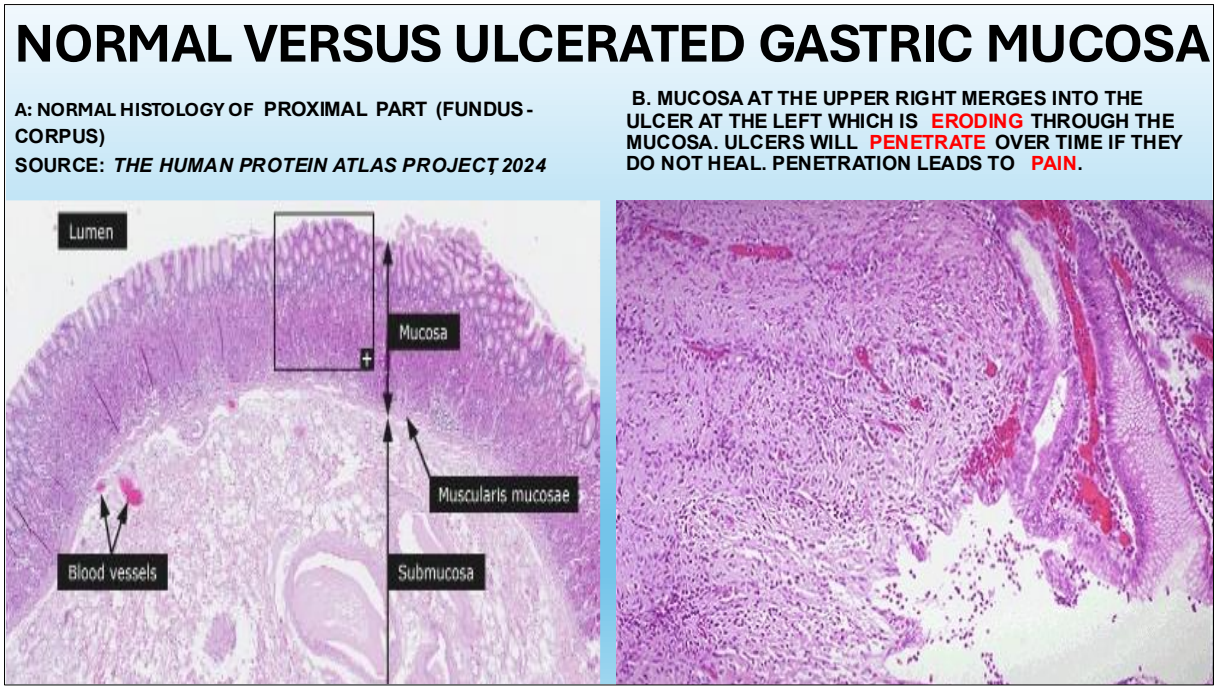

Fig. 14: Micrograph of Normal (A) and Ulcerated Gastric Mucosal tissue (The Human Protein Project, 2024)

8.0 WHAT IS THE PHYSIOLOGICAL BASIS OF THE TREATMENT OF GASTRIC ULCER?

Orthodox medical treatment of gastric ulcer involves the novel triple therapy. At other times, surgical intervention is applied. The tables below (Table 3and 4) summaries the various approaches in the treatment of peptic ulcer (gastric or duodenal ulcer).

Table 3: Physiologic basis of Treatment of peptic Ulcer (A)

Physiologic Basis of Treatment for Gastric Ulcer

S/N	THERAPY	OPTION	EXAMPLE
1.	ERADICATION OF *H. pylori* INFECTION	TRIPLE THERAPY	A.PROTON PUMP INHIBITORS,, PPIs e.g. OMEPRAZOLE B. ANTIBIOTICS 1: CLARITHROMYCIN OR AMOXICILLIN C. ANTIBIOTICS 2: METRONIDAZOLE
2.	SUPPRESSION OF GASTRIC ACID SECRETION	HISTAMINE, H2, BLOCKERS MUSCURANIC,M2, BLOCKERS H-K-ATPase BLOCKERS	CIMETIDINE, RANITIDINE, FAMOTIDINE ATROPINE, PIREZIPINE PPI e.g. OMEPRAZOLE
3.	NEUTRALIZATION OF ACIDITY	ANTACIDS	AMMONIUM HYDROXIDE MAGNESIUM HYDROXIDE CALCIUM CARBONATE
4.	INCREASING MUCOSAL RESISTANCE TO ACID	ALUMINON SALT OF SUCROSE OCTASULFATE	SUCRALFATE

