

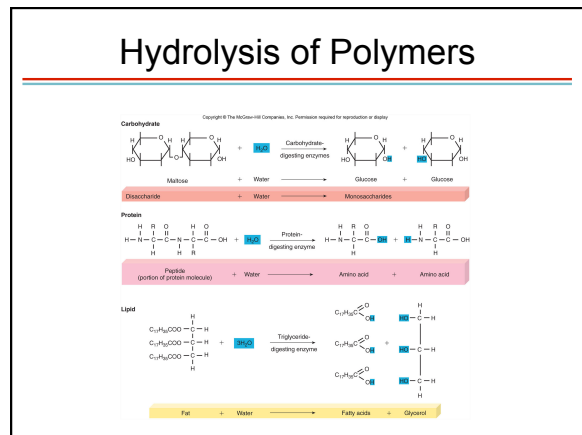
Chapter 18  
The Digestive System

Lecture PowerPoint

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# I. Introduction to the Digestive System

- ## Digestion
- From food, humans must get basic organic molecules to make ATP, build tissues, and serve as cofactors and coenzymes.
    - Digestion breaks polymers (carbohydrates, fats, and proteins) into monomer building blocks.
      - Via hydrolysis reactions
    - Absorption takes these monomers into the bloodstream to be allocated.



- ## Digestive Tract
- Open at both ends and continuous with the environment
    - Considered “outside” the body
    - Materials that cannot be digested (cellulose) never actually “enter” the body.

- ## Digestive Tract Functions
- Motility
    - Ingestion: taking food into the mouth
    - Mastication: chewing
    - Deglutination: swallowing
    - Peristalsis: one-way movement through tract
    - Segmentation: churning/mixing

## Digestive Tract Functions

### 2. Secretion

- Exocrine: digestive enzymes, acid, mucus
- Endocrine: hormones to regulate digestion

### 3. Digestion

- Breaking food down into smaller units

### 4. Absorption

- Passing broken-down food into blood or lymph

## Digestive Tract Functions

### 5. Storage and elimination

- Temporary storage and elimination of undigested food

### 6. Immune barrier

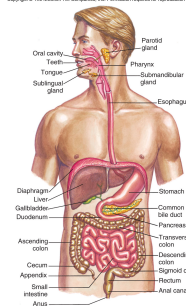
- Simple columnar epithelium with tight junctions prevents swallowed pathogens from entering body.

## Digestive System Divisions

- Gastrointestinal tract: 30 feet long, from mouth to anus
  - Mouth →
  - Stomach →
  - Pharynx →
  - Esophagus →
  - Stomach →
  - Small intestines →
  - Large intestines →
  - Anus
- Accessory organs: teeth, tongue, salivary glands, liver, gallbladder, pancreas

## Digestive System Divisions

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## GI Tract Layers

- Also called tunics
- There are four tunics:
  1. Mucosa: inner secretory and absorptive layer; may be folded to increase surface area
  2. Submucosa: very vascular, to pick up nutrients; also has some glands
  3. Muscularis: smooth muscle; responsible for peristalsis and segmentation
  4. Serosa: outer binding and protective layer

## Regulation of the GI Tract

- Parasympathetic division:
  - Stimulates esophagus, stomach, small intestine, pancreas, gallbladder, and first part of large intestine via vagus nerve
  - Spinal nerves in sacral region stimulate lower large intestine.
  - Preganglionic neurons synapse on submucosal and myenteric plexi.

## Regulation of the GI Tract

- Sympathetic division:
  - Inhibits peristalsis and secretion
  - Stimulates contraction of sphincters
- Hormones:
  - From brain or other digestive organs

## Regulation of the GI Tract

- Intrinsic regulation:
  - Intrinsic sensory neurons in gut wall help in intrinsic regulation via separate **enteric nervous system**
  - Paracrine signals

## II. From Mouth to Stomach

## Mouth

- Mastication: Chewing breaks food down into smaller pieces for deglutition and mixes it with saliva.
- Saliva: contains mucus, an antimicrobial agent, and **salivary amylase** to start digestion of starch.

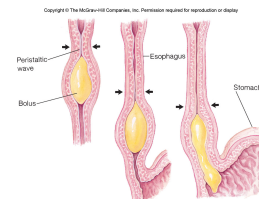
## Deglutition

- Involves coordinated contraction of 25 pairs of muscles
- Three parts:
  1. Oral: voluntary; muscles of mouth and tongue mix food with saliva to form **abolus**.
  2. Pharyngeal: initiated by receptors in the posterior oral cavity and oropharynx
    - Uvula lifts to cover nasopharynx, and esophagus covers vocal cords.
    - Upper esophageal sphincter relaxes.

## Deglutition

3. Esophageal: automatic; controlled by **swallowing center** of brain stem

- Bolus is moved down esophagus to stomach via peristalsis



## Deglutition

- Mouth, pharynx, and upper esophagus lined with skeletal muscles innervated by somatic motor neurons
- Lower esophagus lined with smooth muscle controlled by autonomic nervous system

## Esophagus

- ~10 inches long
- Passes through the diaphragm via the esophageal hiatus
- Lined with nonkeratinized stratified squamous epithelium
- Upper portion has skeletal muscle; lower portion smooth muscle
- Lower esophageal sphincter opens to allow food to pass into stomach. It stays closed to prevent regurgitation.

