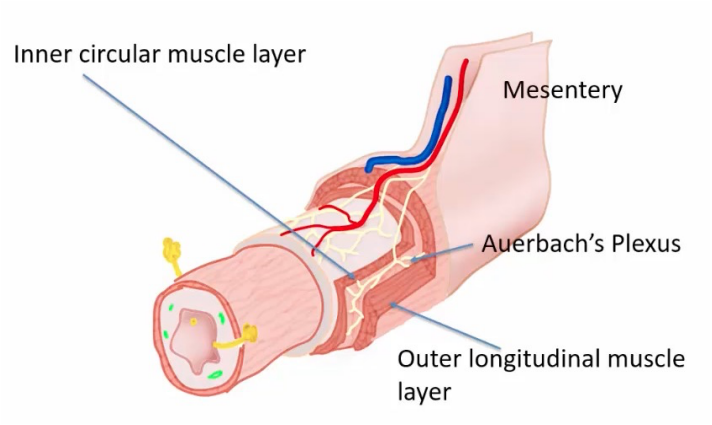
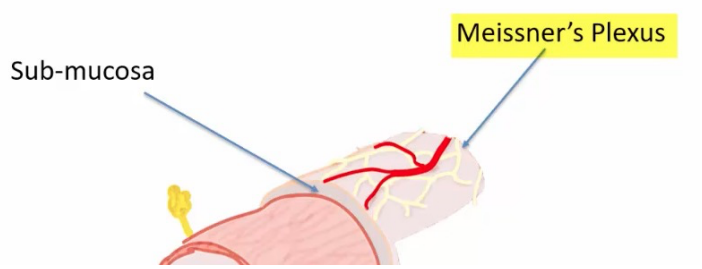
GI





Meissner's plexus

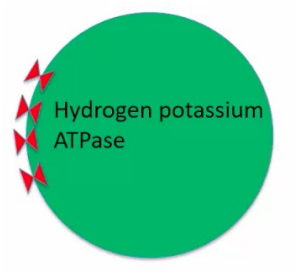
- Some sensory sensation in gut
- Gland innervation

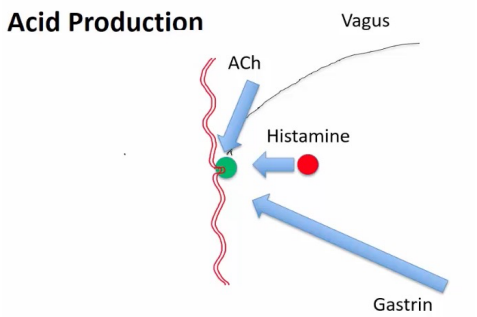
Parotid gland: serous and amylase secretions.
Submandibular gland: 80% of saliva - serous and mucous secretions
Sublingual gland: serous and mucous

Cell type	Substance secreted	Function of secretion
Parietal cells	Hydrochloric acid	Kills microbes and activates pepsinogen
Parietal cells	Intrinsic factor	Binds to vitamin B12 and facilitates it's absorption
Chief cells	Pepsinogen	Protein digestion
Chief cells	Gastric lipase	Fat digestion
G-cells	Gastrin	Stimulates gastric acid secretion
Enterochromaffin-like cells (ECL cells)	Histamine	Stimulates gastric acid secretion
Mucous-neck cells	Mucous and bicarbonate	Protects stomach epithelium from acid
D-cells	Somatostatin	Inhibits gastric acid secretion

