24

The Digestive System

Learning Outcomes

These Learning Outcomes correspond by number to this chapter's sections and indicate what you should be able to do after completing the chapter.

24-1 Identify the **organs of the digestive system**, list their **major functions**, describe the **functional histology of the digestive tract**, and outline the **mechanisms that regulate digestion**.

24-2 Discuss the **anatomy of the oral cavity**, and list the **functions of its major structures and regions**.

24-3 Describe the **structure and functions of the pharynx**.

24-4 Describe the **structure and functions of the esophagus**.

24-5 Describe the **anatomy of the stomach**, including its **histological features**, and discuss its **roles in digestion and absorption**.

24-6 Describe the anatomical and histological **characteristics of the small intestine**, explain the **functions and regulation of intestinal secretions**, and describe the structure, functions, and regulation of the **accessory digestive organs**.

24-7 Describe the gross and histological **structure of the large intestine**, including its regional specializations and **role in nutrient absorption**.

24-8 List the **nutrients required by the body**, describe the **chemical events responsible for the digestion of organic nutrients**, and describe the mechanisms involved in the **absorption of organic and inorganic nutrients**.

24-9 Summarize the **effects of aging** on the digestive system.

24-10 Give examples of **interactions between the digestive system and other organ systems** studied so far.

Clinical Notes

Spotlights

An Introduction to the Digestive System

The digestive system may not have the visibility of the integumentary system or the glamour of the reproductive system, but it is certainly just as important. All living organisms must get nutrients from their environment to sustain life. These nutrients are used as raw materials for synthesizing essential compounds (anabolism). They are also broken down to provide the energy that cells need to continue functioning (catabolism). pp. 34–37, 305–306 Catabolic reactions require two essential ingredients: oxygen and organic molecules (such as carbohydrates, fats, or proteins) that can be broken down by enzymes inside cells. Obtaining oxygen and organic molecules can be straightforward for a single-celled organism like an amoeba. The process is much more complicated for animals as large and complex as humans. Along with increasing size and complexity come a division of labor within the body and the need to coordinate organ system activities.

In this chapter we discuss the structure and function of the digestive tract and several digestive glands, notably the liver and pancreas. We look at the process of digestion and how it breaks down large and complex organic molecules into smaller fragments that can be absorbed by the digestive epithelium. We also see how a few organic wastes are removed from the body.

24-1 The digestive system, consisting of the digestive tract and accessory organs, has overlapping food utilization functions

In our bodies, the respiratory system works with the cardiovascular system to supply the oxygen needed for catabolism. The *digestive system*, working with the cardiovascular and lymphatic systems, provides the needed organic molecules. In effect, the digestive system supplies both the fuel that keeps all the body's cells running and the building blocks needed for cell growth and repair.

The **digestive system** consists of a muscular tube, the **digestive tract,** also called the *gastrointestinal (GI) tract* or *alimentary canal*, and various **accessory organs.** The *oral cavity* (mouth), *pharynx* (throat), *esophagus*, *stomach*, *small intestine*, and *large intestine* make up the digestive tract. Accessory digestive organs include the teeth, tongue, and various *glandular organs*, such as the salivary glands, liver, and pancreas. The glandular organs secrete their products into ducts that empty into the digestive tract. These secretions contain water, enzymes, buffers, and other substances. Food enters the digestive tract and passes along its length. Along the way, the secretions of the glandular organs prepare organic and inorganic nutrients for absorption across the epithelium of the digestive tract.

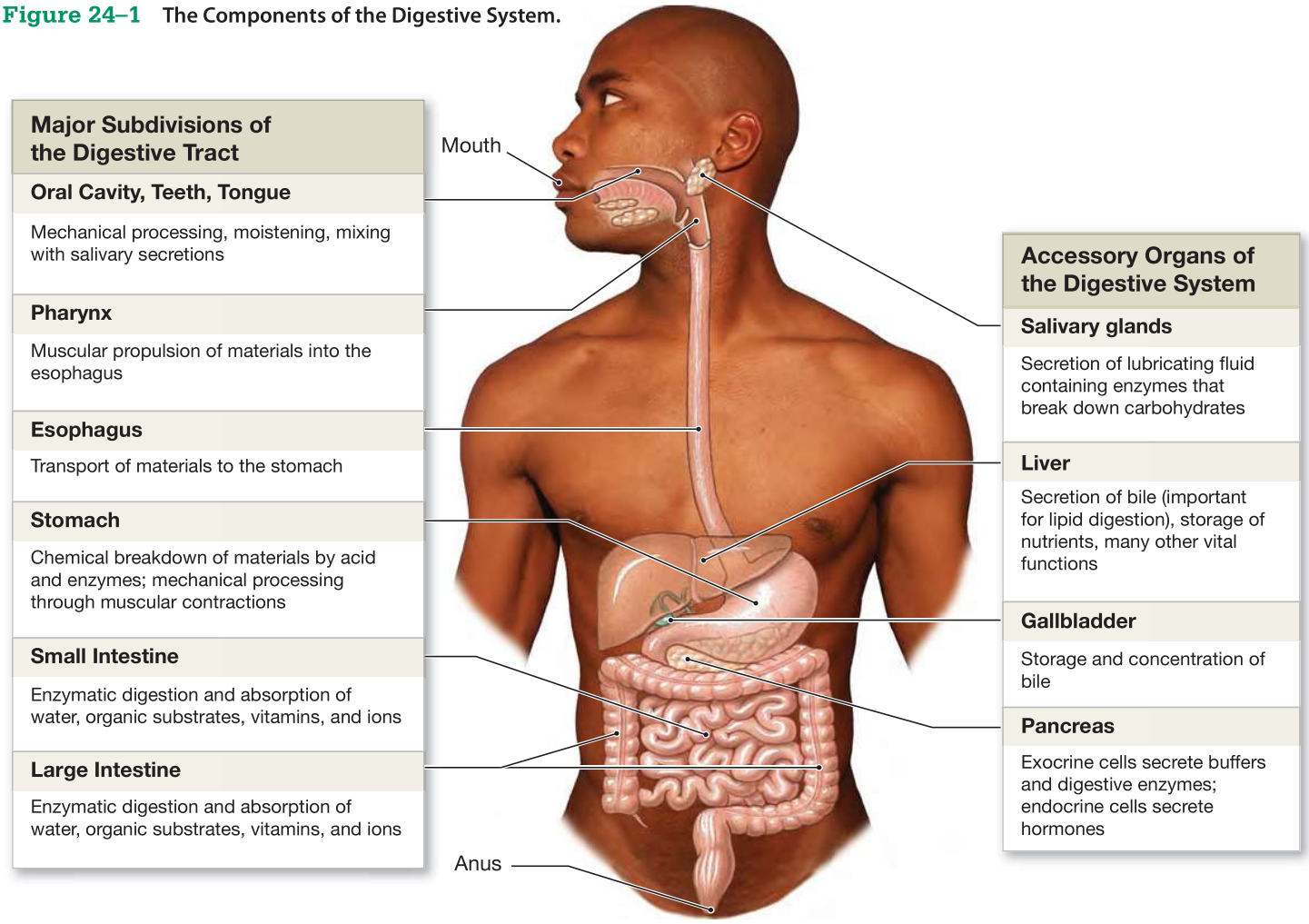
Figure 24–1 shows the major parts of the digestive system. The digestive tract begins at the oral cavity. It continues through the pharynx, esophagus, stomach, small intestine, and large intestine, which opens to the exterior at the anus. These structures have overlapping functions, but each has certain areas of specialization and shows distinctive histological characteristics.

Functions of the Digestive System

We can regard digestive functions as a series of six integrated steps:

1. **Ingestion** takes place when materials enter the digestive tract through the mouth. Ingestion is an active process involving conscious choice and decision making.
2. **Mechanical processing** is crushing and shearing that makes materials easier to propel along the digestive tract. It also increases their surface area, making them more susceptible to attack by enzymes. Mechanical processing may or may not be required before ingestion. You can swallow liquids immediately, but you must chew most solids first. Tearing and mashing with the teeth, followed by squashing and compacting by the tongue, are examples of preliminary mechanical processing. Swirling, mixing, and churning motions of the stomach and intestines provide mechanical processing after ingestion.
3. **Digestion** refers to the chemical breakdown of food into small organic fragments suitable for absorption by the digestive epithelium. Simple molecules in food, such as glucose, can be absorbed intact. However, epithelial cells have no way to absorb molecules with the size and complexity of proteins, polysaccharides, or triglycerides. Digestive enzymes must first disassemble these molecules. For example, the starches in a potato are of no nutritional value until enzymes have broken them down to simple sugars that the digestive epithelium can absorb for distribution to body cells.
4. **Secretion** is the release of water, acids, enzymes, buffers, and salts by the epithelium of the digestive tract and by glandular organs.
5. **Absorption** is the movement of organic molecules, electrolytes (inorganic ions), vitamins, and water across the digestive epithelium and into the interstitial fluid of the digestive tract.
6. **Excretion** is the removal of waste products from body fluids. The digestive tract and glandular organs discharge waste products in secretions that enter the lumen of the tract. Most of these waste products mix with the indigestible residue of the digestive process and then leave the body. The process called **defecation** (def-e-KĀ-shun), or *egestion*, ejects materials from the digestive tract, eliminating them as **feces.**

Figure 24–1 The Components of the Digestive System.



The lining of the digestive tract also plays a protective role. It safeguards surrounding tissues against (1) the corrosive effects of digestive acids and enzymes; (2) mechanical stresses, such as abrasion; and (3) bacteria that either are swallowed with food or live in the digestive tract. The digestive epithelium and its secretions provide a nonspecific defense against these bacteria. When bacteria reach the underlying layer of areolar tissue, the *lamina propria*, macrophages and other cells of the immune system attack them.

We explore specific functions in more detail as we consider the individual regions and components of the system. First, however, let's look at several structural and functional characteristics of the system as a whole.

The Digestive Organs and the Peritoneum

The abdominopelvic cavity contains the *peritoneal cavity*, which is lined by a serous membrane. This membrane consists of a superficial mesothelium covering a layer of areolar tissue. ↩ pp. 22, 131 We can divide this serous membrane into two parts. The serosa or *visceral peritoneum* covers organs which project into the peritoneal cavity. The *parietal peritoneum* lines the inner surfaces of the body wall.

The serous membrane lining the peritoneal cavity continuously produces peritoneal fluid, which provides essential lubrication. Because a thin layer of peritoneal fluid separates them, the parietal and visceral surfaces can slide without friction and resulting irritation. The membrane secretes and reabsorbs about 7 liters (7.4 quarts) of fluid each day, but the volume within the peritoneal cavity at any one time is very small. Liver disease, kidney disease, and heart failure can cause an increase in the rate at which fluids move into the peritoneal cavity. The buildup of fluid creates a characteristic abdominal swelling called **ascites** (a-SĪ-tēz). This fluid can distort internal organs and cause symptoms such as heartburn, indigestion, and lower back pain.

Mesenteries

Portions of the digestive tract are suspended within the peritoneal cavity by sheets of serous membrane that connect the

parietal peritoneum with the visceral peritoneum. These **mesenteries** (MEZ-en-ter-ēz) are double sheets of peritoneal membrane. The areolar tissue between the mesothelial surfaces provides a route to and from the digestive tract for blood vessels, nerves, and lymphatic vessels. Mesenteries stabilize the positions of the attached organs. The mesenteries also prevent the intestines from becoming entangled during digestive movements or sudden changes in body position.

During embryonic development, the digestive tract and accessory organs are suspended within the peritoneal cavity by *dorsal* and *ventral mesenteries* (**Figure 24–2a**). The ventral mesentery later disappears along most of the digestive tract. It persists in adults in only two places: on the ventral surface of the stomach, between the stomach and the liver (the *lesser omentum)*, (**Figure 24–2b,d**); and between the liver and the anterior abdominal wall (the *falciform ligament)*, (**Figure 24–2c,d**). The **lesser omentum** (ō-MEN-tum; *omentum*, fat skin) stabilizes the position of the stomach and provides an access route for blood vessels and other structures entering or leaving the liver. The **falciform** (FAL-si-form; *falx*, sickle + *forma*, form) **ligament** helps stabilize the position of the liver relative to the diaphragm and abdominal wall. ATLAS: Embryology Summary 19: The Development of the Digestive System.

As the digestive tract elongates, it twists and turns within the crowded peritoneal cavity. The dorsal mesentery of the stomach becomes greatly enlarged and forms an enormous pouch that extends inferiorly between the body wall and the anterior surface of the small intestine. This pouch is the **greater omentum** (**Figure 24–2b,d**). It hangs like an apron from the lateral and inferior borders of the stomach. Adipose tissue in the greater omentum conforms to the shapes of the surrounding organs, providing padding and protection across the anterior and lateral surfaces of the abdomen. When an individual gains weight, this adipose tissue contributes to the characteristic "beer belly." The lipids in the adipose tissue are an important energy reserve. The greater omentum also provides insulation that reduces heat loss across the anterior abdominal wall.

All but the first 25 cm (10 in.) of the small intestine is suspended by the **mesentery proper,** a thick mesenterial sheet. It provides stability, but permits some independent movement. The mesentery associated with the initial portion of the small intestine (the *duodenum*) and the pancreas fuses with the posterior abdominal wall and locks those structures in place. Only their anterior surfaces remain covered by peritoneum. We describe these organs as **retroperitoneal** (*retro*, behind) because their mass lies posterior to, rather than surrounded by the peritoneal cavity.

A **mesocolon** is a mesentery associated with a portion of the large intestine. During normal development, the mesocolon of the *ascending colon*, the *descending colon*, and the *rectum* of the large intestine fuse to the posterior body wall. These regions become locked in place. Thereafter, these organs are retroperitoneal. The visceral peritoneum covers only their anterior surfaces and portions of their lateral surfaces (**Figure 24–2b,c,d**). The **transverse mesocolon,** which supports the transverse colon, and the **sigmoid mesocolon,** which supports the sigmoid colon, are all that remain of the original embryonic mesocolon.

Clinical Note

Peritonitis An inflammation of the peritoneal membrane (peritoneum) is called **peritonitis** (per-i-tō-NĪ-tis). This painful condition interferes with the normal functioning of the affected organs. Physical damage, chemical irritation, and bacterial invasion of the peritoneum can lead to severe and even fatal cases of peritonitis. In untreated appendicitis, the appendix may rupture and release bacteria into the peritoneal cavity. This event may cause peritonitis. Peritonitis can also be a complication of any surgery that opens the peritoneal cavity. Any disease or injury that perforates the stomach or intestines carries the danger of peritonitis.

Histological Organization of the Digestive Tract

The four major layers of the digestive tract are (1) the *mucosa*, (2) the *submucosa*, (3) the *muscularis externa*, and (4) the *serosa*. The structure of these layers varies by region. **Figure 24–3** is a composite view. It most closely resembles the small intestine, the longest segment of the digestive tract.

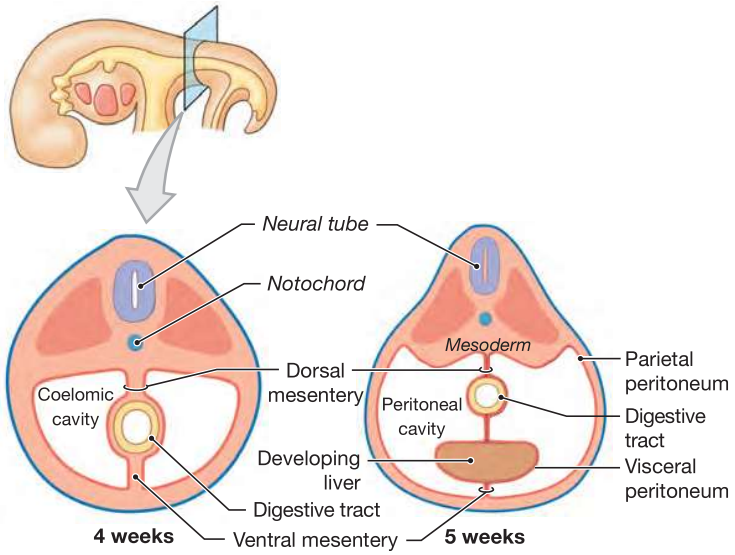
The Mucosa

The inner lining, or **mucosa,** of the digestive tract is a *mucous membrane.* It consists of an epithelium, moistened by glandular secretions, and a *lamina propria* of areolar tissue.

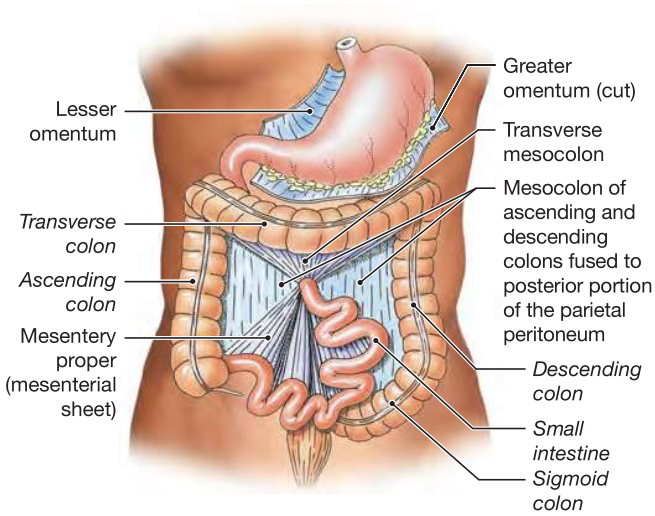
The Digestive Epithelium. The mucosal epithelium is either simple or stratified, depending on its location and the stresses placed on it. Mechanical stresses are most severe in the oral cavity, pharynx, and esophagus. These structures are lined by a stratified squamous epithelium. In contrast, the stomach, the small intestine, and almost the entire length of the large intestine (where absorption occurs) have a simple columnar epithelium that contains mucous cells. Scattered among the columnar cells are **enteroendocrine cells.** They secrete hormones that coordinate the activities of the digestive tract and the accessory glands.

The lining of the digestive tract appears as longitudinal folds, which disappear as the tract fills. The lining also has permanent transverse folds, or *plicae* (PLĪ-sē; folds; singular, *plica*) *circulares* (**Figure 24–3**). The folding dramatically increases the surface area available for absorption. The secretions of gland

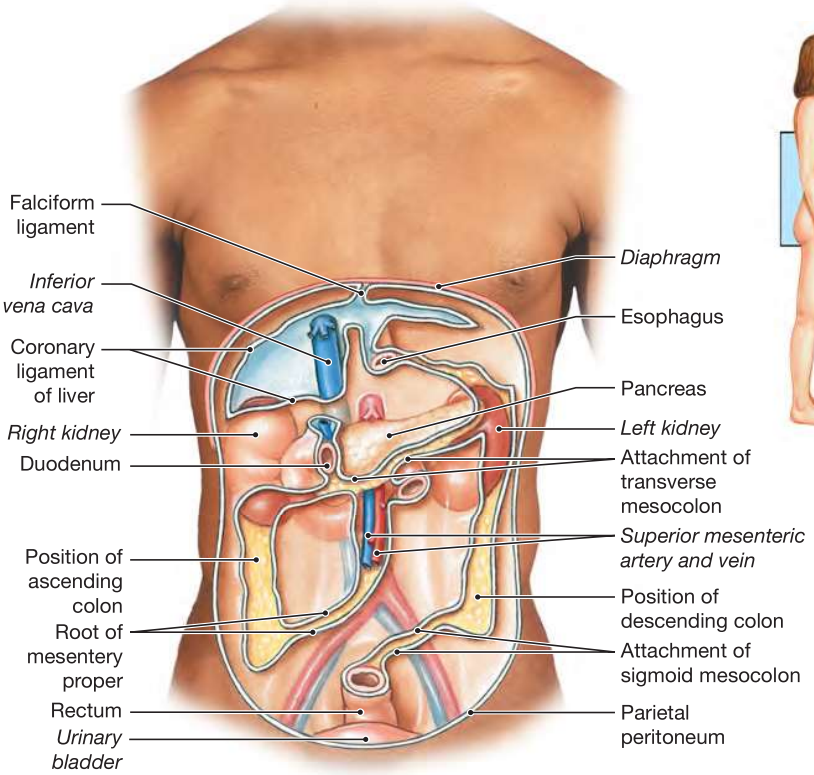
Figure 24–2 Mesenteries.