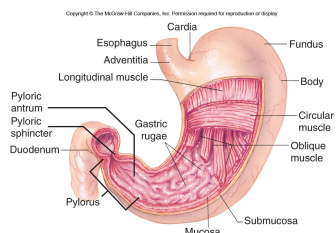
## Stomach: Functions

- Stores food
- Churns food to mix with gastric secretions
- Begins protein digestion
- Kills bacteria in the food (acid)
- Moves food into small intestine in the form of chyme

## Stomach Structure

- Food is delivered to cardiac region.
- Upper region = fundus
- Lower region = body
- Distal region = pylorus
  - Ends at pyloric sphincter
- Lining has folds called rugae.

## Stomach Structure



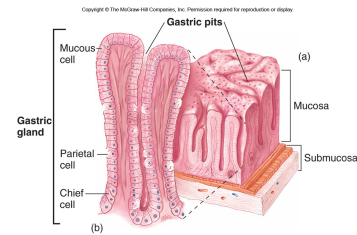
## Stomach Structure

- Gastric pits at base of folds lead to gastric glands with secretory cells:
  - Mucus neck cells secrete mucus to help protect stomach lining from acid.
  - Parietal cells secrete HCl acid and intrinsic factor (helps small intestine absorb vitamin B<sub>12</sub>).
  - Chief cells secrete pepsinogen.

## Stomach Structure

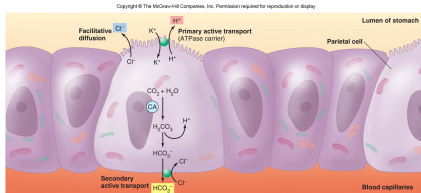
- Enterochromaffin-like (ECL) cells secrete **histamine** and **serotonin** (paracrine signals).
- G cells secrete **gastrin** (hormone).
- D cells secrete **somatostatin** (hormone).

## Stomach Structure



## HCl Secretion

- Primary active transport of  $H^+$  via  $H^+/K^+$  ATPase pumps
- Facilitated diffusion of  $Cl^-$



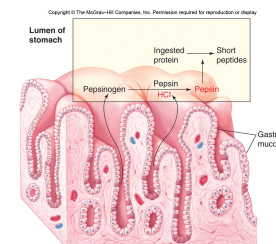
## Stimulation of HCl Secretion

- Gastrin: made in G cells; carried to parietal cells in blood
  - Also stimulates ECL cells to make histamine
- Histamine: also stimulates parietal cells via  $H_2$  histamine receptors
  - Examples: Tagamet and Zantac block  $H_2$  receptors.
- Parasympathetic neurons: stimulate parietal and ECL cells

## Function of HCl

- Drops pH to 2
  - Proteins are denatured (allows enzymes access).
  - Pepsinogen is converted to active pepsin (digests proteins).
  - Serves as the optimal pH for pepsin activity

## Function of HCl



## Stomach Defenses

- Acid and pepsin could eat the stomach lining.
- Defenses that help prevent this:
  - Adherent layer of mucus with bicarbonate
  - Tight junctions between epithelial cells
  - Rapid epithelial mitosis that replaces epithelium every three days

## Digestion and Absorption in the Stomach

- Proteins begin digestion in the stomach.
  - Starches begin digestion in the mouth, but salivary amylase is not active at pH 2, so this activity stops in the stomach.
- Alcohol and NSAIDs (aspirin) are the only common substances absorbed in the stomach (due to high lipid solubility).

## Peptic Ulcers

- Peptic ulcers: erosions of the mucosa of the stomach or duodenum
  - *Helicobacter pylori*: bacterium that reduces mucosal barriers to acid
  - Treatment for ulcers combines K<sup>+</sup>/H<sup>+</sup> pump inhibitors (Prilosec) and antibiotics.

## Gastritis

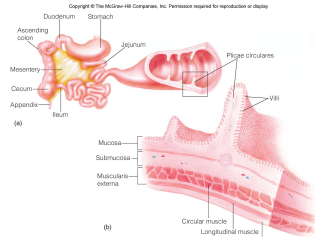
- Inflammation of the submucosa caused by acid eating at it
  - Histamine released as part of the inflammatory response can stimulate more acid release.
  - Prostaglandins are needed to stimulate protective alkaline mucus production.
  - NSAIDs inhibit prostaglandin activity and can lead to gastritis.
  - Tagamet and Zantac inhibit H<sub>2</sub> receptors.

