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Processes Discussed in the Digestive System:

Ingestion – food intake

Mastication – chewing (provides mechanical or physical digestion)

Deglutition – swallowing

Propulsion – (motility) movement through digestive tract via muscular action.

 circular (transverse) muscle – segmentation

 longitudinal muscle – peristalsis

Secretion – release of mucus, enzymes and other substances along with water.

Processes associated with the digestive system: **ingestion** - intake of food, **mastication**- chewing, a component of physical digestion, **deglutition** – swallowing, **propulsion** - movement of materials along the alimentary canal. Occurs mostly in the alimentary canal as muscular movements producing **segmentation** and **peristalsis**, **secretion** - the release of substances from cells in the digestive tract, e.g. mucus, enzymes, hormones, etc.



Digestive Processes (contd.)

Absorption – the transport of digestive endproducts into the blood or lymph.

Exfoliation – the shedding of mucosal lining cells.

Defecation – elimination of waste containing bacteria, exfoliated cells, and undigested materials.

Digestion

physical digestion – breaks down food mass exposing food to enzymes

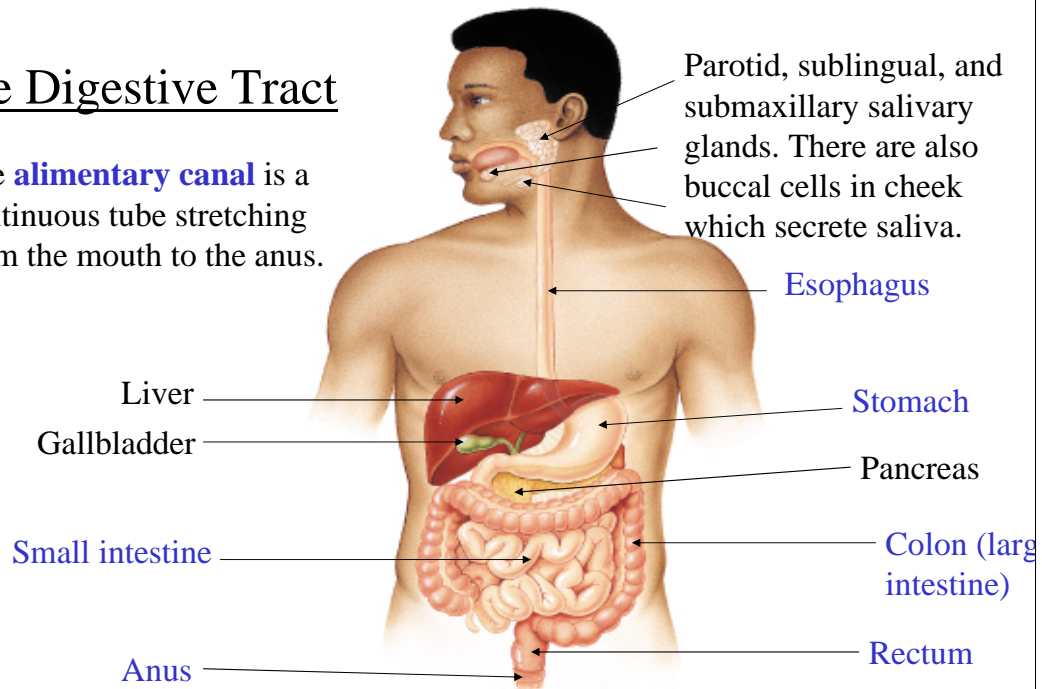
chemical digestion – enzymatic hydrolysis which breaks complex molecules into their subunits.

Absorption - the transport of digestive endproducts into the blood or the lymph; **defecation** - the removal of waste from the GI tract including undigested materials, exfoliated cells and bacteria; **exfoliation** - the constant shedding of the mucosal lining cells and their replacement by mitosis; **digestion** - consists of **physical (mechanical) digestion**, and **chemical digestion**; Physical digestion is the reduction in bulk and increase in surface area of ingested food.; Chemical digestion is **enzymatic hydrolysis** in which large complex molecules are broken down to their subunits. Physical digestion makes chemical digestion possible.



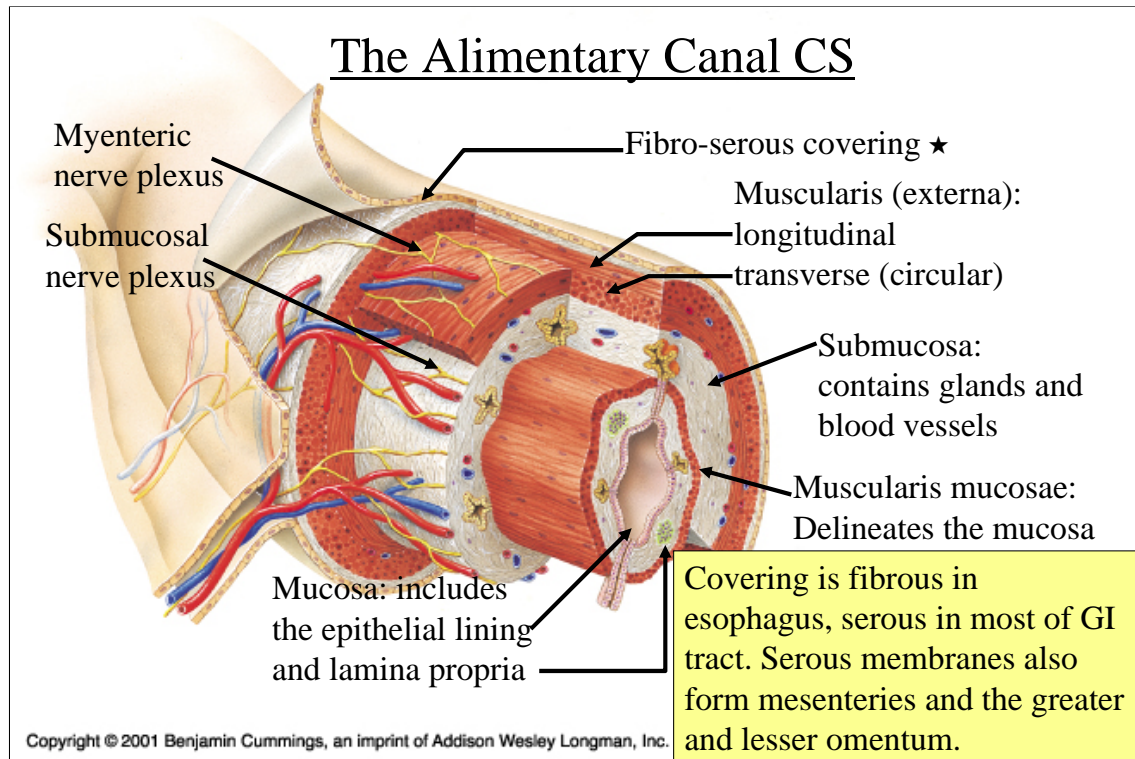
The Digestive Tract

The **alimentary canal** is a continuous tube stretching from the mouth to the anus.



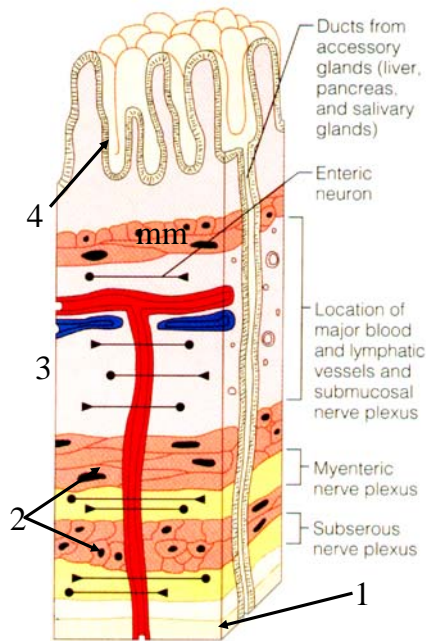
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The digestive tract is composed mostly of the alimentary canal (see next frame), together with accessory glands and organs. The alimentary canal is the continuous tube stretching from the mouth to the anus. Components of this tube, the various organs of the system, are specialized to perform particular functions. The stomach and intestines are commonly referred to as the GI (gastrointestinal) tract, but this designation is also often used to include the entire alimentary canal.



The **alimentary canal** is composed of four layers, each layer typically composed of certain tissues. But these layers can vary somewhat within the canal. **mucosa** - this is the lining tissue, mostly made of simple columnar epithelium (the mucosa of the esophagus is non-keratinized stratified squamous epithelium). Goblet cells within this layer secrete mucus for lubrication and protection and other cells may secrete enzymes, hormones etc. The lining through much of the alimentary canal exfoliates on a 3 to 5 day cycle. The gastrointestinal mucosa is also responsible for absorption of digestive endproducts. Beneath the epithelial surface is a connective-like component called the **lamina propria**. This layer contains blood and lymph capillaries for absorption. The boundary of the mucosa is the **muscularis mucosae**, the "muscle of the mucosa" which contracts to increase exposure of the mucosal lining to contents of the alimentary canal. The **submucosa** - this layer lies beneath the mucosa and is basically areolar connective tissue containing major blood vessels, nerves, and lymph nodes serving the alimentary canal. The submucosal nerve plexus controls the function of mucosal cells and digestive functions. The **muscularis** (or **muscularis externae**) - this is mostly smooth muscle (the esophagus has partly skeletal muscle) in two or three layers. In most of the GI tract two layers exist, the **longitudinal smooth muscle** layer and the **circular** or **transverse smooth** muscle layer. The circular layer squeezes to produce segments in the intestines, while the longitudinal layer causes the repeated shortening and lengthening called peristalsis. Segmentation contractions are mostly mixing actions, but work together with peristalsis in propulsion.

The **serosa** or **fibroserous layer** - this is the covering, a serous membrane in the portions of the alimentary canal in the peritoneal cavity and a fibrous covering in portions not in the peritoneal cavity or considered retroperitoneal.

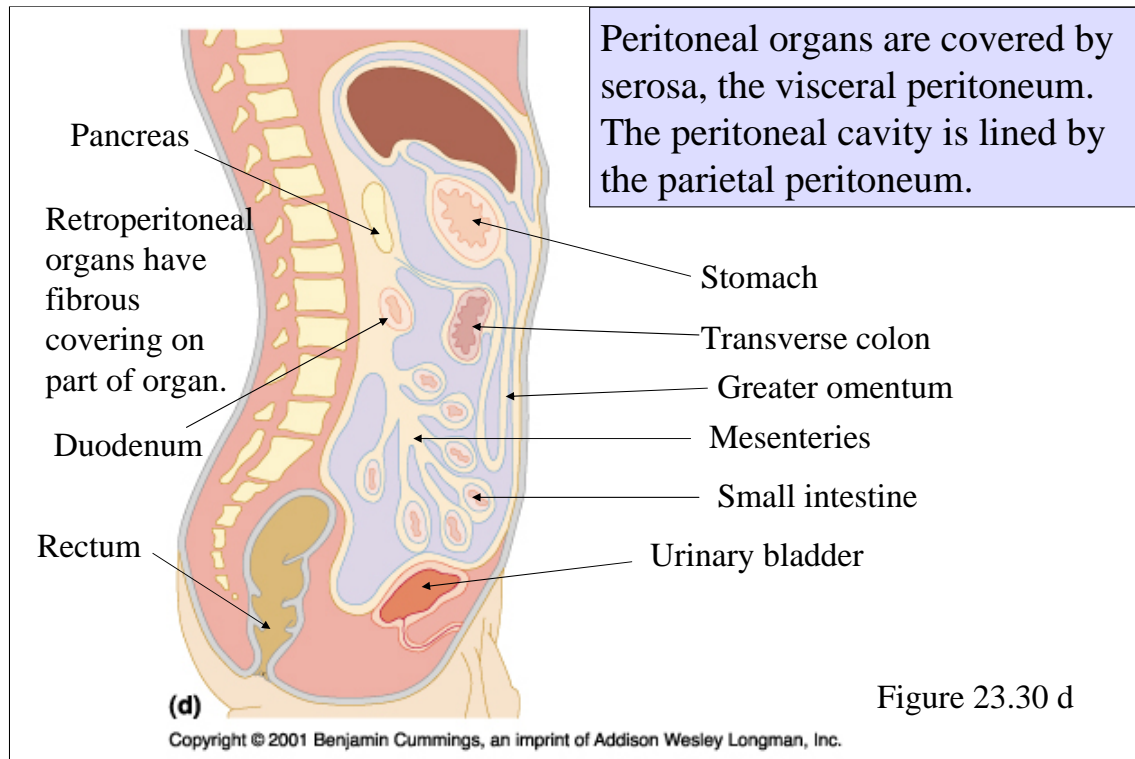


Wall of the Alimentary Canal

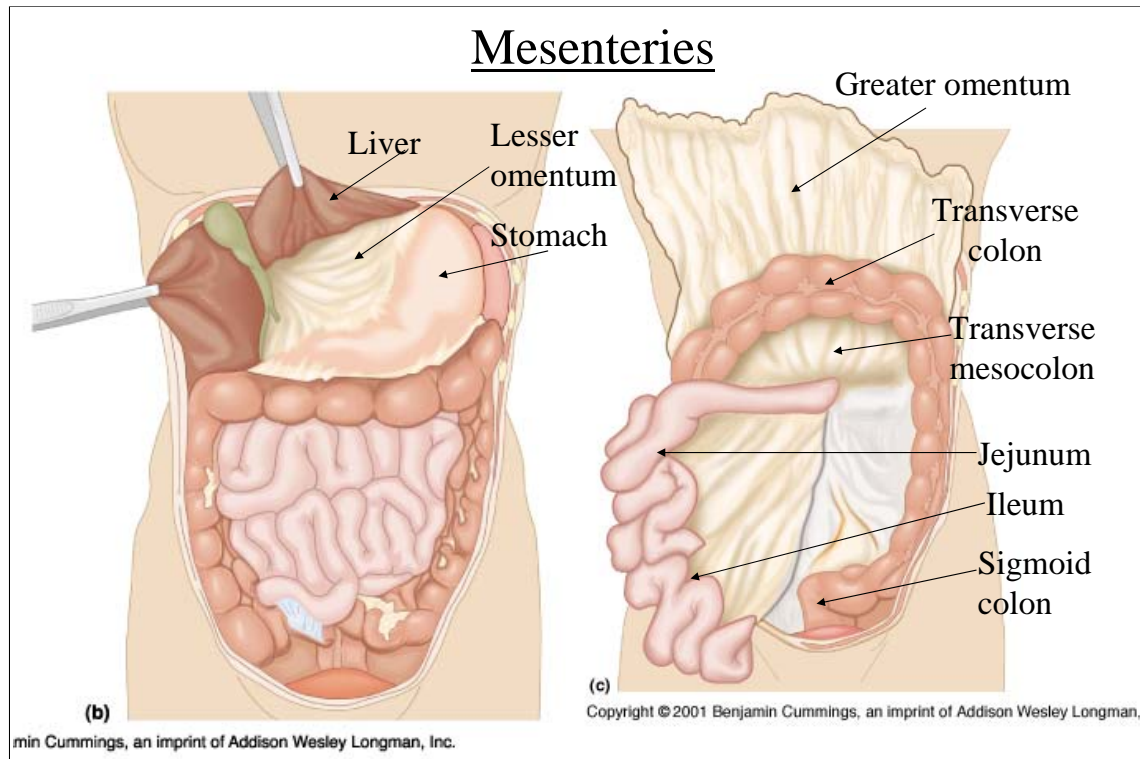
The alimentary canal consists of four layers. Listed from outside to inside: 1) the **fibroserous** outer covering; serous in most of GI tract, fibrous in esophagus. 2) the **muscularis** (externa), smooth muscle (except in upper esophagus) in two or three layers; 3) the **submucosa**, containing glands, nerves, and blood vessels; 4) the **mucosa**, the epithelial secretory and absorptive lining, bounded by the *muscularis mucosae* (mm).

Organs and Regions of the Alimentary Canal: (See previous frame)

Note that in this view the lining is shown with villi. This arrangement is seen in the small intestine.



