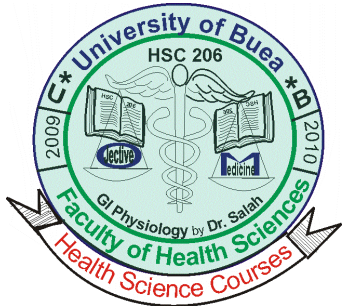


HSC 206. GI PHYSIOLOGY



Monday, October 25, 2010

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1

Introduction

- GI Tract **Digest** and **Absorb** Nutrients.
- This is achieved by its
 - **Motility** and,
 - **Secretions**
- To remain protected, GI tract has various **defence** mechanisms

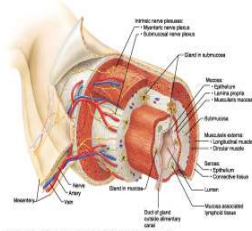
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Structure of the GI Tract

- **Epithelial Cells** specialized for **secretion** and **absorption**
- **Muscularis Mucosa** where **contractions** result in **change of surface area** of the GI tract
- **Circular Muscles** **decreases diameter** of the GI tract
- **Longitudinal Muscles** **shortens GI segments**
- **Submucosal and Myenteric plexuses** comprising the **enteric Nervous system**



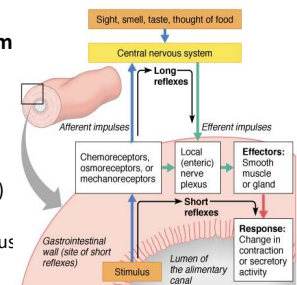
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Nervous Control of the GI Tract

- **Extrinsic Innervations (Autonomic Nervous System)**
 - Parasympathetic
 - Sympathetic
- **Intrinsic Innervations (Enteric Nervous System)**
 - Myenteric (Auebach's Plexus) controls Motility
 - Submucosal (Meissner's plexus) controls secretion



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Endocrine Regulation of the GI Tract

- **Gastrin** Secreted by **G-cells** of the stomach **increases gastric H⁺ secretion** and **growth of gastric mucosa**.
- **Cholecystokinin (CCK)** secreted by the **I-cells** of the small intestine **stimulates secretion of HCO₃⁻**, **gallbladder contraction** and **inhibits gastric emptying**.
- **Secretin** secreted by the **S-cells** of the duodenum **stimulates HCO₃⁻ secretion** and **inhibit gastric H⁺ secretion**
- **Gastric Inhibitory Peptide (GIP)** secreted by small intestine **inhibits gastric H⁺ secretion** and **promotes insulin secretion**

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Paracrine Regulation of the GI Tract

- **Somatostatin**
 - secreted in response to **H⁺** in the lumen
 - Secretion **inhibited** by **vagal stimulation**
 - Inhibits release of **all GI hormones** and also **inhibits gastric H⁺ secretion**
- **Histamines**
 - Secreted by **mast cells** of gastric mucosa
 - Increases **gastric H⁺ secretions** by potentiating **gastrin** and **vagal stimulations**

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Neurocrine Regulation of the GI Tract

- **Vasoactive Intestinal Peptide (VIP)**
 - Produces **relaxation** of GI **smooth muscles**
 - **Stimulates HCO_3^- secretion** and **inhibits gastric H^+ secretion**
- **Gastric Relasing Peptide (Bombesin)**
 - Released from vagal nerves that innervate **G-cells**
 - **Stimulates gastrin release** from G-cells
- **Enkephalins**
 - Stimulate the **contraction of GI smooth muscles**
 - **Inhibit intestinal secretion of fluids and electrolytes**

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Gastrointestinal Motility

- GI Motility is the **contraction** and **relaxation** of the **walls** and **sphincters** of the GI Tract
- Motility **grinds, mixes** and **fragments** ingested food to prepare it for **digestion** and **absorption**, and
- It **propels** food along the GI tracts

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GI Muscles and Contractions

- Contractile tissues are the **muscles** of the GI tract
- **Phasic** or **tonic** contractions
- **Phasic contractions** are periodic for mixing and propelling food.
- **Tonic contractions** are consistent used for mixing food

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Slow Waves

- Oscillating membrane potentials
- Determine pattern of action potential thus contraction

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Frequency of Slow Waves

- Originate in the interstitial cells of **Cajal** (pacemaker) in **Myenteric plexus**
- Cyclic **depolarization** and **repolarization** then spontaneously spread to adjacent smooth muscles

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Frequency of Slow Waves

- Averagely 2-12 Waves per minute
- The vary depending on the section of the gut
 - Slowest in stomach (3/min)
 - Highest in the duodenum (12/min)
- They are not influenced by hormonal or neural input except for the frequency

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Mechanism of Slow Waves Production

- Caused by **cyclic opening of Ca^{2+} channels** which produce **inward Ca^{2+} current**.
- This depolarizes the cell membrane and eventually generate an **action potential**.
- The can also be caused by **cyclic activation and deactivation of the cell membrane Na^+-K^+ pump**.