## III. Small Intestine

## Small Intestine Structure

- Three sections:
  - Duodenum
  - Jejunum
  - Ileum
- Mucosa and submucosa folded into *plicae circulares*; mucosa further folded into *villi*; and epithelial plasma membranes folded into *microvilli*

## Small Intestine Structure



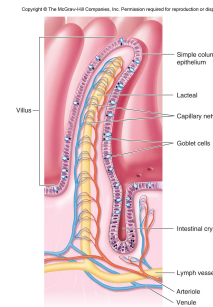
## Small Intestine Functions

- Complete digestion of carbohydrates, proteins, and fats
- Absorption of nutrients
  - Sugars, lipids, amino acids, calcium, and iron absorbed in duodenum and jejunum
  - Bile salts, vitamin B<sub>12</sub>, water, and electrolytes in ileum
  - Very rapid due to villi and microvilli

## Villi and Microvilli

- Columnar epithelium with goblet cells (mucus)
- Capillaries absorb sugars and amino acids, and lacteals absorb fatty acids.
- Intestinal crypts with Paneth cells (secrete antibacterial molecules) and mitotic stem cells

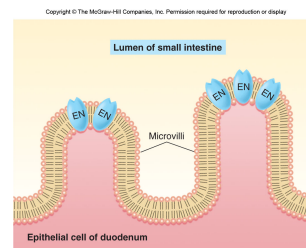
## Villi and Microvilli



## Intestinal Enzymes

- Called brush border enzymes
  - Not released into lumen, but stay attached to plasma membrane with active site exposed to chyme

## Intestinal Enzymes



## Intestinal Enzymes

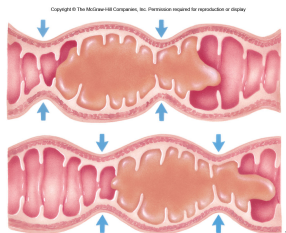
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**Table 18.1 | Brush Border Enzymes Attached to the Cell Membrane of Microvilli in the Small Intestine**

Category	Enzyme	Comments
Disaccharidase	Sucrase	Digests sucrose to glucose and fructose; deficiency produces gastrointestinal disturbances
	Maltase	Digests maltose to glucose
	Lactase	Digests lactose to glucose and galactose; deficiency produces gastrointestinal disturbances (lactose intolerance)
Peptidase	Aminopeptidase	Produces free amino acids, dipeptides, and tripeptides
	Enterokinase	Activates trypsin (and indirectly other pancreatic juice enzymes); deficiency results in protein malnutrition
Phosphatase	Ca <sup>2+</sup> , Mg <sup>2+</sup> -ATPase	Needed for absorption of dietary calcium; enzyme activity regulated by vitamin D
	Alkaline phosphatase	Removes phosphate groups from organic molecules; enzyme activity may be regulated by vitamin D

## Intestinal Contractions/Motility

- Peristalsis is weak. Movement of food is much slower due to pressure at pyloric end.
- Segmentation is stronger and serves to mix the chyme.

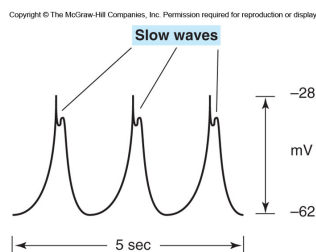
## Intestinal Contractions/Motility



## Intestinal Contractions/Motility

- Smooth muscle contractions occur automatically.
  - Graded depolarizations called **slow waves** produced by pacemaker cells called **interstitial cells of Cajal** produce action potentials in muscle cells.

## Intestinal Contractions/Motility



## Intestinal Contractions/Motility

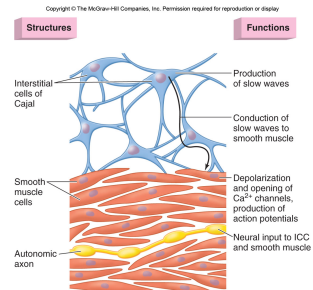
- Depolarization from the interstitial cells of Cajal opens voltage-gated Ca<sup>2+</sup> channels in muscle cells → action potential and contraction.
- Stimulation travels short distances through gap junctions but must be regenerated in neighboring pacemaker regions.
  - Produces contractions needed for segmentation



## Regulation of Contraction

- Autonomic nerves influence enteric nervous system to stimulate or inhibit cells ofCajal.
- Acetylcholine from parasympathetic system interacts with muscarinicACh receptors to increase amplitude and duration of slow waves.

## Regulation of Contraction

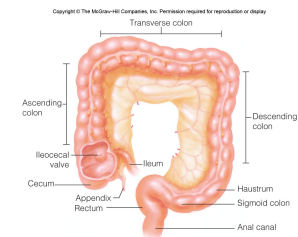


## IV. Large Intestine

## Large Intestine Structure

- Chyme from ileum passes through ileocecal valve into:

- Cecum →
- Ascending colon →
- Transverse colon →
- Descending colon →
- Sigmoid colon →
- Rectum →
- Anal canal →
- Anus



## Large Intestine Function

- Absorption of water, electrolytes, vitamin K, and some B vitamins
- Production of vitamin K and B vitamins via microbial organisms
- Storage of feces

## Large Intestine Function



## Microbial Biota

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- Several hundred different species of bacteria live in the large intestine.
  - Some are commensal. The bacteria benefit, and we aren't harmed.
  - Others are mutualistic. We benefit too.

## Benefits from Microbes

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- Microbes make vitamin K and some B vitamins.
- They also make fatty acids from cellulose. Some of these are used for energy by large intestine epithelial cells. We can't absorb the fatty acids, but they help absorb electrolytes such as sodium, calcium, bicarbonate, magnesium, and iron.
- They outcompete harmful species of bacteria.
- Disruption of normal microflora can lead to irritable bowel disease.