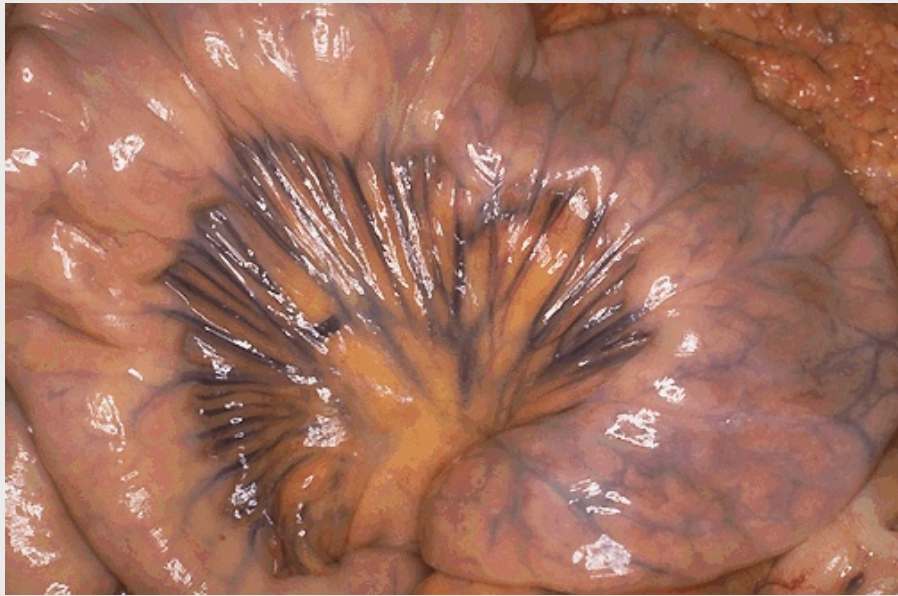
The **peritoneal cavity** (blue area in above slide) houses most of the digestive organs. It is lined with a serous membrane, the **parietal peritoneum**, which is continuous with the **visceral peritoneum** that covers these organs. Within the **abdomino-pelvic** region there are also organs not in the peritoneal cavity or **retroperitoneal**, which are covered with **fibrosa** rather than **serosa**.



The **mesenteries** are double layers of serous membrane, composed of peritoneal membranes which have folded against each other. These mesenteries connect and hold gastrointestinal organs in place and attach blood vessels and nerves. They also, with their fatty coverings, protect and insulate the organs. The **greater omentum**, for instance, hangs in front of the intestines acting as an insulator and shock absorber.



The Mesenteries



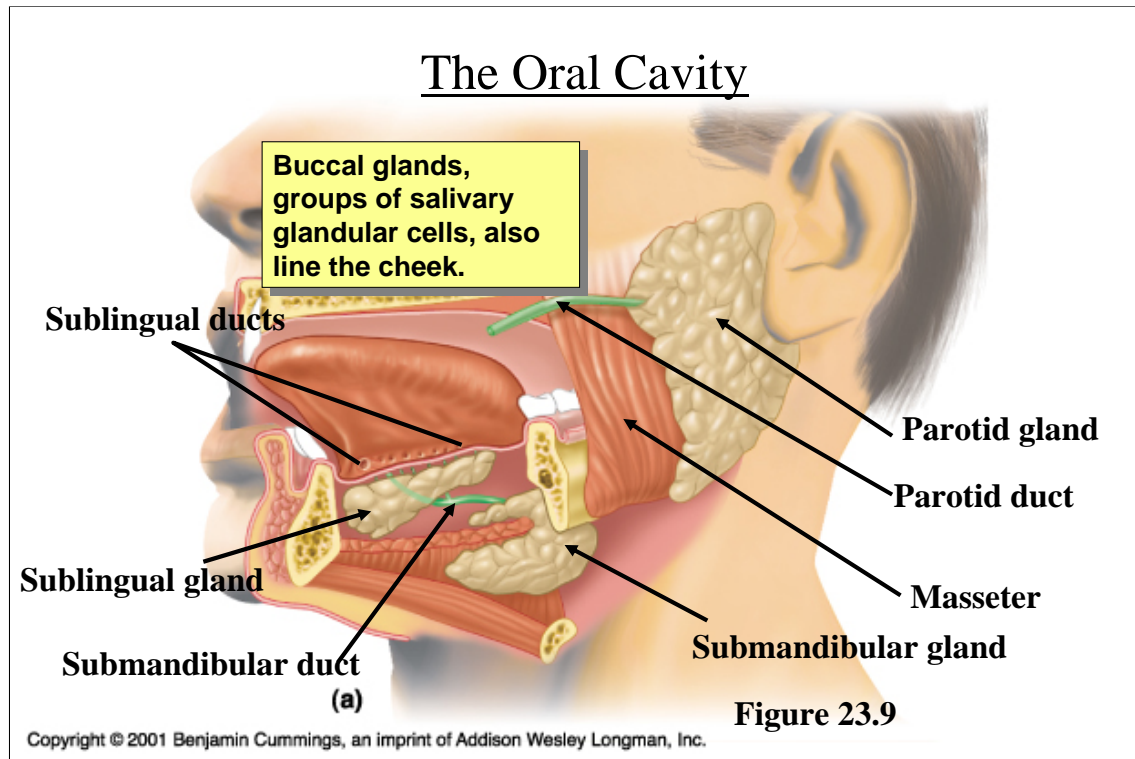
Seen here is a loop of bowel attached via the mesentery. Note the extent of the veins and arteries. There is an extensive anastomosing arterial blood supply to the bowel, making it more difficult to infarct. Also, the extensive venous drainage is incorporated into the portal venous system heading to the liver.



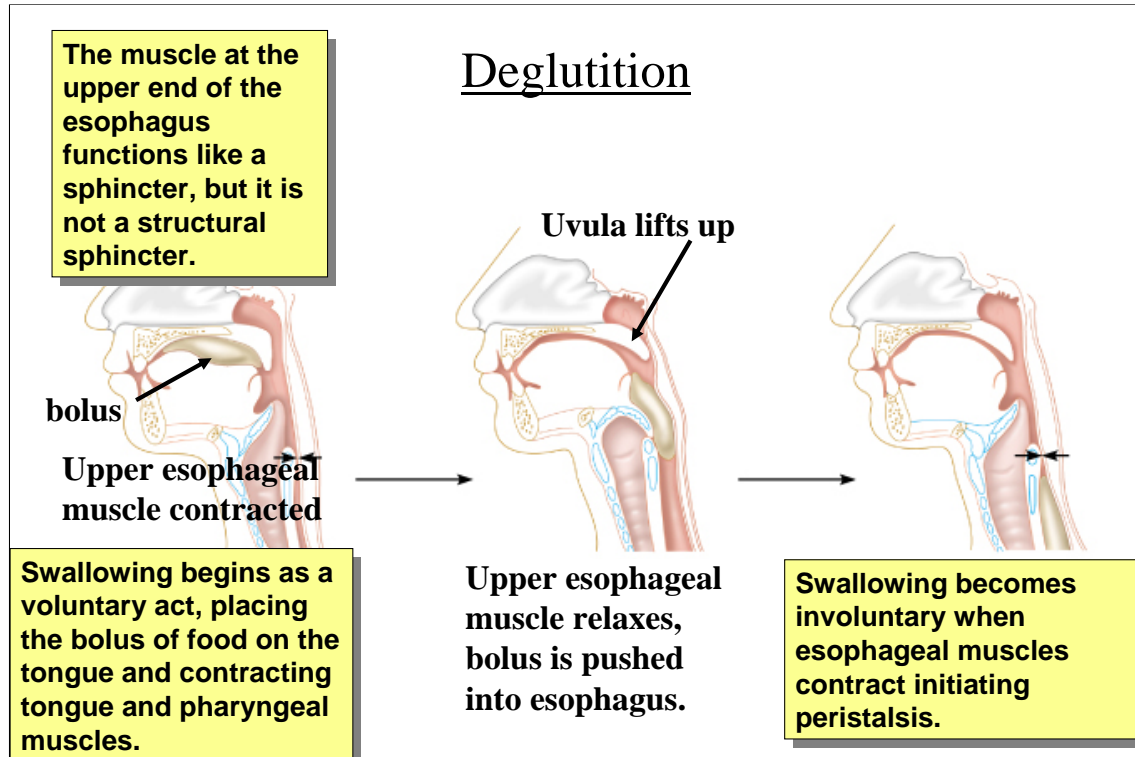
Digestive Chart: The Mouth

AREA	PROCESSES	SECRETIONS	CONTROLS	HISTOLOGY
Mouth	mechanical digestion chemical digestion: starches->shorter chains	saliva: salivary amylase (ptyalin) Saliva also contains water, electrolytes, mucus and serous fluid, lysozyme.	cephalic physical contact Presso- receptors and chemo- receptors respond to presence of substances in the mouth.	non-keratinized stratified squamous; salivary glands

The mouth: The mucosa of the mouth is composed of mostly non-keratinized stratified squamous epithelium. This mucosa continues through the esophagus. Three salivary glands on each side, plus buccal glands in the mucosa, provide the fluid known as saliva. Saliva contains water, salts, mucin, serous fluid, lysozyme, IgA, growth factors, and amylase.



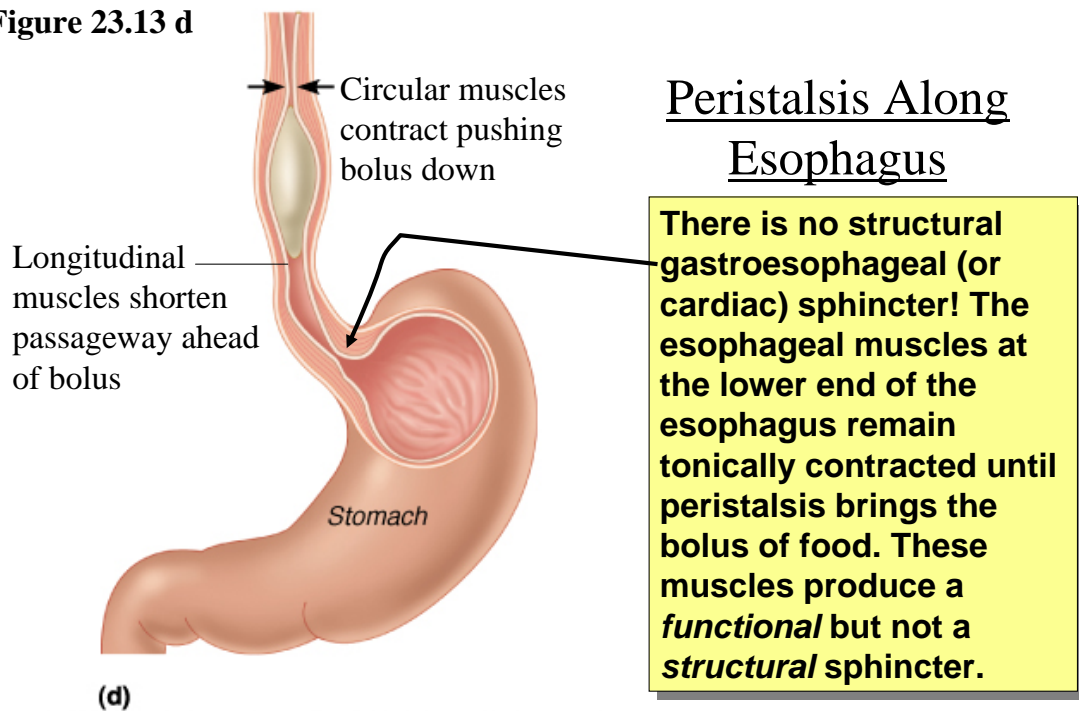
The three glands (**parotid**, **submandibular**, and **sublingual**) produce varying amounts of salivary components. The pH of this fluid is from 6.35 to 6.85, supporting the action of salivary amylase to begin the breakdown of **polysaccharides to shorter chains**. The action does not normally progress very far due to the short exposure to active enzyme.



Chewing is a form of mechanical digestion which reduces the bulk of the food and, especially, exposes it to the enzyme. The bolus of food is swallowed in a process called **deglutition** which begins as voluntary and becomes involuntary. At first the bolus is lodged on the tongue and pushed voluntarily into the pharynx. Then pharyngeal muscles contract pushing the bolus into the esophagus where peristalsis begins. Peristaltic waves move food down the esophagus into the stomach.



Figure 23.13 d

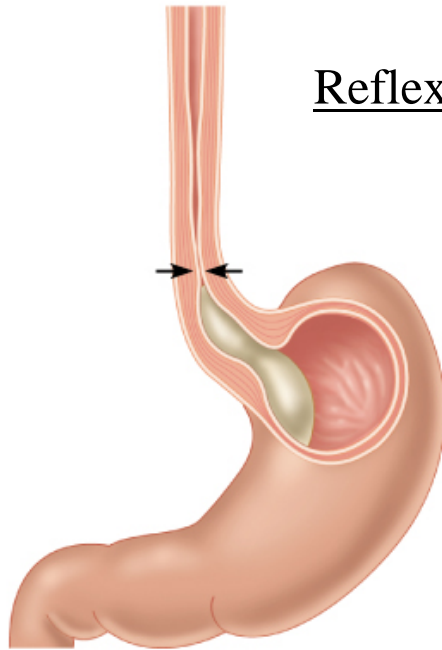


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Peristalsis begins in the esophagus and moves the bolus into the stomach. The muscle at the lower end of the esophagus remains contracted until the bolus arrives, then briefly relaxes to allow the bolus to pass, then tonically contracts again. Although traditionally referred to as the *cardiac sphincter* (now called the **gastroesophageal region**) of the stomach, there is **no structural sphincter** or valve in this region, and the region has nothing to do with the heart.



Reflexive Relaxation



The esophageal muscles at the junction with the stomach reflexively relax to admit the bolus of food, then return to tonic contraction. It is this tonic contraction which leads some to consider this a sphincter.

(e)

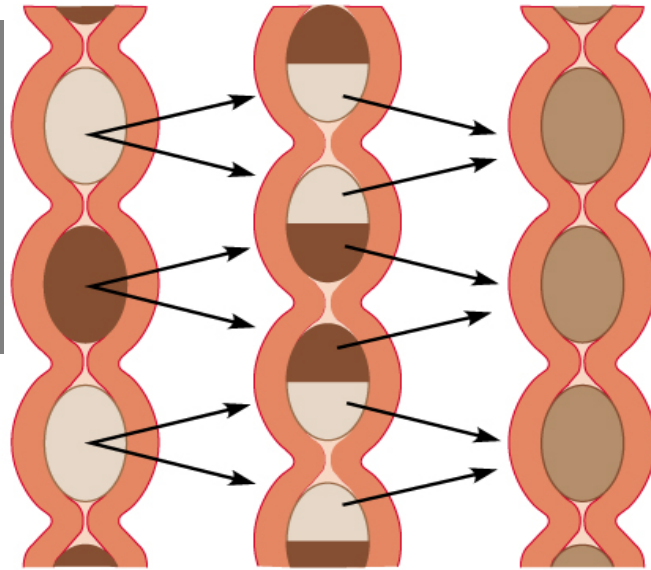
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As the bolus enters the stomach the muscle at the gastroesophageal junction first relaxes, then contracts behind the bolus.



Segmentation

Circular muscles alternately contract and relax along the alimentary canal. This mixes and liquefies the food as well as helping to propel it along.



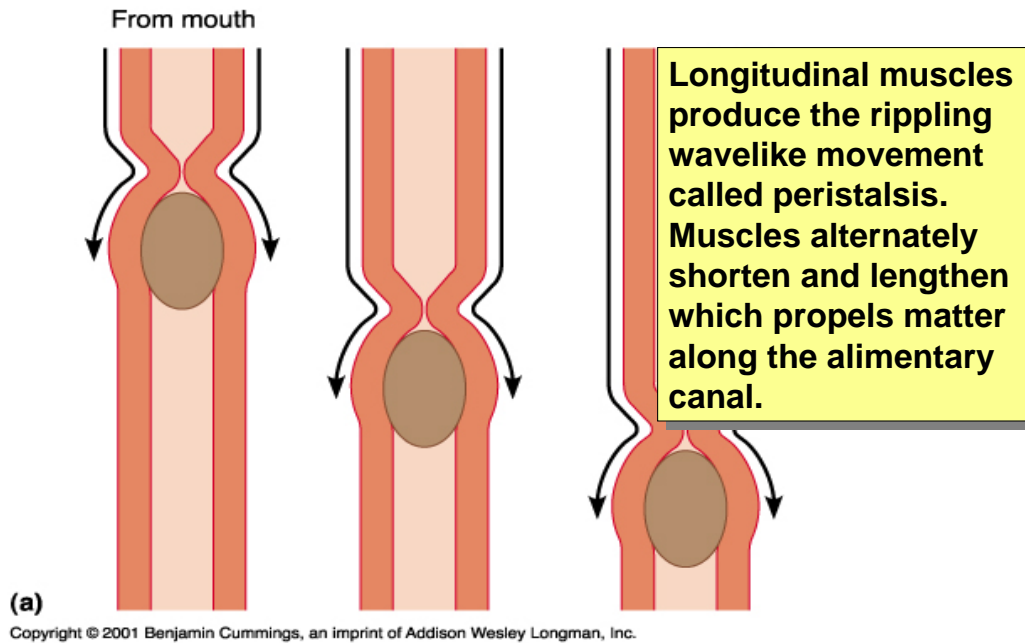
(b)

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Segmentation is one of two muscular actions in the esophagus and it continues throughout most of the GI tract. It produces mixing of the contents and works together with peristalsis to move them along.