Table 4: Physiologic Basis of treatment of Peptic Ulcer Disease (B)

Physiologic Basis of Treatment for Gastric Ulcer (2)

S/N	THERAPY	OPTIONS	EXAMPLES
5	WITHDRAWAL OF OFFENDING SUBSTANCES	NSAIDs REMOVAL AVOID TOBACCO SHUN ALCOHOLISM CAFFEINE ADJUSTMENT	ASPIRIN, INDOMETHACIN CIGARETTE SMOKING ETHANOL DRINKING COFFEE
6	PREVENTION AND TREATMENT OF MUCOSAL DAMAGE	PROSTAGLANDIN AGONISTS	PGE ANALOGUES e.g. MISOPROSTOL
7	SURGICAL INTERVENTION	PARALYSIS OF PARASYMPATHETIC DISCHARGE REMOVAL OF GASTRIN-SECRETING MUCOSA	VAGOTOMY GASTRECTOMY

9.0 GASTRIC ULCER TREATMENT: RECENT TRENDS IN PHYTO-MEDICINAL RESEARCH

The adverse side effects of conventional drugs, the economic cost implication and challenges in accessing medical attention have attracted recent trends in phyto-medicinal research. Figure 15 shows result of investigations in Malaysia: *M. pruriens* was found to ameliorate ethanol-induced gastric mucosal injuries.

There is growing interest in trado-medical and phyto-medicinal management to peptic ulcer disease, in Nigeria, and some other parts of the world.