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Chewing

- **Lubricates** food by mixing it with **saliva**.
- **Decreases size of food** particles rendering large surface area for **salivary amylase**
- Chewing movements is under **voluntary control**.

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Swallowing

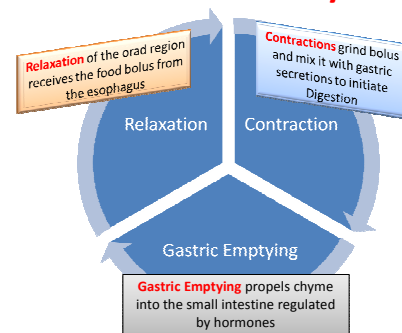
- **Initiated voluntary** in the mouth and thereafter become **involuntary**.
- Involuntary is controlled by **Swallowing Center** in **Medulla**.
- Swallowing is done in the phases:
 - Oral Phase
 - Pharyngeal Phase &
 - Esophagus Phase

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Gastric Motility



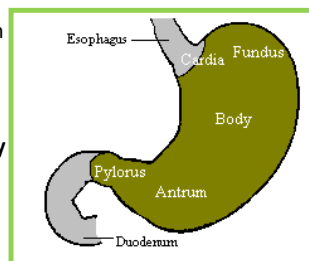
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Structure of The Stomach

- Three layers of smooth muscles: **longitudinal, circular and oblique**
- Three anatomical divisions: **fundus, body and antrum**

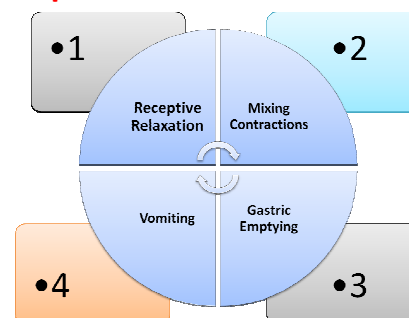


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Components of Gastric Motility



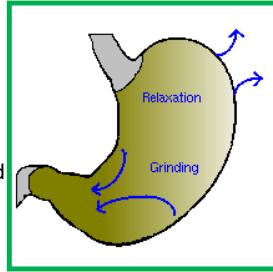
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Receptive Relaxation

- Receives food bolus
- Distention of lower **esophageal sphincter** and **simultaneous relaxation of orad stomach**
- **Vagovagal** reflex mediated by **VIP**.
- **CCK** participates in receptive relaxation



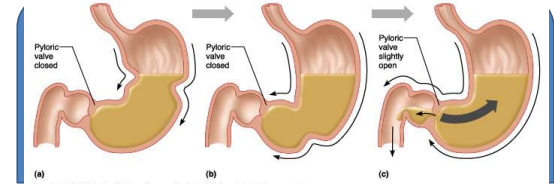
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Mixing Contractions

- Grinds and Mixes food with gastric secretions and begins the process of digestion
- Mixing contractions are determined by the frequency of slow waves.



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Gastric Emptying

- Takes about 3 hrs regulated to neutralize H^+ in the duodenum.
- Rate is fastest (**isotonic**) and slowed in either **hypertonic** or **hypotonic** stomach contents.
- Inhibited by **fats** through the intermediary of **CCK** and by H^+ in the duodenum.

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Vomiting

- Rapid expulsion of stomach contents through the mouth.
- Preceded by the CNS, PNS and Enteric NS.
- Mechanism involves respiratory movements.
- Integrated in the **vomiting centers** in Medulla influenced by **chemoreceptor trigger zone** in **area postrema** that induces vomiting.
- Vomiting can be induced by **Morphine, sickness, motion** and stimulation of the back of the tongue.

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Small Intestinal Motility

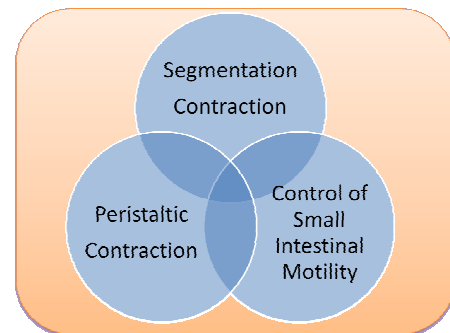
- **Mixes** nutrients with digestive enzymes and exposes them for **absorption** and **propels** the rest to large intestine.
- Slow waves (12/min) generate conditioned by **parasympathetic** (increases) and **Sympathetic** (decreases) intestinal smooth muscle contractions.
- There are two patterns of contractions coordinated by the **enteric NS**

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Patterns of Small Intestinal Motility



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Segmentation Contractions

- Segment **contracts** and then **relaxes** and contents move back to the segment.
- **Back-and-Forth** movements causes segmentation contractions
- This **mixes** food **without net movement** of the chyme

