# Gastric acid secretion

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Secretion of gastric acid and pepsin tends to run in parallel, although the understanding of the mechanisms of gastric acid secretion is considerably greater than that of pepsin. Numerous factors are involved to some degree in the production of the gastric acid. These include neurotransmitters, neuropeptides and peptide hormones. This complexity need not detract from the fact that there are basic principles that are relatively easily understood ( Figure 67.4 ). Hydrogen ions are produced in the parietal cell by the proton pump. Although numerous factors can act on the parietal cell, the most important of these is hista - mine, which acts via the H -receptor. Histamine is produced, in 2 turn, by the ECL cells of the stomach and acts in a paracrine (local) fashion on the parietal cells. These relationships explain why PPIs can abolish gastric acid secretion, as they act on the final common pathway – hydrogen ion secretion. H -receptor 2 - antagonists have profound e ff ects on gastric acid secretion, but this is not insurmountable. The ECL cell produces histamine - - -

Distension of stomach M 1 Chemical in food Release of acetylcholine HCI Gastric ECL cell G cell Release of Release of gastrin histamine M 3 G H 2 Figure 67.4 The parietal cell in relation to the mechanism of gastric acid secretion. ECL, enterochromaf /f_i n-like; G, gastrin receptor; H, histamine receptor; HCl, hydrochloric acid; M, muscarinic receptor.

nerve and gastrin. Gastrin is released by the G cells in response to the presence of food in the stomach. The production of gastrin is inhibited by acid, creating a negative feedback loop. Various other peptides, including secretin, inhibit gastric acid secretion. Classically , three phases of gastric secretion are described. The cephalic phase is mediated by vagal activity , secondary to sensory arousal as first demonstrated by Pavlov . The tric phase is a response to food within the stomach, which is mediated principally , but not exclusively , by gastrin. In the intestinal phase , the presence of chyme in the duodenum and small bowel inhibits gastric emptying, and acidification of the duodenum leads to the production of secretin, which inhibits gastric acid secretion, along with numerous other peptides originating from the gut. The stomach also possesses somatostatin-containing D cells. Somatostatin is released in response to a number of factors, including acidification. This peptide acts probably on the G cell, the ECL cell and the pari etal cell itself to inhibit the production of acid.

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