Peristalsis



Longitudinal muscle contraction produces peristalsis, the wave-like movements which, together with segmentation, propel the contents along the esophagus and GI tract. Most people use the term peristalsis to include both processes.



Digestive Chart: Esophagus

AREA	PROCESSES	SECRETIONS	CONTROLS	HISTOLOGY
Esophagus	peristalsis begins	mucus	involuntary reflex	non-keratinized stratified squamous esophageal glands skeletal & smooth muscle

Lining tissue is a continuation of that in the oropharynx and laryngopharynx

Submucosal esophageal glands secrete mucus in place of goblet cells.

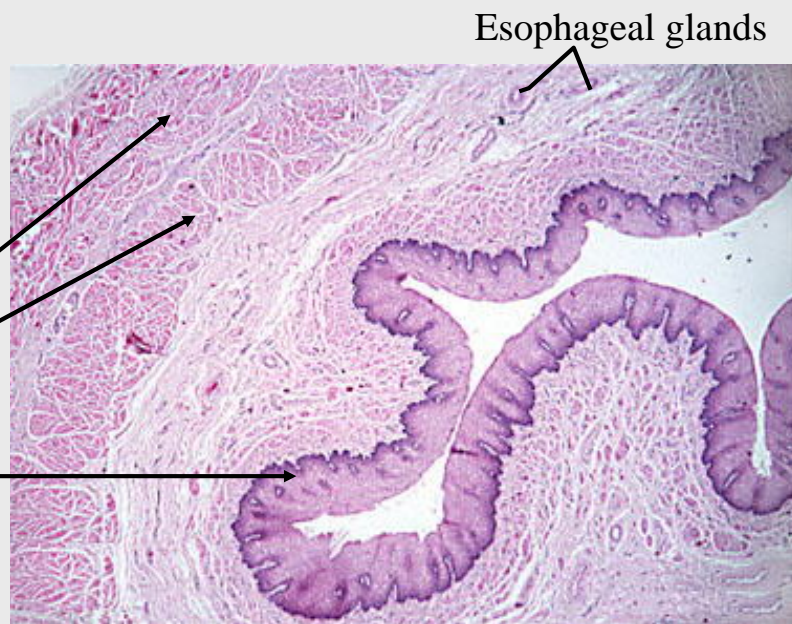
The upper 1/3 is skeletal, middle 1/3 is mixed, lower 1/3 is smooth muscle

The esophagus: The esophagus is about 10" long and is also lined with non-keratinized stratified squamous epithelium.

Esophageal glands located in the submucosa produce mucus for lubrication. The first third of the muscularis is skeletal, the last third is smooth muscle, and the middle of the esophagus is mixed smooth and skeletal muscle.



The upper third of the esophagus is marked by skeletal muscle. Notice the outer longitudinal and inner circular muscle layers. The lining is non-keratinized stratified squamous epithelium.



Esophageal glands

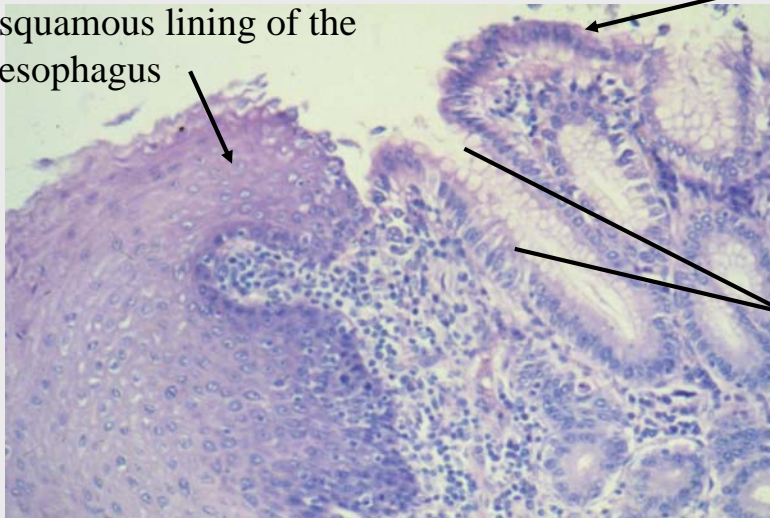
The Esophagus

Because the esophagus has stratified squamous epithelium as a lining and does not have goblet cells, it must have glands to secrete lubricating serous fluid and mucus.



Gastroesophageal Junction

Note the stratified squamous lining of the esophagus

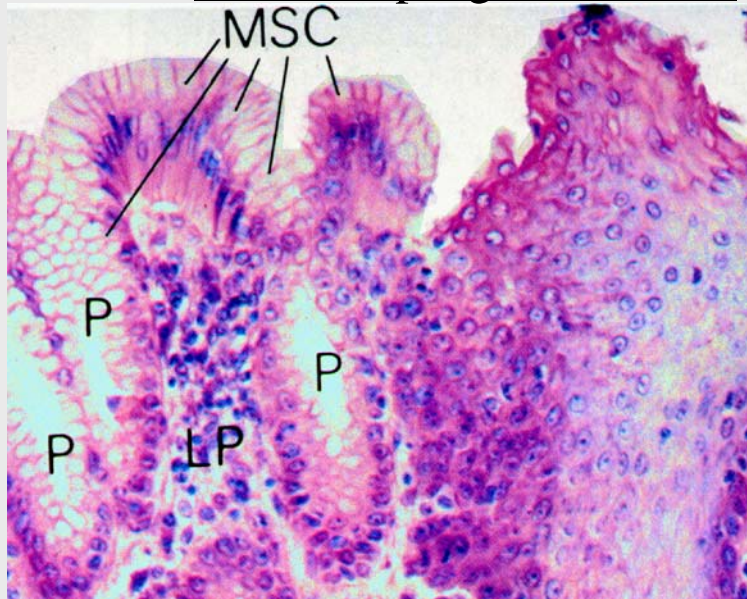


Simple columnar epithelial lining in the stomach. Virtually all the cells seen in this lining are mucus surface cells.

The lining abruptly changes at the gastroesophageal junction from the stratified squamous epithelium of the esophagus to the simple columnar epithelium of the stomach. The stomach mucosa is heavily populated with mucus secreting cells.



Gastroesophageal Junction

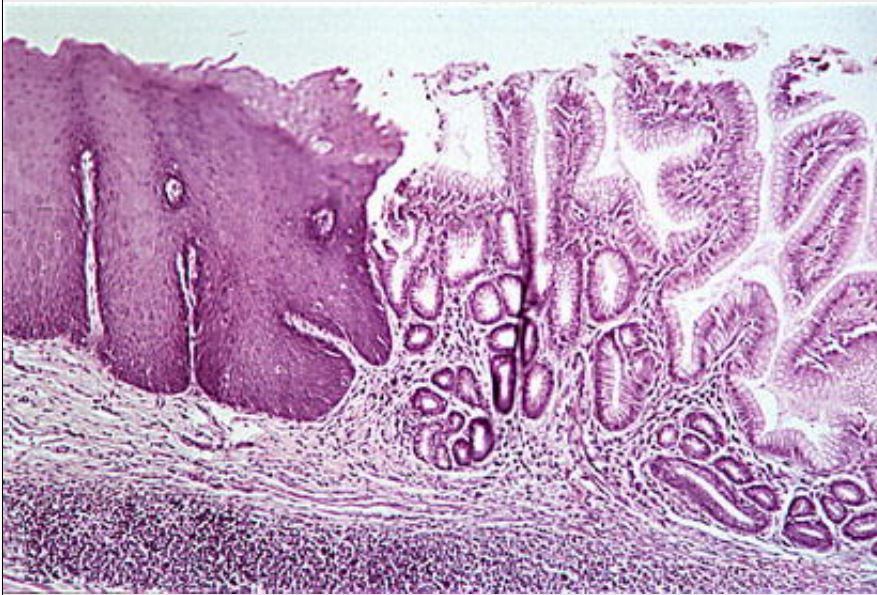


mucus
surface
cells
(MSC),
gastric
pits (P),
lamina
propria
(LP)

Most of the cells within the epithelial lining near the upper portion of the gastric pits are mucus surface cells. Although they look like goblet cells, and are sometimes called goblet cells, they are in fact from a different cell line.

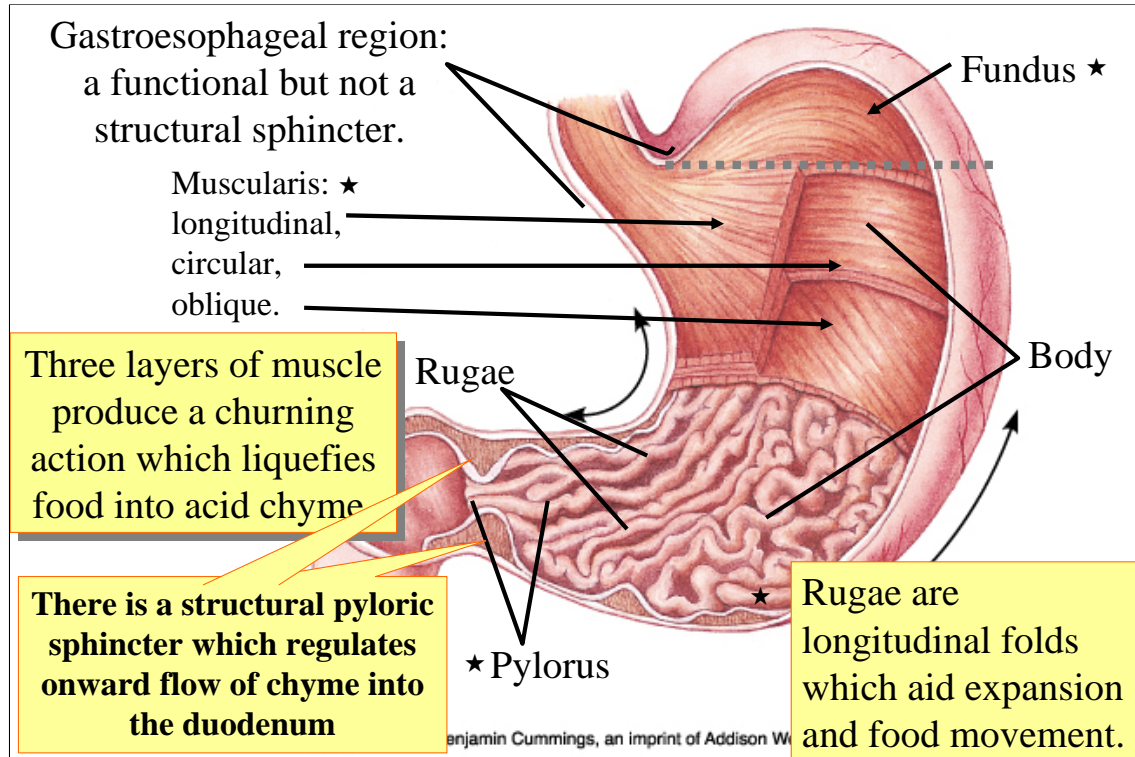


Gastroesophageal Junction



**Which side
is the
stomach,
which is the
esophagus?**

The stratified squamous epithelium on the left differs considerably from the gastric pits lined with columnar epithelium seen on the right.



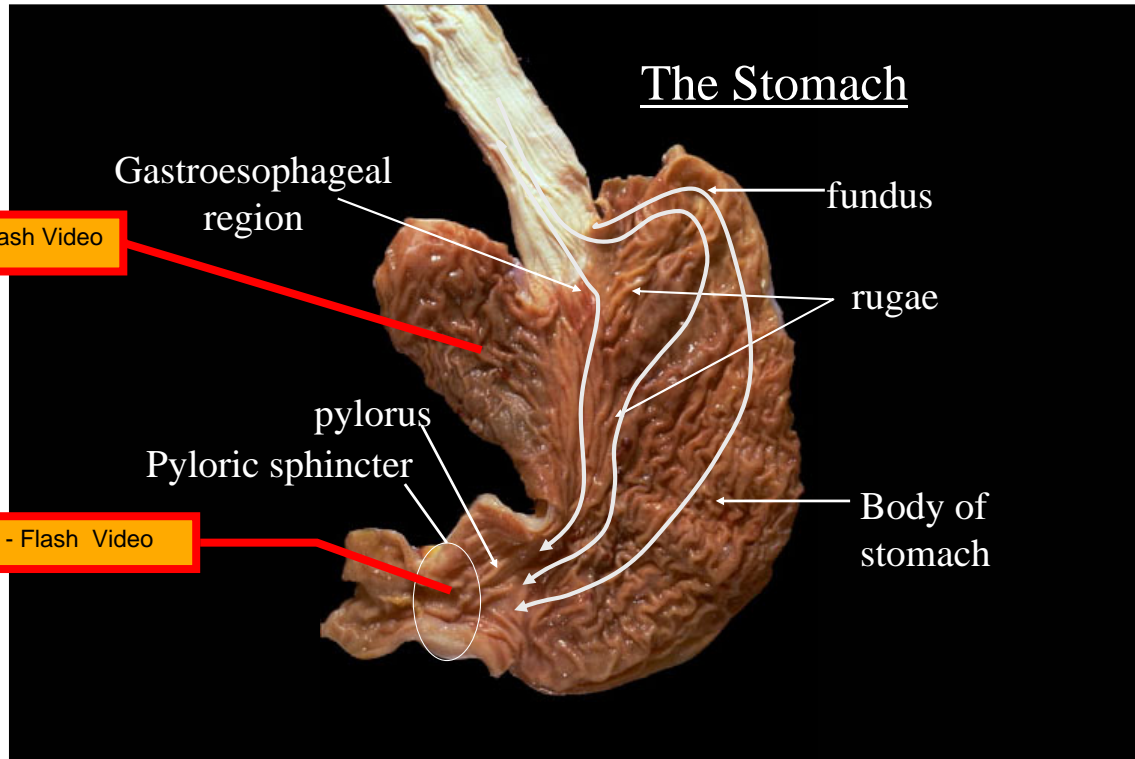
The stomach: The stomach is composed of several regions and structures -
1) The **gastroesophageal** region (a.k.a. cardia) mentioned above.

2) The **fundus**, the blind portion of the stomach above its junction with the esophagus. This portion is thin walled compared to the rest of the stomach and has few secretory cells. As the bolus of food enters this area first some action of salivary amylase may continue briefly.

3) The **body** of the stomach. This is where extensive **gastric pits** are located which possess the secretory cells of the stomach.

4) The **pylorus**. This narrowed region leads through the **pyloric** sphincter into the **duodenum**.

3-layered muscularis - an **oblique** layer in addition to the **longitudinal** and **transverse** layers. The three layers produce a churning and liquefying effect on the **chyme** in the stomach.



Rugae are the extensive folds in the stomach lining. These folds can stretch to accommodate an increase in stomach volume with consumption of a meal. They also help direct the food downward toward the pylorus as a result of stomach motility.



Digestive Chart: The Stomach

AREA	PROCESSES	SECRETIONS	CONTROLS	HISTOLOGY	
Stomach	storage (up to 4 hrs) mechanical digestion some absorption	pepsinogen + HCl ★ mucus	cephalic; contact; gastric phase: <i>gastrin</i>	simple columnar gastric pits mucus neck cells	
	chemical digestion: polypeptides --> shorter chains	<i>gastrin</i>	intestinal phase: <i>gastrin, GIP,</i> enterogastric reflex	3-layered smooth muscle	
Mostly on an empty stomach substances absorbed include: alcohol, water, electrolytes, glucose, fat-soluble molecules		<div><div>Pepsinogen + H⁺ → pepsin</div><div>↓ pH 1.5-3.5</div><div>Polypeptides → shorter chains</div></div>			Gastrin is a hormone involved in control of gastric activity.

- Processes occurring in the stomach:**
- 1) Storage - the stomach allows a meal to be consumed and the materials released incrementally into the duodenum for digestion. It may take up to four hours for food from a complete meal to clear the stomach.
 - 2) Chemical digestion - pepsin begins the process of protein digestion cleaving large polypeptides into shorter chains .
 - 3) Mechanical digestion - the churning action of the muscularis causes liquefaction and mixing of the contents to produce **acid chyme**.
 - 4) Some absorption - water, electrolytes, monosaccharides, and fat soluble molecules including alcohol are all absorbed in the stomach to some degree.