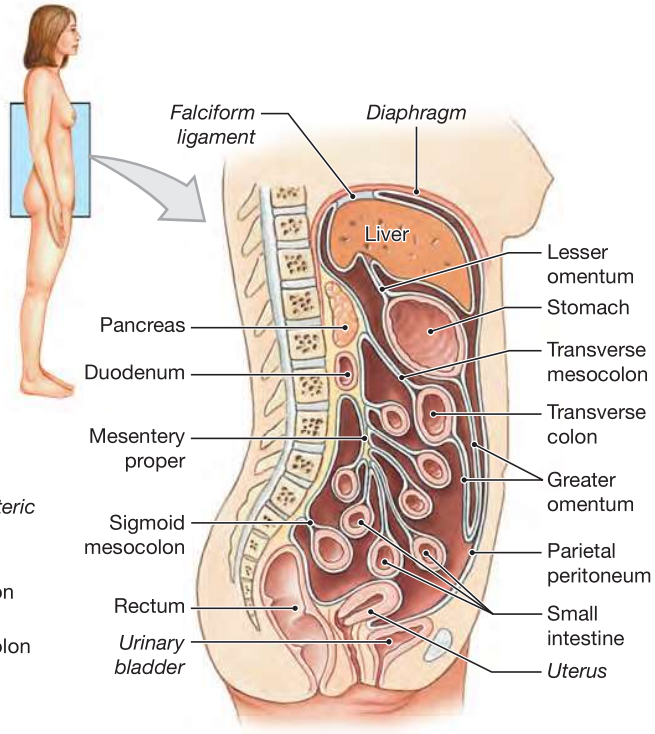
a During embryonic development, the digestive tube is initially suspended by dorsal and ventral mesenteries. In adults, the ventral mesentery is lost, except where it connects the stomach to the liver (at the lesser omentum) and the liver to the anterior body wall and diaphragm (at the falciform ligament).



b A diagrammatic view of the organization of mesenteries in an adult. As the digestive tract enlarges, mesenteries associated with the proximal portion of the small intestine, the pancreas, and the ascending and descending portions of the colon fuse to the body wall.



c An anterior view of the empty peritoneal cavity, showing the attachment of mesenteries to the posterior body wall. Some visceral organs that were originally suspended within the peritoneal cavity are now retroperitoneal due to fusion of the serosa with the parietal peritoneum.



d A sagittal section showing the mesenteries of an adult. Notice that the pancreas, duodenum, and rectum are retroperitoneal.

Clinical Note

Epithelial Renewal and Repair

The life span of a typical epithelial cell varies from two to three days in the esophagus to six days in the large intestine. The divisions of epithelial stem cells continuously renew the lining of the entire digestive tract. These divisions normally keep pace with the rates of cell destruction and loss at epithelial surfaces. This high rate of cell division explains why radiation and anticancer drugs that inhibit mitosis have drastic effects on the digestive tract. Lost epithelial cells are no longer replaced. The cumulative damage to the epithelial lining quickly leads to problems in absorbing nutrients. In addition, the exposure of the lamina propria to digestive enzymes can cause internal bleeding and other serious problems.

cells in the mucosa and submucosa—or in accessory glandular organs—are carried to the epithelial surfaces by ducts.

The Lamina Propria. The lamina propria is a layer of areolar tissue that also contains blood vessels, sensory nerve endings, lymphatic vessels, smooth muscle cells, and scattered lymphoid tissue. In the oral cavity, pharynx, esophagus, stomach, and *duodenum* (the proximal portion of the small intestine), the lamina propria also contains the secretory cells of mucous glands.

In most areas of the digestive tract, the lamina propria contains a narrow sheet of smooth muscle and elastic fibers. This sheet is called the **muscularis** (mus-kū-LAIR-is) **mucosae** (mū-KŌ-sē) (**Figure 24–3**). The smooth muscle cells in the muscularis mucosae are arranged in two concentric layers. The inner layer encircles the lumen (the *circular muscle*), and the outer layer contains muscle cells oriented parallel to the long axis of the tract (the *longitudinal layer*). Contractions in these layers alter the shape of the lumen and move the epithelial pleats and folds.

The Submucosa

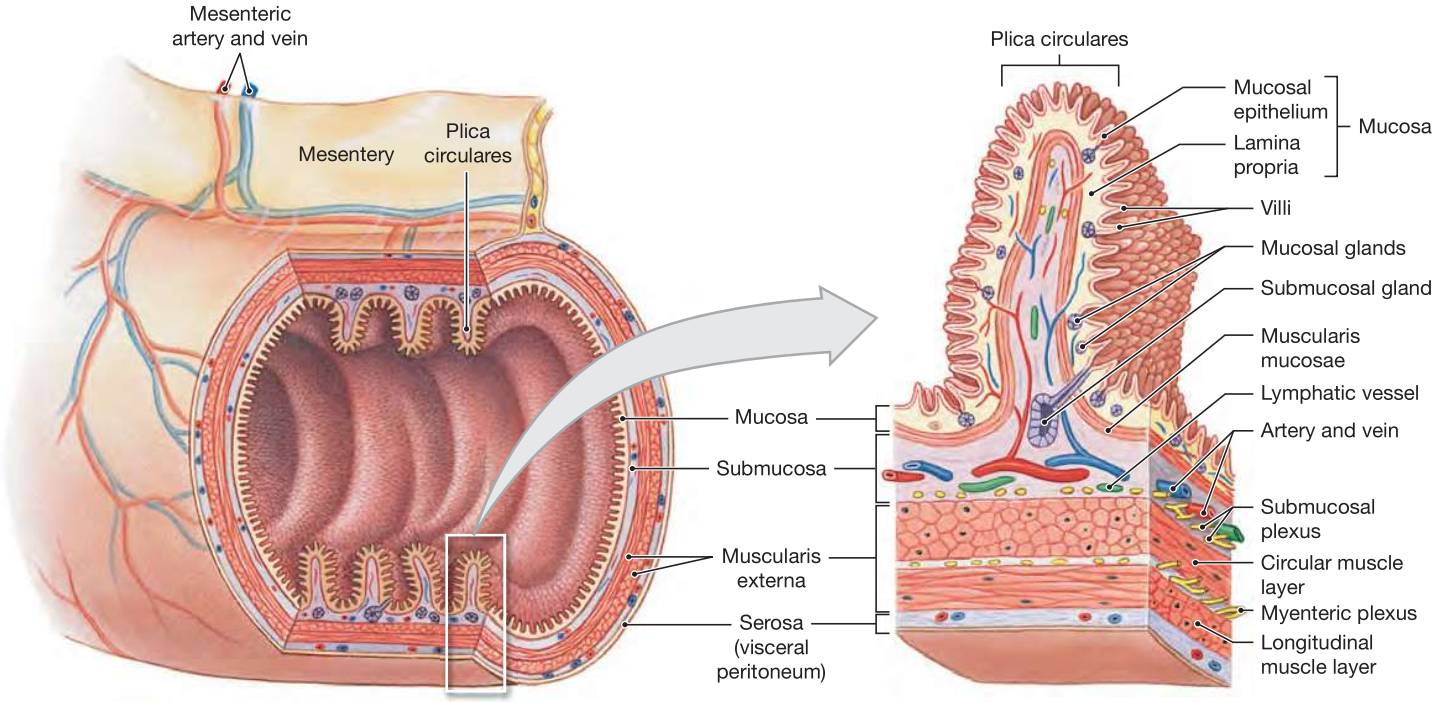
The **submucosa** is a layer of dense irregular connective tissue that binds the mucosa to the muscularis externa (**Figure 24–3**). The submucosa has numerous blood vessels and lymphatic vessels. In some regions it also contains exocrine glands that secrete buffers and enzymes into the lumen of the digestive tract.

Along its outer margin, the submucosa contains a network of intrinsic nerve fibers and scattered neurons. This network is the **submucosal plexus,** or *plexus of Meissner*. It contains sensory neurons, parasympathetic ganglionic neurons, and sympathetic postganglionic fibers that innervate the mucosa and submucosa.

The Muscularis Externa

The submucosal plexus lies along the inner border of the **muscularis externa,** also called the **muscularis.** Smooth muscle

Figure 24–3 The Structure of the Digestive Tract. A diagrammatic view of a representative portion of the digestive tract. The features illustrated are typical of those of the small intestine.



cells dominate this region. Like the smooth muscle cells in the muscularis mucosae, those in the muscularis externa are arranged in an inner circular layer and an outer longitudinal layer. These layers play an essential role in mechanical processing and in moving materials along the digestive tract.

The movements of the digestive tract are coordinated primarily by the sensory neurons, interneurons, and motor neurons of the enteric nervous system (ENS) p. 519. The ENS is primarily innervated by the parasympathetic division of the ANS. Sympathetic postganglionic fibers also synapse here. Many of these fibers continue onward to innervate the mucosa and the **myenteric** (mī-en-TER-ik) **plexus** (*mys*, muscle + *enteron*, intestine), or *plexus of Auerbach*. This plexus is a network of parasympathetic ganglia, sensory neurons, interneurons, and sympathetic postganglionic fibers. It lies sandwiched between the circular and longitudinal muscle layers. In general, parasympathetic stimulation increases muscle tone and activity. Sympathetic stimulation promotes muscular inhibition and relaxation.

Tips & Tricks

Because the parasympathetic nervous system plays a dominant role in the digestive process, it is often referred to as the "rest and digest" division.

The Serosa

A serous membrane known as the **serosa** covers the muscularis externa along most portions of the digestive tract inside the peritoneal cavity (**Figure 24–3**).

There is no serosa covering the muscularis externa of the oral cavity, pharynx, esophagus, and rectum. Instead, a dense network of collagen fibers firmly attaches the digestive tract to adjacent structures. This fibrous sheath is called an *adventitia* (ad-ven-TISH-uh).

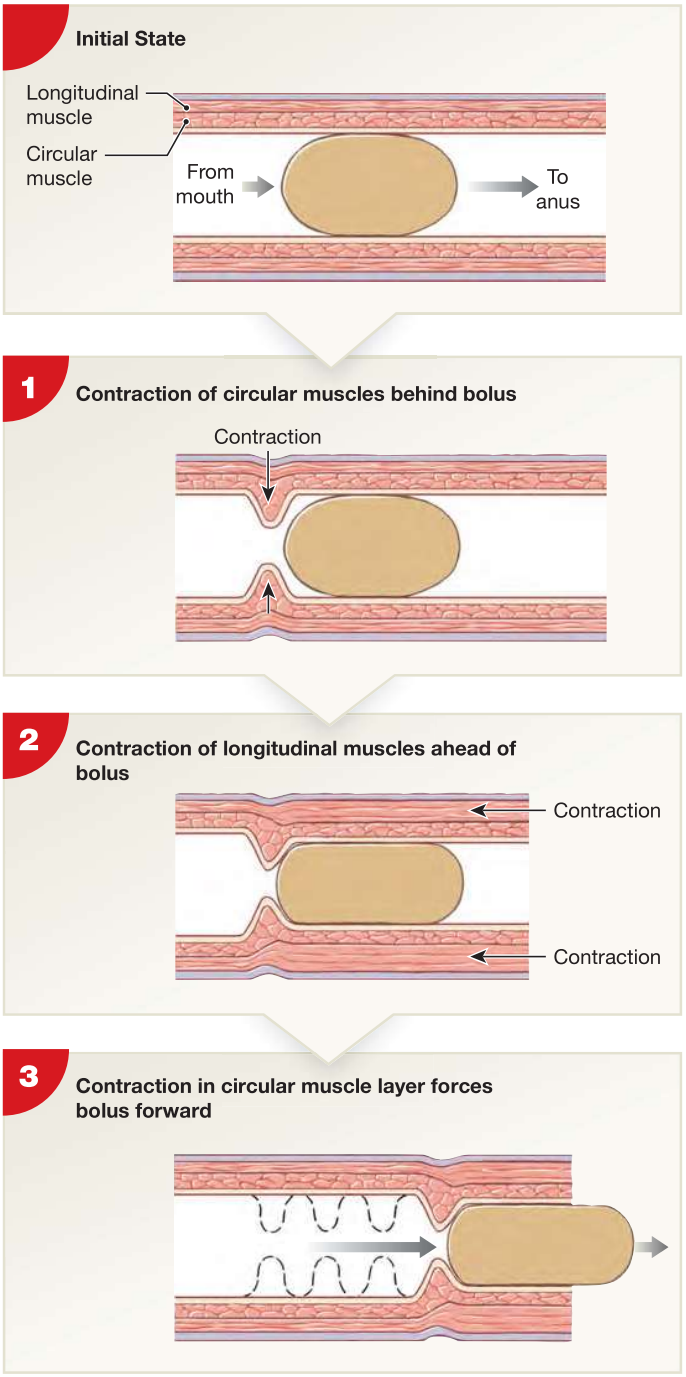
The Movement of Digestive Materials

The muscular layers of the digestive tract consist of *visceral smooth muscle tissue*. We introduced this type of smooth muscle in Chapter 10. p. 316 The smooth muscle along the digestive tract has rhythmic cycles of activity due to *pacesetter cells*. These smooth muscle cells undergo spontaneous depolarization, triggering a wave of contraction that spreads throughout the entire muscular sheet. Pacesetter cells are located in the muscularis mucosae and muscularis externa, which surround the lumen of the digestive tract. The coordinated contractions of the muscularis externa play a vital role in moving materials along the tract, through *peristalsis*, and in mechanical processing, through *segmentation*.

Peristalsis

The muscularis externa propels materials from one portion of the digestive tract to another by contractions known as **peristalsis** (per-i-STAL-sis). Peristalsis consists of waves of muscular contractions that move a **bolus** (BŌ-lus), or soft rounded ball of digestive contents, along the length of the digestive tract (**Figure 24–4**). During a peristaltic movement, the circular muscles contract behind the bolus while circular muscles ahead of the bolus relax. Longitudinal muscles ahead of the bolus then contract, shortening adjacent segments. A wave of contraction in the circular muscles then forces the bolus forward.

Figure 24–4 Peristalsis. Peristalsis propels materials along the length of the digestive tract.



Tips & Tricks

Squeezing toothpaste out of a tube is similar to peristalsis: Your squeezing hand (contracting circular muscles) forces toothpaste (the bolus) along and out of the tube (the digestive tract).

Segmentation

Most areas of the small intestine and some portions of the large intestine undergo cycles of contraction that churn and fragment the bolus, mixing the contents with intestinal secretions. This activity, called **segmentation,** does not follow a set pattern. For this reason, segmentation does not push materials along the tract in any one direction.

Control of Digestive Functions

Local factors interact with neural and hormonal mechanisms to regulate the activities of the digestive system (**Figure 24–5**).

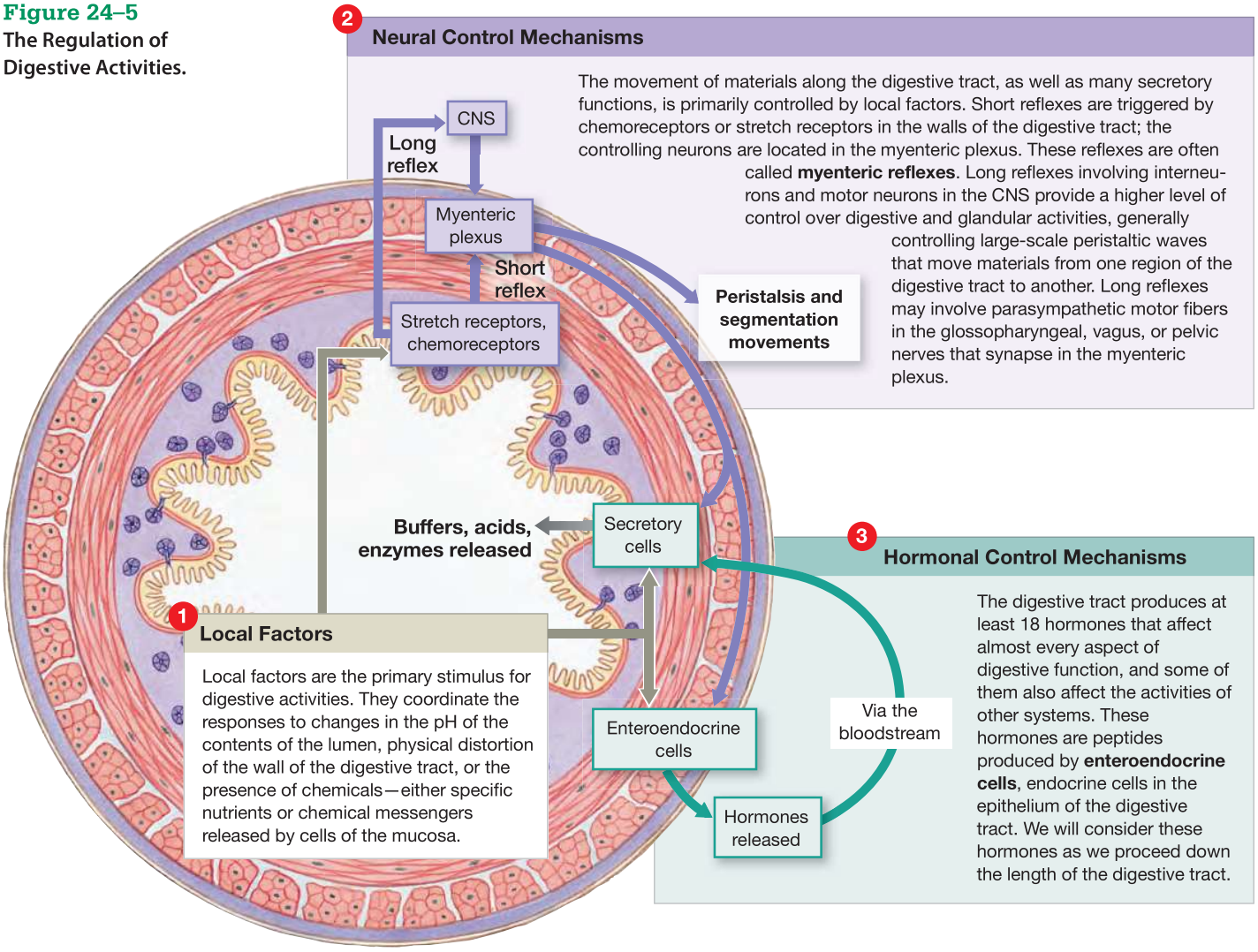
Local Factors

The initial regulation of digestive function occurs at the local level. Local environmental factors such as the pH, volume, or chemical composition of the intestinal contents can have a direct affect on digestive activity in that segment of the digestive tract. Some of these local factors have a direct effect on local digestive activities; for example, stretching of the intestinal wall can stimulate localized contractions of smooth muscles. In other cases, the local factors stimulate the release of chemical messengers. Prostaglandins, histamine, and other chemicals released into interstitial fluid may affect adjacent cells within a small segment of the tract. For example, the release of histamine in the lamina propria of the stomach stimulates the secretion of acid by cells in the adjacent epithelium.