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Peristaltic Contractions

- **Highly coordinated** and **propel chyme** through small intestine to the large intestine.
- **Contraction** occurs **behind** the bolus and **relaxation** occurs **in front** of the chyme
- Contraction is conditioned by **ACh, Substance P**, and Relaxation by **VIP** and **NO**.
- **Peristaltic reflexes** is coordinated by the **enteric Nervous System**

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Control of Small Intestinal Motility

- Done by reflexes mediated by **extrensic ANS** and **gastrin**.
- Presence of food in the stomach

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Large Intestinal Motility

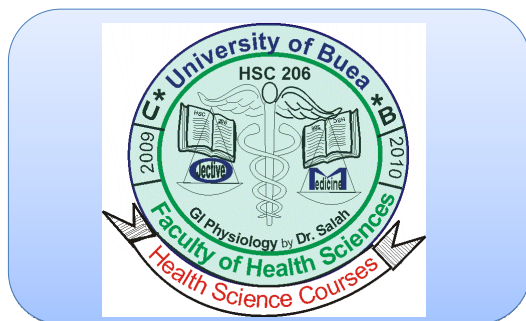
- **Stores undigested** food before defecation and regulate **salt** and **water balance** with **bacteria** that **breaks down glucose**.
- **Segmentation, peristalsis and bulk movement** occurs 2-3 times/day and pushes contents towards **rectum** where defecation occurs.
- Dependent on **myogenic** and **neural** mechanisms involving **gastrocolic reflexes, CCK** and **gastrin**.
- Defecation is voluntary

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Secretions of the GI Tract



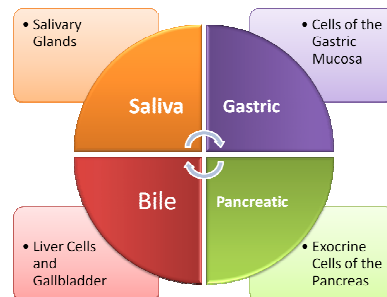
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Gastrointestinal Secretion

Secretion adds fluids, enzymes and mucus



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Salivary Secretion

- Produced by **salivary glands** into the mouth at a rate of **1 liter/day**.
- Starts **Starch** and **lipid digestion**
- Digest, dilute, buffer and lubricate** ingested food with mucus to aid swallowing

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Functions of Saliva

- α -amylase (ptylin)** initiate starch digestion and triglyceride digestion by **lingual lipase**.
- Protects mouth and esophagus by **lubrication** by mucus

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Composition of Saliva

- High $[K^+]$ and $[HCO_3^-]$
- Low $[Na^+]$ and $[Cl^-]$
- α -amylase (ptylin)
- lingual lipase, and
- Kallikrein
- Composition varies with salivary rate.**

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Formation of Saliva

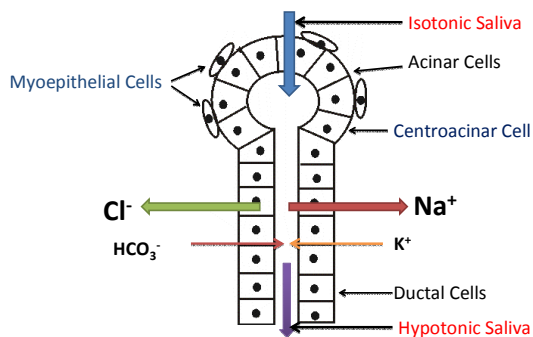
- Saliva is formed by three major glands
 - **Parotid**
 - **Submaxillary and**
 - **Sublingual glands**
- The structure of each gland is made up of **acinus** and a **duct**

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Formation of Saliva



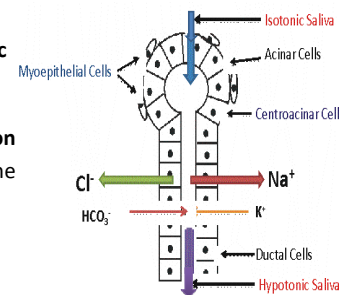
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Formation of Saliva

- Acinus** is lined with **acinar cells** that secrete initial **isotonic** saliva
- Myoepithelial cells** assist in **saliva ejection**
- Ductal cells** modify the saliva producing a **hypotonic** saliva



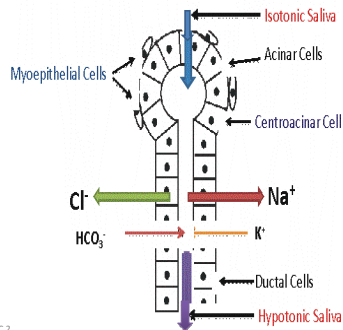
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The Acinus