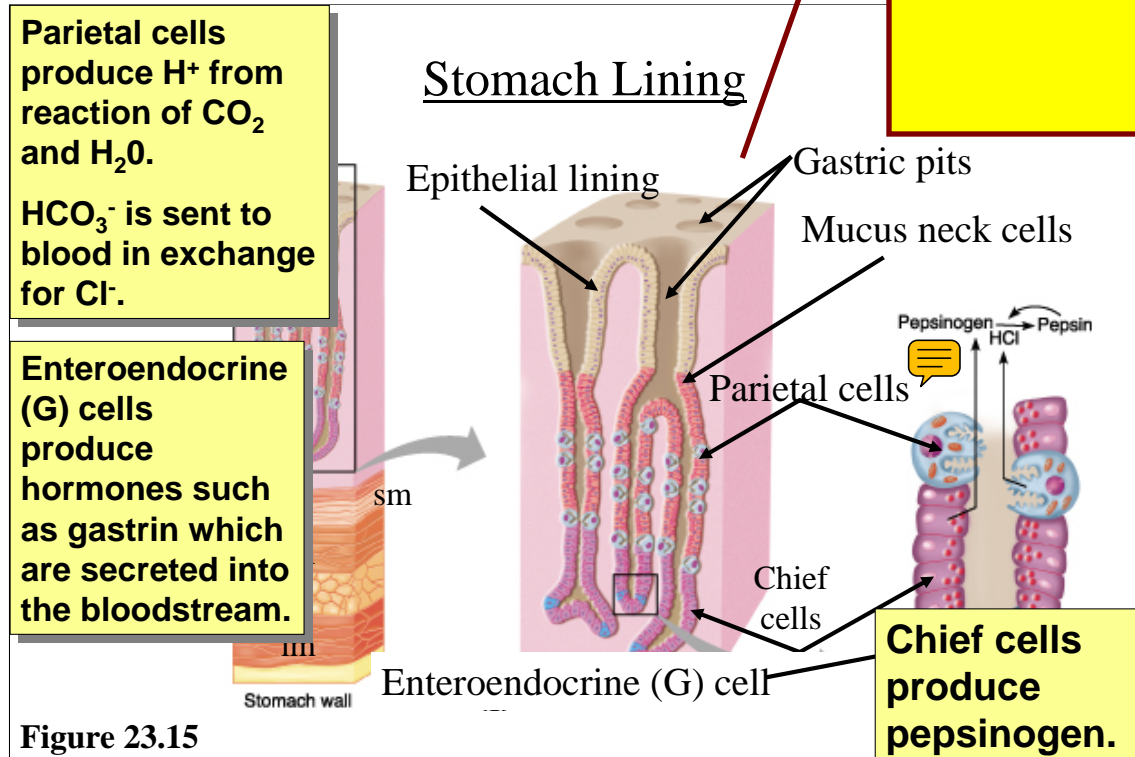
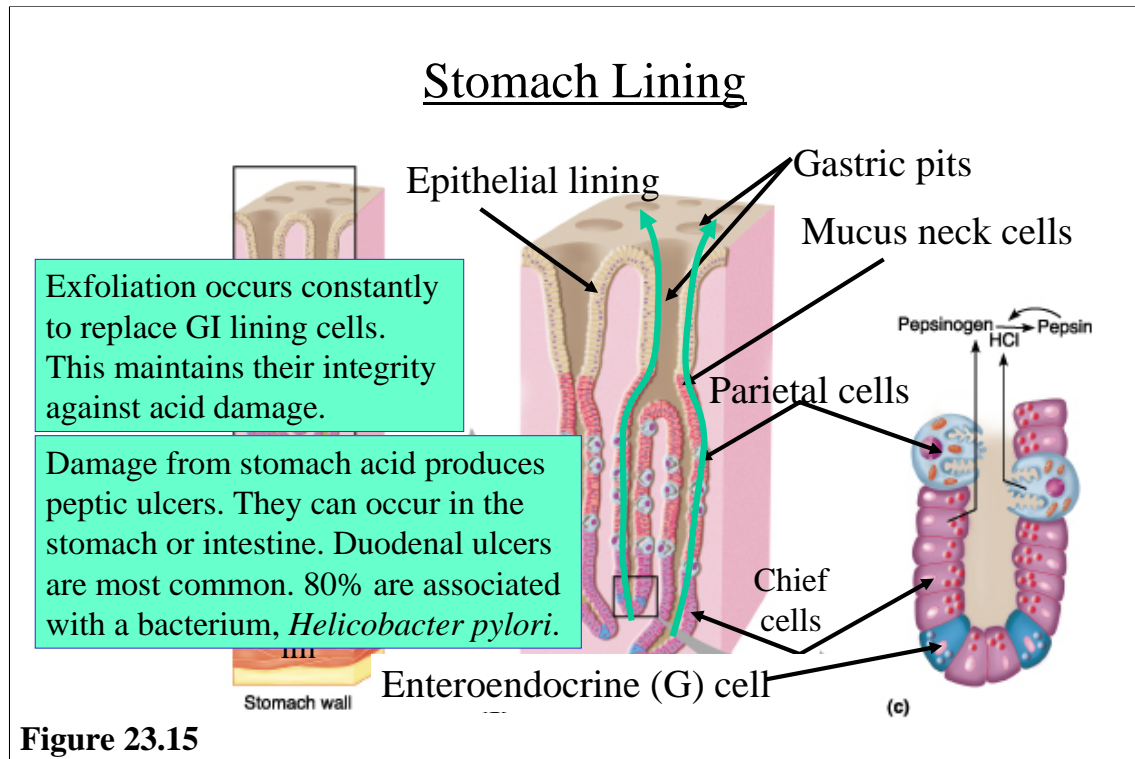


Figure 23.15

Gastric pits increase the surface mucosa for secretion and absorption. Specialized columnar epithelial cells release enzymes and other substances: **zymogen (chief) cells** release **pepsinogen** and **parietal cells** release **hydrochloric acid**. **[IMPORTANT NOTE:** Actually these cells secrete H^+ , derived from the same chemical reaction of CO_2 and water which produces carbonic acid in the blood. The bicarbonate ions are retained and transported into the blood and the chloride ions are exchanged for them and pass into the stomach.] The H^+ causes activation of the pepsinogen to produce the protease **pepsin**. **Mucous neck cells** and **mucous surface cells** (there are no true goblet cells in the stomach) produce an alkaline mucus which helps protect the lining from the acidity, which in the stomach reaches a pH from 1.5 to 3.5. Enteroendocrine cells produce a number of hormone substances including gastrin, histamine, endorphins, serotonin and somatostatin. Cells lining the gastric pits are arranged in circular acini in the stomach called gastric glands. These glands are found throughout the stomach and vary from one area to another with regard to their complement of cells.

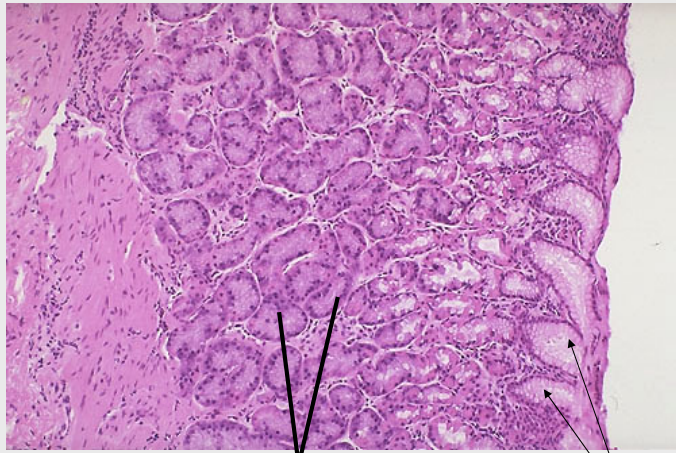


Protection from the acid produced by the stomach is afforded by ¹⁾ the tight junctions of the mucosal lining cells, ²⁾ the alkaline mucus secreted by the mucous neck cells and surface mucous cells, and by ³⁾ constant **exfoliation** of lining cells and their replacement by mitosis. On average the stomach lining has a **3 day turnover**. Acid does occasionally make its way into the esophagus causing a burning sensation of the esophageal lining, formerly called "heartburn" and now called acid reflux disease. Peptic ulcer is the name given to damage to lining cells due to stomach acid.

The greatest proportion of peptic ulcers actually occur in the duodenum. A bacterium, *Helicobacter pylori*, has been associated with many ulcers and treatment has often focused on this bacteria. Other causative agents such as increased histamine secretion may reflect the relationship of ulcers to stress.



Stomach Fundus



Gastric glands

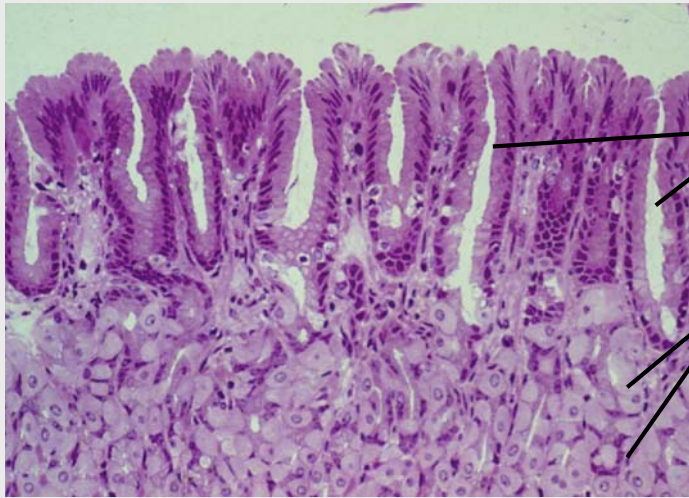
Gastric pits

The fundus is marked by deep glands and shorter pits. The fundus tends to be thinner than other stomach areas and exhibits less secretion.

The **fundus** of the stomach is comparatively thinner than the other areas. Its pits are shallower and it has fewer cells which secrete enzyme of acid. It does have abundant mucus secreting cells.



Body of the Stomach

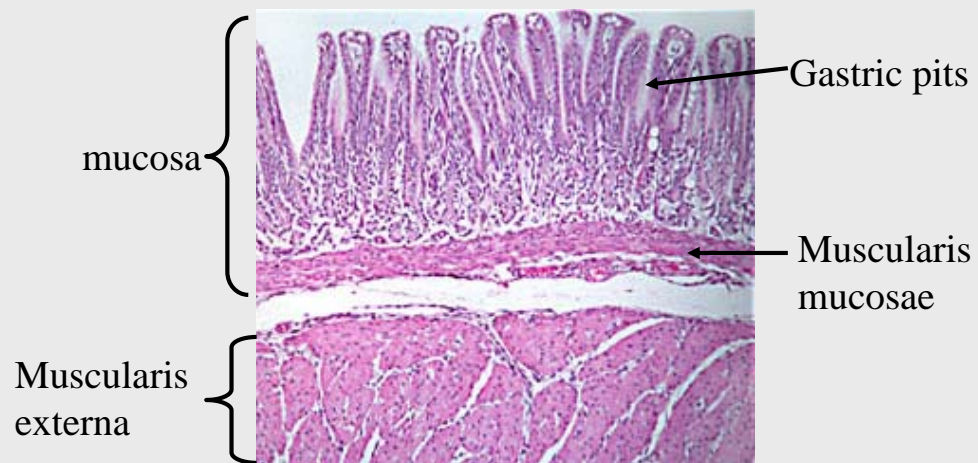


Note the deep gastric pits and the numerous mucus-secreting glands. The cells seen in these glands are mucous secreting cells.

The **body** of the stomach is where most enzyme (precursor) and acid is secreted and its pits are much deeper. The body is noticeably thicker in gross examination than the fundus.



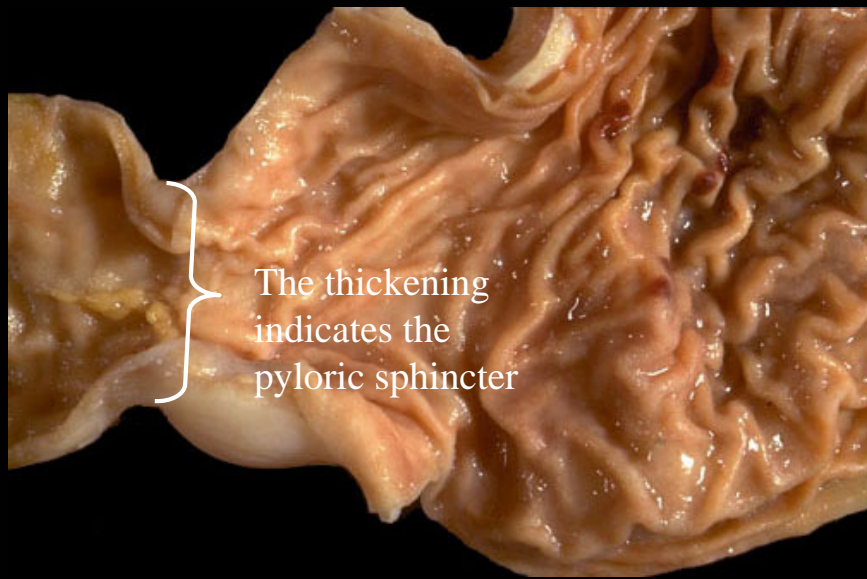
Stomach Pylorus



The **pylorus** has thick muscle, including a **pyloric sphincter**. Also found here in the mucosa are **enteroendocrine cells** which secrete **gastrin**.



Pyloric Sphincter



The **pyloric sphincter** is a structural sphincter which regulates the onward progression of materials from the stomach into the duodenum, and helps to prevent their return to the stomach.



Control of Stomach Functions

Cephalic phase – prepares stomach to receive food, results from stimulation by the Vagus n.

Gastric phase

Secretion of pepsinogen, H^+ and Cl^- secretion from parietal cells

acetylcholine – from Vagus nerve

histamine – from ECL cells, secreted in response to gastrin and ACh.

gastrin – hormone from the G cells, stimulates ECL cells to release histamine

Control of processes in the stomach: The stomach, like the rest of the GI tract, receives input from the autonomic nervous system. Positive stimuli come from the parasympathetic division through the vagus nerve. This stimulates normal secretion and motility of the stomach. Control occurs in several phases: the **cephalic phase** stimulates secretion in anticipation of eating to prepare the stomach for reception of food. The secretions from cephalic stimulation are watery and contain little enzyme or acid.

The **gastric phase** of control begins with a direct response to the contact of food in the stomach and is due to stimulation of pressoreceptors in the stomach lining which result in ACh and histamine release triggered by the vagus nerve. The secretion and motility which result begin to churn and liquefy the chyme and build up pressure in the stomach. Chyme surges forward as a result of muscle contraction but is blocked from entering the duodenum by the pyloric sphincter. A phenomenon called **retropulsion** occurs in which the chyme surges backward only to be pushed forward once again into the pylorus. The presence of this acid chyme in the pylorus causes the release of a hormone called **gastrin** into the bloodstream. Gastrin has a positive feedback effect on the motility and acid secretion of the stomach. This causes more churning, more pressure, and eventually some chyme enters the duodenum. There the **intestinal phase** of stomach control occurs.

At first this involves more gastrin secretion from duodenal cells which acts as a "go" signal to enhance the stomach action already occurring. But as more acid chyme enters the duodenum the decreasing pH inhibits gastrin secretion and causes the release of negative or "stop" signals from the duodenum.

These take the form of chemicals called **enterogastrones** which include **GIP (gastric inhibitory peptide)**.

GIP inhibits stomach secretion and motility and allows time for the digestive process to proceed in the duodenum before it receives more chyme. The enterogastric reflex also reduces motility and forcefully closes the pyloric sphincter. Eventually as the chyme is removed, the pH increases and gastrin and the "go" signal resumes and the process occurs all over again. This series of "go" and "stop" signals continues until stomach emptying is complete.

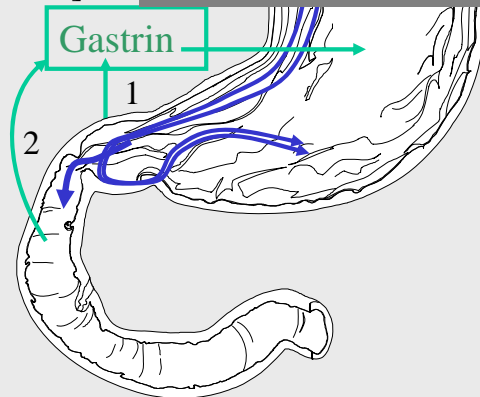


Excitatory Stimuli

1) **Gastric phase:** Increasing motility builds pressure of chyme into the pylorus. Gastrin is secreted into the blood, which increases motility and secretion of H^+ in the stomach. [+FB]

2) **Intestinal phase:** Pressure forces acid chyme into the duodenum and, at first, causes more gastrin secretion. [+FB]

At first chyme is unable to pass through the pyloric sphincter and retropulsion occurs.



Local excitatory stimuli occur in two phases: First in the **gastric phase** as materials enter the stomach pylorus, then again as they move from the pylorus into the duodenum in the **intestinal phase**. The excitatory stimulus is **gastrin**.



Control of Stomach Functions (Contd.)

Intestinal phase

Positive Feedback: due to intestinal gastrin – when pH is above a setpoint.