Neural Mechanisms

Neural mechanisms control digestive tract movement. For example, sensory receptors in the walls of the digestive tract trigger

Figure 24–5
The Regulation of Digestive Activities.



peristaltic movements that are limited to a few centimeters. The motor neurons that control smooth muscle contraction and glandular secretion are located in the myenteric plexus. These neurons are usually considered parasympathetic, because some of them are innervated by parasympathetic preganglionic fibers. However, the plexus also contains sensory neurons, motor neurons, and interneurons responsible for local reflexes that operate entirely outside the control of the central nervous system. As we noted in Chapter 16, local reflexes are called *short reflexes*. ↩ p. 534

In general, short reflexes control localized activities that involve small segments of the digestive tract. For example, they may coordinate local peristalsis and trigger secretion by digestive glands in response to the arrival of a bolus. Many neurons are involved. The enteric nervous system has about as many neurons as the spinal cord, and as many neurotransmitters as the brain. The specific functions and interactions of these neurotransmitters in the enteric nervous system remain largely unknown.

Sensory information from receptors in the digestive tract is also distributed to the CNS. There it can trigger *long reflexes*, which involve interneurons and motor neurons in the CNS. ↩ p. 534

Hormonal Mechanisms

Digestive hormones can enhance or inhibit the sensitivity of the smooth muscle cells to neural commands. These hormones, produced by enteroendocrine cells in the digestive tract, travel through the bloodstream to reach their target organs.

Checkpoint

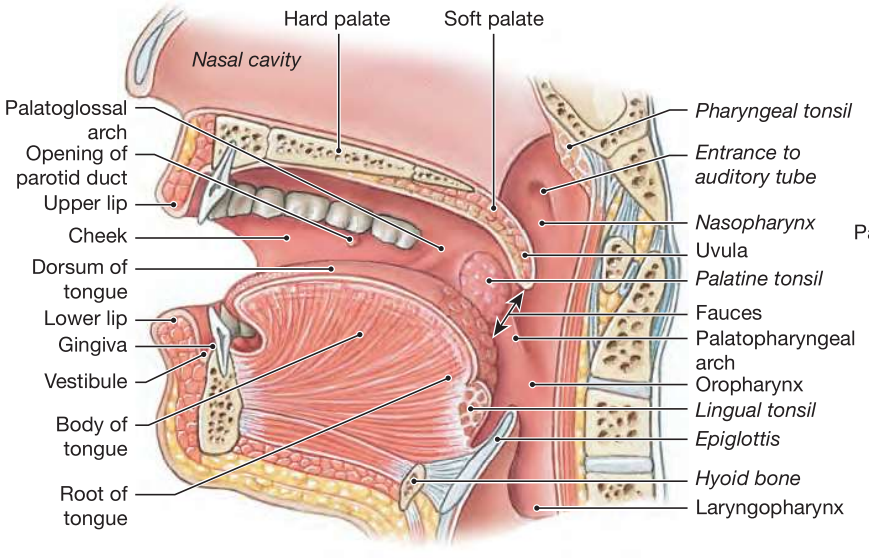
1. Identify the organs of the digestive system.
2. List and define the six primary functions of the digestive system.
3. What is the importance of the mesenteries?
4. Name the layers of the gastrointestinal tract from superficial to deep.
5. Which is more efficient in propelling intestinal contents from one place to another: peristalsis or segmentation?
6. What effect would a drug that blocks parasympathetic stimulation of the digestive tract have on peristalsis?

See the blue Answers tab at the back of the book.

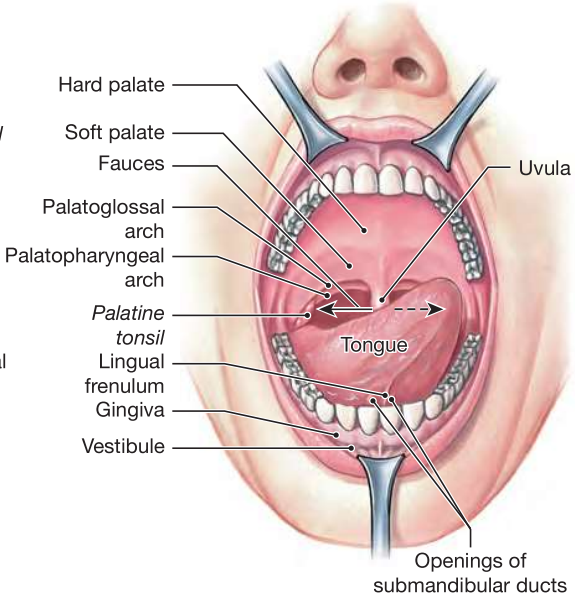
24-2 The oral cavity contains the tongue, salivary glands, and teeth, each with specific functions

Let's continue our exploration of the digestive tract by following the path of ingested materials. We begin at the mouth, which opens into the **oral cavity,** or **buccal** (BUK-ul) **cavity** (**Figure 24–6**). The functions of the oral cavity include (1) *sensory analysis* of material before swallowing; (2) *mechanical processing* through the actions of the teeth, tongue, and palatal surfaces; (3) *lubrication* by mixing with mucus and salivary gland secretions; and (4) limited *digestion* of carbohydrates and lipids.

Figure 24–6 The Oral Cavity. ATLAS: Plates 11a; 19



a A sagittal section of the oral cavity



b An anterior view of the oral cavity, as seen through the open mouth

The oral cavity is lined by the **oral mucosa,** which has a stratified squamous epithelium. A layer of keratinized cells covers regions exposed to severe abrasion, such as the superior surface of the tongue and the opposing surface of the hard palate (part of the roof of the mouth). The epithelial lining of the cheeks, lips, and inferior surface of the tongue is relatively thin and nonkeratinized. Nutrients are not absorbed in the oral cavity, but the mucosa inferior to the tongue is thin enough and vascular enough to permit the rapid absorption of lipid-soluble drugs. *Nitroglycerin* may be administered by this route to treat acute angina attacks. p. 683

The mucosae of the **cheeks,** or lateral walls of the oral cavity, are supported by pads of fat and the buccinator muscles. Anteriorly, the mucosa of each cheek is continuous with that of the lips, or **labia** (LĀ-bē-uh; singular, *labium*). The **vestibule** is the space between the cheeks (or lips) and the teeth. The **gingivae** (JIN-ji-vē), or *gums*, are ridges of oral mucosa that surround the base of each tooth on the alveolar processes of the maxillary bones and mandible. In most regions, the gingivae are firmly bound to the periostea of the underlying bones.

The hard and soft palates form the roof of the oral cavity. The tongue dominates its floor (**Figure 24–6b**). The floor of the mouth inferior to the tongue gets extra support from the geniohyoid and mylohyoid muscles. p. 337 The palatine processes of the maxillary bones and the horizontal plates of the palatine bones form the *hard palate*. A prominent central ridge, or *raphe* (RĀ-fee), extends along the midline of the hard palate. The mucosa lateral and anterior to the raphe is thick, with complex ridges. When your tongue compresses food against the hard palate, these ridges provide traction. The *soft palate* lies posterior to the hard palate. A thinner and more delicate mucosa covers the posterior margin of the hard palate and extends onto the soft palate.

The posterior margin of the soft palate supports the **uvula** (Ū-vū-luh), a dangling process that helps prevent food from entering the pharynx too soon (**Figure 24–6a**). On either side of the uvula are two pairs of muscular *pharyngeal arches* (**Figure 24–6b**). The more anterior **palatoglossal** (pal-a-tō-GLOS-al) **arch** extends between the soft palate and the base of the tongue. A curving line that connects the palatoglossal arches and uvula forms the boundaries of the **fauces** (FAW-sēz), the arched opening between the soft palate and the base of the tongue. The fauces serve as the passageway between the oral cavity and the oropharynx. The more posterior **palatopharyngeal** (pal-a-tō-fa-RIN-jē-al) **arch** extends from the soft palate to the pharyngeal wall. A palatine tonsil lies between the palatoglossal and palatopharyngeal arches on either side.

The Tongue

The **tongue** (**Figure 24–6**) manipulates materials inside the mouth and occasionally brings in foods (such as ice cream on a cone). The primary functions of the tongue are (1) mechanical processing by compression, abrasion, and distortion; (2) manipulation to assist in chewing and to prepare material for swallowing; (3) sensory analysis by touch, temperature, and taste receptors; and (4) secretion of mucins and the enzyme *lingual lipase*.

We can divide the tongue into an anterior **body,** or *oral portion*, and a posterior **root,** or *pharyngeal portion*. The superior surface, or *dorsum*, of the body contains a forest of fine projections, the *lingual papillae*. p. 551 The thickened epithelium covering each papilla assists the tongue in moving materials. A V-shaped line of circumvallate papillae roughly marks the boundary between the body and the root of the tongue, which is located in the oropharynx (**Figure 24–6a**).

The epithelium covering the inferior surface of the tongue is thinner and more delicate than that of the dorsum. Along the inferior midline is the **lingual frenulum** (FREN-ū-lum; *frenulum*, a small bridle), a thin fold of mucous membrane that connects the body of the tongue to the mucosa covering the floor of the oral cavity (**Figure 24–6a**). Ducts from two pairs of salivary glands open on either side of the lingual frenulum, which serves to prevent extreme movements of the tongue. However, an overly restrictive lingual frenulum hinders normal eating or speech. Properly diagnosed, this condition, called *ankyloglossia* (ang-ki-lō-GLOS-ē-uh), can be corrected surgically.

The tongue's epithelium is flushed by the secretions of small glands that extend into the underlying lamina propria. These secretions contain water, mucins, and the enzyme **lingual lipase.** This enzyme works over a broad pH range (3.0–6.0), enabling it to start lipid digestion immediately. Because lingual lipase tolerates an acid environment, it can continue to break down lipids—specifically, triglycerides—for a considerable time after the food reaches the stomach.

The tongue contains two groups of skeletal muscles. The large **extrinsic tongue muscles** perform all gross movements of the tongue. p. 336 The smaller **intrinsic tongue muscles** change the shape of the tongue and assist the extrinsic muscles during precise movements, as in speech. Both intrinsic and extrinsic tongue muscles are under the control of the hypoglossal cranial nerves (N XII).

Salivary Glands

Three pairs of salivary glands secrete into the oral cavity (**Figure 24–7a**). Each pair has a distinctive cellular organization and produces *saliva*, a mixture of glandular secretions, with slightly different properties:

1. The large **parotid** (pa-ROT-id) **salivary glands** lie inferior to the zygomatic arch deep to the skin covering the lateral and posterior surface of the mandible. Each gland has an

irregular shape. It extends from the mastoid process of the temporal bone across the outer surface of the masseter muscle. The parotid salivary glands produce a serous secretion containing large amounts of *salivary amylase*. This enzyme breaks down starches (complex carbohydrates). The secretions of each parotid gland are drained by a **parotid duct,** which empties into the vestibule at the second upper molar.

2. The **sublingual** (sub-LING-gwal) **salivary glands** are covered by the mucous membrane of the floor of the mouth. These glands produce a mucous secretion that acts as a buffer and lubricant. Numerous **sublingual ducts** open along either side of the lingual frenulum.
3. The **submandibular salivary glands** are in the floor of the mouth along the inner surfaces of the mandible within a depression called the *mandibular groove*. Cells of the submandibular glands (**Figure 24–7b**) secrete a mixture of buffers, glycoproteins called *mucins*, and salivary amylase. The **submandibular ducts** open into the mouth on either side of the lingual frenulum immediately posterior to the teeth (**Figure 24–6b**).

Saliva

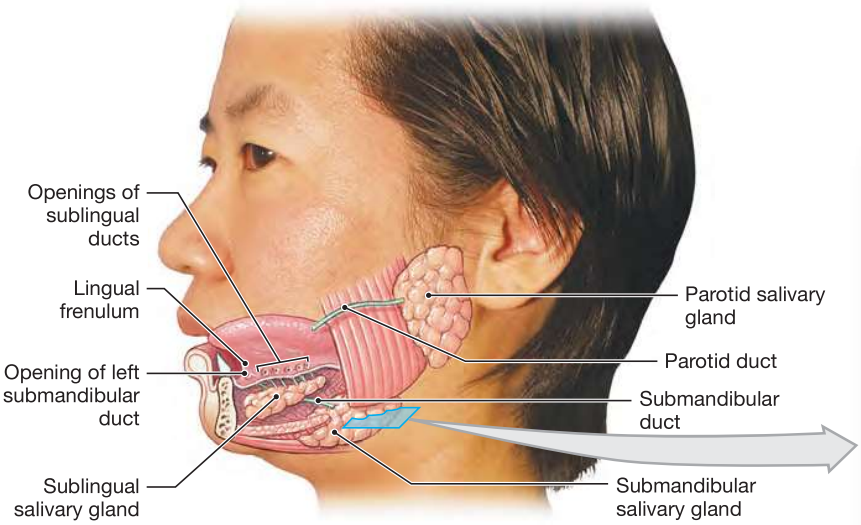
The salivary glands produce 1.0–1.5 liters of saliva each day. Saliva is 99.4 percent water. The remaining 0.6 percent includes electrolytes (principally Na^+, Cl^-, and HCO_3^-), buffers, glycoproteins, antibodies, enzymes, and waste products. The glycoproteins, called **mucins,** give saliva its lubricating action. About 70 percent of saliva comes from the submandibular salivary glands. Another 25 percent comes from the parotids, and 5 percent from the sublingual salivary glands.

Saliva continuously flushes the oral surfaces, helping to keep them clean. Buffers in the saliva keep the pH of your mouth near 7.0. They prevent the buildup of acids produced by bacteria. In addition, saliva contains antibodies (IgA) and *lysozyme.* Both

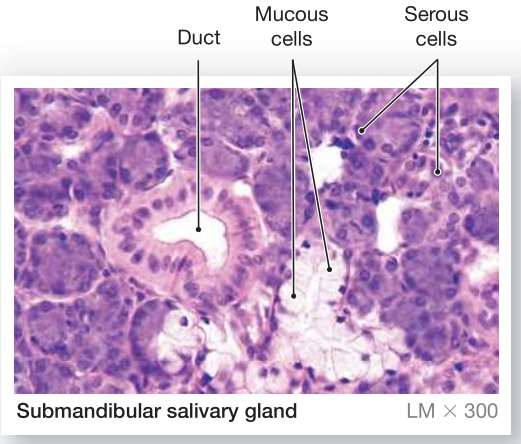
Clinical Note

Mumps The *mumps virus* most often targets the salivary glands, especially the parotid salivary glands, although other organs can also become infected. Infection typically occurs at 5–9 years of age. The first exposure stimulates the production of antibodies and, in most cases, confers permanent immunity. In postadolescent males, the mumps virus can also infect the testes and cause sterility. Infection of the pancreas by the mumps virus can produce temporary or permanent diabetes. Other organ systems, including the central nervous system, are affected in severe cases. A mumps vaccine effectively confers active immunity. Widespread administration of that vaccine has almost eliminated the incidence of the disease in the United States.

Figure 24–7 The Salivary Glands. ATLAS: Plates 3c,d; 18a,b



a A lateral view, showing the relative positions of the salivary glands and ducts on the left side of the head. For clarity, the left ramus and body of the mandible have been removed. For the positions of the parotid and submandibular ducts in the oral cavity, see *Figure 24-6*.



b The submandibular gland secretes a mixture of mucins, produced by mucous cells, and enzymes, produced by serous cells.

help control populations of oral bacteria. A reduction in or elimination of salivary secretions—caused by radiation, emotional distress, certain drugs, sleep, or other factors—triggers a bacterial population explosion. This proliferation rapidly leads to recurring infections and progressive erosion of the teeth and gums.

The saliva produced when you eat has a variety of functions, including the following:

- Lubricating the mouth.
- Moistening and lubricating materials in the mouth.
- Dissolving chemicals that can stimulate the taste buds and provide sensory information about the food.
- Beginning the digestion of complex carbohydrates before the food is swallowed. The enzyme involved is **salivary amylase,** also known as *ptyalin* or *alpha-amylase*. Saliva also contains a small amount of lingual lipase secreted by the glands of the tongue. The digestive process begins in the oral cavity, but it is not completed there. No absorption of nutrients takes place across the lining of the oral cavity.

Control of Salivary Secretions

The autonomic nervous system normally controls salivary secretions. Each salivary gland has parasympathetic and sympathetic innervation. The parasympathetic outflow originates in the **salivatory nuclei** of the medulla oblongata and synapses in the submandibular and otic ganglia. ↩ pp. 483, 485 Any object in your mouth can trigger a salivary reflex. It stimulates receptors monitored by the trigeminal nerve (N V) or taste buds innervated by cranial nerve VII, IX, or X. Parasympathetic stimulation speeds up secretion by all the salivary glands. As a result, you produce large amounts of saliva. The role of sympathetic innervation is unclear. Evidence suggests that it provokes the secretion of small amounts of very thick saliva.

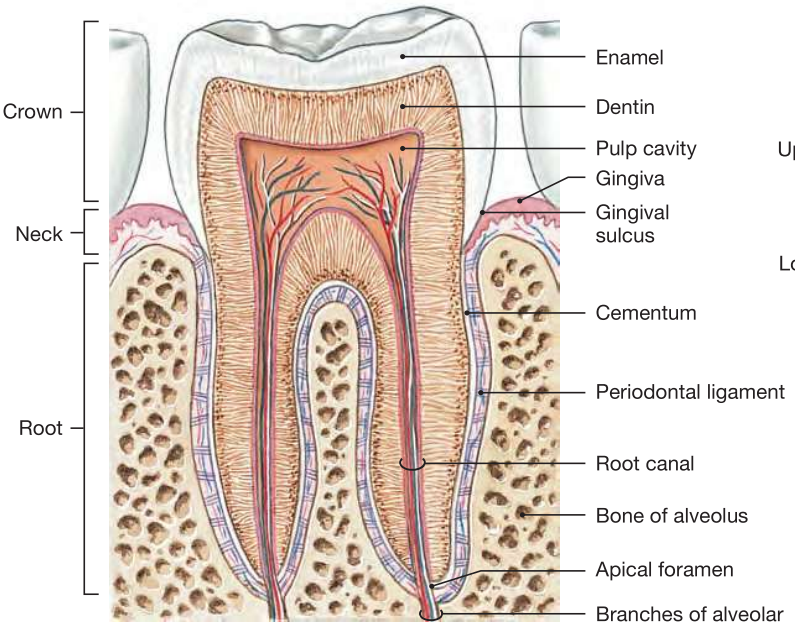
The salivatory nuclei are also influenced by other brain stem nuclei, as well as by the activities of higher centers. For example, chewing with an empty mouth, smelling food, or even thinking about food begins an increase in salivary secretion rates. That is why chewing gum keeps your mouth moist. Irritating stimuli in the esophagus, stomach, or intestines also speed up production of saliva, as does nausea. Increased saliva production in response to unpleasant stimuli helps reduce the stimulus by dilution, by rinsing, or by buffering strong acids or bases.