## Absorption of Fluids

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- Most absorption occurs in small intestine, but some is left for large intestine.
- Not all water is absorbed; about 200 ml is left per day to be excreted with feces.
- Water is absorbed passively following an osmotic gradient set up by active  $\text{Na}^+/\text{K}^+$  pumps.
  - Aldosterone stimulates greater salt and water absorption here.

## Defecation

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- As material passes to the rectum, pressure there increases, the internal anal sphincter relaxes, and the need to defecate rises.
- The external anal sphincter controls defecation voluntarily.
- During defecation, longitudinal rectal muscles contract to increase pressure as the anal sphincters relax.

## V. Liver, Gallbladder, and Pancreas

## Liver

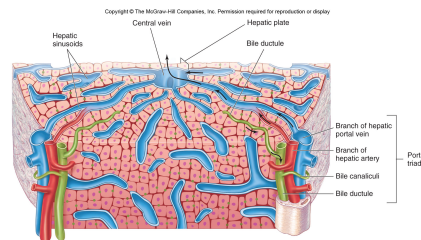
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- Largest abdominal organ
- Has amazing regenerative abilities due to mitosis of **hepatocytes**
- Composed of hepatocytes that form **hepatic plates** separated by capillaries called **sinusoids**
  - Very permeable, allowing passage of blood proteins, fat, and cholesterol

## Hepatic Portal System

- Products of digestion absorbed in intestines are delivered to the liver via the hepatic portal vein.
- After circulating through liver capillaries, the blood leaves via the hepatic vein.

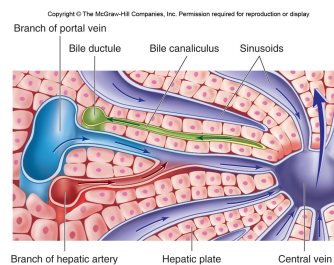
## Hepatic Portal System



## Liver Lobules

- Hepatic plates are arranged as liver lobules with hepatic arteries, hepatic portal veins, and a central vein.
  - Bile secreted by the hepatocytes is released into bile canaliculi, which drain into bile ducts.

## Liver Lobules



## Secretion of Drugs into Bile

- Aside from bile, the liver secretes other substances into the bile ducts to clear them from the blood.
  - These are then excreted in feces.

## Secretion of Drugs into Bile

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**Table 18.2 | Compounds Excreted by the Liver into the Bile Ducts**

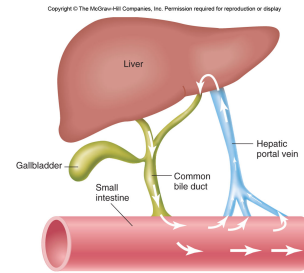
Category	Compound	Comments
Endogenous (Naturally occurring)	Bile salts, urobilinogen, cholesterol	High percentage reabsorbed and has an enterohepatic circulation*
	Lecithin	Small percentage reabsorbed and has an enterohepatic circulation
	Bilirubin	No enterohepatic circulation
Exogenous (Drugs)	Ampicillin, streptomycin, tetracycline	High percentage reabsorbed and has an enterohepatic circulation
	Sulfonamides, penicillin	Small percentage reabsorbed and has an enterohepatic circulation

\*Compounds with an enterohepatic circulation are absorbed to some degree by the intestine and are returned to the liver in the hepatic portal vein.

## Enterohepatic Circulation

- Some of the molecules released into the bile are absorbed again in the small intestine and returned to the liver.
- These molecules are part of enterohepatic circulation.

## Enterohepatic Circulation



## Liver Functions

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**Table 19.3 | Major Categories of Liver Function**

Functional Category	Actions
Detoxification of Blood	Phagocytosis by Kupfer cells Chemical alteration of biologically active molecules (hormones and drugs) Production of urea, uric acid, and other molecules that are less toxic than their parent compounds Excretion of molecules in bile
Carbohydrate Metabolism	Conversion of blood glucose to glycogen and fat Production of glucose from liver glycogen and from other molecules (amino acids, lactic acid) by gluconeogenesis Secretion of glucose into the blood
Lipid Metabolism	Synthesis of triglycerides and cholesterol Excretion of cholesterol in bile Production of ketone bodies from fatty acids
Protein Synthesis	Production of albumin Production of plasma transport proteins Production of clotting factors (thrombogen, prothrombin, and fibrin)
Secretion of Bile	Synthesis of bile salts Conjugation and excretion of bile pigment (bilirubin)