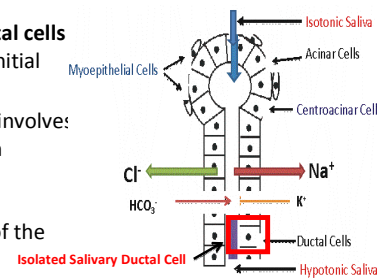
- Produces initial saliva isotonic to plasma.
- Has same Na^+ , K^+ , Cl^- and HCO_3^- concentration as plasma



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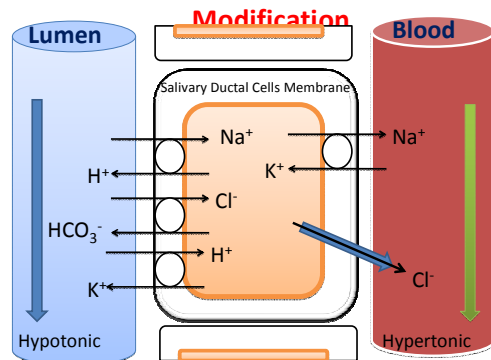
The Duct

- Contains **ductal cells** that **modify** initial saliva.
- Modification involves mechanism in **luminal** and **basolateral** membranes of the ductal cell



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Ductal Cell Mechanism of Saliva Modification



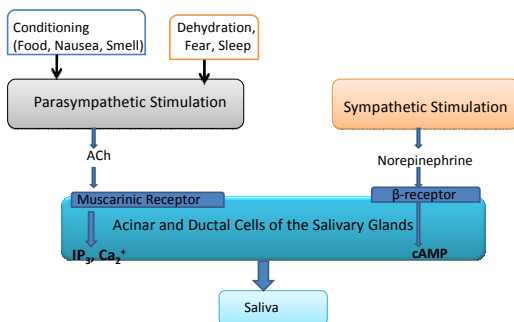
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Effect of Flow Rate on Saliva Composition

- Influenced by changes in contact time for reabsorption and secretion in the ducts
- Higher flow rate (isotonic) and slower (hypotonic)
- HCO_3^- is controlled otherwise

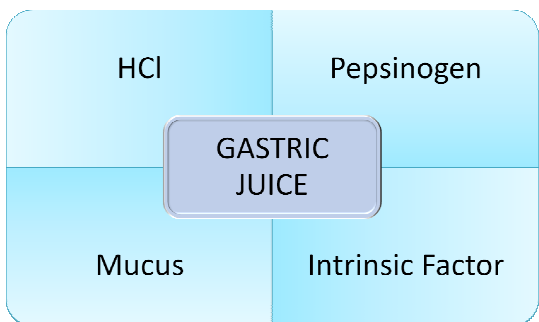
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Regulation of Salivary Secretion



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Gastric Secretions

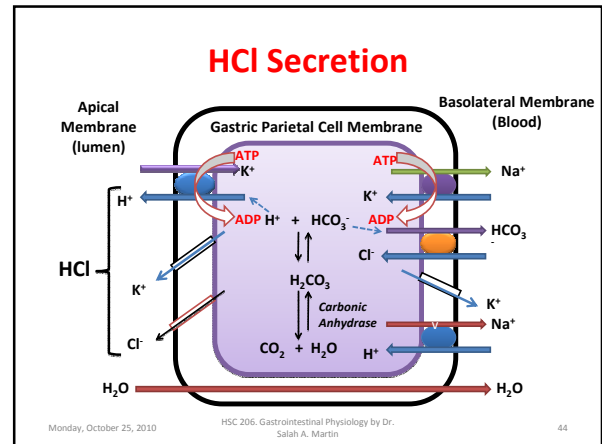


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Cell Types of Gastric Mucosa

Cell Types	Location	Secretion
Parietal Cell	Body	HCl and Intrinsic Factor
Chief Cells	Body	Pepsinogen
G-Cells	Antrum	Gastrin
Mucus Cells	Antrum	Mucus Pepsinogen

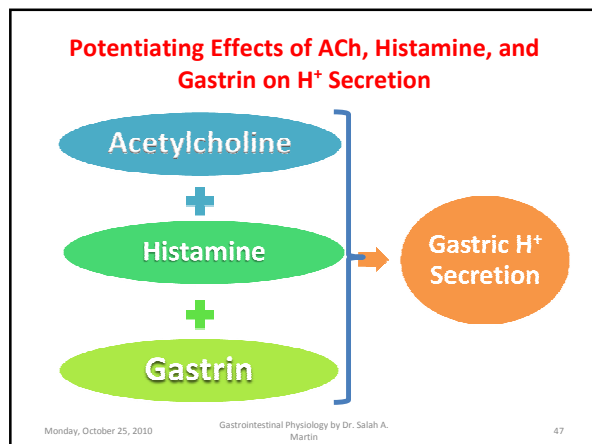
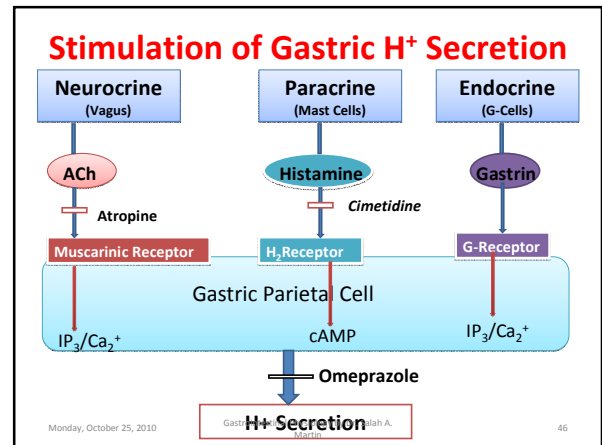
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Stimulation of Gastric H⁺ Secretion