The Teeth

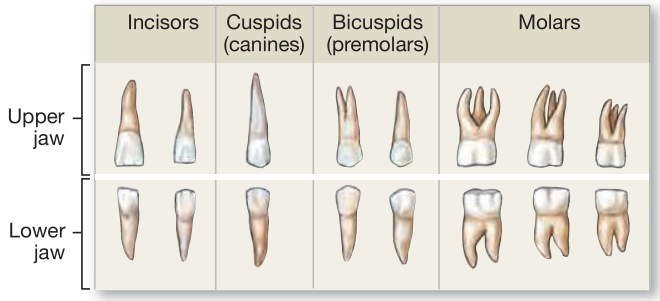
Movements of the tongue are important in passing food across the opposing surfaces, or *occlusal surfaces*, of the **teeth.** These surfaces carry out chewing, or **mastication** (mas-ti-KĀ-shun), of food. Mastication breaks down tough connective tissues in meat and the plant fibers in vegetable matter. It also helps saturate the materials with salivary secretions and enzymes.

Figure 24–8a is a sectional view through an adult tooth. The bulk of each tooth consists of a mineralized matrix similar to that of bone. This material, called **dentin,** differs from bone in that it

Figure 24–8 Teeth.



a A diagrammatic section through a typical adult tooth.



b The adult teeth from the right side of the upper and lower jaws. *Figure 24–9a,b* provides a view of the occlusal surfaces.

does not contain cells. Instead, cytoplasmic processes extend into the dentin from cells in the central **pulp cavity,** an interior chamber. The pulp cavity receives blood vessels and nerves through the **root canal,** a narrow tunnel located at the **root,** or base, of the tooth. Blood vessels and nerves enter the root canal through an opening called the **apical foramen** to supply the pulp cavity.

The root of each tooth sits in a bony socket called an *alveolus*. Collagen fibers of the **periodontal ligament** extend from the dentin of the root to the bone of the alveolus, creating a strong articulation known as a gomphosis. ↩ p. 255 A layer of **cementum** (se-MEN-tum) covers the dentin of the root. Cementum provides protection and firmly anchors the periodontal ligament. Cementum is histologically similar to bone and is less resistant to erosion than is dentin.

The **neck** of the tooth marks the boundary between the root and the **crown,** the exposed portion of the tooth that projects beyond the soft tissue of the gingiva. A shallow groove called the **gingival sulcus** surrounds the neck of each tooth. The mucosa of the gingival sulcus is very thin and is not tightly bound to the periosteum. The epithelium is bound to the tooth at the base of the sulcus. This epithelial attachment prevents bacterial access to the lamina propria of the gingiva and the relatively soft cementum of the root. When you brush and massage your gums, you stimulate the epithelial cells and strengthen the attachment. A condition called *gingivitis*, a bacterial infection of the gingivae, can occur if the attachment breaks down.

A layer of **enamel** covers the dentin of the crown. Enamel, which contains calcium phosphate in a crystalline form, is the hardest biologically manufactured substance. Adequate amounts of calcium, phosphates, and vitamin D during childhood are essential if the enamel coating is to be complete and resistant to decay.

Tooth decay generally results from the action of bacteria that live in your mouth. Bacteria adhering to the surfaces of the teeth produce a sticky matrix that traps food particles and creates deposits known as *dental plaque*. Over time, this organic material can become calcified, forming a hard layer of *tartar*, or *dental calculus*, which can be difficult to remove. Tartar deposits most commonly develop at or near the gingival sulcus, where brushing cannot remove the soft plaque deposits.

24

Types of Teeth

The alveolar processes of the maxillae and the mandible form the *maxillary* and *mandibular arcades*, or upper and lower dental arches, respectively. These arcades contain four types of teeth, each with specific functions (**Figure 24–8b**):

1. **Incisors** (in-SĪ-zerz) are blade-shaped teeth located at the front of the mouth. Incisors are useful for clipping or cutting, as when you nip off the tip of a carrot stick. These teeth have a single root.
2. The **cuspids** (KUS-pidz), or *canines*, are conical, with a sharp ridgeline and a pointed tip. Also known as the "eyeteeth," because they lie directly under the eye, the cuspids are used for tearing or slashing. You might weaken a tough piece of celery using the clipping action of the incisors and then take advantage of the shearing action provided by the cuspids. Cuspids have a single root.
3. **Bicuspids** (bī-KUS-pidz), or **premolars,** have flattened crowns with prominent ridges. They crush, mash, and grind. Bicuspids have one or two roots.
4. **Molars** have very large, flattened crowns with prominent ridges adapted for crushing and grinding. You can usually shift a tough nut to your bicuspids and molars for successful crunching. Molars typically have three or more roots.

Dental Succession

Two sets of teeth form during development. The first to appear are the **deciduous teeth** (de-SID-ū-us; *deciduus*, falling off), the temporary teeth of the **primary dentition.** Deciduous teeth are also called *primary teeth*, *milk teeth*, or *baby teeth*. Most children have 20 deciduous teeth (**Figure 24–9a**). On each side of the upper or lower jaw, the primary dentition consists of two incisors, one cuspid, and a pair of deciduous molars. These teeth are later replaced by the **secondary dentition,** or *permanent dentition* of the larger adult jaws (**Figure 24–9b**). Three additional molars appear on each side of the upper and lower jaws as the individual ages. These molars extend the rows of teeth posteriorly and bring the permanent tooth count to 32.

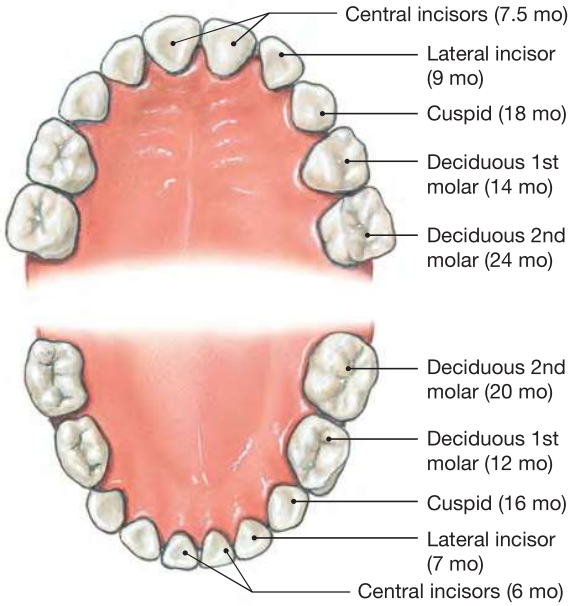
Tips & Tricks

Here's how to remember the timing of dental succession: The deciduous (primary) teeth are present during the primary grades, and the secondary (permanent) teeth are present during and after the secondary grades.

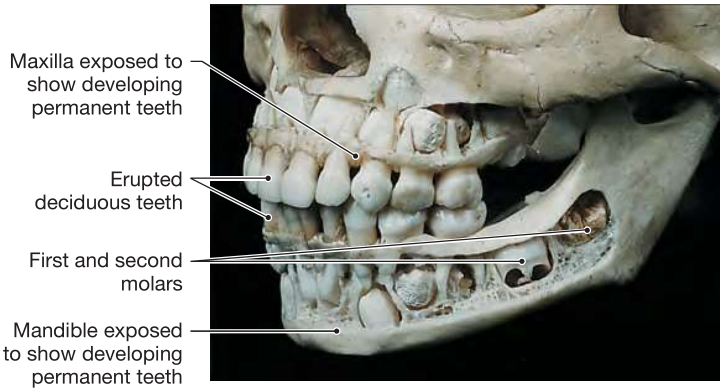
As replacement proceeds, the periodontal ligaments and roots of the primary teeth erode. The deciduous teeth either fall out or are pushed aside by the **eruption,** or emergence, of the secondary teeth (**Figure 24–9c**). The adult premolars take the place of the deciduous molars. The adult molars extend the tooth rows as the jaw enlarges.

The third molars, or *wisdom teeth*, may not erupt before age 21. Wisdom teeth may fail to erupt because they develop in inappropriate positions or because space on the dental arcade is inadequate. Any teeth that develop in locations that do not permit their eruption are called *impacted teeth*. Impacted wisdom teeth can be surgically removed to prevent the formation of abscesses.

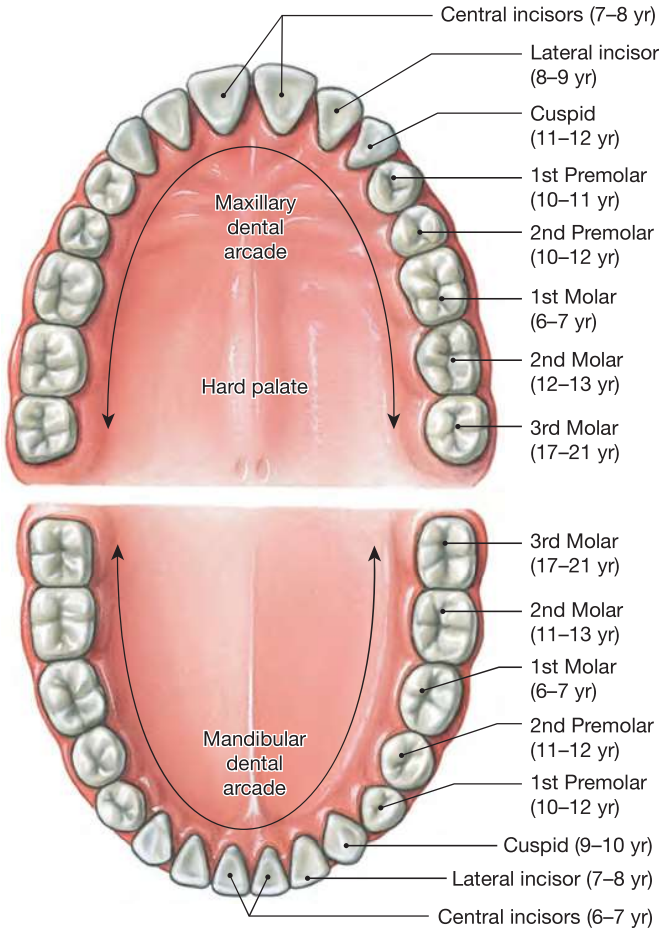
Figure 24–9 Primary and Secondary Dentitions.



a The primary teeth, with the age at eruption given in months.



c Maxilla and mandible with unerupted teeth exposed.



b The adult teeth, with the age at eruption given in years.

Mastication

The *muscles of mastication* close your jaws and slide or rock your lower jaw from side to side. ↩ p. 332 Chewing is not a simple process. It can involve any combination of mandibular elevation/depression, protraction/retraction, and medial/lateral movement. (Try classifying the movements involved the next time you eat.)

During mastication, you force food from the oral cavity to the vestibule and back, crossing and recrossing the **occlusal** (biting) **surfaces.** This movement results in part from the action of the muscles of mastication. Control would be impossible, however, without help from the muscles of the cheeks, lips, and tongue. Once you have shredded or torn the material to a satisfactory consistency and have moistened it with salivary secretions, your tongue compacts the debris into a moist, cohesive bolus that is fairly easy to swallow.

Checkpoint

7. **Name the structures associated with the oral cavity.**
8. **Which type of epithelium lines the oral cavity?**
9. **The digestion of which nutrient would be affected by damage to the parotid salivary glands?**
10. **Which type of tooth is most useful for chopping off bits of rigid foods?**
11. **Where are the fauces located?**

See the blue Answers tab at the back of the book.

24-3 The pharynx is a passageway between the oral cavity and esophagus

The **pharynx** (FAR-ingks), or throat, is an anatomical space that serves as a common passageway for solid food, liquids, and air. We described the epithelial lining and regions of the pharynx—the nasopharynx, the oropharynx, and the laryngopharynx—in Chapter 23. p. 819 Food normally passes through the oropharynx and laryngopharynx on its way to the esophagus. Both of these regions have a stratified squamous epithelium similar to that of the oral cavity. The lamina propria contains scattered mucous glands and the lymphoid tissue of the pharyngeal, palatal, and lingual tonsils. Deep to the lamina propria lies a dense layer of elastic fibers, bound to the underlying skeletal muscles.

We described the specific pharyngeal muscles involved in swallowing in Chapter 11. p. 336

- The *pharyngeal constrictor muscles* push the bolus toward and into the esophagus.
- The *palatopharyngeus* and *stylopharyngeus muscles* elevate the larynx.
- The *palatal muscles* elevate the soft palate and adjacent portions of the pharyngeal wall.

These muscles work with muscles of the oral cavity and esophagus to start swallowing, which pushes the bolus along the esophagus and into the stomach.

Checkpoint

12. **Describe the structure and function of the pharynx.**
13. **Identify the muscles associated with the pharynx.**

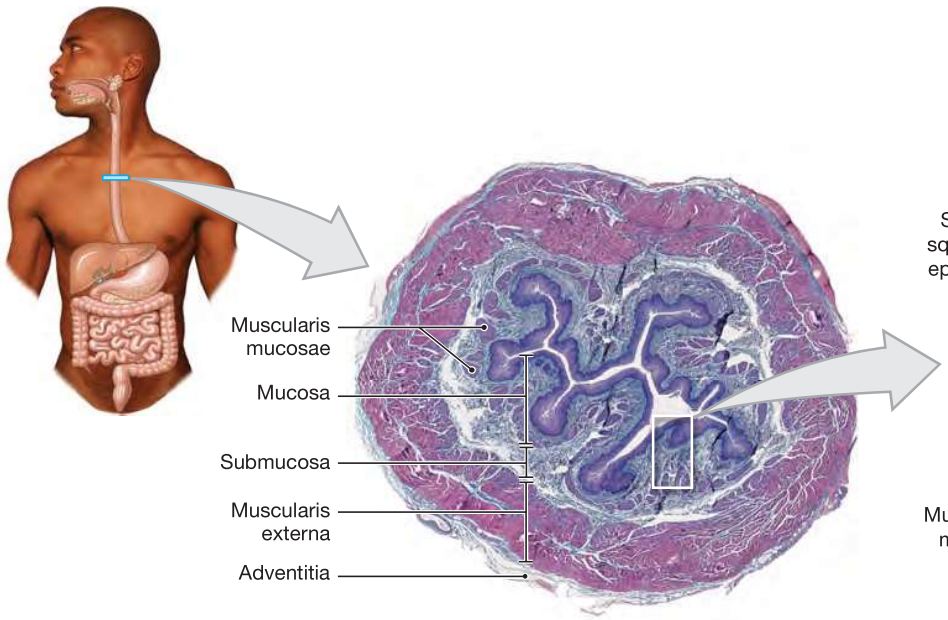
See the blue Answers tab at the back of the book.

24-4 The esophagus is a muscular tube that transports solids and liquids from the pharynx to the stomach

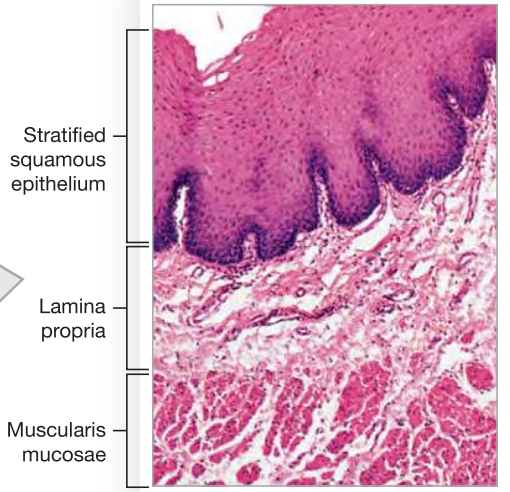
The **esophagus** (**Figure 24–10**) is a hollow muscular tube that conveys solid food and liquids to the stomach. Its length is approximately 25 cm (10 in.) and its diameter is about 2 cm (0.80 in.) at its widest point.

The esophagus begins posterior to the cricoid cartilage, at the level of vertebra C_6. It is narrowest at this point. The esophagus descends toward the thoracic cavity posterior to the trachea, continuing inferiorly along the posterior wall of the

Figure 24–10 The Esophagus.



a A transverse section through an empty esophagus.



b This light micrograph illustrates the extreme thickness of the epithelial portion of the esophageal mucosal layer.

mediastinum. It then enters the abdominopelvic cavity through the **esophageal hiatus** (hī-Ā-tus), an opening in the diaphragm. The esophagus empties into the stomach anterior to vertebra T_7.

The esophagus is innervated by parasympathetic and sympathetic fibers from the esophageal plexus. ↩ p. 531 Resting muscle tone in the circular muscle layer in the superior 3 cm (1.2 in.) of the esophagus normally prevents air from entering the esophagus. A comparable zone at the inferior end of the esophagus normally remains in a state of active contraction. This state prevents the backflow of materials from the stomach into the esophagus. Neither region has a well-defined sphincter muscle. Nevertheless, we often use the terms *upper esophageal sphincter* and *lower esophageal sphincter (cardiac sphincter)* to describe these regions, which are similar in function to other sphincters.

Tips & Tricks

Just as security gates control the passage of people by opening and closing, sphincters control the passage of material through them by dilating and constricting.

Histology of the Esophagus

The wall of the esophagus contains mucosal, submucosal, and muscularis layers comparable to those shown in **Figure 24–3**. Distinctive features of the esophageal wall include the following (**Figure 24–10**):

- The mucosa of the esophagus contains a nonkeratinized, stratified squamous epithelium similar to that of the pharynx and oral cavity.
- The mucosa and submucosa are packed into large folds that extend the length of the esophagus. These folds allow for expansion during the passage of a large bolus. Muscle tone in the walls keeps the lumen closed, except when you swallow.
- The muscularis mucosae consists of an irregular layer of smooth muscle.
- The submucosa contains scattered *esophageal glands*. They produce a mucous secretion that reduces friction between the bolus and the esophageal lining.
- The muscularis externa has the usual inner circular and outer longitudinal layers. However, in the superior third of the esophagus, these layers contain skeletal muscle fibers. The middle third contains a mixture of skeletal and smooth muscle tissue. Along the inferior third, only smooth muscle occurs.
- There is no serosa, but an adventitia of connective tissue outside the muscularis externa anchors the esophagus to the posterior body wall. Over the 1–2 cm (0.4–0.8 in.) between the diaphragm and stomach, the esophagus is retroperitoneal. Peritoneum covers the anterior and left lateral surfaces.

Swallowing

Swallowing, or **deglutition** (dē-gloo-TISH-un), is a complex process that can be initiated voluntarily but proceeds automatically once it begins. You take conscious control over swallowing when you eat or drink, but swallowing is also controlled at the subconscious level. The **swallowing reflex** begins when tactile receptors on the palatal arches and uvula are stimulated by the passage of the bolus. The information is relayed to the **swallowing center** of the medulla oblongata over the trigeminal (CN V) and glossopharyngeal (CN IX) nerves. Motor commands from this center then signal the pharyngeal musculature, producing a coordinated and stereotyped pattern of muscle contraction. It takes less than a second for the pharyngeal muscles to propel the bolus into the esophagus. During this period, the respiratory centers are inhibited and breathing stops. Swallowing takes place at regular intervals as saliva collects at the back of the mouth. Each day you swallow approximately 2400 times.

We can divide swallowing into buccal, pharyngeal, and esophageal phases, detailed in **Figure 24–11**.