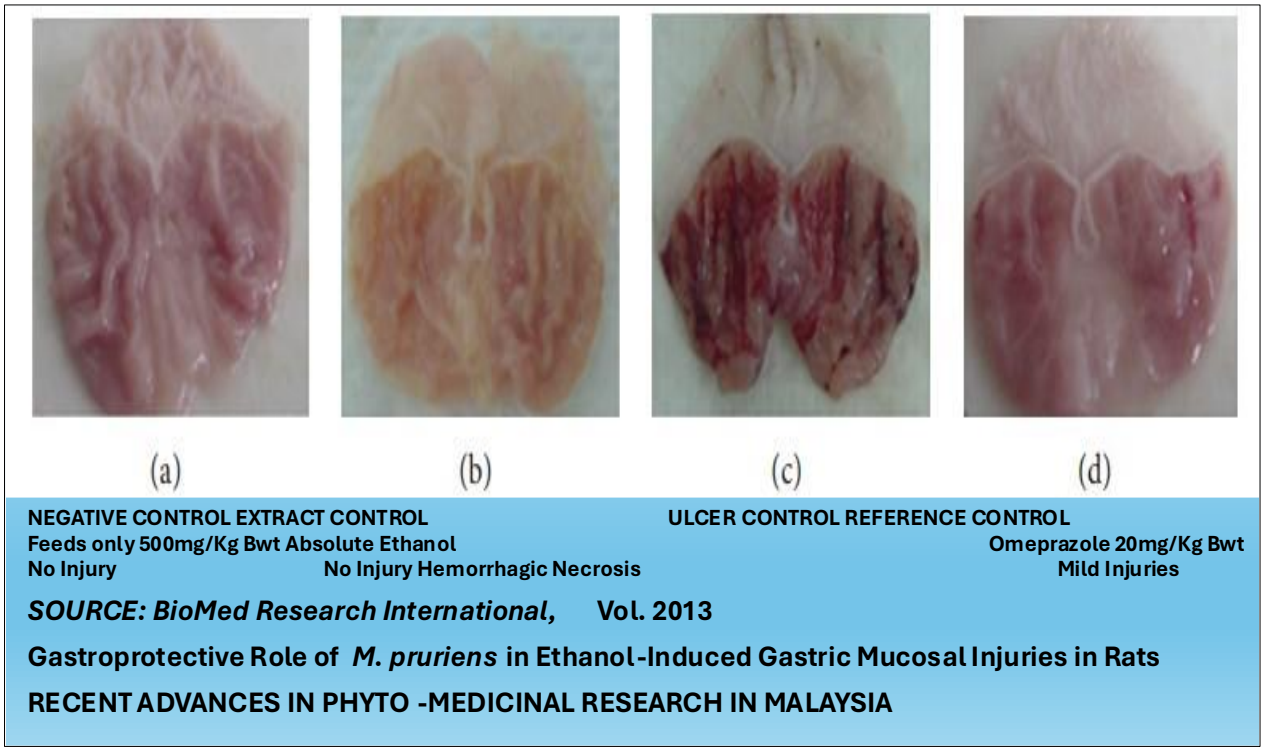

Fig. 15A: Gastroprotective Role of *M. pruriens in Ethanol induced* gastric mucosal injuries in rats (BMRI, 2013)

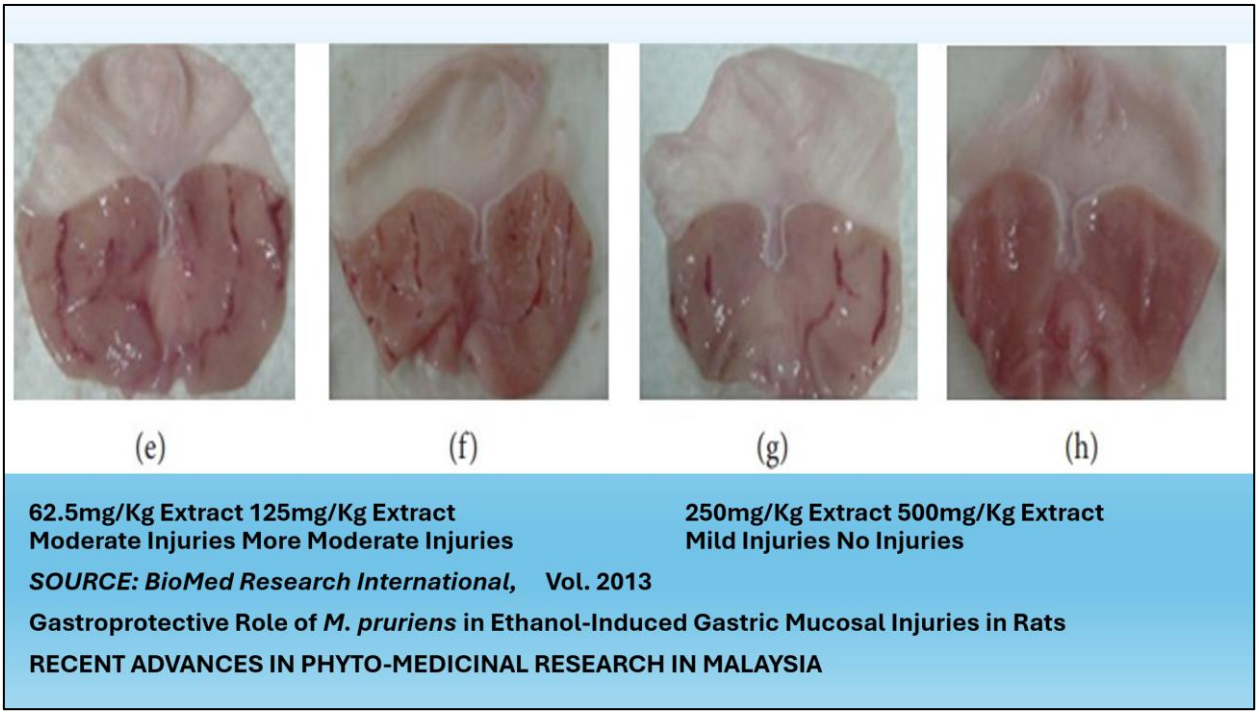

Fig 15B Gastroprotective Role of M. pruriens in Ethanol induced gastric mucosal injuries in rats (BMRI, 2013)

11.0 CONCLUSION

The gastric acid is corrosive enough to dissolve metals. It is also true that the stomach does not digest itself under normal circumstances. Yet, the gastric mucosal lining could break under otherwise untoward conditions. Gastrointestinal Tract Science is evolving by the day. There is therefore more to learn, to investigate, and to unravel.

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Authors' Contribution: All authors contributed equally to the data acquisition. K W drafted the paper. All authors read and approved the final paper.

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