Negative Feedback: when ↓ pH reaches the setpoint.

enterogastric reflex –

inhibition of parasympathetic n.s. inhibits stomach motility.

stimulation of sympathetic n.s forcefully contracts pyloric sphincter.

enterogastrones: GIP, CCK, Secretin

In the **intestinal phase** first gastrin continues the excitatory stimuli which began in the stomach. Then inhibitory stimuli take over as the acid levels reach their low points. Inhibitory stimuli via GIP and other hormones as well as the nervous system reduce stomach emptying.

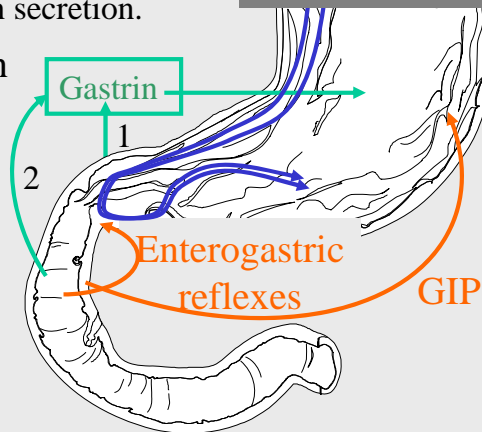


Excitatory/inhibitory

- 1) Gastric phase: Increasing motility builds pressure of chyme into the pylorus. Gastrin is secreted into the blood, which increases motility and secretion of H^+ in the stomach.
- 2) Intestinal phase: Acid chyme entering the duodenum at first results in more gastrin secretion.
- 3) As acidity increases the gastrin secretion ends and inhibitory processes begin: secretion of GIP and the enterogastric reflexes.

GIP (Gastric Inhibitory Peptide) inhibits motility and H^+ secretion in the stomach.

Enterogastric reflex causes forceful contraction of the pyloric sphincter and inhibition of other muscles.



The excitatory and inhibitory stimuli act as stop and go signals to direct stomach emptying into the duodenum for mixing with digestive enzymes. This stop and go processing continues as long as food remains in the stomach, which can be up to four hours for a full meal.



Stop and Go in Gastric→Duodenal Emptying

Go signal: gastrin - Positive feedback signals to the stomach to keep up secretion and motility.

Stop signal: GIP and other enterogastrones – Negative feedback signals which reduce stomach motility and secretion to provide time for digestion.

Summary of the events in gastric function: 1) Signals from vagus nerve begin gastric secretion in **cephalic phase**.

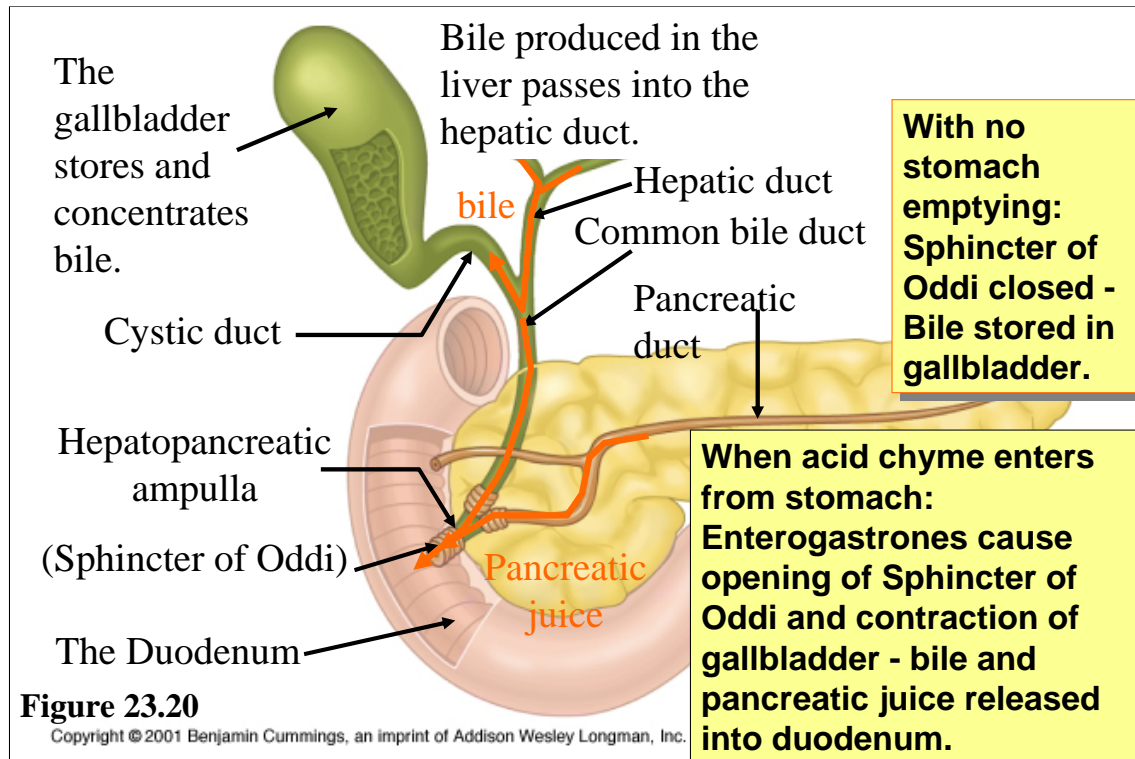
2) Physical contact by food triggers release of pepsinogen and H^+ in **gastric phase**.

3) Muscle contraction churns and liquefies chyme and builds pressure toward pyloric sphincter.

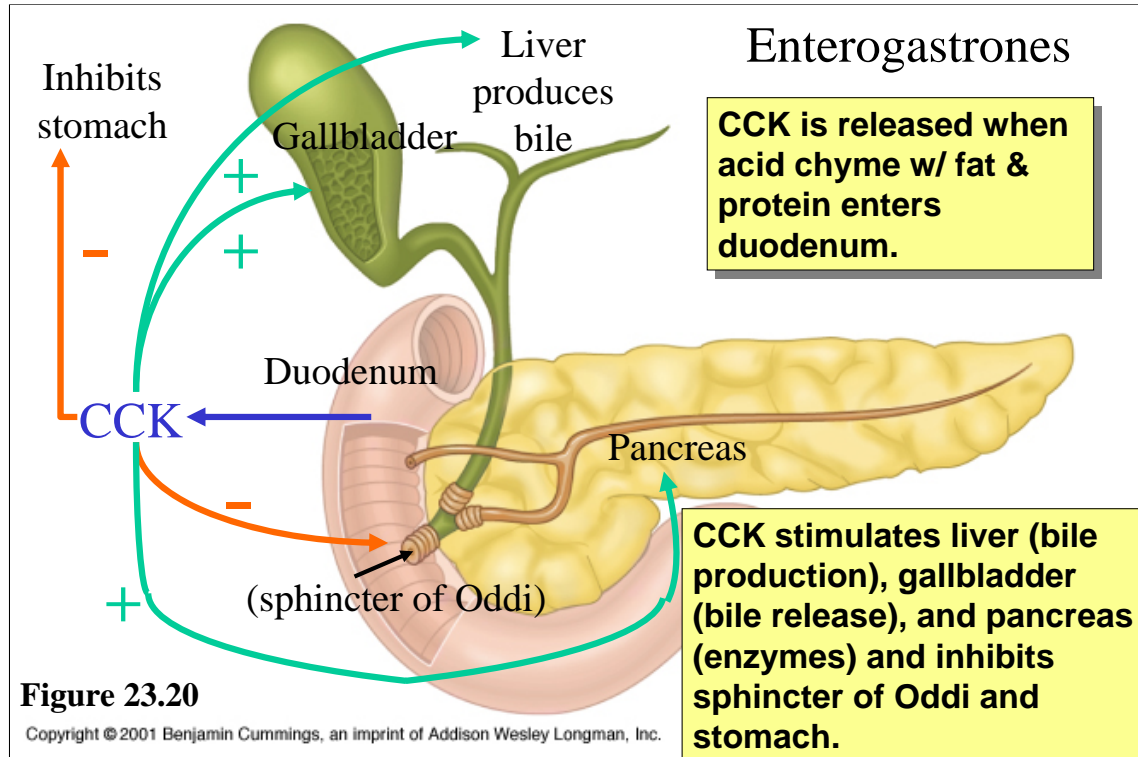
4) Gastrin is released into the blood by cells in the pylorus. Gastrin reinforces the other stimuli and acts as a positive feedback mechanism for secretion and motility.

5) The **intestinal phase** begins when acid chyme enters the duodenum. First more gastrin secretion causes more acid secretion and motility in the stomach.

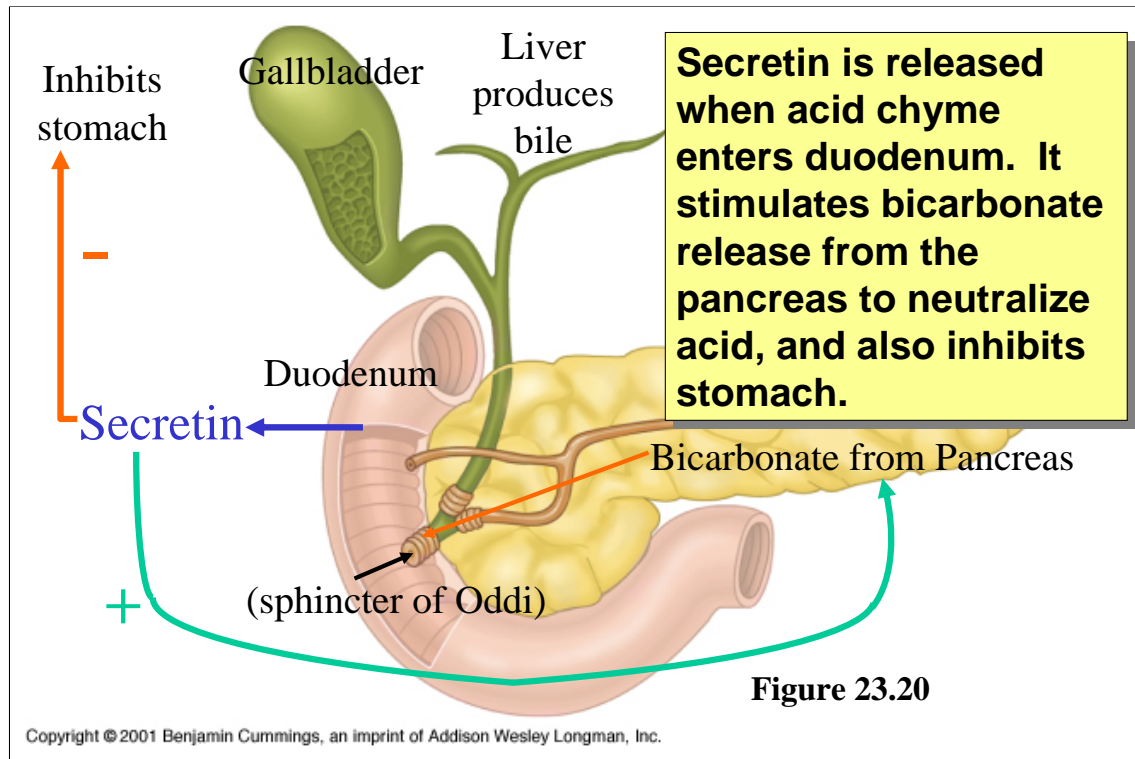
6) Low pH inhibits gastrin secretion and causes the release of enterogastrones such as GIP into the blood, and causes the enterogastric reflex. These events stop stomach emptying and allow time for digestion in the duodenum before gastrin release again stimulates the stomach.



The duodenum is the site of most digestive enzyme release. Intensive digestion begins here. The duodenum is the first 10" of the small intestine, and receives secretions from the **pancreas**, from the **intestinal mucosal cells**, and from the **liver** and **gallbladder**. Secretions from the pancreas and bile from the gallbladder enter the duodenum through the **hepatopancreatic ampulla** and the **sphincter of Oddi**. These lie where the **pancreatic duct** and **common bile duct** join before entering the duodenum. The presence of fatty chyme in the duodenum causes release of the hormone **CCK** into the bloodstream. CCK is one of the enterogastrones and its main function, besides inhibiting the stomach, is to stimulate the release of enzymes by the pancreas, and the contraction of the gallbladder to release bile. The acid in the chyme stimulates the release of secretin which causes the pancreas to release **bicarbonate** which neutralizes the acidity.



CCK is an **enterogastrone** which inhibits the stomach, but its major function is to stimulate the release of **bile** and **pancreatic juice** and relax the **sphincter of Oddi**. CCK is released into the bloodstream from duodenal endocrine cells in response to fatty and protein-rich chyme entering the duodenum



Secretin is also an enterogastrone. Its major function, however, is to stimulate the release of **bicarbonate** from the pancreas. It is released into the bloodstream in response to acid chyme entering the duodenum.



AREA	PROCESSES	SECRETIONS	CONTROLS	HISTOLOGY
small intestine: duodenum	polysaccharides -> maltose	from pancreas : amylase	<i>CCK</i>	simple columnar Brunner's glands
	polypeptides-> shorter & dipeptides	trypsin, chymotrypsin, carboxy-peptidase		goblet cells
	dipeptides - amino a.			
	fats->glycerol & f.a.	lipase from intestine : amino- & carboxy-peptidases		
	disaccharides --> monosaccharides	sucrase, maltase, lactase <i>secretin</i> <i>cholecystokinin (CCK)</i>		plicae circularis
jejunum		bicarbonate	<i>secretin</i>	
ileum	absorption over 4 to 6 hours	from gallbladder : bile		villi and microvilli

Summary of secretions into the duodenum and their actions: **Bile** - produced in the liver and stored in the gallbladder, released in response to CCK . Bile salts (salts of cholic acid) act to emulsify fats, i.e. to split them so that they can mix with water and be acted on by lipase.

Pancreatic juice: **Lipase** - splits fats into glycerol and fatty acids. **Trypsin**, and **chymotrypsin** - protease enzymes which break polypeptides into dipeptides. **Carboxypeptidase** - splits dipeptide into amino acids. **Bicarbonate** - neutralizes acid. **Amylase** - splits polysaccharides into shorter chains and disaccharides.

Intestinal enzymes (brush border enzymes): **Aminopeptidase** and **carboxypeptidase** - split dipeptides into amino acids. **Sucrase**, **lactase**, **maltase** - break disaccharides into monosaccharides. **Enterokinase** - activates trypsinogen to produce trypsin. Trypsin then activates the precursors of chymotrypsin and carboxypeptidase. Other carbohydrases: **dextrinase** and **glucoamylase**. These are of minor importance.