- Neurocrine** • Vagus (ACh)
- Paracrine** • Mast Cells (Histamine)
- Endocrine** • G-Cells (Gastrin)

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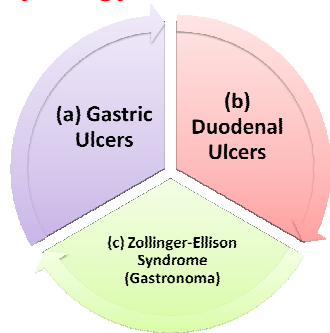


Inhibition of Gastric H⁺ Secretion

- Decrease **pH** of gastric contents
- Food **Buffers**
- Exit of to the duodenum
- **Somatostatin** and **GIP** are leased in response to the low **gastric pH** and inhibit **gastrin** release and thus **gastric H⁺** secretion.

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Pathophysiology of Gastric H⁺ Secretion



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Pepsinogen Secretion

- **Pepsinogen** secreted by **chief cells** is an **inactive precursor to pepsin**
- Pepsinogen is **activated** by H⁺ to **pepsin**
- Pepsin **begins protein digestion**
- In the **Cephalic** and **Gastric phases** of H⁺ secretion, **vagal stimulation** is the stimulus for pepsinogen secretion.
- H⁺ also **triggers reflexes** that stimulate the **chief cells to secrete pepsinogen**

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Intrinsic Factor Secretion

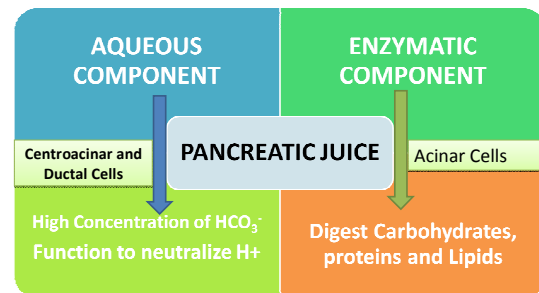
- A **mucoprotein** and secreted by the **parietal cells**.
- Required for **Vitamin B₁₂ absorption** in the **ileum**, deficiency results in **pernicious anemia**
- Only **essential** stomach secretion
- **Gastrectomy** (removal of the stomach), patients must receive **vitamin B₁₂ injections** to bypass absorption defect.

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Pancreatic Secretion



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Structure of Pancreatic Exocrine Glands

- Resembles **salivary glands** with **acinus, ductal cells** and **centroacinar cells**
- **Acinus** is lined with **acinar cells** that **secrete the enzymatic portion**
- The **ducts** are lined with **ductal cells** which extend into special region of **centroacinar cells** in the **acinus**.
- The **centroacinar** and **ductal cells** secrete the **aqueous HCO₃⁻ containing** component of the pancreatic secretion