Primary peristaltic waves are peristaltic movements coordinated by afferent and efferent fibers in the glossopharyngeal (CN IX) and vagus (CN X) nerves. For a typical bolus, the entire trip along the esophagus takes about 9 seconds. Liquids may make the journey in a few seconds, flowing ahead of the peristaltic contractions with the assistance of gravity.

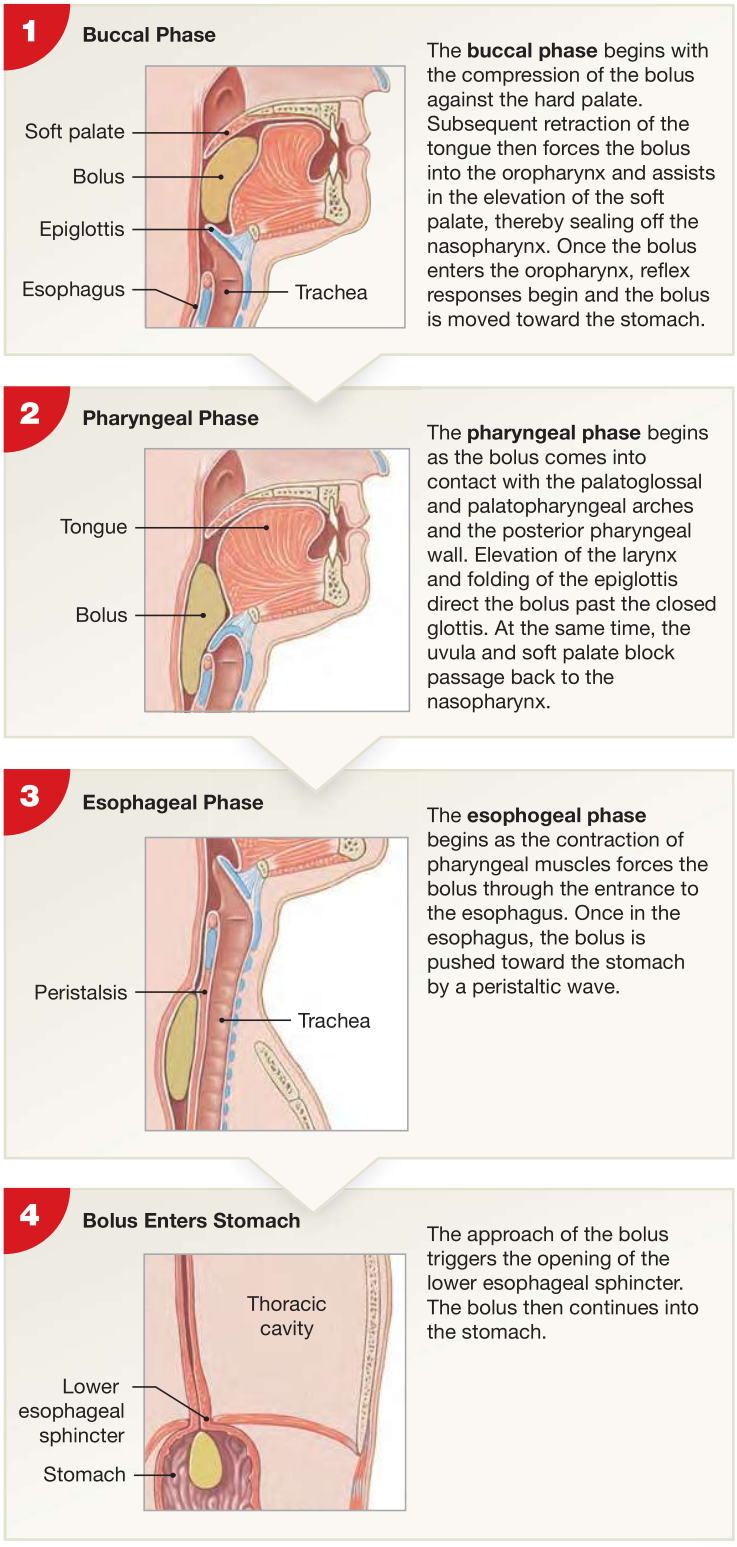
A dry or poorly lubricated bolus travels much more slowly. A series of *secondary peristaltic waves* may be required to push it all the way to the stomach. Secondary peristaltic waves are local reflexes triggered by the stimulation of sensory receptors in the esophageal walls.

Checkpoint

14. **Name the structure connecting the pharynx to the stomach.**
15. **Compared to other segments of the digestive tract, what is unusual about the muscularis externa of the esophagus?**
16. **What is occurring when the soft palate and larynx elevate and the glottis closes?**

See the blue Answers tab at the back of the book.

Figure 24–11 The Swallowing Process. This sequence, based on a series of x-rays, shows the phases of swallowing and the movement of a bolus from the mouth to the stomach.



24-5 The stomach is a J-shaped organ that receives the bolus from the esophagus and aids in chemical and mechanical digestion

The **stomach** performs four major functions: (1) storage of ingested food; (2) mechanical breakdown of ingested food; (3) disruption of chemical bonds in food through the action of acid and enzymes; and (4) production of *intrinsic factor*, a glycoprotein needed in the digestive tract for the absorption of vitamin B_{12} by the small intestine. Ingested substances combine with the secretions of the glands of the stomach, producing a viscous, highly acidic, soupy mixture of partially digested food called **chyme** (KĪM).

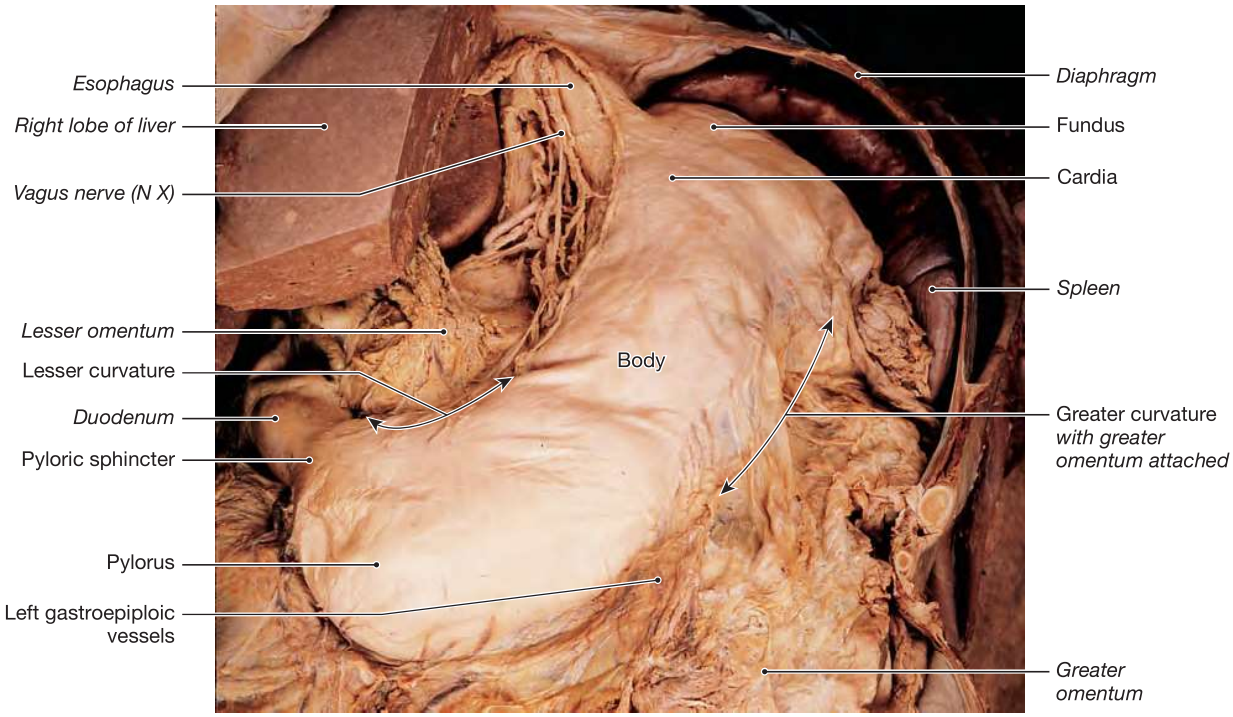
Anatomy of the Stomach

The stomach has the shape of an expanded J (**Figure 24–12**). A short **lesser curvature** forms the medial surface of the organ, and a long **greater curvature** forms the lateral surface. The anterior and posterior surfaces are smoothly rounded. The shape and size of the stomach can vary greatly from individual to individual and even from one meal to the next. In an "average" stomach, the lesser curvature is approximately 10 cm (4 in.) long, and the greater curvature measures about 40 cm (16 in.). The stomach typically extends between the levels of vertebrae T_7 and L_3.

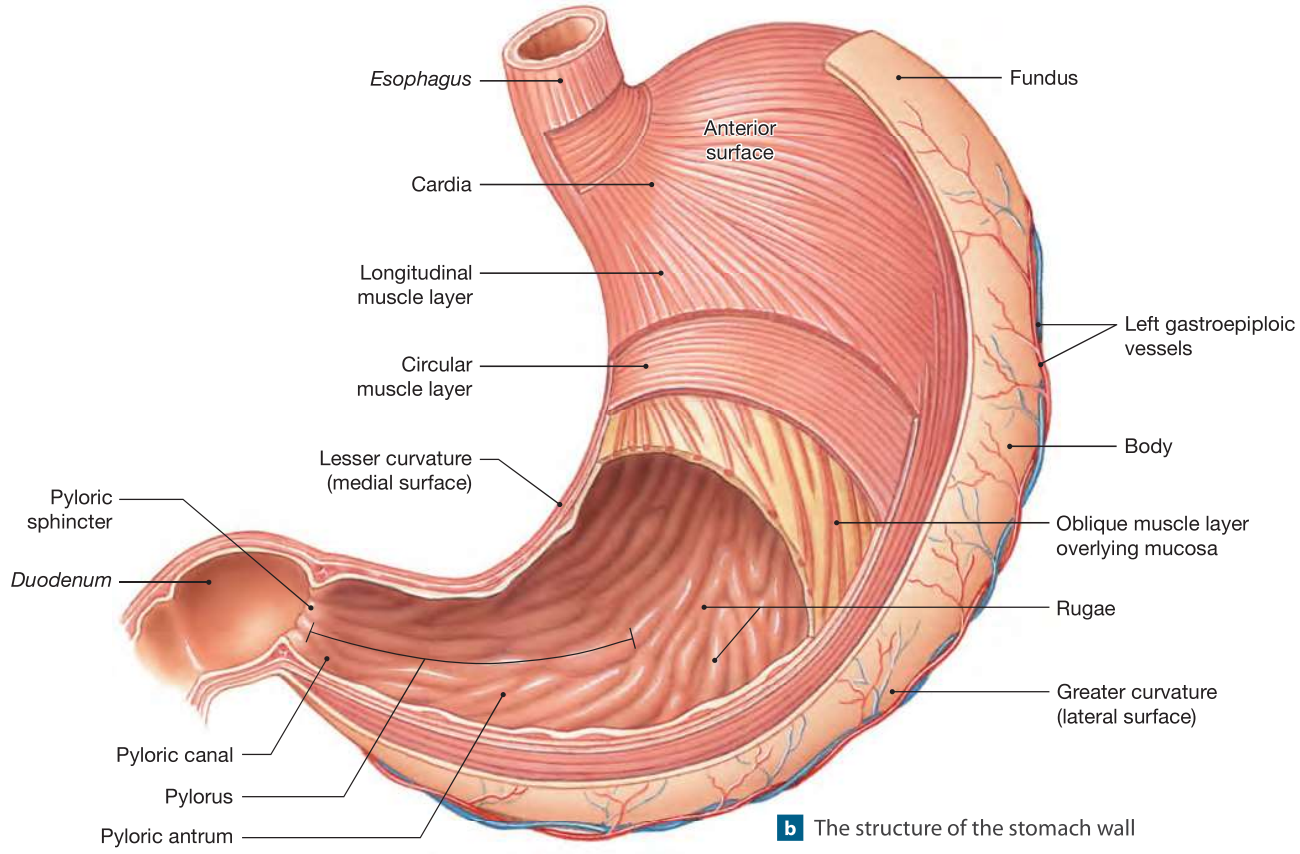
We can divide the stomach into four regions (**Figure 24–12**):

1. *The Cardia.* The **cardia** (KAR-dē-uh) is the smallest part of the stomach. It consists of the superior, medial portion of the stomach within 3 cm (1.2 in.) of the junction between the stomach and the esophagus. The cardia contains abundant mucous glands. Their secretions coat the connection with the esophagus and help protect that tube from the acid and enzymes of the stomach.
2. *The Fundus.* The **fundus** (FUN-dus) is the portion of the stomach that is superior to the junction between the stomach and the esophagus. The fundus contacts the inferior, posterior surface of the diaphragm (**Figure 24–12a**).
3. *The Body.* The area of the stomach between the fundus and the curve of the J is the **body,** the largest region of the stomach. The body acts as a mixing tank for ingested food and secretions produced in the stomach. *Gastric* (*gaster*, stomach) *glands* in the fundus and body secrete most of the acid and enzymes involved in gastric digestion.
4. *The Pylorus.* The **pylorus** (pī-LOR-us) forms the sharp curve of the J. The pylorus is divided into a **pyloric antrum** (*antron*, cavity), which is connected to the body, and a **pyloric canal,** which empties into the

Figure 24–12 The Stomach. ATLAS: Plates 49a–c; 50a–c



a The position and external appearance of the stomach, showing superficial landmarks



b The structure of the stomach wall

duodenum, the proximal segment of the small intestine. As mixing movements take place during digestion, the pylorus frequently changes shape. A muscular **pyloric sphincter** regulates the release of chyme into the duodenum. Glands in the pylorus secrete mucus and important digestive hormones, including *gastrin*, a hormone that stimulates gastric glands.

Tips & Tricks

The stomach squeezes chyme into the small intestine just as you squeeze cake frosting out of a pastry bag.

The stomach's volume increases while you eat and then decreases as chyme enters the small intestine. When the stomach is relaxed (empty), the mucosa has prominent folds called **rugae** (ROO-gē; wrinkles). These temporary features let the gastric lumen expand (**Figure 24–12b**). The stomach can stretch up to 50 times its empty size. As the stomach fills, the rugae gradually flatten out until, at maximum distension, they almost disappear. (The world record, set in 2009, for eating hot dogs with buns in 10 minutes is 68!) When empty, the stomach resembles a muscular tube with a narrow, constricted lumen. When full, it can contain 1–1.5 liters of material.

The muscularis mucosae and muscularis externa of the stomach contain extra layers of smooth muscle cells in addition to the usual circular and longitudinal layers. The muscularis mucosae generally contain an outer, circular layer of muscle cells. The muscularis externa has an inner, **oblique layer** of smooth muscle (**Figure 24–12b**). The extra layers of smooth muscle strengthen the stomach wall and assist in the mixing and churning essential to the formation of chyme.

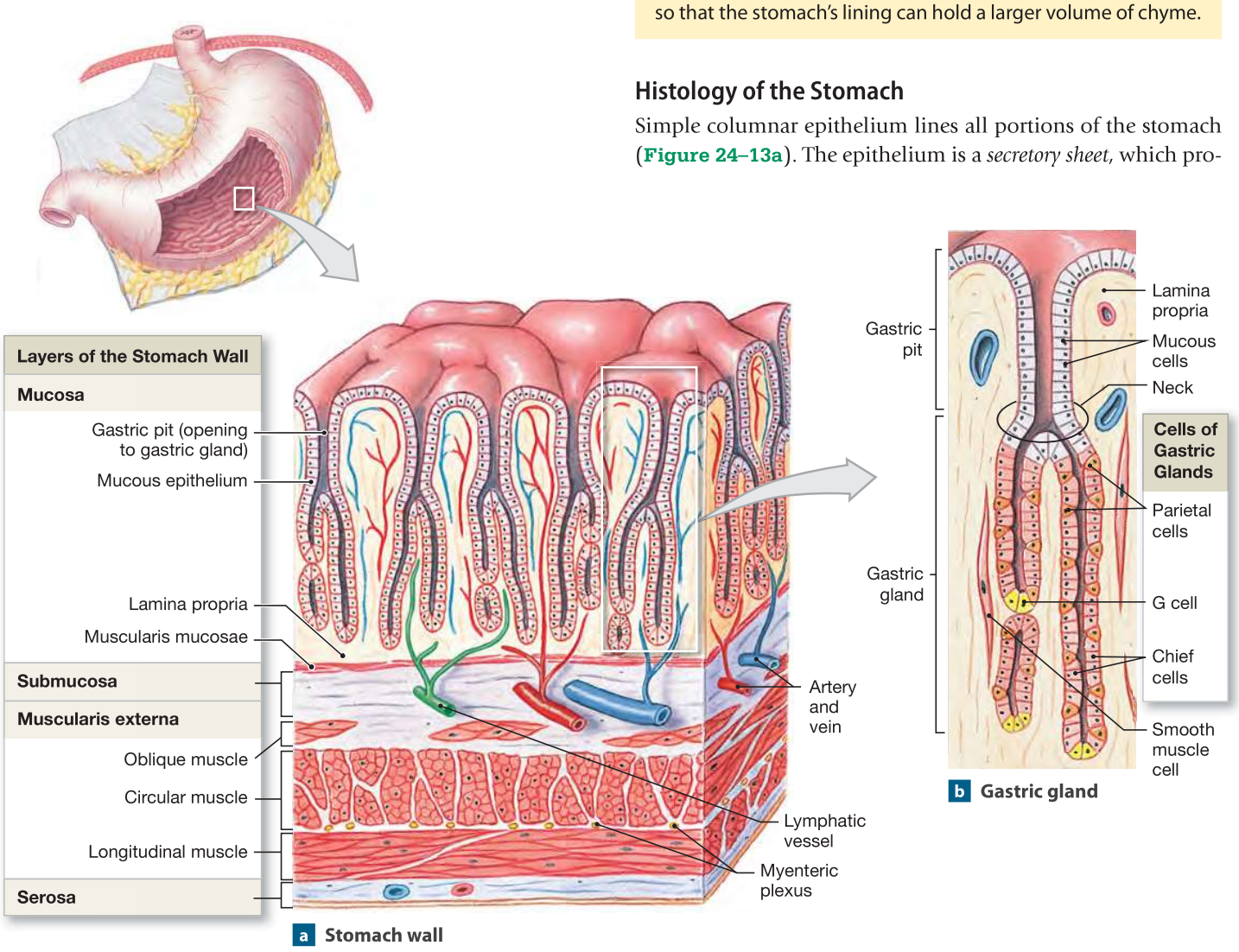
Tips & Tricks

Both plicae circulares and rugae allow a large surface area to fit within a small volume, just as crumpling a sheet of paper rearranges its surface area so that it occupies a smaller space. When the stomach fills, its rugae allow its volume to expand, so that the stomach's lining can hold a larger volume of chyme.

Histology of the Stomach

Simple columnar epithelium lines all portions of the stomach (**Figure 24–13a**). The epithelium is a *secretory sheet*, which pro-

Figure 24–13 The Stomach Lining.



duces a carpet of mucus that covers the interior surface of the stomach. The alkaline mucous layer protects epithelial cells against the acid and enzymes in the gastric lumen.