Pancreatic Enzymes

Amylase: polysaccharides → disaccharides

Trypsin, chymotrypsin: polypeptides → dipeptides

Carboxypeptidase: dipeptides → amino acids

Lipase: fats → glycerol and fatty acids

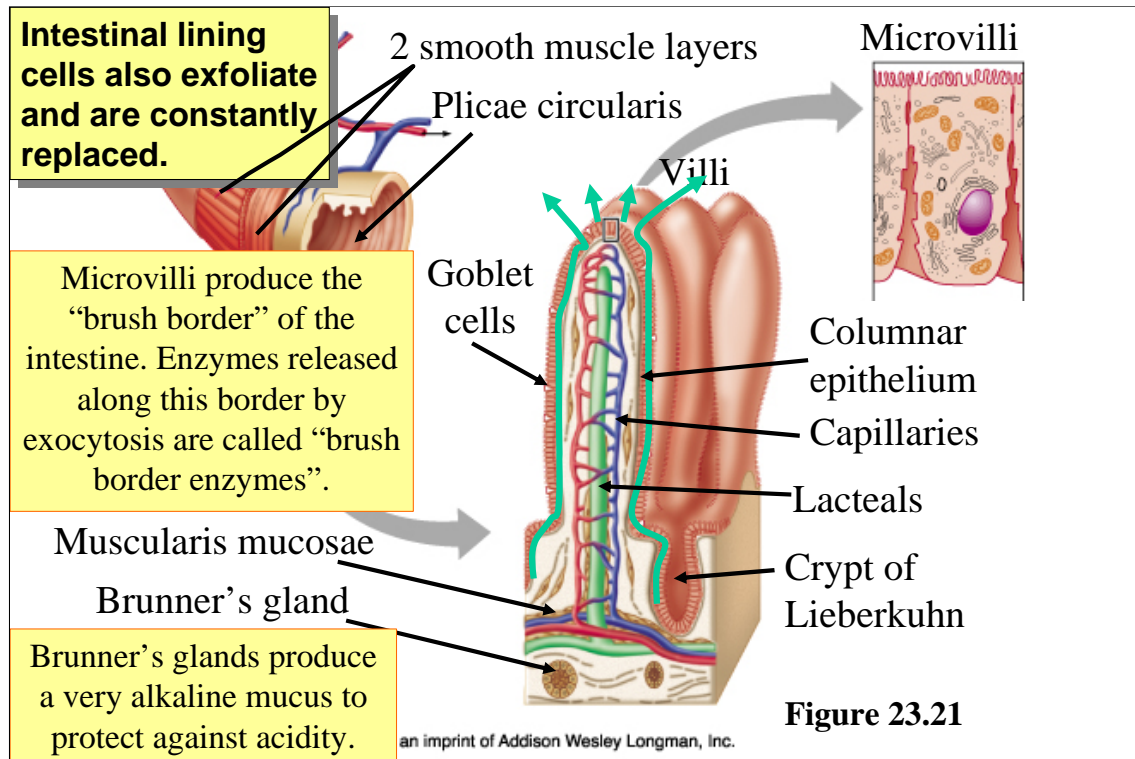
Brush border enzymes (intestinal):

Aminopeptidase, carboxypeptidase: dipeptides → amino acids

Sucrase, lactase, maltase:

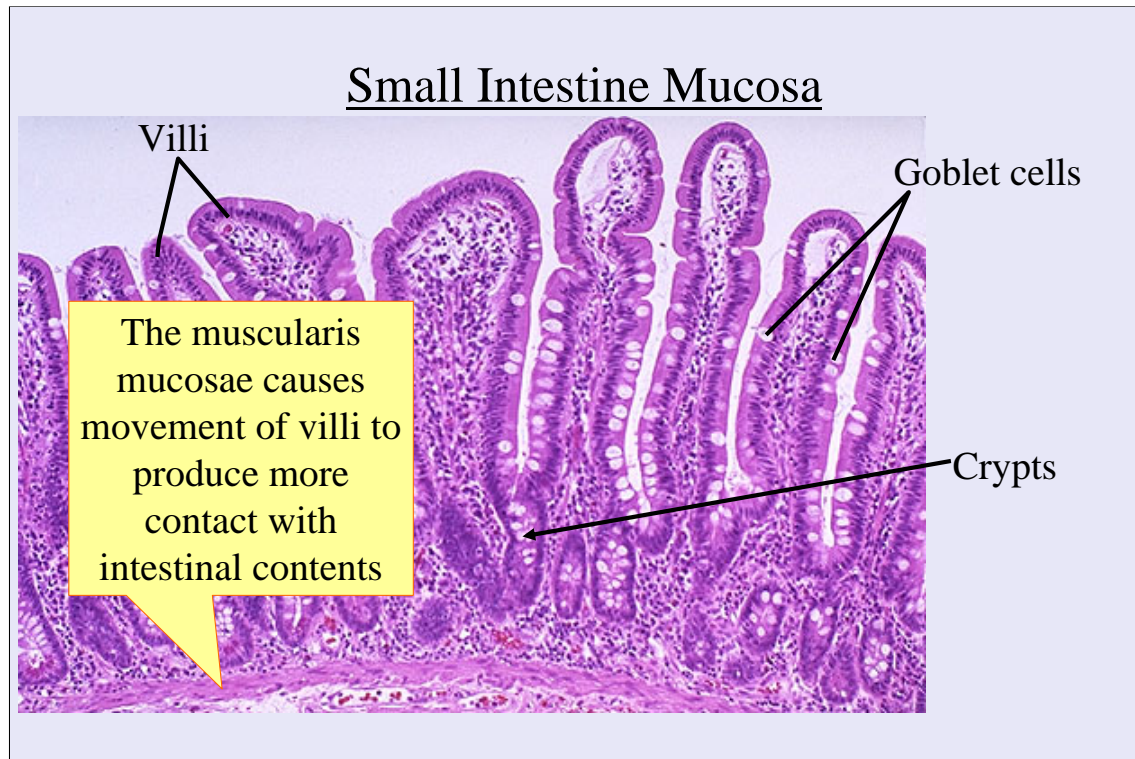
disaccharides → monosaccharides

A summary of enzymes released into the duodenum from the pancreas, and from the intestinal lining (brush border).



Structure and functions of the small intestine: The small intestine stretches nearly 20 feet, including the **duodenum**, **jejunum** and **ileum**. The surface area is increased by circular folds (the **plicae circularis**), finger-like **villi**, and the presence of **microvilli (brush border)** on the cell surfaces. At the base of the villi are the **intestinal crypts**, also called the intestinal glands because they are the source of the secretory cells of the mucosa. These cells are constantly renewed by mitosis and push up along the villi until they exfoliate from the surface. They cycle with about a 5-day turnover. Intestinal enzymes are released from the surface of the mucosal cells by exocytosis. These enzymes are called brush border enzymes because they cling to the microvilli. The villi possess a **lamina propria** beneath the epithelial lining which contains both blood and lymph capillaries for the absorption of materials. The **muscularis mucosae** contracts to move the villi and increase their exposure to the contents of the lumen. The three portions of the small intestine differ in subtle ways - the duodenum is the only portion with **Brunner's glands** in its submucosa which produce an alkaline mucus. The ileum has **Peyer's Patches**, concentrated lymph tissue in the submucosa. Goblet cells are progressively more abundant the further one travels along the intestine.

Virtually all remaining digestion occurs in the small intestine as well as all absorption of the digestive endproducts. In addition 95% of water absorption also occurs in the small intestine. Segmentation and peristalsis propel materials through the small intestine in 4 to 6 hours.

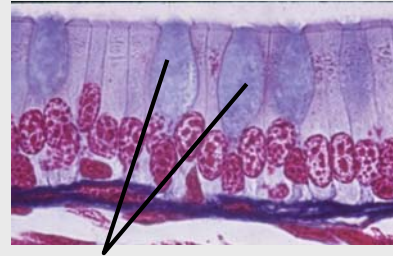
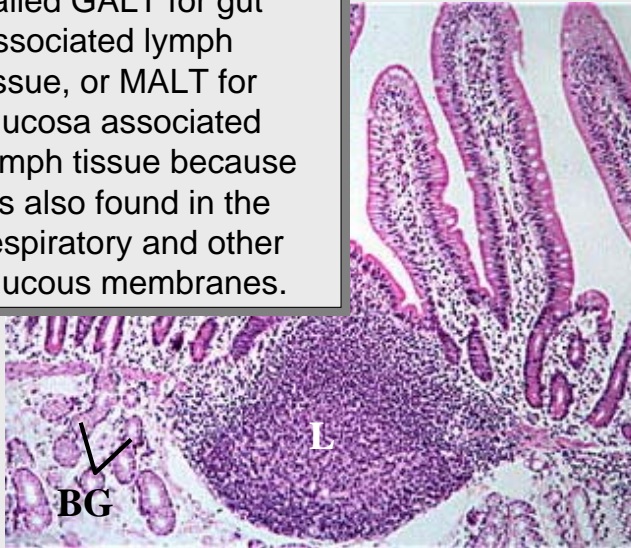


Note the abundant goblet cells among the columnar epithelial lining. In the duodenum these are supplemented by alkaline mucus from Brunner's glands.



The Duodenum

Extensive lymph tissue is found in the intestine, called GALT for gut associated lymph tissue, or MALT for mucosa associated lymph tissue because it's also found in the respiratory and other mucous membranes.



Goblet cells and villi confirm that this is the small intestine. The small Brunner's glands (BG) under the muscularis mucosa are found only in the duodenum. The large lymphocyte infiltrate (L) is common to the GI tract.

The duodenum has many mucus secreting glands called **Brunner's glands** in its submucosa. The alkaline mucus produced by these glands helps neutralize the acid from the stomach. The intestine also has many lymphocyte infusions, part of the **GALT** (Gut Associated Lymph Tissue) which helps to protect against ingested pathogens.



Brunner's Glands

Muscularis mucosae

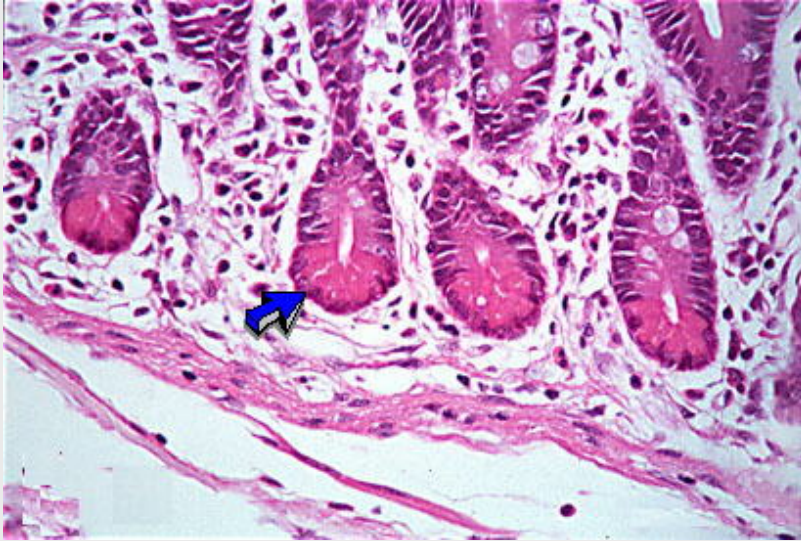


The highly alkaline secretions from the Brunner's glands serve to change the acidic chyme to an alkaline pH.

Close-up view of Brunner's glands, located in the submucosa. At the boundary of the mucosa is the **muscularis mucosae**.



Crypts of Lieberkühn

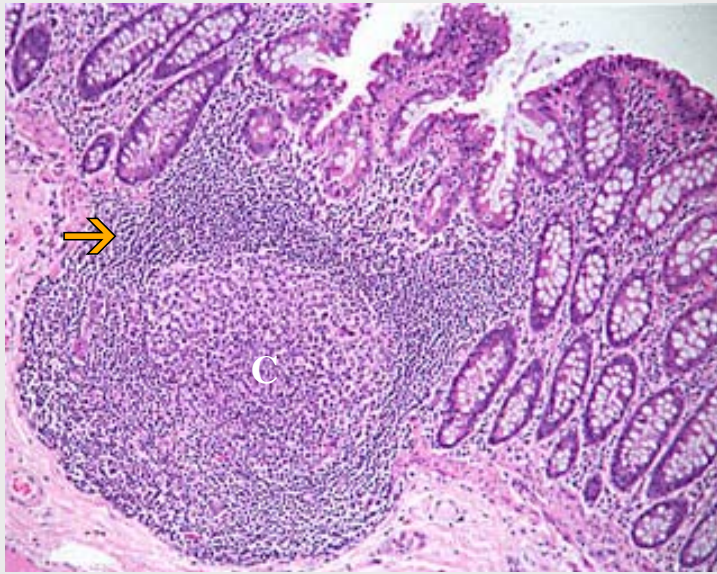


Deep clefts between villi, called Crypts of Lieberkühn, are shown here in cross section (arrow). They empty into the intestinal lumen.

The **Crypts of Lieberkuhn** are the source of new lining cells. They are sometimes called **intestinal glands** because the cells they produce are the secretory cells for enzymes and other substances.



Peyer's Patch



Peyer's patches are large lymph nodules found in the latter portion of the small intestine, visible to the naked eye. Notice the germinal center (C) where B-cells proliferate. These are a major source of antibody production

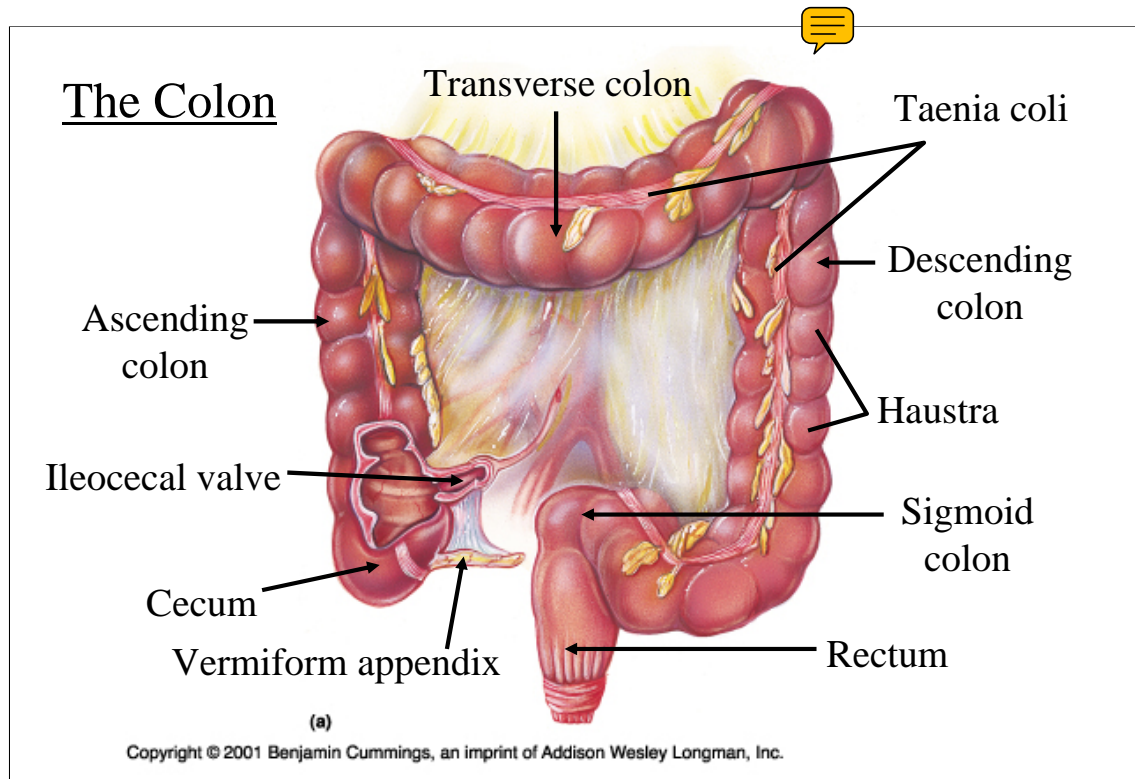
Peyer's patches are more organized lymphoid tissue that was seen a few slides previously in the duodenum. Peyer's patches are nearly exclusive to the **ileum**.



Terminal Ileum



Peyer's patches are large and can be seen with the naked eye.



Structure and functions of the colon (large intestine): the colon is much shorter in length while larger in diameter than the small intestine. The longitudinal muscle of the colon is arranged into three distinct bands, the **taenia coli**, which cause the colon to buckle producing the **haustra**. These are pouches which increase the surface area of the colon for absorption of water and electrolytes. The colon also has deep clefts which increase its surface area. The first part of the colon is a blind pouch called the **cecum**. The ileum enters the cecum at the **ileocecical sphincter** (valve). Attached to the cecum is the **vermiform** (wormlike) **appendix**, a vestigial remnant of the larger cecum seen in other mammals. The appendix has a concentration of lymph tissue and is filled with lymphocytes, but its removal has not been demonstrated to have any negative effect on the immune system.

The cecum leads in sequence to the **ascending colon**, then the **transverse colon**, the **descending colon**, and the **sigmoid colon** before entering the **rectum**. The rectum possesses skeletal muscle which functions during the defecation reflex.



Comparison of Large Intestine to Small Intestine)

Structural differences:

taenia coli – distinct bands of longitudinal smooth muscle.

haustra – pouches produced by the contraction of the taenia coli.

deep clefts rather than villi increase the surface lining.

Similarities: abundant goblet cells

See previous slide.



Functional Differences in the Large Intestine (Compared to the Small)

small peristaltic waves – play little roll in propulsion

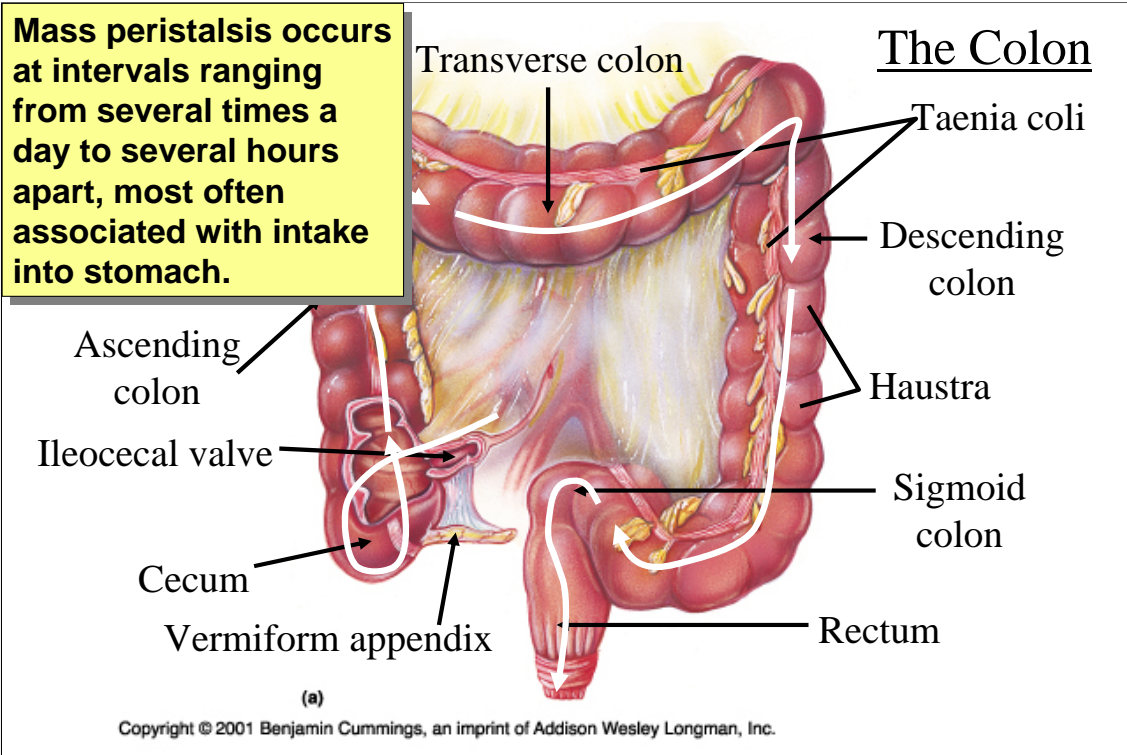
haustral churning – Contraction of circular muscle serves to mix contents and expose to lining tissue.

mass peristalsis – large peristaltic waves which occur at intervals resulting from the **gastrocolic** or **gastroileal** reflexes.

