Regulation of gastric acid secretion: Acetylcholine, gastrin, and histamine.

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Introduction

Gastric acid secretion

The acid-secreting pump hydrogen-potassium-adenosine triphosphatase (ATPase) is found in the parietal cell. Acetylcholine, gastrin, and histamine are the major cues that control gastric acid secretion by the parietal cell. Several processes are involved in the production of gastric acid output. Separately from the cytoplasm of parietal cells, chloride and hydrogen ions are secreted and combined in the canaliculi. The gastric acid is subsequently produced into the gastric gland lumen, where it eventually reaches the main stomach lumen. Because acid must first traverse the relatively pH-neutral gastric mucus layer, the exact way released acid reaches the stomach lumen is debatable.

Acetylcholine

The current review highlights the clinical and fundamental scientific literature from the previous year on the physiologic and pharmacologic regulation of stomach acid secretion in health and sickness. The hormone gastrin, released by antral G cells, the paracrine agent histamine, released by oxyntic enterochromaffin-like cells, and the neuropeptide acetylcholine, released by antral and oxyntic intramural neurons, are the principal stimulants of acid secretion. Gastrin is a trophic hormone that plays a role in cancer development. Depending on the intensity and major anatomic focus of infection, Helicobacter pylori can increase or reduce acid output; most patients experience hypochlorhydria. Despite the fact that proton pump inhibitors (PPIs) are among the most commonly prescribed medications, they are underused in individuals who are at high risk of UGI bleeding [1].

PPIs have been linked to dementia, kidney disease, myocardial infarction, pneumonia, osteoporosis, dysbiosis, small intestinal damage, vitamin deficiency, and abdominal and pelvic gland polyps, despite the fact that they are normally well tolerated [1].

Gastrin and Control of gastric acid secretion

The discovery of histamine H(2)-receptors and the development of histamine H(2)-receptor antagonists, the identification of H(+)K(+)-ATPase as the parietal cell proton pump and the development of proton pump inhibitors, and the identification of Helicobacter pylori as the major cause of duodenal ulcer and the development of effective eradication regimens are all recent milestones in the understanding of gastric acid secretion and The importance and relevance of stomach acid secretion and its regulation in health and sickness are highlighted in this study. The physiology and pathology of acid secretion, as well as the evidence for inhibiting it in the treatment of acid-related clinical disorders, are discussed [2].

Control of protein digestion

Protein digestion, as well as the absorption of iron, calcium, vitamin B(12), and several medicines, are aided by gastric acid (e.g. thyroxin). It also kills ingested bacteria and protects against bacterial overgrowth, intestinal illness, and maybe spontaneous bacterial peritonitis. Histamine, gastrin, acetylcholine, and ghrelin are all acid secretion stimulants. Somatostatin, nefstatin-1, interleukin-11, and calcitonin gene-related peptide are all inhibitors. Depending on the time course of infection and the location of the stomach that is primarily infected, Helicobacter pylori stimulates or inhibits acid secretion. Acute infection activates calcitonin gene-related peptide sensory neurons, inhibiting histamine and acid production at the same time. In patients using proton pump inhibitors, serum chromogranin A, a marker for neuroendocrine tumours, is raised [3].

Control of histamine regulation

For almost 20 years, it was thought to be the same as the antral factor gastrin. It stimulates the parietal cells, which produce acid, via an H2-receptor in a dynamic way, meaning that histamine is continuously exchanged at the receptor site. The stimulation of receptors produces cyclic AMP, however it is unclear how this is linked to proton transport. It's perhaps plausible that the major function of histamine action is to kickstart the parietal cells' dramatic morphologic alteration that occurs in response to acid secretion. Some acid inhibitors' possible locations have been identified [4].

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