Digestive System
BIO220 Human Anatomy & Physiology
Refer to Websites listed on Daly’s Webpage under Exam 3 Digestive System

PARIETAL CELL ACID PRODUCTION: Go to the GERD weblink and note especially Parietal Cell and Acid Production Pathways via ACh, Gastrin, and Histamine.

Acetylcholine Receptor Pathway

The sight, smell, and taste of food cause salivation and the stimulation of the vagus nerve to release acetylcholine.

When acetylcholine binds to its receptor, the parietal cell's permeability to calcium ions (Ca++) is altered so that the ions move into the cell. The intracellular increase in Ca++ activates the intracellular protein phosphokinases.

In the nonsecreting state, the proton acid pump (H+-K+-ATPase) is situated in tubulovesicles in the cell cytoplasm bordering the secretory canaliculi. The increase in protein phosphokinases results in the translocation of H+-K+-ATPase to the secretory canaliculus, where the extracellular aspect of the pump is exposed to potassium ions (K+).

The proton pump exchanges K+ for hydrogen ions (H+). Chloride ions (Cl-) diffuse across the parietal cell from the bloodstream to the secretory canaliculus, where they combine with H+ to form hydrochloric acid (HCl).

Gastrin Receptor Pathway

Digested food in the stomach chemically stimulates the release of gastrin from G cells located in the antrum of the stomach. Distention of the stomach causes release of acetylcholine from the vagus nerve, and this further stimulates the G cells to produce gastrin. Gastrin travels through the bloodstream and binds to the gastrin receptor on the parietal cells, located in the gastric body and fundus.

When gastrin binds to its receptor, the parietal cell's permeability to calcium ions (Ca++) is altered so that the ions move into the cell. The intracellular increase in Ca++ activates the intracellular protein phosphokinases.

In the nonsecreting state, the proton pump (H+-K+-ATPase) is situated in tubulovesicles in the cell cytoplasm bordering the secretory canaliculi. The increase in protein phosphokinases results in the translocation of H+-K+-ATPase to the secretory canaliculus where the extracellular aspect of the pump is exposed to potassium ions (K+).

The proton pump exchanges K+ for hydrogen ions (H+). Chloride ions (Cl-) diffuse across the parietal cell from the bloodstream to the secretory canaliculus, where they combine with H+ to form hydrochloric acid (HCl).
Histamine Receptor Pathway
The sight, smell, and taste of food cause acetylcholine to be released from the vagus nerve. Digested food and distention also cause gastrin to be released from G cells in the stomach. Acetylcholine and gastrin both stimulate the ECL cells to release histamine.

When histamine binds to its receptor, the parietal cell's permeability to calcium ions (Ca++) is altered, and Ca++ move into the cell. The intracellular increase in Ca++ activates the intracellular protein phosphokinases. Simultaneously, a membrane-bound adenylate cyclase leads to the generation of cAMP. cAMP acts as a second messenger to activate protein phosphokinases.

In the nonsecreting state, the proton pump (H+-K+-ATPase) is situated in tubulovesicles in the cell cytoplasm bordering the secretory canaliculi. The increase in protein phosphokinases results in the translocation of H+-K+-ATPase to the secretory canaliculus where the extracellular aspect of the pump is exposed to potassium ions (K+).

The proton pump exchanges K+ for hydrogen ions (H+). Chloride ions (Cl-) diffuse across the parietal cell from the bloodstream to the secretory canaliculus, where they combine with H+ to form hydrochloric acid (HCl).