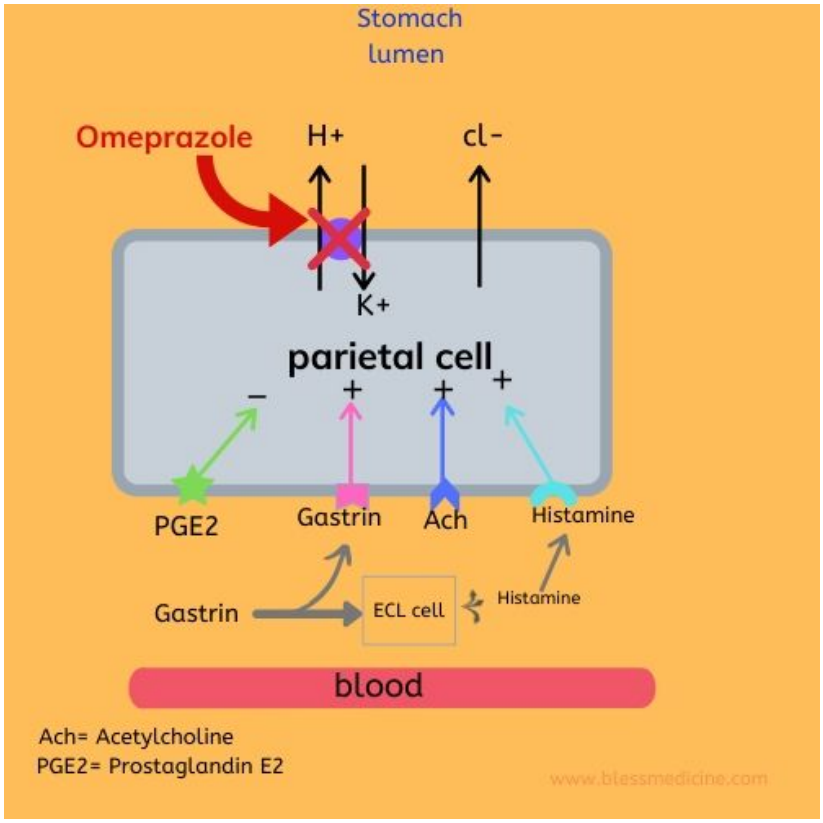
Stomach

PARIETAL cells

1. Intrinsic factor - produced here —> binds to B12 —> carried to terminal ileum
2. Acid production
    - 

      acid produced by hydrogen potassium ATPase on parietal cell surface. PPIs work here
    - 

        1. Vagus n —> ACh
        2. Enterochromaffin like cells - adjacent to parietal cells —> histamine —> H2 receptors —> acid production
        3. Gastrin from G cells



Chief cell

- Pepsinogen —> pepsin (stimulated by acidic pH)
- Acid —> denatures protein —> pepsinogen to pepsin by acidic environment —> continued breakdown of proteins
- Vagus n, gastrin and lowered pH stimulate chief cell

G cell

- Located in antrum of stomach. Stretch = ↑ gastrin production
- Gastrin stimulates parietal and chief cells

Gastric wall protection

Mechanisms

1. Mucus lining the stomach wall
2. Bicarb produced by endothelial layer

Other

- H-pylori lives in mucus layer —> causes ↑ inflammation —> reduced mucus layer and ↓ protection from acid —> ulcer formation
- Arachidonic acid —> prostaglandins —> ↑ mucus production
    - NSAIDs and aspirin inhibit COX-1 (which converts arachidonic acid to prostaglandins) = ↓ mucus production = ↓ protection
    - Naproxen and ibuprofen = more selective for COX-2 (involved in pain + inflammation) and less so for COX-1

Gastric juice contents (throughout whole GI tract)

1. Hydrochloric acid (HCL)
2. Digestive enzymes
    - Pepsinogen
    - gastric lipase
3. Mucus
4. Intrinsic factor

Factors that inhibit gastric emptying:

- Hormones:
    - Cholecystekinin
    - Secretin
- ↓ pH in duodenum
- Distension of duodenum
- Fats in the duodenum
- ↑ osmolality in the duodenum

Intestines

Commensal intestinal bacterial flora have a role in:

- Keeping pathogenic bacteria at bay by competing for space and nutrient
- Converting conjugated bilirubin to urobilinogen (some of which is reabsorbed and excreted in urine) and stercobilinogen which is excreted in the faeces
- The synthesis of vitamins K, B12, thiamine and riboflavin
- The breakdown of primary bile acids to secondary bile acids
- The breakdown of cholesterol, some food additives and drugs

CCK

- From i-cells in duodenum and jejunum
- Actions
  1. Slows stomach emptying
  2. ↑ pancreatic secretions
  3. ↑ gallbladder emptying
- Stimulated by ↑ presence of lipids, proteins in intestines
- Inhibited by - SOMATOSTATIN

SECRETIN

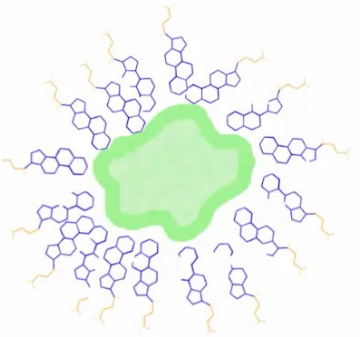
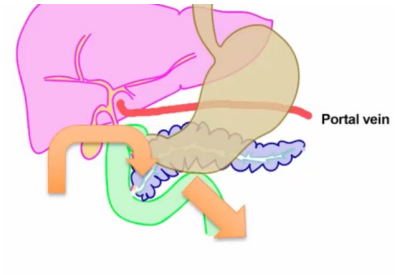
- From S-cells
- Actions
  - Reduces parietal cell acid production
  - ↑ bicarbonate secretion
  - Inhibit gastric emptying
  - ↑ pepsinogen release by chief cells
  - Potentiate CCK release
- Stimulated by
  - ↑ acidity / ↓ pH
  - ↑ fatty acids in duodenum
- Inhibited by - SOMATOSTATIN