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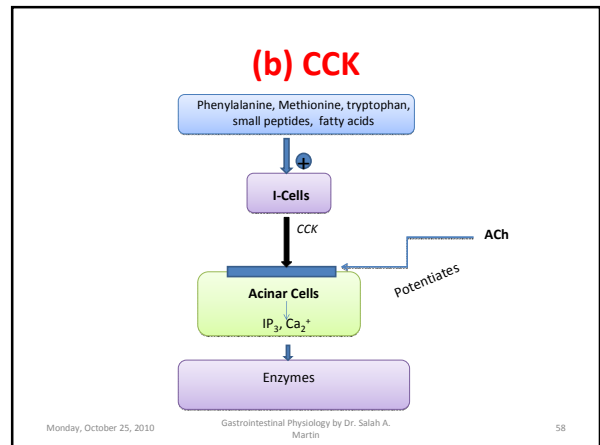
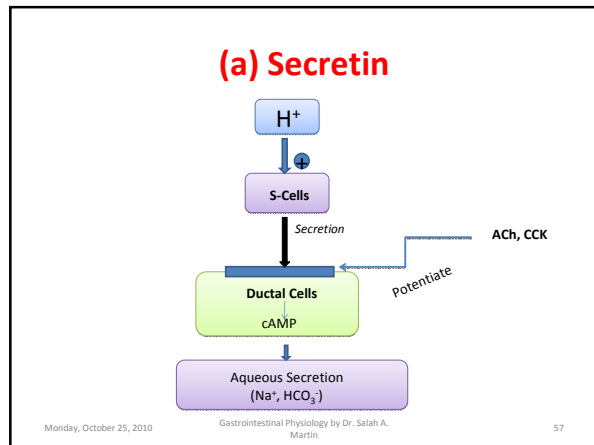
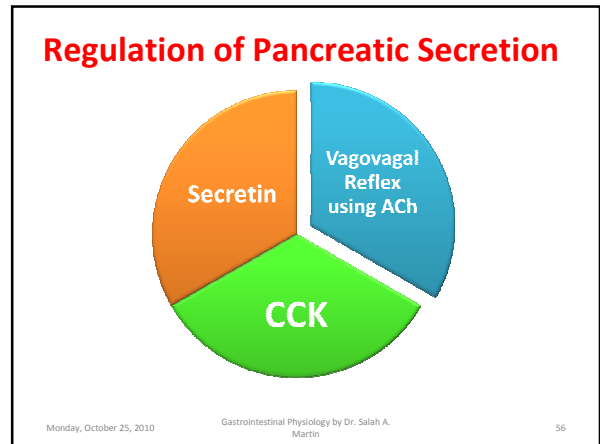
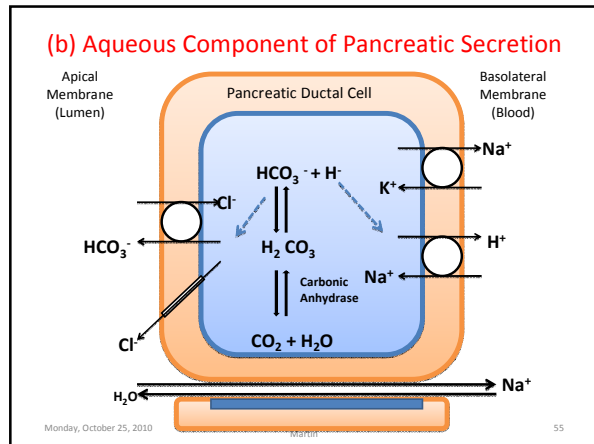
Formation of Pancreatic Secretion (a) Enzymatic Component

- Pancreatic **amylase** and **lipases** are secreted as **active enzymes**.
- Pancreatic **proteases** are secreted **inactive forms**
- Synthesize in the **rough endoplasmic reticulum** of **acinar cells**, transferred to the **Golgi apparatus**
- There are condensed into **Zymogen Granules**
- Release is stimulated by **parasympathetic stimulation** or **CCK**

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(c) Cystic Fibrosis

- **Disorder of Pancreatic Secretions**
- Caused by **defect in Cl⁻ channels** as a result of **mutation on cyctic fibrosis transmembrane conductance regulator gene**.
- Associated with **deficiency in pancreatic enzyme** resulting in **malabsorption** and **steatorrhea**.

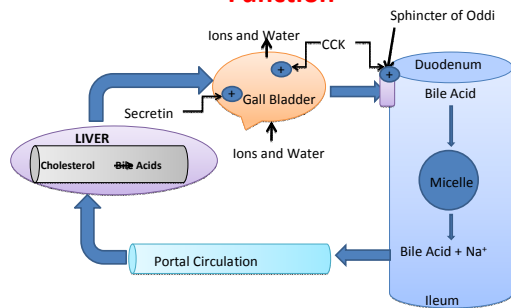
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Bile Secretions and Gallbladder Function

- Bile is necessary for digestion and absorption of lipids in small intestine through the intermediary of **micelles**.

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Biliary System and Bile Composition and Function



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Formation of Bile

- **Primary bile acids** are **Cholic acid** and **chenodeoxycholic acid** synthesized from **cholesterol** by **hepatocytes**.
- **Secondary bile acids** are composed **deoxycholic acid** and **lithocholic acid**.

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Contraction of the Gallbladder

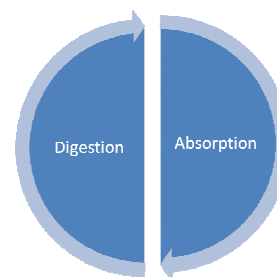
- Regulated by **CCK** and **Ach**
- **CCK** caused **relaxation** of **Oddi's sphincter**.