## Bile Production

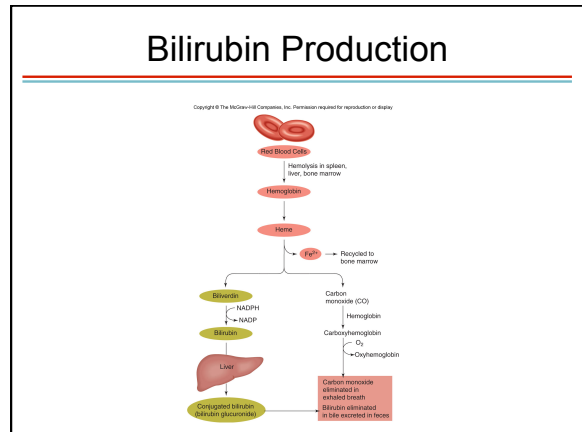
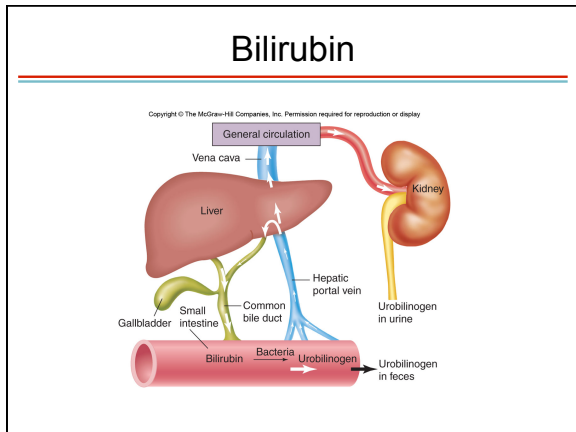
- The liver makes 250–1,500 ml of bile per day.
- Bile is composed of:
  - Bile pigments (bilirubin)
  - Bile salts
  - Phospholipids (lecithin)
  - Cholesterol
  - Inorganic ions

## Bilirubin

- Produced in spleen, liver, and bone marrow
  - Derived from heme (– iron) from hemoglobin
  - Not water-soluble
    - Carried on albumin in the blood
    - Not directly filtered by kidneys or secreted into bile
    - Conjugated with **glucuronic acid** to make it water-soluble

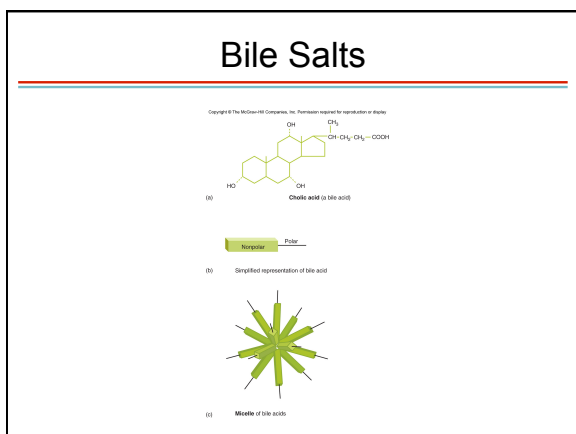
## Bilirubin

- Conjugated bilirubin is secreted into the bile, where it is taken to the small intestine.
  - Bacteria there turn it into **urobilinogen**, which makes feces brown.
  - 30–50% is absorbed by the intestines and taken back to the liver.
  - Some is used to make bile, and some remains in blood to be filtered by the kidneys.



- ### Bile Salts
- Made from bile acids conjugated with glycine or taurine
  - Bile acids: derived from cholesterol
    - Four polar groups on each molecule
    - Cholic acid and chenodeoxycholic acid
    - Most is recycled in enterohepatic circulation.
    - ½ gram of cholesterol is broken down and lost in the feces through this pathway.

- ### Bile Salts
- Form micelles with polar groups toward water
    - Fats enter the micelle and are emulsified.



- ### Detoxification of Blood
- The liver can remove hormones, drugs, and other substances in three ways:
    - Secreted into bile
    - Phagocytized by Kupffer cells lining sinusoids
    - Chemically altered by hepatocytes
      - Ammonia is converted into urea.
      - Urea is returned to the blood to be filtered by the kidneys.
      - Steroids are altered and then secreted into bile.

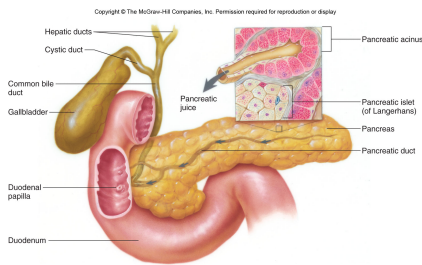
### Secretion of Glucose

- The liver helps balance blood glucose levels by removing glucose and storing it as glycogen (glycogenesis)/triglycerides (lipogenesis) or by breaking down glycogen (glycogenolysis) and releasing it into the blood.
- The liver can also make glucose from amino acids (gluconeogenesis) and convert fatty acids into ketones (ketogenesis).

### Gallbladder

- Stores and concentrates bile from the liver:
  - Liver →
  - Bile ducts →
  - Hepatic duct →
  - Cystic duct →
  - Gallbladder →
  - Cystic duct →
  - Common bile duct →
  - (Sphincter of Ampulla) duodenum

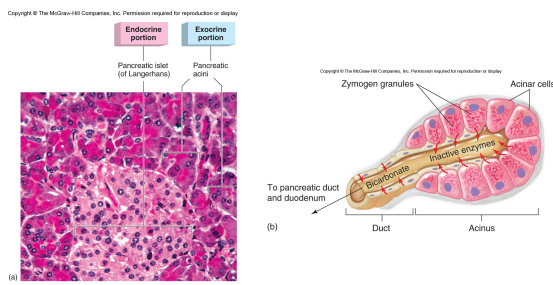
### Gallbladder



### Pancreas

- Has endocrine and exocrine functions
  - Endocrine: Islets of Langerhans cells (alpha and beta) make insulin and glucagon.
  - Exocrine: Acini cells make pancreatic juice, which is delivered to the duodenum via the pancreatic duct.