Shallow depressions called **gastric pits** open onto the gastric surface (**Figure 24–13b**). The mucous cells at the base, or *neck*, of each gastric pit actively divide, replacing superficial cells that are shed into the chyme. A typical gastric epithelial cell has a life span of three to seven days. Exposure to alcohol or other chemicals that damage or kill epithelial cells increases cell turnover.

Gastric Glands

In the fundus and body of the stomach, each gastric pit communicates with several **gastric glands,** which extend deep into the underlying lamina propria (**Figure 24–13b**). Gastric glands are dominated by two types of secretory cells: *parietal cells* and *chief cells*. Together, they secrete about 1500 mL (1.6 qt.) of **gastric juice** each day.

Parietal cells are especially common along the proximal portions of each gastric gland (**Figure 24–13b**). These cells secrete **intrinsic factor.** This glycoprotein helps the absorption of **vitamin B_{12}** across the intestinal lining. (Recall from Chapter 19 that this vitamin is essential for normal erythropoiesis.) ↩ p. 649

Parietal cells also secrete *hydrochloric acid* (HCl). They do not produce HCl in the cytoplasm, however. This acid is so strong that it would erode a secretory vesicle and destroy the cell. Instead, H^+ and Cl^-, the two ions that form HCl, are transported independently by different mechanisms (**Figure 24–14**).

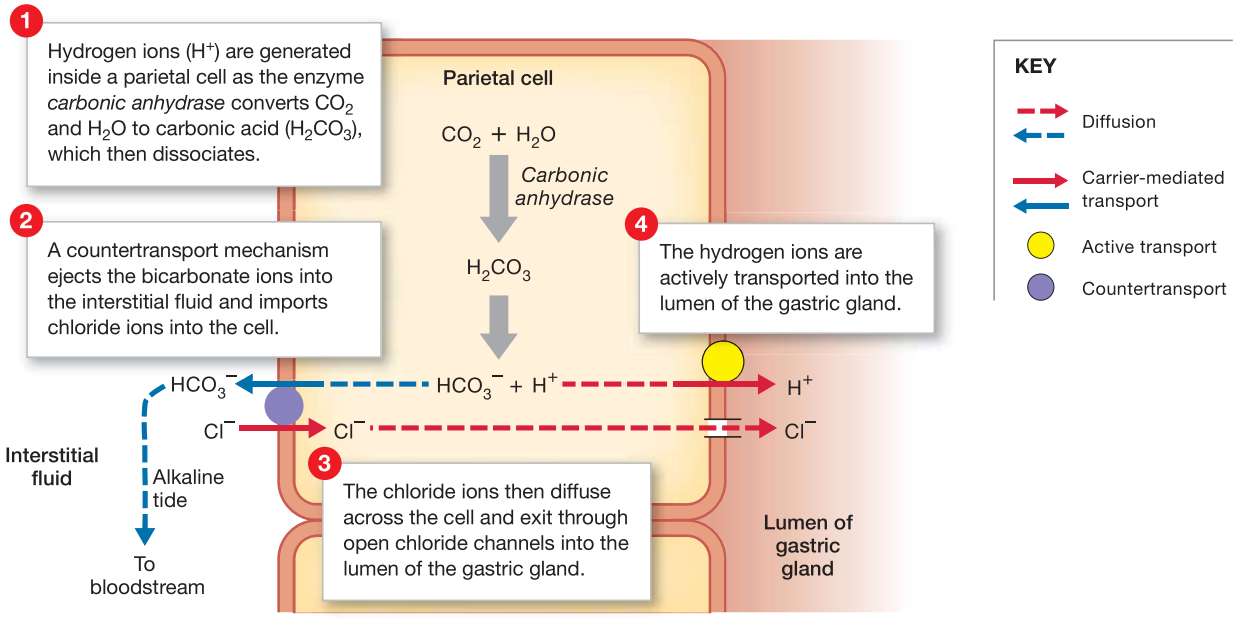
The initial step in HCl production is the formation of carbonic acid within parietal cells. The dissociation of releases bicarbonate and hydrogen ions. The H^+ are actively transported into the lumen of the gastric gland. The bicarbonate ions are exchanged for chloride ions from the interstitial fluid. When gastric glands are actively secreting, enough bicarbonate ions diffuse into the bloodstream from the interstitial fluid to increase the pH of the blood significantly. This sudden influx of bicarbonate ions has been called the *alkaline tide*.

The secretions of the parietal cells can keep the stomach contents at pH 1.5–2.0. Although this highly acidic environment does not by itself digest chyme, it has four important functions:

1. The acidity of gastric juice kills most of the microorganisms ingested with food.
2. The acidity denatures proteins and inactivates most of the enzymes in food.
3. The acidity helps break down plant cell walls and the connective tissues in meat.
4. An acidic environment is essential for the activation and function of *pepsin*, a protein-digesting enzyme secreted by chief cells.

Chief cells are most abundant near the base of a gastric gland (**Figure 24–13b**). These cells secrete **pepsinogen** (pep-SIN-ō-jen), an inactive proenzyme. Acid in the gastric lumen converts pepsinogen to **pepsin,** an active *proteolytic,* or protein-digesting, enzyme. Pepsin functions most effectively at a strongly acidic pH of 1.5–2.0. In addition, the stomachs of newborn infants (but not of adults) produce **rennin,** also known as *chymosin,* and **gastric lipase.** These enzymes are important for the digestion of milk. Rennin coagulates milk proteins. Gastric lipase initiates the digestion of milk fats.

Figure 24–14 The Secretion of Hydrochloric Acid.



Clinical Note

Gastritis and Peptic Ulcers

A superficial inflammation of the gastric mucosa is called *gastritis* (gas-TRĪ-tis). The condition can develop after a person has swallowed drugs, including beverage alcohol and aspirin. Gastritis is also associated with smoking, severe emotional or physical stress, bacterial infection of the gastric wall, or ingestion of strongly acidic or alkaline chemicals. Over time, gastritis can lead to the erosion of the gastric lining. *Gastric* or *peptic ulcers* may develop.

Pyloric Glands

Glands in the pylorus produce primarily a mucous secretion, rather than enzymes or acid. In addition, several types of enteroendocrine cells are scattered among the mucus-secreting cells. These enteroendocrine cells produce at least seven hormones, most notably **gastrin** (GAS-trin). Gastrin is produced by *G cells*, which are most abundant in the gastric pits of the pyloric antrum. Gastrin stimulates secretion by both parietal and chief cells, as well as contractions of the gastric wall that mix and stir the gastric contents.

The pyloric glands also contain *D cells*, which release **somatostatin,** a hormone that inhibits the release of gastrin. D cells continuously release their secretions into the interstitial fluid adjacent to the G cells. Neural and hormonal stimuli can override this inhibition of gastrin production when the stomach is preparing for digestion or is already engaged in digestion.

Several other hormones play a role in hunger and satiety. Levels of *ghrelin*, a hormone produced by *P/D1 cells* lining the fundic region of the stomach, rise before meals to initiate hunger. Ghrelin levels decline shortly after eating to curb appetite. Ghrelin is also antagonistic to *leptin*, a fat-tissue-derived hormone that induces satiety. Another hormone from the stomach and small intestine, *obestatin*, decreases appetite. The same gene encodes both ghrelin and obestatin.

Regulation of Gastric Activity

The production of acid and enzymes by the gastric mucosa can be (1) controlled by the CNS; (2) regulated by short reflexes of the enteric nervous system, coordinated in the wall of the stomach; and (3) regulated by hormones of the digestive tract. Gastric control proceeds in three overlapping phases. They are named according to the location of the control center: the *cephalic phase*, the *gastric phase*, and the *intestinal phase* (**Spotlight Figure 24–15**).

Digestion and Absorption in the Stomach

The stomach carries out preliminary digestion of proteins by pepsin. For a variable period, it permits the digestion of carbohydrates and lipids by salivary amylase and lingual lipase. Until the pH throughout the contents of the stomach falls below 4.5, these enzymes continue to work on carbohydrates and lipids. They generally remain active one to two hours after a meal.

As the stomach contents become more fluid and the pH approaches 2.0, pepsin activity increases. Protein disassembly begins. Protein digestion is not completed in the stomach, because time is limited and pepsin attacks only specific types of peptide bonds, not all of them. However, pepsin generally has enough time to break down complex proteins into smaller peptide and polypeptide chains before the chyme enters the duodenum.

Although digestion takes place in the stomach, nutrients are not absorbed there, for several reasons: (1) The epithelial cells are covered by a blanket of alkaline mucus and are not directly exposed to chyme; (2) the epithelial cells lack the specialized transport mechanisms of cells that line the small intestine; (3) the gastric lining is relatively impermeable to water; and (4) digestion has not been completed by the time chyme leaves the stomach. At this stage, most carbohydrates, lipids, and proteins are only partially broken down.

Some drugs can be absorbed in the stomach. For example, ethyl alcohol can diffuse through the mucous barrier and penetrate the lipid membranes of the epithelial cells. As a result, beverage alcohol is absorbed in your stomach before any nutrients in a meal reach the bloodstream. Meals containing large amounts of fat slow the rate of alcohol absorption. Why? The reason is that alcohol is lipid soluble, and some of it will be dissolved in fat droplets in the chyme. Aspirin is another lipid-soluble drug that can enter the bloodstream across the gastric mucosa. Such drugs alter the properties of the mucous layer and can promote epithelial damage by stomach acid and enzymes. Prolonged use of aspirin can cause gastric bleeding, so individuals with stomach ulcers usually avoid aspirin.

Checkpoint

17. **Name the four major regions of the stomach.**
18. **Discuss the significance of the low pH in the stomach.**
19. **How does a large meal affect the pH of blood leaving the stomach?**
20. **When a person suffers from chronic gastric ulcers, the branches of the vagus nerves that serve the stomach are sometimes cut in an attempt to provide relief. Why might this be an effective treatment?**

See the blue Answers tab at the back of the book.

24-6 The small intestine digests and absorbs nutrients, and associated glandular organs assist with the digestive process

The stomach is a holding tank in which food is saturated with gastric juices and exposed to stomach acid and the digestive effects of pepsin. Most of the important digestive and absorptive steps of digestion take place in the small intestine, where chemical digestion is completed and the products of digestion are absorbed. The mucosa of the small intestine produces only a few of the enzymes involved. The pancreas provides digestive enzymes, as well as buffers that help neutralize chyme. The liver secretes *bile*, a solution stored in the gallbladder for discharge into the small intestine. Bile contains buffers and *bile salts*, compounds that facilitate the digestion and absorption of lipids.

The Small Intestine

The **small intestine** plays the key role in the digestion and absorption of nutrients. Ninety percent of nutrient absorption takes place in the small intestine. Most of the rest occurs in the large intestine. The small intestine averages 6 m (19.7 ft) in length (range: 4.5–7.5 m; 14.8–24.6 ft). Its diameter ranges from 4 cm (1.6 in.) at the stomach to about 2.5 cm (1 in.) at the junction with the large intestine. It occupies all abdominal regions except the right and left hypochondriac and epigastric regions (see **Figure 1–6b**, p. 17). The small intestine has three segments: the duodenum, the jejunum, and the ileum (**Figure 24–16a**).

The **duodenum** (doo-ō-DĒ-num), 25 cm (10 in.) in length, is the segment closest to the stomach. This portion of the small intestine is a "mixing bowl." It receives chyme from the stomach and digestive secretions from the pancreas and liver. From its connection with the stomach, the duodenum curves in a C that encloses the pancreas. Except for the proximal 2.5 cm (1 in.), the duodenum is in a retroperitoneal position between vertebrae L_1 and L_4 (**Figure 24–2d**).

A rather abrupt bend marks the boundary between the duodenum and the **jejunum** (je-JOO-num). At this junction, the small intestine reenters the peritoneal cavity, supported by a sheet of mesentery. The jejunum is about 2.5 meters (8.2 ft) long. The bulk of chemical digestion and nutrient absorption occurs there.

The **ileum** (IL-ē-um), the final segment of the small intestine, is also the longest. It averages 3.5 meters (11.5 ft) in length. The ileum ends at the **ileocecal** (il-ē-ō-SĒ-kal) **valve.** This sphincter controls the flow of material from the ileum into the *cecum* of the large intestine.

Tips & Tricks

To remember the order of the small intestine segments, beginning at the stomach, use this mnemonic: ***D***on't ***j***ump ***i***n—***d***uodenum, ***j***ejunum, and ***i***leum. Also, do not confuse ileum, the last segment of the small intestine, with ilium, which is a bone.

The small intestine fills much of the peritoneal cavity. Its position is stabilized by the mesentery proper, a broad mesentery attached to the posterior body wall (**Figure 24–2c,d**). The stomach, large intestine, abdominal wall, and pelvic girdle restrict movement of the small intestine during digestion. Blood vessels, lymphatic vessels, and nerves reach the segments of the small intestine within the connective tissue of the mesentery. The primary blood vessels involved are branches of the superior mesenteric artery and the superior mesenteric vein. pp. 745, 755

The segments of the small intestine—the duodenum, jejunum, and ileum—are distinguished by both histological specialization and primary function.

Histology of the Small Intestine

The intestinal lining bears a series of transverse folds called **plicae circulares** (**Figure 24–16b**). Unlike the rugae in the stomach, the plicae circulares are permanent features. They do not disappear when the small intestine fills. The small intestine contains roughly 800 plicae circulares—roughly 2 per centimeter. They greatly increase the surface area available for absorption.

Intestinal Villi

The mucosa of the small intestine is thrown into a series of fingerlike projections, the **intestinal villi** (**Figure 24–17a,b**). The villi are covered by simple columnar epithelium that is carpeted with microvilli. The cells are said to have a *brush border* because the microvilli project from the epithelium like the bristles on a brush (**Figure 24–17d**).

If the small intestine were a simple tube with smooth walls, it would have a total absorptive area of about 3300 cm^2 (3.6 ft^2). Instead, the mucosa contains plicae circulares; each plica circulares supports a forest of villi; and epithelial cells bearing microvilli cover each villus. This arrangement increases the total area for absorption by a factor of more than 600, to approximately 2 million cm^2 (more than 2200 ft^2). This area is roughly the floor space of a spacious four-bedroom home.

The lamina propria of each villus contains an extensive network of capillaries that originate in a vascular network within the submucosa (**Figure 24–17c**). These capillaries carry absorbed nutrients to the hepatic portal circulation for delivery to the liver. The liver then adjusts the nutrient concentrations of blood before the blood reaches the general systemic circulation.

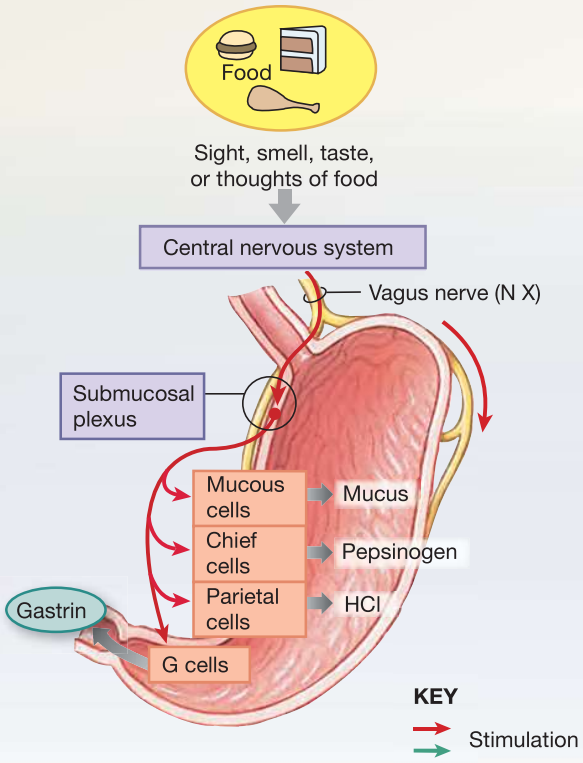
Spotlight

Figure 24–15
Regulation of Gastric Activity

The duodenum plays a key role in controlling digestive function because it monitors the contents of the chyme and adjusts the activities of the stomach and accessory glands to protect the delicate absorptive surfaces of the jejunum. This pivotal role of the duodenum is apparent when you consider the three phases of gastric secretion.

1 CEPHALIC PHASE

The **cephalic phase** of gastric secretion begins when you see, smell, taste, or think of food. This phase, which is directed by the CNS, prepares the stomach to receive food. The neural output proceeds by way of the parasympathetic division of the autonomic nervous system. The vagus nerves innervate the submucosal plexus of the stomach. Next, postganglionic parasympathetic fibers innervate mucous cells, chief cells, parietal cells, and G cells of the stomach. In response to stimulation, the production of gastric juice speeds up, reaching rates of about 500 mL/h, or about 2 cups per hour. This phase generally lasts only minutes.



Emotional states can exaggerate or inhibit the cephalic phase. For example, anger or hostility leads to excessive gastric secretion. On the other hand, anxiety, stress, or fear decreases gastric secretion and gastric contractions, or *motility.*

2 GASTRIC PHASE

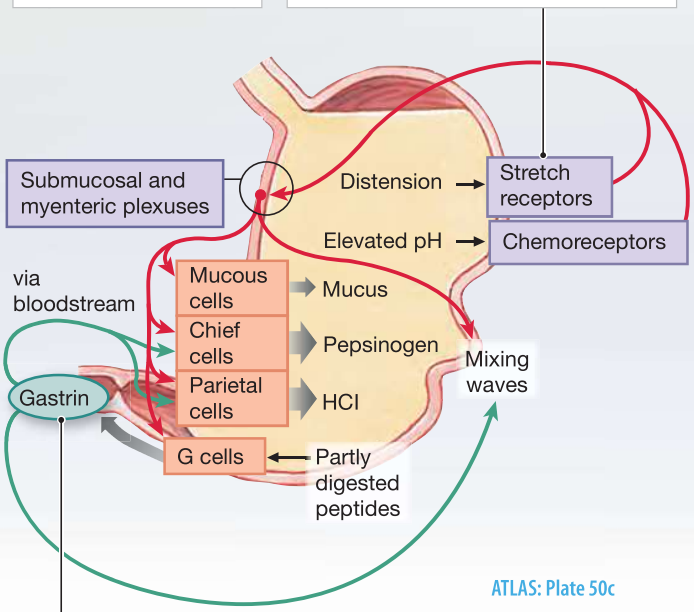
The **gastric phase** begins with the arrival of food in the stomach and builds on the stimulation provided during the cephalic phase. This phase may continue for three to four hours while the acid and enzymes process the ingested materials. The stimuli that initiate the gastric phase are (1) distension of the stomach, (2) an increase in the pH of the gastric contents, and (3) the presence of undigested materials in the stomach, especially proteins and peptides. The gastric phase consists of the following mechanisms:

Local Response

Distention of the gastric wall stimulates the release of histamine in the lamina propria, which binds to receptors on the parietal cells and stimulates acid secretion.

Neural Response

The stimulation of stretch receptors and chemoreceptors triggers short reflexes coordinated in the submucosal and myenteric plexuses. This in turn activates the stomach's secretory cells. The stimulation of the myenteric plexus produces powerful contractions called **mixing waves** in the muscularis externa.