Unlike the nearly constant propulsion in the small intestine, in the large intestine materials move at intervals by **mass peristalsis**. In between these intervals **segmentation**, called **haustral churning**, stirs the contents so that water can be absorbed more efficiently and bacteria, an important component in fecal processing, can be mixed with the contents. (See next slide)



Mass peristalsis occurs at intervals ranging from several times a day to several hours apart, most often associated with intake into stomach.



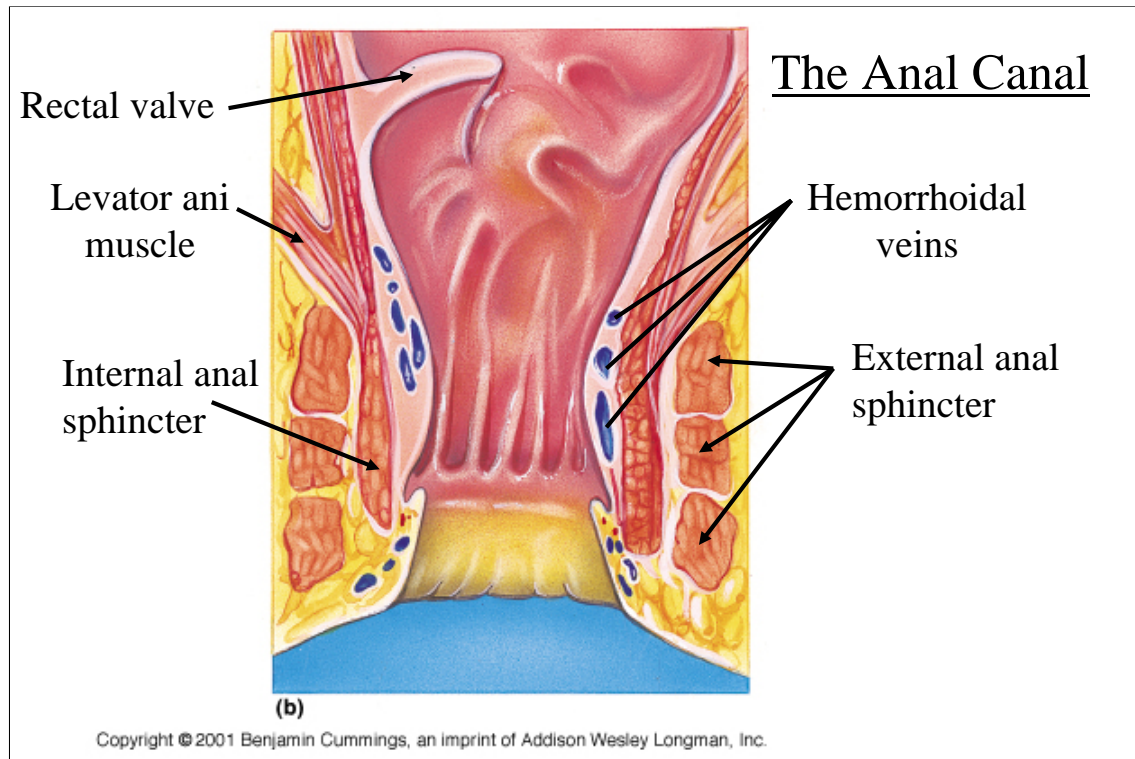
The colon absorbs the remaining water and produces the feces. The process takes about 12 hours. Liquid chyle enters the colon through the ileocecal sphincter, whose pursed lips protrude into the cecum to help prevent backflow of chyme under pressure. This valve relaxes only when peristalsis arrives from the ileum. Muscle movements in the colon consist of: 1) minor peristaltic waves, 2) **haustral churning**, and 3) **mass peristalsis**. Haustral churning is produced by segmentation contractions which serve to mix the contents to enhance absorption. Mass peristalsis consists of large movements which occur at intervals, usually associated with meals. These movements are often initiated by the **gastrocolic reflex** (or gastroileal or duodenocolic reflexes) which stimulates the colon in response to food entering the stomach. This reflex is especially active after fasting and when the food is hot or cold. It causes mass peristalsis in about 15 minutes which continues for about 30 minutes. These movements cause the chyme to move in several large steps through the colon, stopping at each step to be further concentrated and converted into feces. The chyme turns from a liquid into a slush and then into firm feces. In the process some vitamins such as vitamin K and certain B vitamins are produced and absorbed, along with water and electrolytes.



The Digestive Chart: The Colon

AREA	PROCESSES	SECRECTIONS	CONTROLS	HISTOLOGY
colon	absorption over 12 hrs. haustral churning mass peristalsis	mucus	gastrocolic reflex	epithelial clefts goblet cells taenia coli

The colon absorbs what's left of the water (about 400 cc. or so) and produces compact feces. The process takes about 12 hours. What results, the feces, is about one third bacteria, one third exfoliated cells, and one third un-digestible materials.

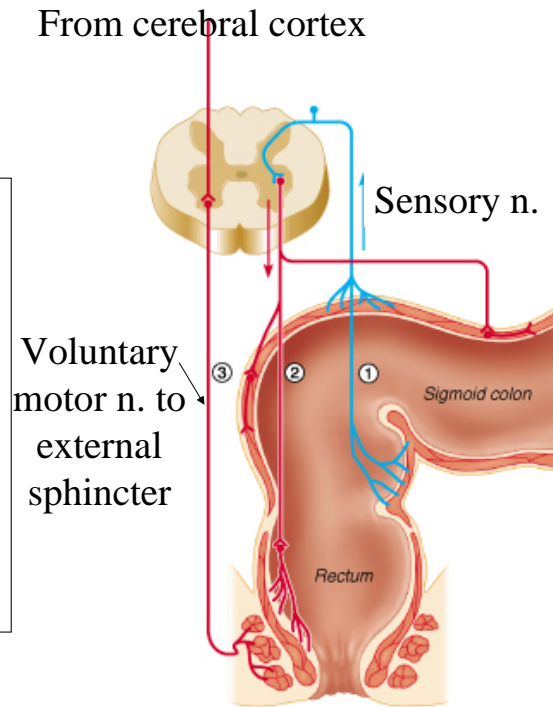


The rectal valve tends to prevent feces from moving into the anal canal until the next mass peristalsis event. Sphincter muscles surrounding the anus must relax in order for defecation to take place. (See the next slide)



Defecation Reflex

- 1) Pressure in rectum from mass peristalsis sends afferent stimuli to spinal cord.
- 2) Parasympathetic stimuli cause contraction of rectal muscle and relaxation of internal anal sphincter.
- 3) Voluntary stimuli relax external sphincter and cause abdominal contraction.



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The defecation reflex: As a result of the mass movements described above, pressure is exerted on the rectum and on the internal anal sphincter, which is smooth muscle, resulting in its involuntary relaxation. Afferent impulses are sent to the brain indicating the need to defecate. The external sphincter is voluntary muscle and is controlled by the voluntary nervous system. This sphincter is relaxed along with contraction of the rectal and abdominal muscles in the defecation reflex.

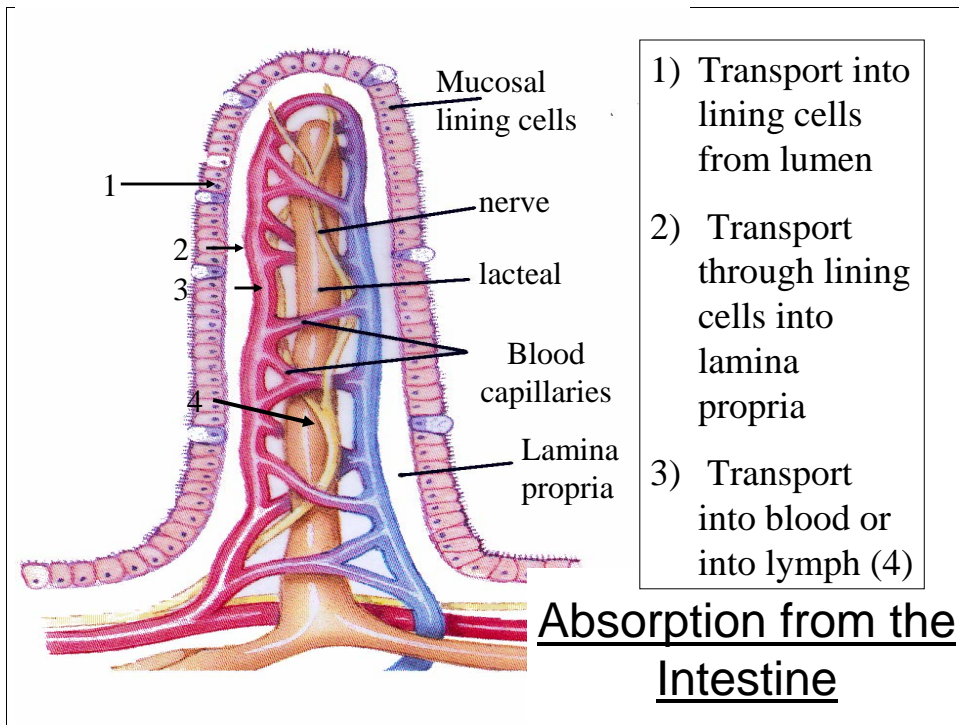


Goblet Cells in the Colon

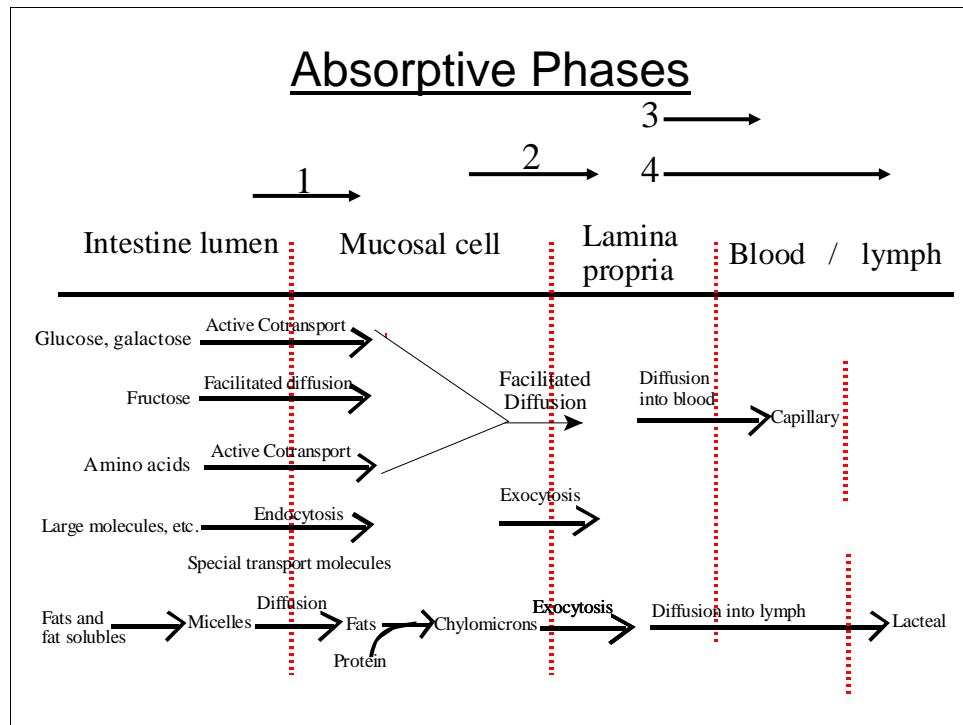


Mucus from the numerous goblet cells is used to lubricate the large intestine to ease the passage of its contents.

The colon has more goblet cells than any other GI region, owing in part to the fact it has no other cells or glands to produce mucus for lubrication.



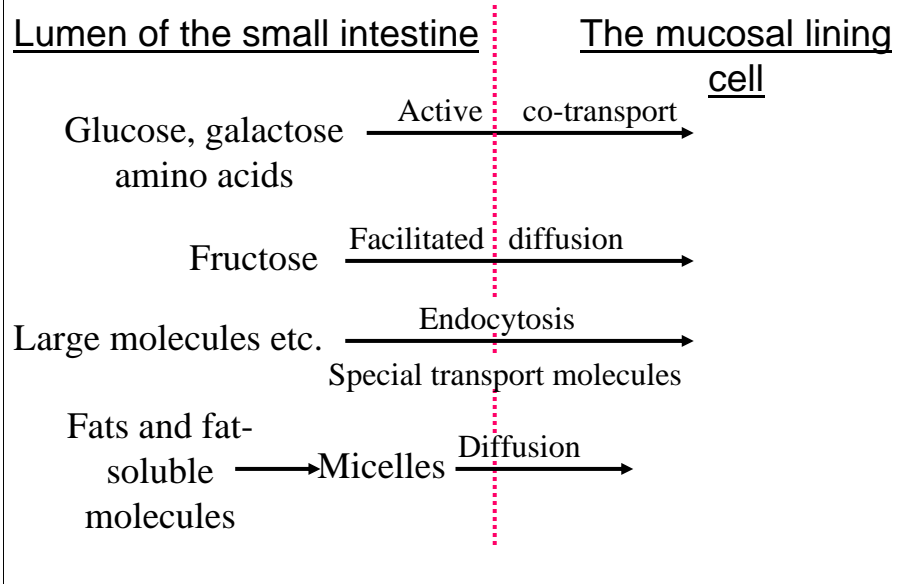
Transport of digestive end products must occur in three steps: 1) into the mucosal lining cells; 2) out of the mucosal cells into the underlying lamina propria; 3) from the lamina propria into the blood or the lymph.



An overview of the absorptive phases by which digestive endproducts are absorbed from the small intestine.