### Pancreas



### Pancreatic Juice

- Bicarbonate + 20 digestive enzymes
  - Enzymes for all three classes of macromolecules

**Table 18.4 | Enzymes Contained in Pancreatic Juice**

Enzyme	Zymogen	Activator	Action
Trypsin	Trypsinogen	Enterokinase	Cleaves internal peptide bonds
Chymotrypsin	Chymotrypsinogen	Trypsin	Cleaves internal peptide bonds
Elastase	Proelastase	Trypsin	Cleaves internal peptide bonds
Carboxypeptidase	Procarboxypeptidase	Trypsin	Cleaves last amino acid from carboxyl-terminal end of polypeptide
Phospholipase	Prophospholipase	Trypsin	Cleaves fatty acids from phospholipids such as lecithin
Lipase	None	None	Cleaves fatty acids from glycerol
Amylase	None	None	Digests starch to maltose and short chains of glucose molecules
Cholesterol esterase	None	None	Releases cholesterol from its bonds with other molecules
Ribonuclease	None	None	Cleaves RNA to form short chains
Deoxyribonuclease	None	None	Cleaves DNA to form short chains

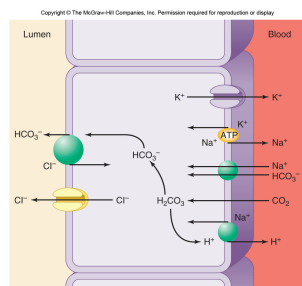
## Bicarbonate

- Made by cells lining ductules
- Made from CO<sub>2</sub> from the blood
  - First, carbonic acid is made.
  - This dissociates to form H<sup>+</sup> and bicarbonate.
  - The bicarbonate is secreted into pancreatic juice, and H<sup>+</sup> goes back into the blood.

## Bicarbonate

- Bicarbonate is countertransported with Cl<sup>-</sup>.
- People with cystic fibrosis have trouble secreting bicarbonate, which can lead to destruction of the pancreas.

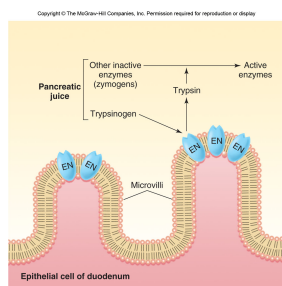
## Bicarbonate



## Pancreatic Enzymes

- Most are inactive until they reach the small intestine.
  - Enterokinase activates trypsinogen → trypsin (to digest protein).
  - Trypsin activates other enzymes.

## Pancreatic Enzymes



## VI. Neural and Endocrine Regulation of the Digestive System

## Neural Control

- Modifies GI tract functioning
  - Sight/smell/thought of food can stimulate salivation and gastric secretions to “prime” the digestive tract for food.
  - Stimulation goes from brain to organ via vagus nerve.

## Intrinsic Gastric Regulation

- Motility and secretion are somewhat automatic.
  - Contractions are stimulated spontaneously by pacesetter cells in greater curvature of stomach.
  - Secretion of HCl and pepsinogen occurs when amino acids enter the stomach.
    - Initiated/regulated by G cells (gastrin), D cells (somatostatin), and ECL cells (histamine)

## Extrinsic Gastric Regulation

- Divided into three phases:
  1. Cephalic phase: control by brain via vagus nerves
    - Stimulates ECL, chief cells, and parietal cells
    - Lasts for the first 30 minutes of a meal

## Extrinsic Gastric Regulation

- Divided into three phases:
  2. Gastric phase: triggered by arrival of food into stomach
    - Gastric secretion is stimulated by stomach distension (amount of food that enters) and amino acids in food.
    - Positive feedback occurs; as more proteins are broken down, more secretions are released to break them down.

## Extrinsic Gastric Regulation

- There is also a negative-feedback system. As pH drops (due to more HCl), **somatostatin** is released. This inhibits gastrin secretion.
- Lots of proteins buffer pH, so secretion matches protein concentration.

## Extrinsic Gastric Regulation

- Divided into three phases:
  3. Intestinal phase: inhibition of gastric activity when chyme enters the small intestine
    - Stretch when food enters the duodenum stimulates a neural reflex that inhibits gastric stimulation via the vagus nerve.
    - The presence of fats stimulates the duodenum to make **enterogastrone**.



## Extrinsic Gastric Regulation

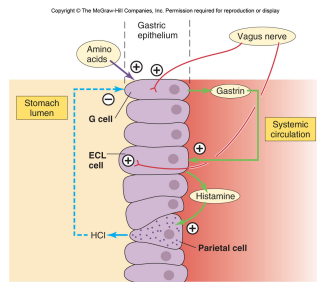
- Enterogastrone inhibits gastric secretions.
- Several specific hormones have been identified with enterogastrone activity (CCK, GIP, GLP-1).