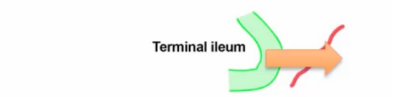
SOMATOSTATIN

- D-cells in pyloric antrum, duodenum and pancreatic islets
- Decreases all GI activity
  - ↓ pancreatic enzymes ↓ gastric motility ↓ gastric emptying ↓ gastric secretions ↓ bile production and release

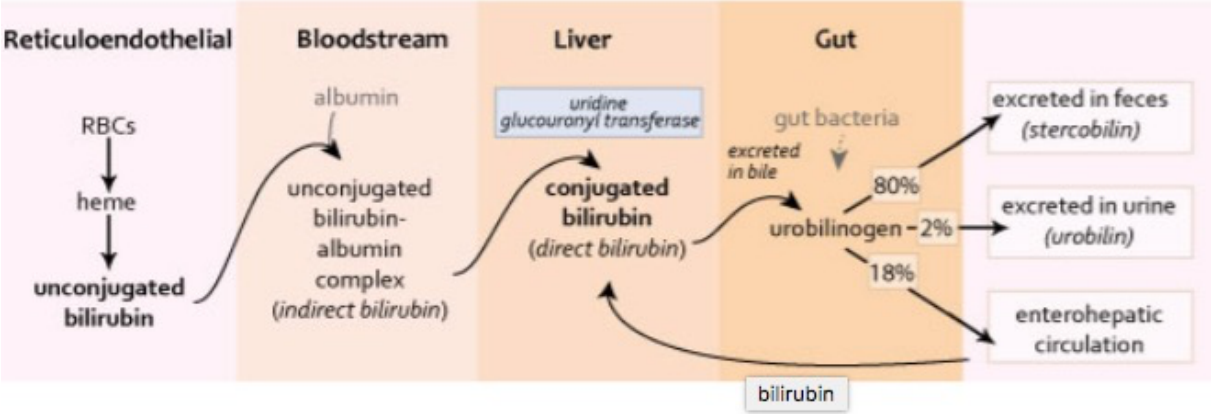
- Decreases ALL GI enzymes
  - Insulin
  - Glucagon
  - Cholecystokinin
  - Secretin
  - Gastric inhibitory polypeptide (GIP)
- Stimulated by ↑ H+ in GI tract
- Inhibited by vagal stimulation

Bile

- Made by hepatocytes —> drains into GB for storage
- 97 % water
  0.7% bile salts
  0.2% bilirubin (bile pigment)
- Stimulated by vagal and CCK
- FUNCTION
  - Neutralise stomach acid in duodenum
  - Emulsify fats —> the further breakdown into monoglycerides and FAs is by pancreatic lipase
  - Elimination of substances from the liver
- *Bile acids/salts*
  - Production
    1. Cholesterol —> cholesterol acid (then binds to water soluble compound e.g. glycine)
    2. —> forms BILE SALTS (lipid and water soluble components)
  - Bile salts gather round fat droplet (green). Lipophilc facing in (blue), hydrophilic facing out (yellow). This then makes fat droplet accessible to other enzymes e.g. lipase
    - 
  - 8 types of bile salts - get recirculated in the body **(95% of bile is recirculated through body)**
    - Enterohepatic circulation recycles the bile salts
    - —> excretion into duodenum —> passage through to terminal ileum —> reabsorption into circulation —> back to liver by PV
    - 