ATLAS: Plate 50c

Hormonal Response

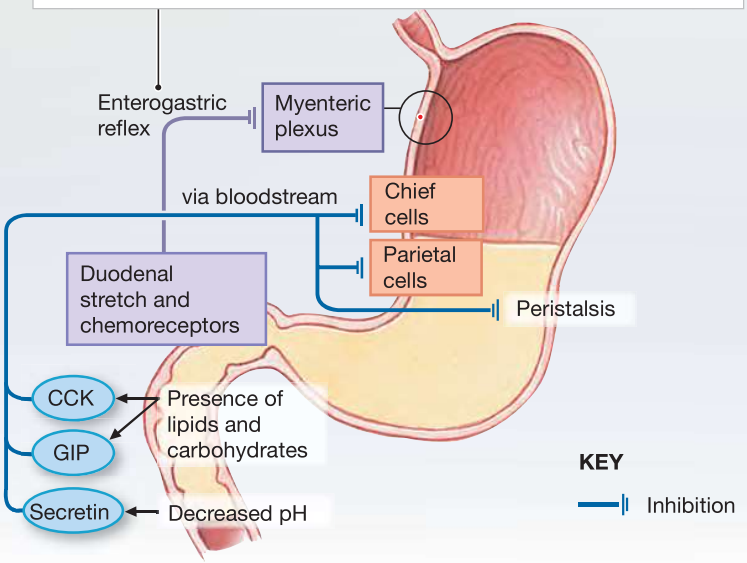
Neural stimulation and the presence of peptides and amino acids in chyme stimulate the secretion of the hormone gastrin, primarily by G cells. Gastrin travels via the bloodstream to parietal and chief cells, whose increased secretions reduce the pH of the gastric juice. In addition, gastrin also stimulates gastric motility.

3 INTESTINAL PHASE

The **intestinal phase** of gastric secretion begins when chyme first enters the small intestine. The function of the intestinal phase is controlling the rate of gastric emptying to ensure that the secretory, digestive, and absorptive functions of the small intestine can proceed with reasonable efficiency. Although here we consider the intestinal phase as it affects stomach activity, the arrival of chyme in the small intestine also triggers other neural and hormonal events that coordinate the activities of the intestinal tract and the pancreas, liver, and gallbladder.

Neural Responses

Chyme leaving the stomach decreases the distension in the stomach, thereby reducing the stimulation of stretch receptors. Distension of the duodenum by chyme stimulates stretch receptors and chemoreceptors that trigger the **enterogastric reflex**. This reflex inhibits both gastrin production and gastric contractions and stimulates the contraction of the pyloric sphincter, which prevents further discharge of chyme. At the same time, local reflexes at the duodenum stimulate mucus production, which helps protect the duodenal lining from the arriving acid and enzymes.



Hormonal Responses

The arrival of chyme in the duodenum triggers hormonal responses:

- Arrival of lipids and carbohydrates stimulates the secretion of cholecystokinin (CCK) and gastric inhibitory peptide (GIP).
- A drop in pH below 4.5 stimulates the secretion of secretin.
- Partially digested proteins in the duodenum stimulates G cells that secrete gastrin, which circulates to the stomach and speeds gastric processing.

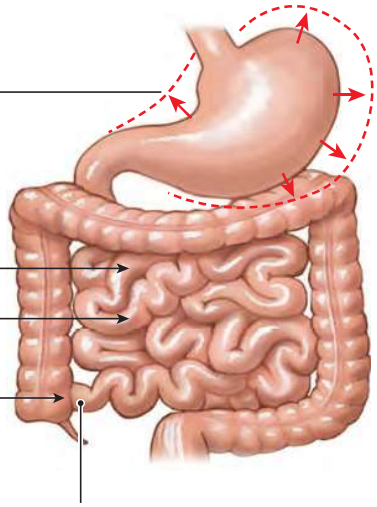
CENTRAL REFLEXES

Two central reflexes, the gastroenteric reflex and the gastroileal reflex, are also triggered by the stimulation of stretch receptors in the stomach wall as it fills. These reflexes accelerate movement along the small intestine while the enterogastric reflex controls the rate of chyme entry into the duodenum.

Central Gastric Reflexes

The **gastroenteric reflex** stimulates motility and secretion along the entire small intestine.

The **gastroileal** (gas-trō-IL-ē-al) **reflex** triggers the opening of the ileocecal valve, allowing materials to pass from the small intestine into the large intestine.

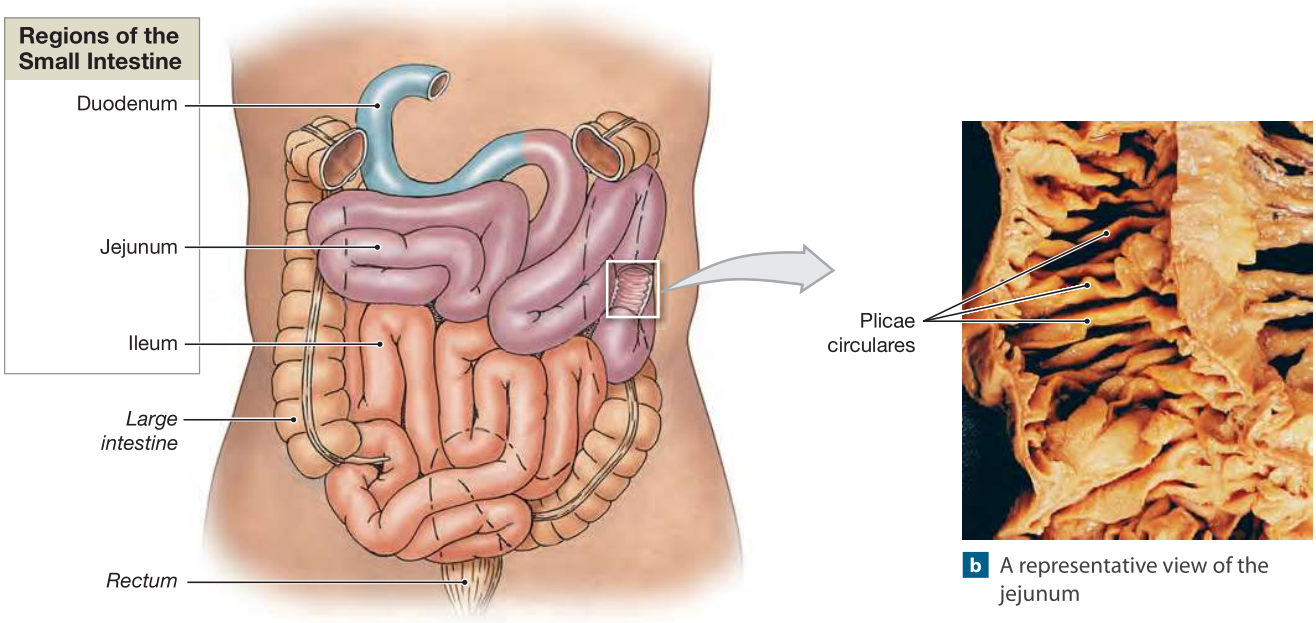


The **ileocecal valve** controls the passage of materials into the large intestine.

In general, the rate of movement of chyme into the small intestine is highest when the stomach is greatly distended and the meal contains little protein. A large meal containing small amounts of protein, large amounts of carbohydrates (such as rice or pasta), wine (alcohol), or after-dinner coffee (caffeine) will leave your stomach very quickly. One reason is that both alcohol and caffeine stimulate gastric secretion and motility.

The *vomiting* reflex occurs in response to irritation of the fauces, pharynx, esophagus, stomach, or proximal segment of the small intestine. These sensations are relayed to the vomiting center of the medulla oblongata, which coordinates motor responses. In preparation for vomiting, the pylorus relaxes and the contents of the duodenum are discharged into the stomach by strong peristaltic waves that travel toward the stomach. Vomiting, or **emesis** (EM-e-sis), then occurs as the stomach regurgitates its contents through the esophagus and pharynx.

Figure 24–16 **Segments of the Intestine.** ATLAS: Plates 49a,b,d; 51a,b



a The positions of the duodenum, jejunum, and ileum in the abdominopelvic cavity

b A representative view of the jejunum

In addition to capillaries and nerve endings, each villus contains a lymphatic capillary called a **lacteal** (LAK-tē-ul; *lacteus*, milky) (**Figure 24–17b,c**). Lacteals transport materials that cannot enter blood capillaries. For example, absorbed fatty acids are assembled into protein–lipid packages that are too large to diffuse into the bloodstream. These packets, called *chylomicrons*, reach the venous circulation through the thoracic duct, which delivers lymph into the left subclavian vein. The name *lacteal* refers to the pale, milky appearance of lymph that contains large quantities of lipids.

The intestinal villi move back and forth, exposing the epithelial surfaces to the liquefied intestinal contents. Contractions of the muscularis mucosae and smooth muscle cells within the villi bring about this movement. The movement makes absorption more efficient by quickly eliminating local differences in nutrient concentration. Movements of the villi also squeeze the lacteals, helping to move lymph out of the villi.

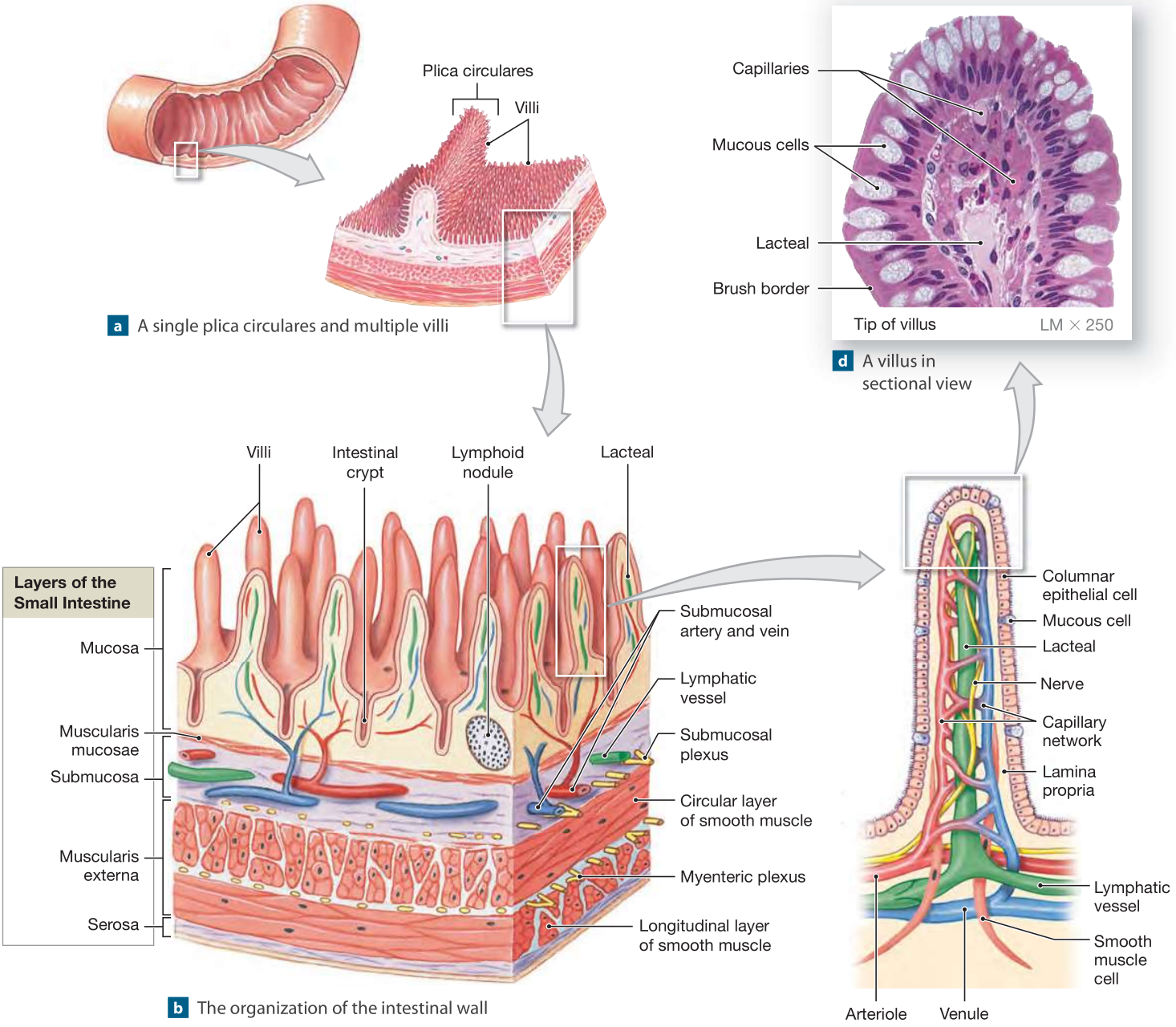
Intestinal Glands

Mucous cells between the columnar epithelial cells eject mucins onto the intestinal surfaces (**Figure 24–17c,d**). At the bases of the villi are the entrances to the **intestinal glands,** or *crypts of Lieberkühn*. These glandular pockets extend deep into the underlying lamina propria. Near the base of each intestinal gland, stem cell divisions produce new generations of epithelial cells, which are continuously displaced toward the intestinal surface. In a few days the new cells reach the tip of a villus and are shed into the intestinal lumen. This ongoing process renews the epithelial surface. The disintegration of the shed cells adds enzymes to the lumen.

The most important of the enzymes introduced into the lumen come from the apical portions of the intestinal cells. *Brush border enzymes* are integral membrane proteins on the surfaces of intestinal microvilli. These enzymes break down materials that come in contact with the brush border. The epithelial cells then absorb the breakdown products. Once the epithelial cells are shed, they disintegrate within the lumen, releasing both intracellular and brush border enzymes. *Enteropeptidase* (previously called *enterokinase*) is one brush border enzyme that enters the lumen in this way. It does not directly participate in digestion. Instead, it activates a key pancreatic proenzyme, trypsinogen. (We consider the functions of enteropeptidase and other brush border enzymes in a later section.) Intestinal glands also contain enteroendocrine cells that produce several intestinal hormones, including gastrin, cholecystokinin, and secretin.

The duodenum has numerous mucous glands, both in the epithelium and deep to it. In addition to intestinal glands, its submucosa contains **duodenal glands,** also called *submucosal glands* or *Brunner's glands*. Duodenal glands produce copious quantities of mucus when chyme arrives from the stomach. The mucus protects the epithelium from the acidity of chyme and also contains bicarbonate ions that help raise the pH of the chyme. As chyme travels the length of the duodenum, its pH increases from 1–2 to 7–8. The duodenal glands also secrete the hormone *urogastrone*, which inhibits gastric acid production and stimulates the division of epithelial stem cells along the digestive tract.

Figure 24–17 The Intestinal Wall. ATLAS: Plate 51a–d



Regional Specializations

The duodenum has few plicae circulares, and their villi are small. The primary function of the duodenum is to receive chyme from the stomach and neutralize its acids before they can damage the absorptive surfaces of the small intestine. Over the proximal half of the jejunum, however, plicae circulares and villi are very prominent. Thereafter, the plicae circulares and villi gradually decrease in size. This reduction parallels a reduction in absorptive activity: Most nutrient absorption takes place before ingested materials reach the ileum. One rather drastic surgical method of promoting weight loss is the removal of a significant portion of the jejunum. The resulting reduction in absorptive area causes a marked weight loss and may not interfere with adequate nutrition, but the side effects can be very serious.

The distal portions of the ileum lack plicae circulares. The lamina propria there contains 20–30 masses of lymphoid tissue

called aggregated lymphoid nodules, or *Peyer's patches*. These lymphoid tissues are most abundant in the terminal portion of the ileum, near the entrance to the large intestine. The lymphocytes in the aggregated lymphoid nodules protect the small intestine from bacteria that normally inhabit the large intestine.

Intestinal Secretions

Roughly 1.8 liters of watery **intestinal juice** enters the intestinal lumen each day. Intestinal juice moistens chyme, helps buffer acids, and keeps both the digestive enzymes and the products of digestion in solution. Much of this fluid arrives by osmosis, as water flows out of the mucosa and into the concentrated chyme. The rest is secreted by intestinal glands, stimulated by the activation of touch receptors and stretch receptors in the intestinal walls.

The duodenal glands help protect the duodenal epithelium from gastric acids and enzymes. These glands increase their secretion in response to (1) local reflexes, (2) the release of the hormone *enterocrinin* by enteroendocrine cells of the duodenum, and (3) parasympathetic stimulation through the vagus nerves. The first two mechanisms operate only after chyme arrives in the duodenum. However, the duodenal glands begin secreting during the cephalic phase of gastric secretion, long before chyme reaches the pyloric sphincter. They do so because vagus nerve activity triggers their secretion. Thus, the duodenal lining has protection in advance.

Sympathetic stimulation inhibits the duodenal glands, leaving the duodenal lining unprepared for the arrival of chyme. This effect probably explains why chronic stress or other factors that promote sympathetic activation can cause duodenal ulcers.

Intestinal Movements

After chyme has arrived in the duodenum, weak peristaltic contractions move it slowly toward the jejunum. The contractions are myenteric reflexes that are not under CNS control. Their effects are limited to within a few centimeters of the site of the original stimulus. Motor neurons in the submucosal and myenteric plexuses control these short reflexes. In addition, some of the smooth muscle cells contract periodically, even without stimulation, establishing a basic contractile rhythm that then spreads from cell to cell.

The stimulation of the parasympathetic system increases the sensitivity of the weak myenteric reflexes and speeds up both local peristalsis and segmentation. More elaborate reflexes coordinate activities along the entire length of the small intestine. The gastroenteric and gastroileal reflexes speed up movement along the small intestine (**Spotlight Figure 24–15**). This effect is the opposite from that of the enterogastric reflex.

Hormones released by the digestive tract can enhance or suppress reflexes. For example, the gastroileal reflex is triggered by stretch receptor stimulation. However, the degree of ileocecal valve relaxation is enhanced by gastrin, which is secreted in large quantities when food enters the stomach.

