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IMBALANCE BETWEEN AGGRESSIVE AND PROTECTIVE FACTORS IN GASTRIC MUCOSAL INJURY

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Peptic ulcers, which include both gastric and duodenal ulcers, are complex gastrointestinal conditions with significant health risks. They primarily arise from a disruption in the balance between protective and harmful factors within the stomach and duodenum, particularly in areas exposed to gastric acid and digestive enzymes. When this equilibrium shifts mainly due to reduced mucus and bicarbonate defenses or increased acid and pepsin activity, the integrity of the mucosal lining deteriorates. This breakdown leads to tissue injury as the protective mechanisms become overwhelmed. Consequently, treatment strategies focus on reducing gastric acid secretion and enhancing mucosal protection to promote healing and prevent further damage.

Nonsteroidal anti-inflammatory drugs (NSAIDs) and Helicobacter pylori infection are recognized as the primary causes of peptic ulcer disease and related gastrointestinal bleeding. Research using mouse models has shown that hecogenin acetate (HA) can markedly reduce ulcerated areas produced by harmful agents such as absolute ethanol or by ischemia reperfusion injury. These findings highlight HA's strong anti-ulcer and tissue-repair properties, demonstrated by both reduced lesion formation and increased collagen deposition. Ulcer development is largely driven by a disruption in the balance between damaging factors mainly gastric acid and pepsin, and the protective mechanisms of the mucosal barrier. When these defenses are weakened, the mucosa becomes more vulnerable to injury. H. pylori further intensifies this imbalance by altering gastrin regulation and stimulating acid secretion, contributing to gastritis and the formation of duodenal ulcers. Earlier treatment strategies focused almost exclusively on reducing gastric acid output, often overlooking the essential roles of mucus and bicarbonate in maintaining mucosal protection. Current understanding emphasizes the need for broader therapeutic approaches, including agents that enhance mucosal defenses or stimulate the release of protective substances. Disturbances in prostaglandin synthesis also play a significant role in ulcer pathology. Prostaglandins are vital for maintaining mucosal integrity, and their deficiency increases susceptibility to acid and pepsin induced damage. Evidence consistently shows that inhibitors of cyclooxygenase, the enzyme responsible for prostaglandin production can promote ulcer formation, reinforcing the importance of preserving adequate prostaglandin levels for gastric protection.

Peptic ulcer formation arises from a complex interplay between systemic regulators such as prostaglandin production, protective elements like mucus and bicarbonate, and damaging influences including gastric acid and pepsin. Emerging insights into healing mechanisms particularly those demonstrated by compounds such as hecogenin acetate suggest promising therapeutic avenues that strengthen mucosal protection and support tissue repair rather than focusing solely on acid suppression. A clear understanding of how these factors interact is essential for developing effective strategies to prevent and manage injuries to the gastric mucosa.