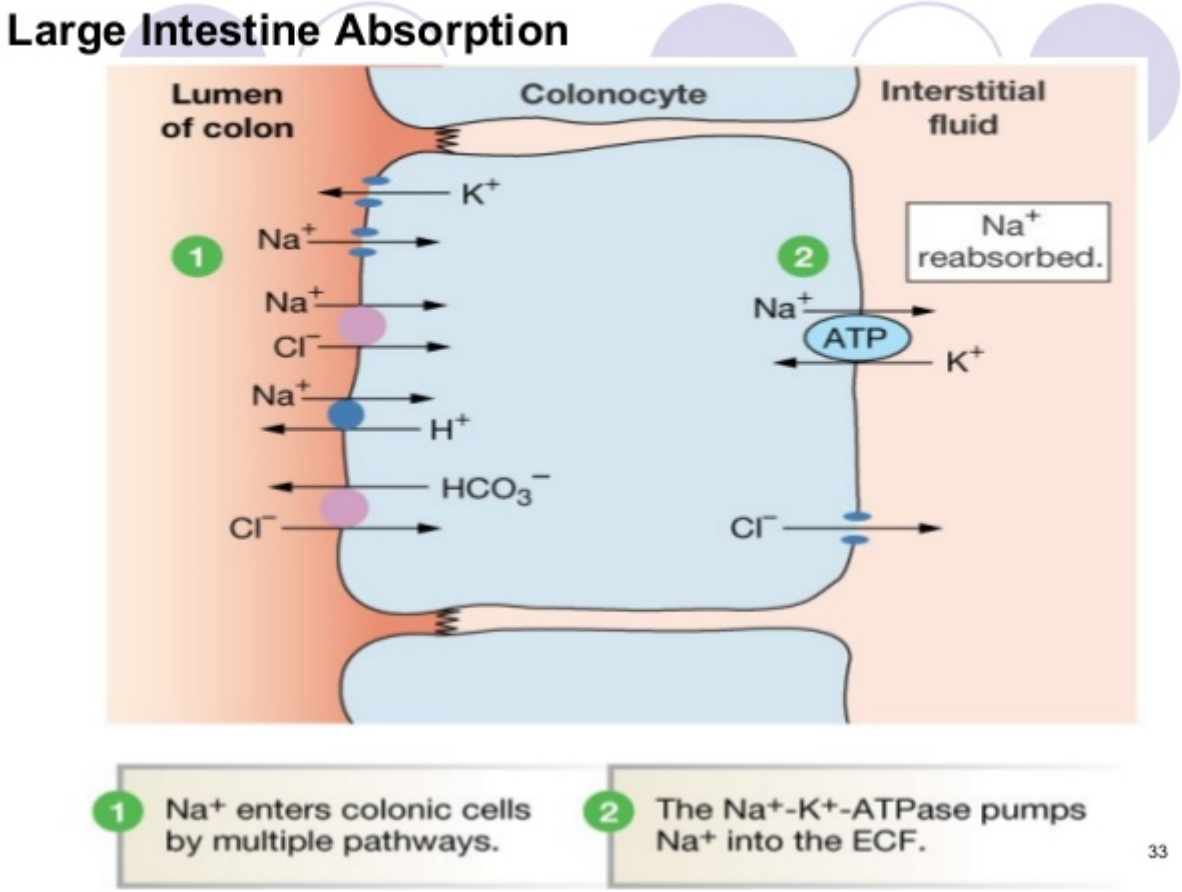


Bilirubin metabolism - gives bile its colour



Colon

- basolateral Na/K ATPase sets up concentration gradient (↓Na and ↑K in colon epithelium)
  - Na diffuses down concentration gradient via multiple transporter types - from lumen —> extracellular
    - *This draws water with it also*
  - K and HCO3- diffuse INTO lumen and out
  - Result is a hypertonic solution in colon (started as isotonic from small intestine)
  -



Nutrient absorption

- Most absorption is in ILEUM
- Iron —> duodenum
- Vit K —> colon
- Bile salts and B12 = ONLY in the terminal ileum
- Water in small and large intestine

Protein digestion

1. HCL denatures protein
2. Pepsin (released by chief cells) turns protein —> polypeptides
3. Trypsin and chymotrypsin (released by pancreas) turns polypeptide —> oligopeptides
4. Carboxypeptidases and aminopeptidases on luminal cells turns oligopeptides —> small peptides and AAs
5. AAs from lumen —> blood stream by facilitated diffusion (e.g. co-transport Na)

Pancreatic enzymes:

The acinar cells secrete a small volume of fluid rich in digestive enzymes.

These enzymes include:

- Pancreatic amylase
- Pancreatic lipase
- Ribonuclease and deoxyribonuclease
- Proteolytic enzymes including trypsin, chymotrypsin, elastase and carboxypeptidase

Most of the proteolytic enzymes are secreted in an inactive proenzyme form to protect the pancreas from autodigestion, and are activated in the duodenum.