### **Gastric secretions**

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## Objectives

- Describe the various type of gastric cells and the secretion of each cell type
- Mention the component of gastric juice and the function of each component
- Role of hormone and other factors influencing gastric secretion
- Describe the different mechanism involved in the control of gastric secretion

## enterogastrone hormones

- Motilin
- Somatostatin
- Glucose dependent insulinotropic peptide
- Vasoactive intestinal peptide
- Cholecystokinin
- Secretin
- somatostatin

#### **Gastric secretions**

Cephalic phase

**Smell** 

**Think** 

Sight

taste

1/3 of gastric juice

## Cephalic phase

Stimulatory

Cerebral cortex, hypothalamus, dorsal nucleus vagus (DNV), stomach parietal and chief cells, HCL and pepsinogen respectively

Inhibitory

Activate the sympathetic (stress, emotion)

T1 to L2 greater splanchnic nerve

Not directly inhibit chief or parietal

- Parietal cells intrinsic factor, HCl anti septic
- Chief cells pepsinogen
- G cells Gastrin
- Mucous glands mucous
- Delta cells somatostatin
- Ecl histamine

## Gastric phase

- 2/3 of gastric juice
   Vagovagal reflex (long reflex)
- Stretch receptors, afferent nerve of vagus,
   DNV, efferent vagus, HCL and pepsinogen

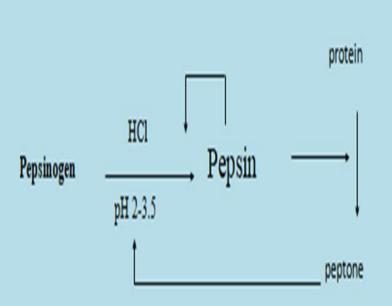
Submucosal plexuses (short reflex)
 HCL And Pepsinogen and myenteric plexuses for contractility

#### Antrum

Patrial digestive protein, Entero endocrine G cells, gastrin (hormone), CCK2 receptors partial cells, increase Ca+, hydrogen potassium pump (H+ lumen and K inside)

 Entire body and antrum
 Chief cells gastrin CCK1, increase Ca+, exocytosis pepsinogen, converted to active pepsin with presence of HCL

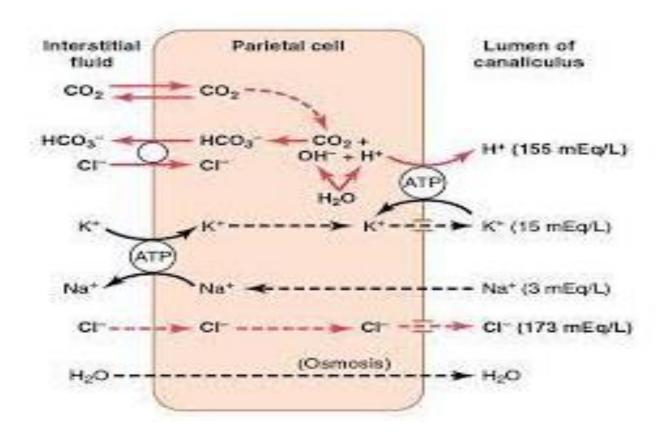
## Pepsinogen



 Concentration of proteins affect H+ the higher the protein the higher the pH

 Mechanically the concentration of protein affect the H+ and thus pH

#### **HCL** secretion



omeprazole Inhibits gastric secretion
 GERD (gastroesophageal reflex disease)
 Alkaline tide

## Continued Gastric phase

Inhibitory
Stimulate sympathetic emotional
( HCL and pepsinogen)

Somatostatin
Antral ( D cells), high Con H+, somatostatin
SST receptors G cells, inhibit gastrin

- Ach M3 receptors D cells, inhibits somatostatin
- Gastrin CCK2 receptors D cells, inhibits somatostatin
- Ach M3 receptor G cells, enhance gastrin
- Gastrin releasing peptide (bombesin) enhance gastrin

# Regulating of Parietal cells secretions

- Somatostatin SST receptors, Inhibit H+ pump
- Ach M3 receptor, stimulate H+ pump
- Histamine H2 receptors, stimulate H+ pump
- Prostaglandin (PGE2) EP3 receptors, inhibit hydrochloric acid production

## Regulating Chief cells secretions

 Histamine H2 receptors, stimulate pepsinogen

Ach M3 receptors, stimulate pepsinogen

Secretin S cells in duodenum to acidic chyme,
 Stimulate pepsinogen

### Enterochromaffin cells

- Ach M3 receptors, stimulate histamine
- Somatostatin SST receptors, inhibit histamine
- Gastrin. stimulate histamine
- Histamine stimulate pepsinogen from chief cells and HCL from G cells
- Somatostatin from the corpus or body of stomach

Ach M3 receptor inhibit the D in body cells and prevent somatostatin from inhibiting the histamine in ECL

#### Mucous barrier

- Foveolar cells
- Mucus neck cells

95% water

**Electrolytes HCO3** 

Phospholipids

Mucin proteins

Prevents corrosive damage by hydrochloric acid and pepsin

#### Gastrin

- G cells antrum stomach and upper part of small intestine
- Mucosa and submucosa secretion and motility increase

Vague etimoulation goethin halooging nontide

- Secretions growth of gastric mucosa sometimes insulin release
- Factors increase

Food protein digest protein

Gastric distention