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Digestion and Absorption



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Carbohydrates

- **Disaccharides** (**sucrose**, **lactose**, **maltose** and **trehose**) are digested to **Monosaccharides** (**glucose**, **galactose** and **Fructose**)

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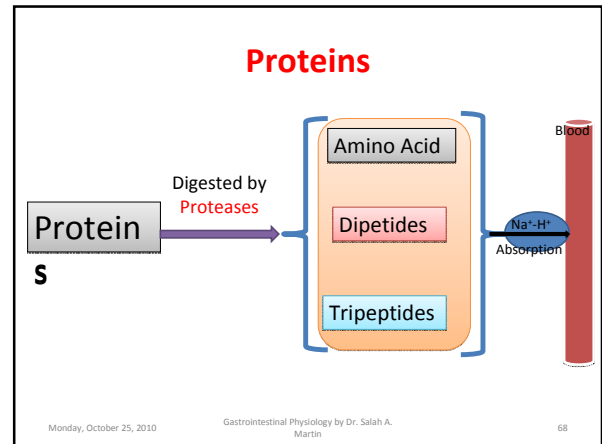
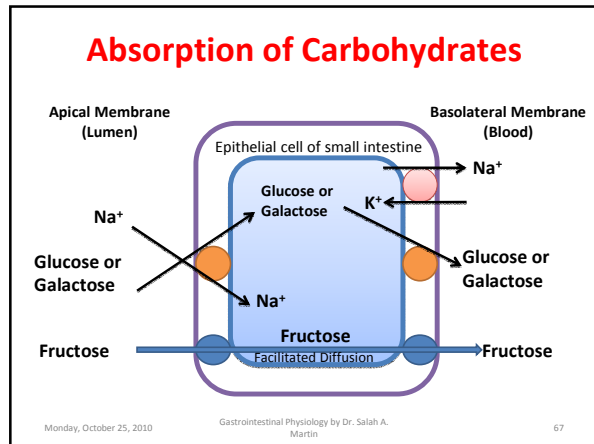
Digestion of Carbohydrates

- **α -amylase** from **saliva** and **pancreatic juice** hydrolyze **1-4 glycosidic bonds** in **starch** yielding **maltose**, **maltotriose**, and **α -limit dextrins**.
- **Maltase**, **α -dextrinase**, and **sucrase** then **hydrolyze** the **oligosaccharides** to **glucose**.
- **Lactase**, **trehalase** and **sucrase** degrade their respective **disaccharides** to **monosaccharides**.
- **Lactase** degrade **Lactose** to **glucose** and **galactose**
- **Trehase** degrade **trehalose** to **glucose**
- **Sucrase** degrade **sucrose** to **glucose** and **fructose**.

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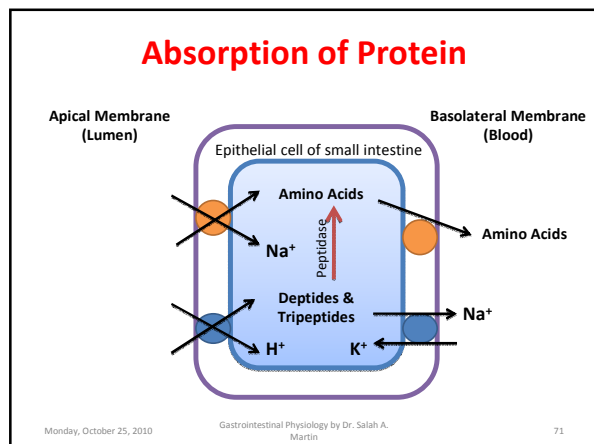
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- ### Digestion of Proteins
- Catalyzed by endopeptidases that break **interior peptide bonds** and, **exopeptidases** at **C-terminal**.
 - **Pepsin** initiates protein digestion but activated by increasing pH of the duodenal fluids.
 - **Trypsin, chymotrypsin, elastase, carboxypeptidase A & B.**
 - **Trypsinogen** is activated to **trypsin** which then activates **chymotrypsinogen, proelastase and procarboxypeptidase A & B** and even **trypsinogen**.
 - After action pancreatic proteases degrade each other and products absorbed by **dietary proteins**.
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- ### Absorption of Proteins
- Free Amino Acids
 - Dipeptides & Tripeptides
 - Disorders of Protein Digestion and Absorption
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- ### Disorders of Protein Digestion and Absorption
- **Deficiency of pancreatic enzymes**
 - **Defect in cell membrane transporters**
 - These are **chronic pancreatitis and cystic fibrosis**.
 - **Proteins cannot be digested and absorbed.**
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Lipids

- **Dietary lipids** such as **triglycerides**, and **phospholipids** are digested into **fatty acids**, **monoglycerides** and **cholesterol**.
- The **digestive products** form **micelles** with **salts** and diffuse into cell.
- **Re-esterification** result in **triglycerides**, and **phospholipids**.
- **Chylomicrons** form assisted by **apoprotein** and are **transferred to the lymph**.