## Summary of Gastric Regulation

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Table 18.6 | The Three Phases of Gastric Secretion

Phase of Regulation	Description
Cephalic Phase	<ol style="list-style-type: none"> <li>1. Sight, smell, and taste of food cause stimulation of vagus nuclei in brain.</li> <li>2. Vagus stimulates acid secretion.                     <ol style="list-style-type: none"> <li>a. Indirect stimulation of parietal cells (major effect)</li> <li>b. Stimulation of gastrin secretion (lesser effect)</li> </ol> </li> </ol>
Gastric Phase	<ol style="list-style-type: none"> <li>1. Distension of stomach stimulates vagus nerve; vagus stimulates acid secretion.</li> <li>2. Amino acids and peptides in stomach lumen stimulate acid secretion.                     <ol style="list-style-type: none"> <li>a. Direct stimulation of parietal cells (lesser effect)</li> <li>b. Stimulation of gastrin secretion; gastrin stimulates acid secretion (major effect)</li> </ol> </li> <li>3. Gastrin secretion inhibited when pH of gastric juice falls below 2.5.</li> </ol>
Intestinal Phase	<ol style="list-style-type: none"> <li>1. Neural inhibition of gastric emptying and acid secretion.                     <ol style="list-style-type: none"> <li>a. Arrival of chyme in duodenum causes distension, increase in osmotic pressure.</li> <li>b. These stimuli activate a neural reflex that inhibits gastric activity.</li> </ol> </li> <li>2. In response to fat in chyme, duodenum secretes a hormone that inhibits gastric acid secretion.</li> </ol>

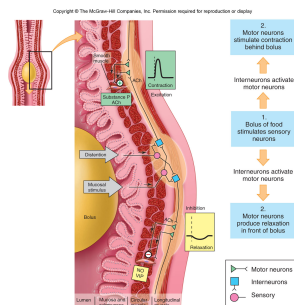
## Summary of Gastric Acid Secretion



## Regulation of Intestinal Function

- Enteric nervous system: neurons and glial cells that innervate the intestines
  - Includes myenteric plexus and submucosal plexus
  - Acts independently from CNS but with some feedback to CNS via vagus nerve
  - Innervates interstitial cells of Cajal

## Enteric Nervous System



## Regulation of Intestinal Function

- Paracrine regulation:
  - Enterochromaffin-like cells in intestinal mucosa secrete serotonin and motilin in response to pressure (filling) and chemicals in the food. This stimulates muscle contractions.
  - Guanylin: made in ileum and colon; stimulates the secretion of water and  $Cl^-$  and inhibits absorption of  $Na^+$ . More water and salt are lost in feces.

## Regulation of Intestinal Function

- Intestinal reflexes:
  - Gastroileal reflex: increased gastric activity = increased ileum activity and movement of food through ileocecal valve
  - Ileogastric reflex: distension of ileum = decrease in gastric motility
  - Intestino-intestinal reflex: Overdistension of one portion of the intestine causes relaxation of other portions.

## CCK and Secretin

- When chyme enters the duodenum, two hormones are produced:
  - Secretin is produced in response to a drop in pH.
    - Production stops with a rise in pH.
  - Cholecystokinin (CCK) is produced in response to the presence of partially digested proteins and fats in chyme.
    - Production stops when food leaves small intestine.

## Regulation of Pancreatic Juice Secretion

- Enzyme production is stimulated by ACh from vagus nerve, CCK, and secretin.
  - ACh and CCK use  $Ca^{2+}$  as a second messenger.
  - Secretin uses cAMP as a second messenger.
- Bicarbonate production is stimulated by secretin.

## Regulation of Bile Secretion

- The liver produces bile continuously, but the arrival of food into the duodenum stimulates increased production of bile.
- Happens when:
  - Bile acids are returned to the liver after intestinal absorption via enterohepatic circulation.
  - Secretin and CCK stimulate increased bicarbonate secretion into bile.
  - CCK (in response to the presence of fat in chyme) stimulates gallbladder contraction.

## Summary of Gastrointestinal Hormones

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**Table 18.5 | Effects of Gastrointestinal Hormones**

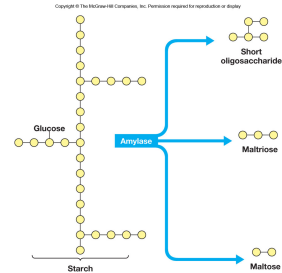
Secreted by	Hormone	Effects
Stomach	Gastrin	Stimulates parietal cells to secrete HCl Stimulates chief cells to secrete pepsinogen Maintains structure of gastric muscles
Small intestine	Secretin	Stimulates water and bicarbonate secretion in pancreatic juice Potentiates actions of cholecystokinin on pancreas
Small intestine	Cholecystokinin (CCK)	Stimulates contraction of gallbladder Stimulates secretion of pancreatic juice enzymes Inhibits gastric motility and secretion Maintains structure of exocrine pancreas (acini)
Small intestine	Gastric inhibitory peptide (GIP)	Inhibits gastric motility and secretion Stimulates secretion of insulin from pancreatic islets
Ileum and colon	Glucagon-like peptide-1 (GLP-1)	Inhibits gastric motility and secretion Stimulates secretion of insulin from pancreatic islets
	Guanylin	Stimulates intestinal secretion of $Cl^-$ , causing elimination of NaCl and water in the feces

## VII. Digestion and Absorption of Carbohydrates, Lipids, and Proteins

## Digestion of Carbohydrates

- Starch digestion begins in mouth with salivary amylase and continues in intestines with pancreatic amylase.
- Brush border enzymes finish breaking down resulting products and other disaccharides (maltose, sucrose, lactose).