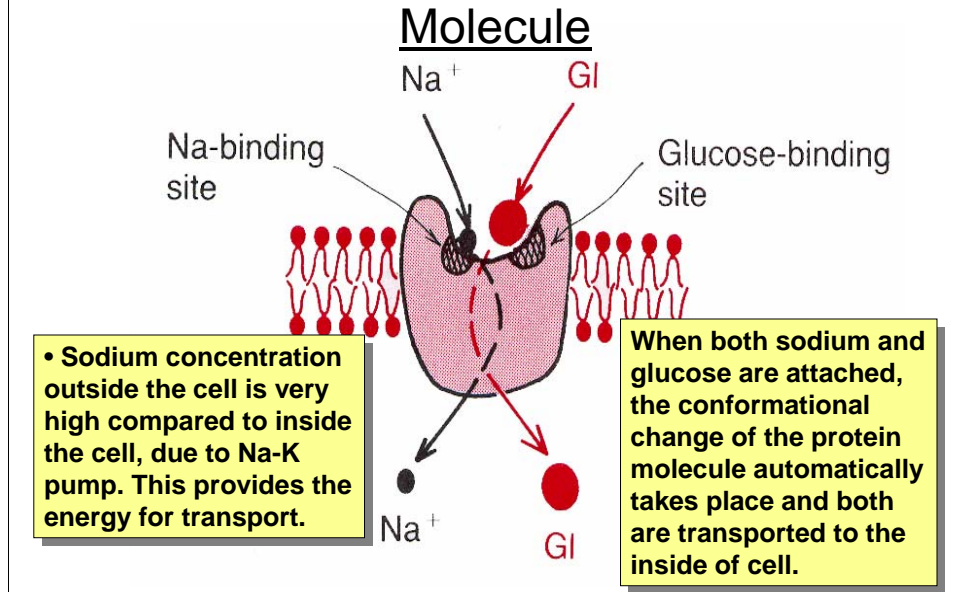
Absorption Into Mucosal Cell



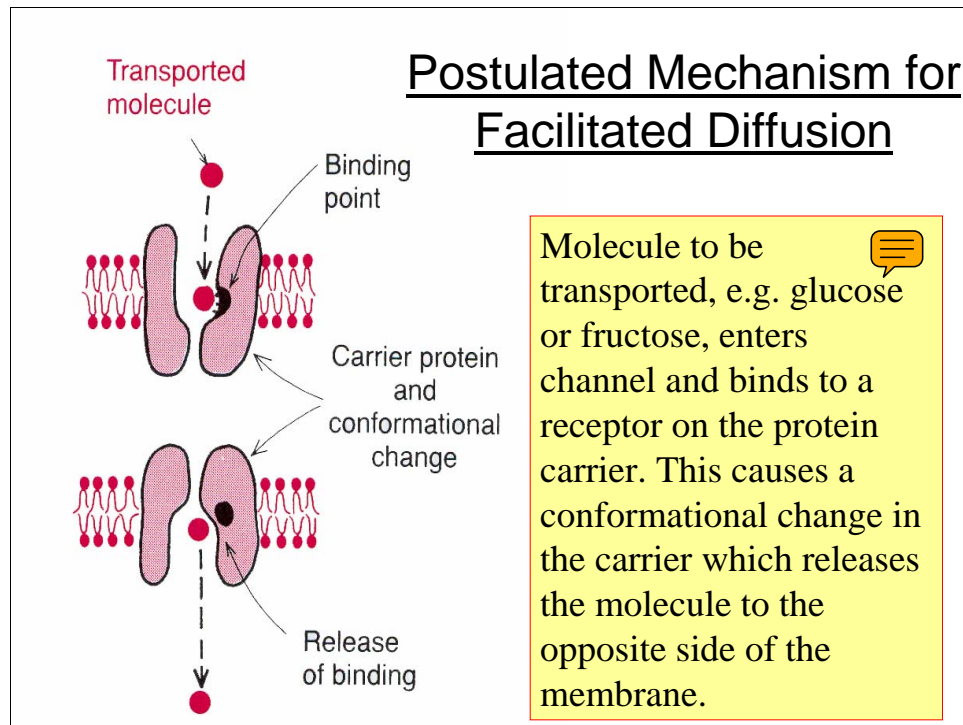
Step 1) Digestive endproducts must be first taken into the mucosal lining cells. See the next few slides for mechanisms of transport.



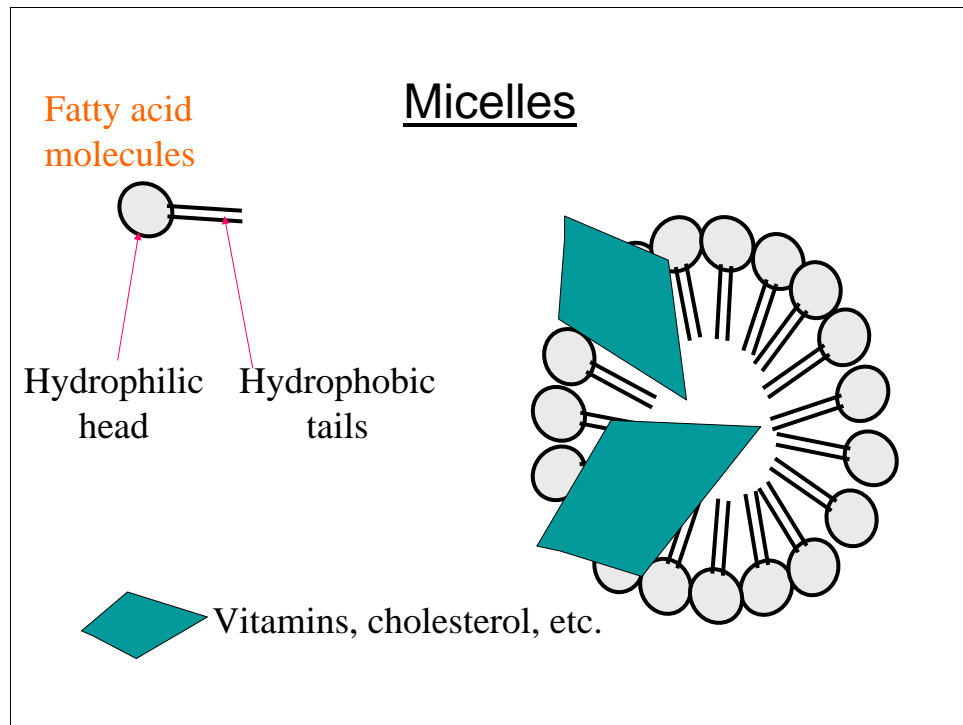
Postulated Mechanism for Sodium Co-transport of Glucose or Other



The force for transport of glucose and other substances by “**co-transport**” comes from the unequal distribution of sodium ions, and therefore indirectly from the sodium-potassium pump. As long as sodium is more concentrated outside the cell it can bind to the transport protein. When glucose is also present and binds it is then transported into the cell.



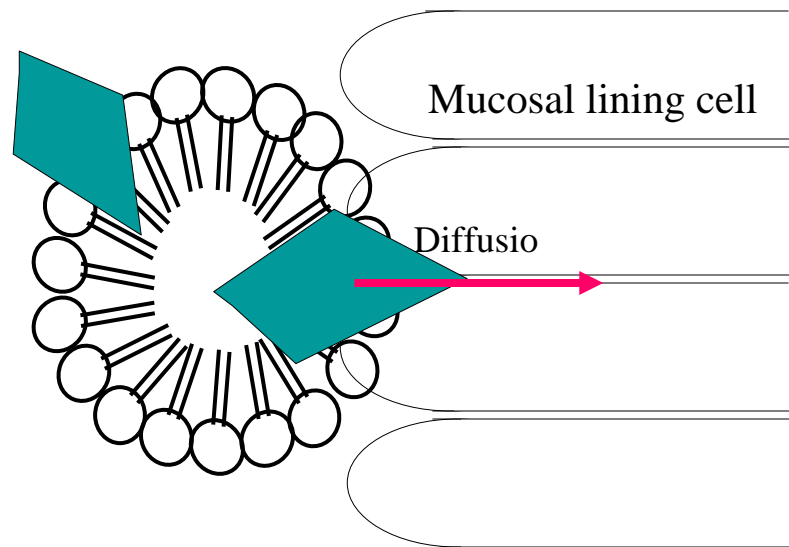
Facilitated diffusion does not require energy. If the substrate, glucose or fructose for instance, is present and binds to the pump protein it triggers a conformational (structural) change which moves the substrate across the membrane. Glucose is transported **into most cells** by this method. However not in the intestine. Instead glucose relies on **co-transport into the mucosal cells** and **facilitated diffusion out of the cells** into the lamina propria.



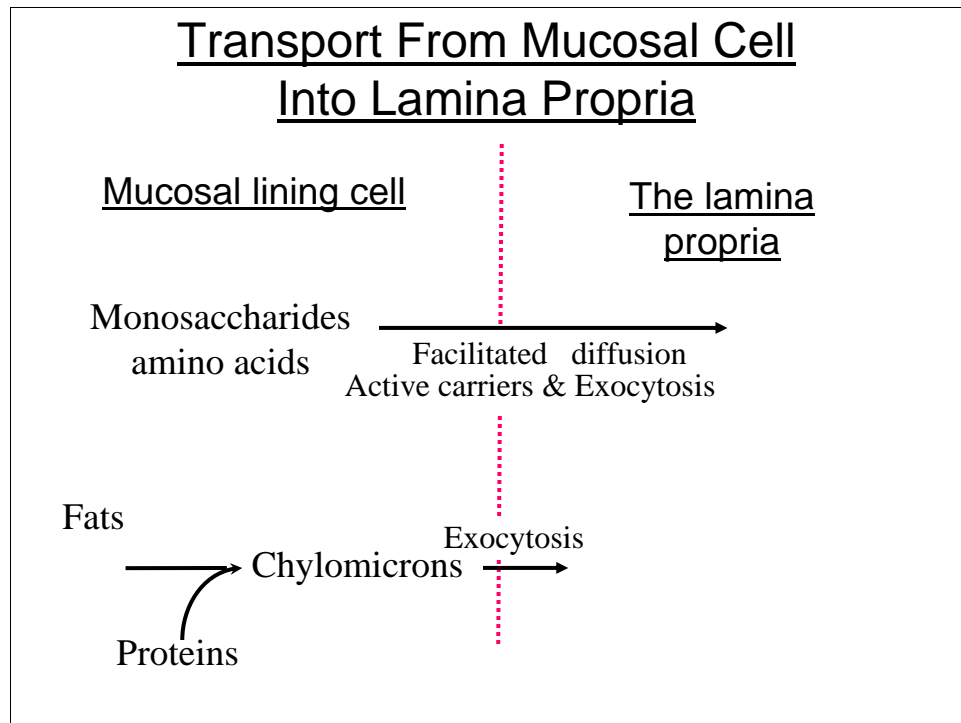
Micelles form because of the polarity of fat molecules. Once formed they incorporate other fat-soluble molecules and aid in their transport across the mucosal membrane. The danger in consuming so-called “fake fats” is that they will incorporate fat-soluble vitamins, but won’t facilitate their transport into mucosal cells, leading to vitamin deficiency.



Diffusion of Fats Into Mucosal Cells



Micelles line up along the mucosal membrane allowing fat-soluble molecules to easily slide into the lipid matrix and be transported into the cells.



Monosaccharides and amino acids are then transported by various means out of the basal end of mucosal cells so that they can be absorbed into capillaries in the villi. Fats are combined with proteins to make **chylomicrons** which must be absorbed into the lymph capillaries (**lacteals**).



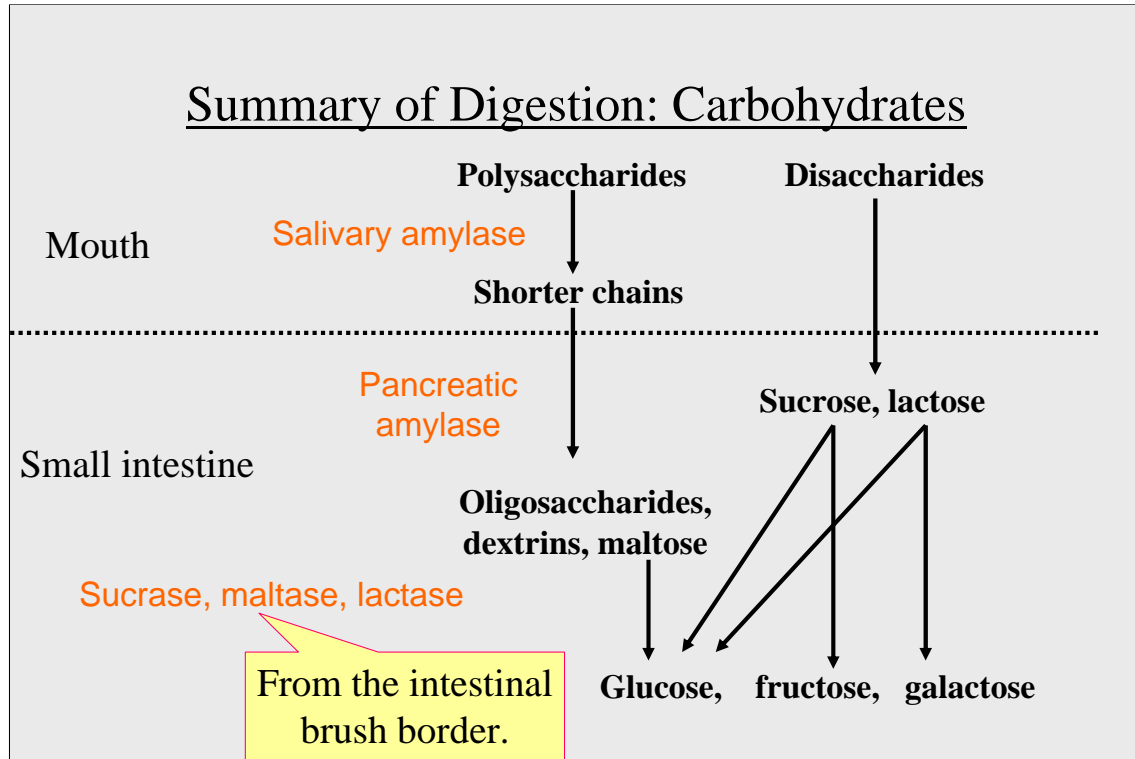
Transport From Lamina Propria Into Blood or Lymph

The lamina propria

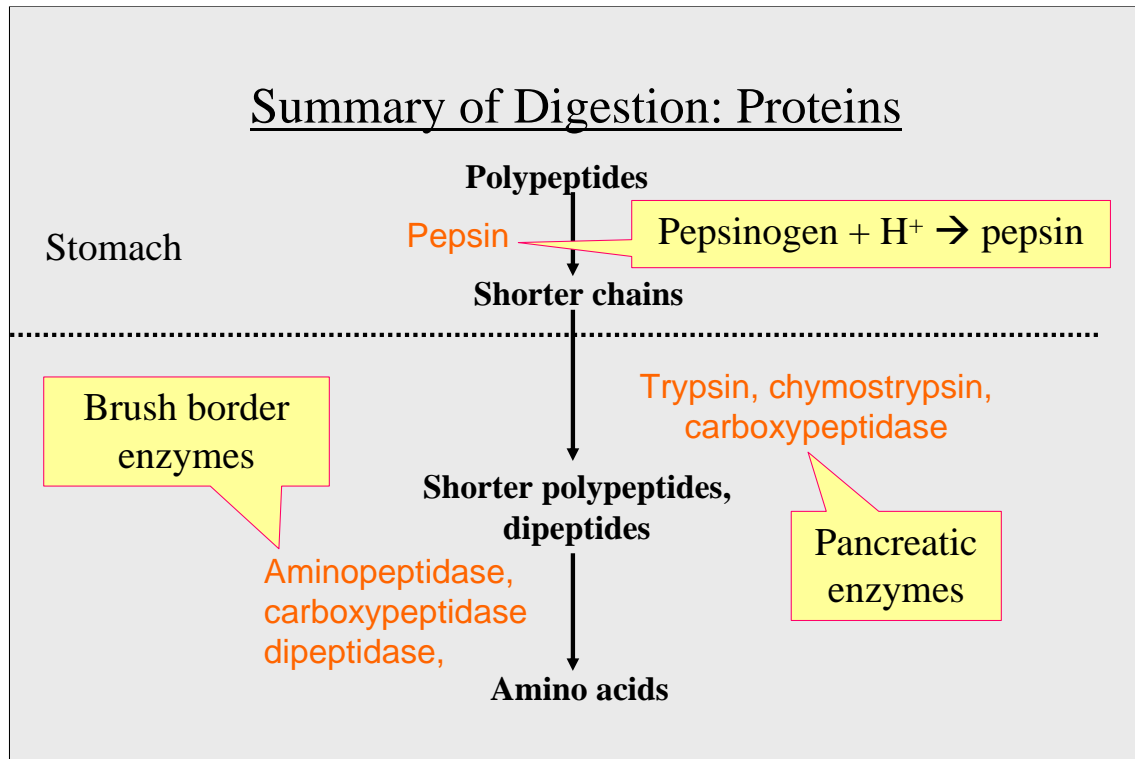
Monosaccharides Diffusion Blood
amino acids → Capillaries

Chylomicrons Diffusion Lacteals

Capillaries in the villi are fenestrated in order to absorb the relatively large monosaccharides and amino acids. But chylomicrons, must be absorbed into the more porous lacteals.



Here is a summary of digestive phases for the carbohydrates.

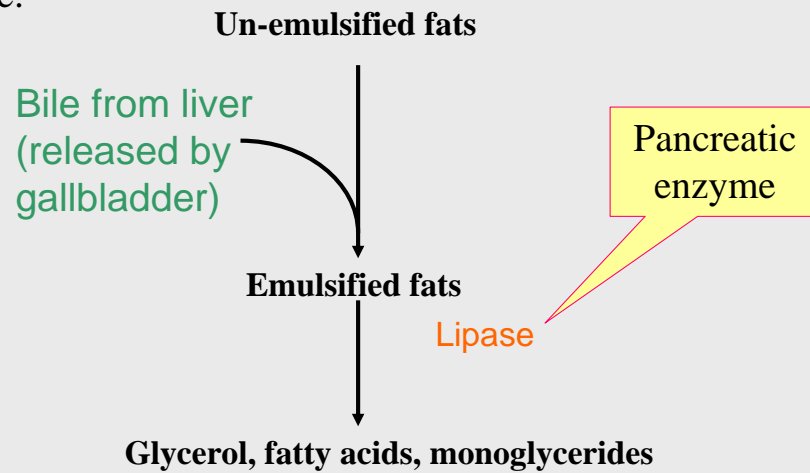


Here is a summary of digestive phases for the proteins.



Summary of Digestion: Fats

Small intestine:



Here is a summary of digestive phases for the fats.