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Digestion of Lipids

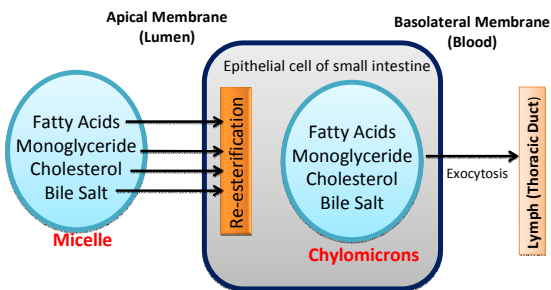
- **(a) In the Stomach**
- **Lingual Lipase** digest **triglycerides** to **monoglycerides** and **fatty acids**.
- **CCK** regulates the process.
- **(b) In the Small Intestines**
- **Pancreatic lipase** hydrolyse lipids to fatty acids, monoglycerides, cholesterol and **lysolecithin**.
- Enzymes are **pancreatic lipase**, **cholesterol ester hydrolase** and **phospholipase A₂**

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Absorption of Lipids



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Absorption and Secretion of Electrolytes and Water

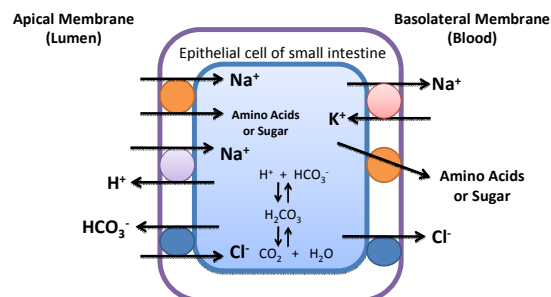
- Cross intestinal epithelium either by
 - Cellular routes
 - Paracellular routes

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Absorption of NaCl in Small Intestine

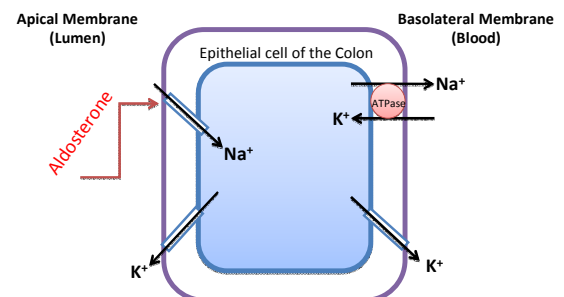


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Absorption of NaCl in the Colon



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Absorption and Secretion of K⁺

- **Dietary K⁺** is absorbed in **small intestine** by **passive diffusion** via **paracellular route**.
- K⁺ is **actively secreted** in the **colon** as in **renal distal tubule**.
- Its secretion is stimulated by **aldosterone**.

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Absorption of Water

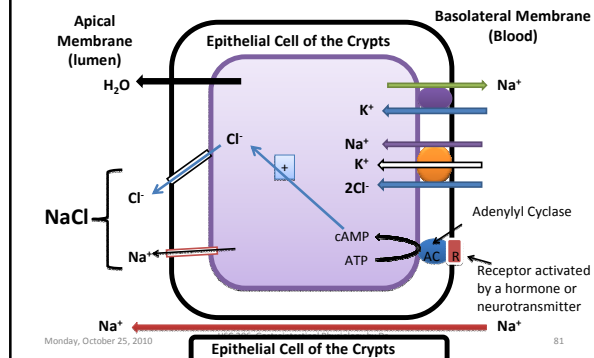
- **Isosmotic** in small intestine
- Couple **water-solute** intake mechanism as in **proximal tubule**
- In colon, water permeability is **much lower**.

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Secretion of Electrolytes and Water by the Intestines



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Absorption of Vitamins

Fat-Soluble Vitamins

- **A, D, E and K**
- Incorporated into micelles

Water-Soluble Vitamins

- **B1, B2, B6, B12, C, biotin, folic acid, nicotinic acid and pantothenic acid**
- Absorbed by Na⁺-dependent co-transport

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Calcium

- Absorbed in small intestine depending on **Vit D**'s active form **1,25 dihydroxycholecalciferol** produced in the **kidney**
- Deficiency results in **Rickets in infants** and **Osteomalacia in adults**.

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Iron

- Absorbed as **heme iron** i.e bound to **hemoglobin** or **myoglobin** as Free Fe₂⁺
- In intestine heme is degraded to free iron and bind to **apoferritin** and transferred to blood
- Free Fe₂⁺ circulate in blood bound to **transferrin**
- Deficiency of iron result in **anemia**

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Gases

- From swallowed air and also generated in the colon by bacterial action.
- The odor generated gas, **hydrogen sulphide (H₂S)** result from food that provide fermentable carbohydrates such as beans

Defence Mechanisms of the GI Tract

- **Mucus, gastric acid** and **proteases** protects against *Vibrio cholerae*.
- **Endotoxins** inhibit fluid absorption and generate **diarrhea**
- **Antibodies** such as **IgA** provides defence.
- **Lymphoid tissues** such as **tonsils, peyer's patches** and **apendix** provide **immune response**.
- **Mast cells** recognise antigens and release **histamines leukotrienes, cytokines** and **prostaglandins** for the defence of the GI tract.