## Digestion of Carbohydrates



## Absorption of Carbohydrates

- Monosaccharides are absorbed across the epithelium via:
  - Secondary active transport with sodium
  - Facilitated diffusion when glucose levels are high

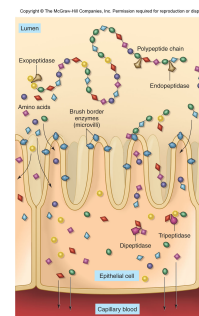
## Digestion of Proteins

- Begins in stomach with pepsin to produce short-chain polypeptides
- Finishes in duodenum and jejunum with pancreatic trypsin, chymotrypsin, elastase, and carboxypeptidase, and the brush border enzyme aminopeptidase.

## Absorption of Proteins

- Free amino acids cotransported with  $\text{Na}^+$
- Dipeptides and tripeptides cross via secondary active transport using a  $\text{H}^+$  gradient.

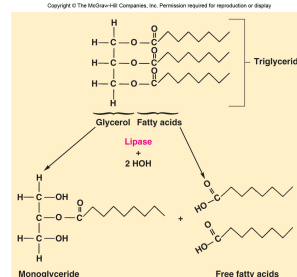
## Absorption of Proteins



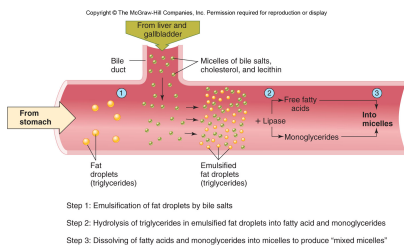
## Digestion of Fats

- Fat digestion begins in duodenum when bile emulsifies the fat and the pancreatic enzyme lipase breaks it down into fatty acids.
- Phospholipase A (from pancreas) digests phospholipids into fatty acids.

## Digestion of Fats



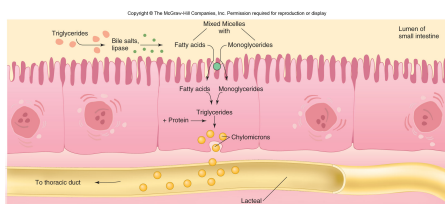
## Fat Emulsification and Digestion



## Absorption of Fats

- Fatty acids and monoglycerides move into bile micelles and are transported to brush border.
- Inside the epithelial cells, they are regenerated into triglycerides, cholesterol, and phospholipids and combined with proteins to form **chylomicrons**.
- These enter the lacteals.

## Fat Absorption



## Transport of Lipids in Blood

- The lymphatic system drops chylomicrons into the bloodstream at the thoracic duct.
- They pick up an **apolipoprotein**, which allows them to bind to receptors on the capillary endothelium within muscles and adipose tissue.
  - Here they are digested by **lipoprotein lipase**, which releases free fatty acids for use by muscle cells or for storage by fat cells.

## Transport of Lipids in Blood

- Cholesterol and triglycerides made in the liver are combined with other apolipoproteins to form very-low-density lipoproteins (VLDLs) to deliver triglycerides to organs.
- Once triglycerides are removed, they are low-density lipoproteins (LDLs), which transport cholesterol to organs.
- Excess cholesterol is returned to the liver on high-density lipids (HDL).

## Lipid Carrier Proteins

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**Table 18.8 | Characteristics of the Lipid Carrier Proteins (Lipoproteins) Found in Plasma**

Lipoprotein Class	Origin	Destination	Major Lipids	Functions
Chylomicrons	Intestine	Many organs	Triglycerides, other lipids	Deliver lipids of dietary origin to body cells
Very-low-density lipoproteins (VLDLs)	Liver	Many organs	Triglycerides, cholesterol	Deliver endogenously produced triglycerides to body cells
Low-density lipoproteins (LDLs)	Intravascular removal of triglycerides from VLDLs	Blood vessels, liver	Cholesterol	Deliver endogenously produced cholesterol to various organs
High-density lipoproteins (HDLs)	Liver and intestine	Liver and steroid-hormone-producing glands	Cholesterol	Remove and degrade cholesterol

## Summary of Major Digestive Enzymes

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**Table 18.7 | Characteristics of the Major Digestive Enzymes**

Enzyme	Site of Action	Source	Substrate	Optimum pH	Product(s)
Salivary amylase	Mouth	Saliva	Starch	6.7	Maltose
Pepsin	Stomach	Gastric glands	Protein	1.6-2.4	Shorter polypeptides
Pancreatic amylase	Duodenum	Pancreatic juice	Starch	6.7-7.0	Maltose, maltotriose, and oligosaccharides
Trypsin, chymotrypsin, carboxypeptidase	Small intestine	Pancreatic juice	Polypeptides	8.0	Amino acids, dipeptides, and tripeptides
Pancreatic lipase	Small intestine	Pancreatic juice	Triglycerides	8.0	Fatty acids and monoglycerides
Maltase	Small intestine	Brush border of epithelial cells	Maltose	5.0-7.0	Glucose
Sucrase	Small intestine	Brush border of epithelial cells	Sucrose	5.0-7.0	Glucose + fructose
Lactase	Small intestine	Brush border of epithelial cells	Lactose	5.8-6.2	Glucose + galactose
Aminopeptidase	Small intestine	Brush border of epithelial cells	Polypeptides	8.0	Amino acids, dipeptides, tripeptides