The Pancreas

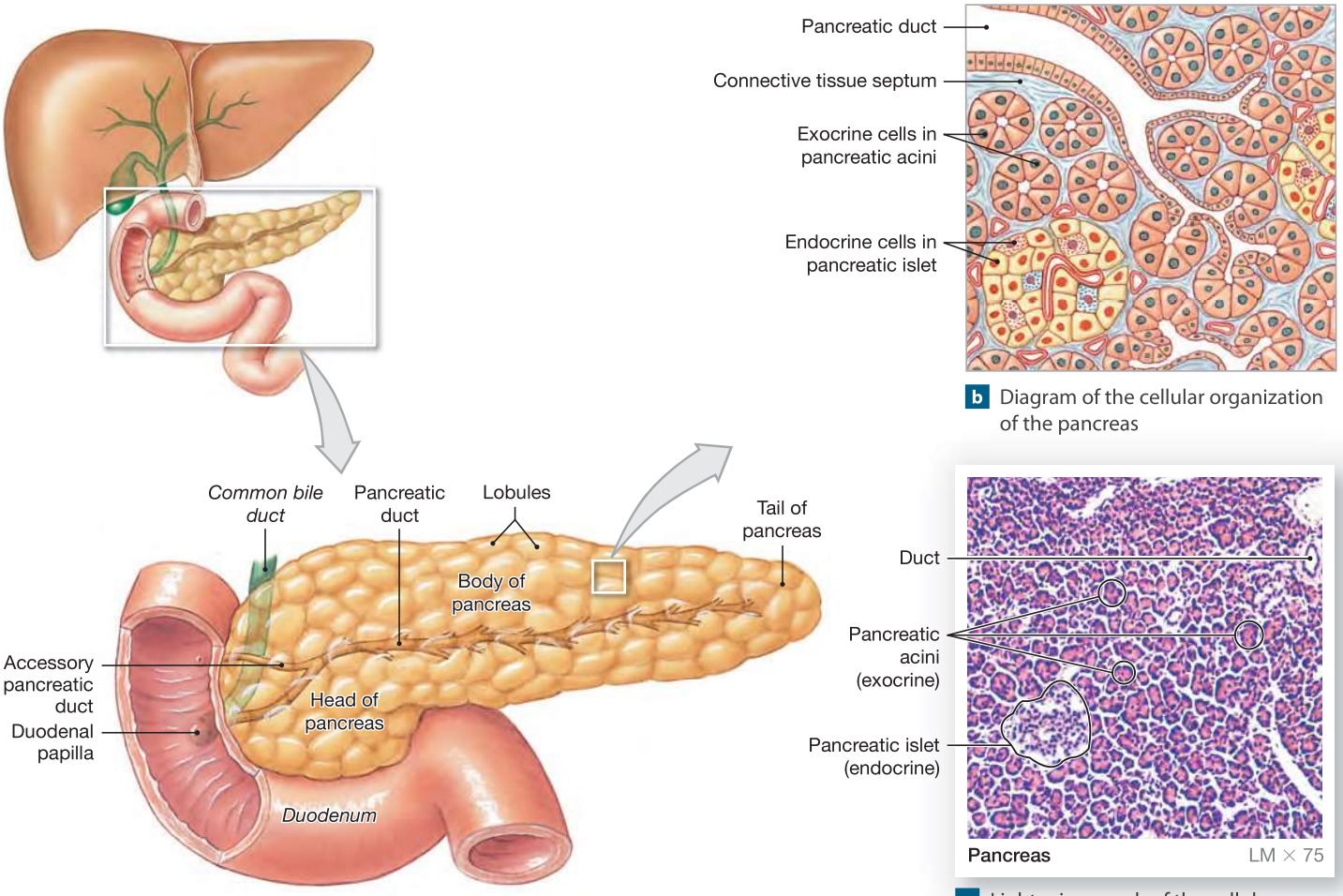
The **pancreas** lies posterior to the stomach. It extends laterally from the duodenum toward the spleen. The pancreas is an elongate, pinkish-gray organ about 15 cm (6 in.) long and weighing about 80 g (3 oz) (**Figure 24–18a**). The broad **head** of the pancreas lies within the loop formed by the duodenum as it leaves the pylorus. The slender **body** of the pancreas extends toward the spleen, and the **tail** is short and bluntly rounded. The pancreas is retroperitoneal and is firmly bound to the posterior wall of the abdominal cavity. The surface of the pancreas has a lumpy, lobular texture. A thin, transparent capsule of connective tissue wraps the entire organ. The pancreatic lobules, associated blood vessels, and excretory ducts are visible through the anterior capsule and the overlying layer of peritoneum. Arterial blood reaches the pancreas by way of branches of the splenic, superior mesenteric, and common hepatic arteries. The pancreatic arteries and pancreaticoduodenal arteries are the major branches from these vessels. The splenic vein and its branches drain the pancreas.

The pancreas is primarily an exocrine organ. It produces digestive enzymes and buffers. The large **pancreatic duct** *(duct of Wirsung)* delivers these secretions to the duodenum. (In 3–10 percent of the population, a small **accessory pancreatic duct** [*duct of Santorini*] branches from the pancreatic duct.) The pancreatic duct extends within the attached mesentery to reach

Clinical Note

Pancreatitis *Pancreatitis* (pan-krē-a-TĪ-tis) is an inflammation of the pancreas. It is an extremely painful condition. A blockage of the excretory ducts, bacterial or viral infections, ischemia, and drug reactions, especially those involving alcohol, are among the factors that may produce it. These stimuli provoke a crisis by injuring exocrine cells in at least a portion of the organ. Then lysosomes in the damaged cells activate the proenzymes, and autolysis begins. The proteolytic enzymes digest the surrounding, undamaged cells, activating their enzymes and starting a chain reaction. In most cases, only a portion of the pancreas is affected, and the condition subsides in a few days. In 10–15 percent of cases, the process does not subside. The enzymes can then ultimately destroy the pancreas. If the islet cells are damaged, diabetes mellitus may result. ↩ p. 622

Figure 24–18 The Pancreas. ATLAS: Plates 54d; 55; 57a



b Diagram of the cellular organization of the pancreas

a The gross anatomy of the pancreas. The head of the pancreas is tucked in to a C-shaped curve of the duodenum that begins at the pylorus of the stomach.

c Light micrograph of the cellular organization of the pancreas

the duodenum, where it meets the *common bile duct* from the liver and gallbladder (**Figure 24–21b**). The two ducts then empty into the *duodenal ampulla (ampulla of Vater)*, a chamber located roughly halfway along the length of the duodenum. When present, the accessory pancreatic duct usually empties into the duodenum independently, outside the duodenal ampulla.

Histological Organization

Partitions of connective tissue divide the interior of the pancreas into distinct lobules. The blood vessels and tributaries of the pancreatic ducts are situated within these connective tissue septa (**Figure 24–18b**). The pancreas is an example of a *compound tubuloalveolar gland*, a structure described in Chapter 4. p. 120 In each lobule, the ducts branch repeatedly before ending in blind pockets called **pancreatic acini** (AS-i-nī). Each pancreatic acinus is lined with simple cuboidal epithelium. *Pancreatic islets*, the endocrine tissues of the pancreas, are scattered among the acini (**Figure 24–18b,c**). The islets account for only about 1 percent of the cell population of the pancreas.

The pancreas has two distinct functions, one endocrine and the other exocrine. The endocrine cells of the pancreatic islets secrete insulin and glucagon into the bloodstream to control blood sugar. The exocrine cells include the acinar cells and the epithelial cells that line the duct system. Together, these exocrine cells secrete **pancreatic juice**—an alkaline mixture of digestive enzymes, water, and ions—into the small intestine. Acinar cells secrete pancreatic enzymes, which do most of the digestive work in the small intestine. Pancreatic enzymes break down ingested materials into small molecules suitable for absorption. The water and ions, secreted primarily by the cells lining the pancreatic ducts, help dilute and neutralize acid in the chyme.

Physiology of the Pancreas

Each day, the pancreas secretes about 1000 mL (1 qt) of pancreatic juice. Hormones from the duodenum control these secretory activities. When chyme arrives in the duodenum, secretin is released. This hormone triggers the pancreatic secretion of a watery buffer solution with a pH of 7.5–8.8. Among its other components, the secretion contains bicarbonate and phosphate buffers that help raise the pH of the chyme.

Another duodenal hormone, cholecystokinin, stimulates the production and secretion of pancreatic enzymes. Stimulation by the vagus nerves also increases the secretion of pancreatic enzymes. Recall that this stimulation takes place during the cephalic phase of gastric regulation, so the pancreas starts to synthesize enzymes before food even reaches the stomach. This head start is important, because enzyme synthesis takes much longer than the production of buffers. By starting early, the pancreatic cells are ready to meet the demand when chyme arrives in the duodenum.

The pancreatic enzymes include the following:

- **Pancreatic alpha-amylase,** a **carbohydrase** (kar-bō-HĪ-drās)—an enzyme that breaks down certain starches. Pancreatic alpha-amylase is almost identical to salivary amylase.
- **Pancreatic lipase,** which breaks down certain complex lipids, releasing products (such as fatty acids) that can be easily absorbed.
- **Nucleases,** which break down RNA or DNA.
- **Proteolytic enzymes,** which break apart certain proteins. These enzymes include **proteases,** which break apart large protein complexes, and **peptidases,** which break small peptide chains into individual amino acids.

Proteolytic enzymes account for about 70 percent of total pancreatic enzyme production. These enzymes are secreted as inactive proenzymes. They are activated only after they reach the small intestine. Proenzymes discussed earlier include pepsinogen, angiotensinogen, plasminogen, fibrinogen, and many of the clotting factors and enzymes of the complement system. ↩ pp. 624, 642, 665, 782 As in the stomach, the release of a proenzyme rather than an active enzyme protects the secretory cells from the destructive effects of their own products. Among the proenzymes secreted by the pancreas are **trypsinogen** (trip-SIN-ō-jen), **chymotrypsinogen** (kī-mo-trip-SIN-ō-jen), **procarboxypeptidase** (prō-kar-bok-sē-PEP-ti-dās), and **proelastase** (pro-ē-LAS-tās).

Inside the duodenum, enteropeptidase in the brush border and the lumen triggers the conversion of trypsinogen to **trypsin,** an active protease. Trypsin then activates the other proenzymes, producing **chymotrypsin, carboxypeptidase,** and **elastase.** Each enzyme attacks peptide bonds linking specific amino acids and ignores others. Together, these enzymes break down proteins into a mixture of dipeptides, tripeptides, and amino acids.

The Liver

The **liver** is the largest visceral organ. It is one of our most versatile organs and the center for metabolic regulation in the body. Most of its mass lies in the right hypochondriac and epigastric regions, but it may extend into the left hypochondriac and umbilical regions as well. The liver weighs about 1.5 kg (3.3 lb). This large, firm, reddish-brown organ performs essential metabolic and synthetic functions.

Anatomy of the Liver

The liver is wrapped in a tough fibrous capsule and is covered by a layer of visceral peritoneum. On the anterior surface, the **falciform ligament** marks the division between the organ's left and right lobes (**Figure 24–19a,b**). A thickening in the posterior margin of the falciform ligament is the **round ligament,** or *ligamentum teres*. This fibrous band marks the path of the fetal umbilical vein.

On the posterior surface of the liver, the impression left by the inferior vena cava marks the division between the right lobe and the small **caudate** (KAW-dāt) **lobe** (**Figure 24–19a,c**). Inferior to the caudate lobe lies the **quadrate lobe,** sandwiched between the left lobe and the gallbladder. Afferent blood vessels and other structures reach the liver by traveling within the connective tissue of the lesser omentum. They converge at a region called the **porta hepatis** ("doorway to the liver").

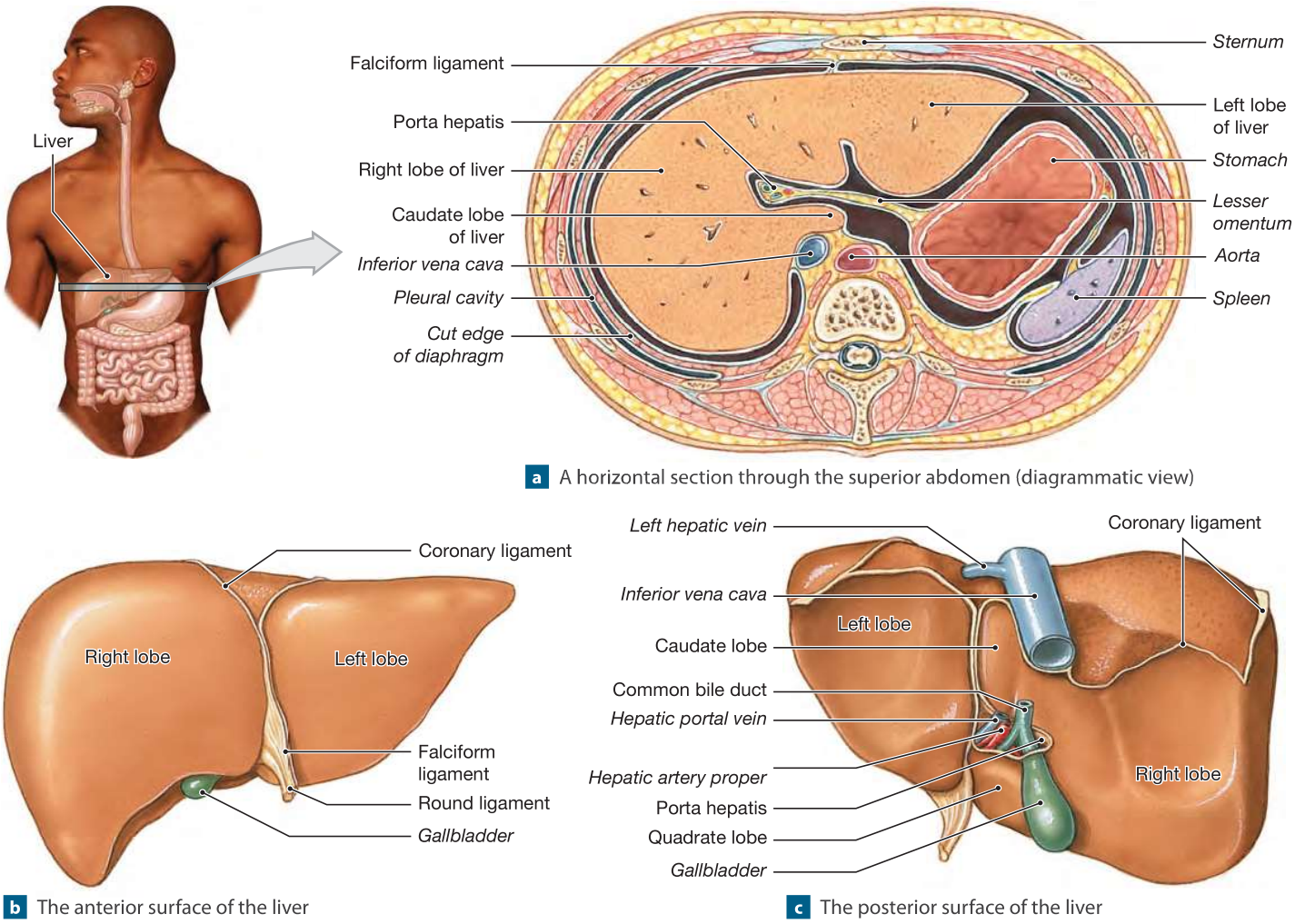
We discussed the circulation to the liver in Chapter 21 and summarized it in **Figures 21–26** and **21–33**, pp. 746, 754. Roughly one-third of the blood supply to the liver is arterial blood from the hepatic artery proper. The rest is venous blood from the hepatic portal vein, which begins in the capillaries of the esophagus, stomach, small intestine, and most of the large intestine. Liver cells, called **hepatocytes** (HEP-a-tō-sīts), adjust circulating levels of nutrients through selective absorption and secretion. The blood leaving the liver returns to the systemic circuit through the hepatic veins. These veins open into the inferior vena cava.

Histological Organization of the Liver

Connective tissue divides each lobe of the liver into approximately 100,000 **liver lobules,** the basic functional units of the liver. The histological organization and structure of a typical liver lobule are shown in **Figure 24–20.**

Each lobule is roughly 1 mm in diameter. Adjacent lobules are separated by an *interlobular septum*. The hepatocytes in a liver lobule form a series of irregular plates arranged like the spokes of a wheel (**Figure 24–20a,b**). The plates are only one cell thick. Exposed hepatocyte surfaces are covered with short microvilli. Within a lobule, sinusoids between adjacent plates empty into

Figure 24–19 **The Anatomy of the Liver.** ATLAS: Plates 49a,b,e; 54a–c; 57a,b



the **central vein.** (We introduced sinusoids in Chapter 21. ↩ p. 713) The liver sinusoids lack a basement membrane, so large openings between the endothelial cells allow solutes—even those as large as plasma proteins—to pass out of the bloodstream and into the spaces surrounding the hepatocytes.

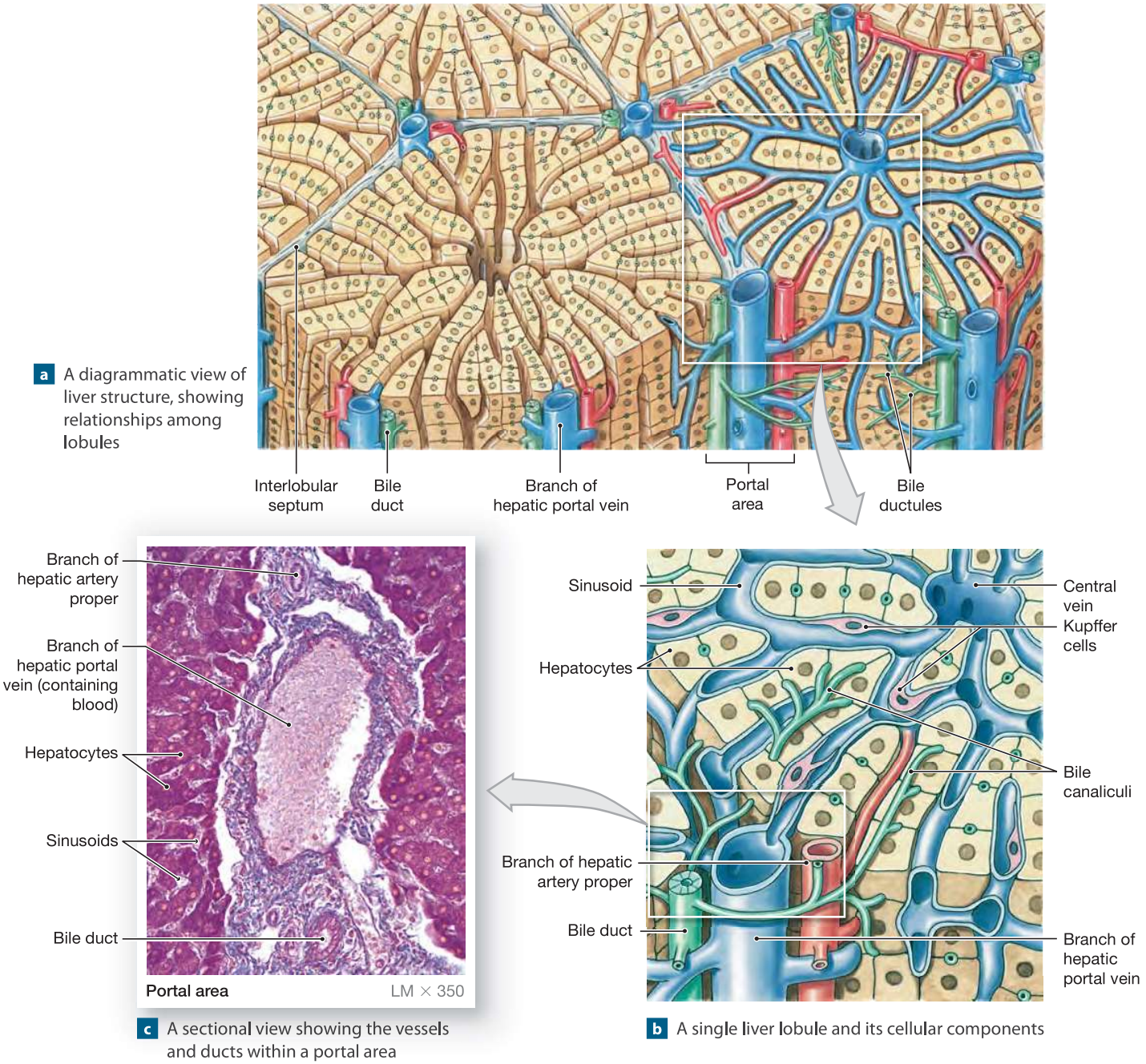
The lining of the sinusoids contains typical endothelial cells and a large number of **Kupffer** (KOOP-fer) **cells,** also known as *stellate reticuloendothelial cells*. ↩ p. 780 These phagocytic cells are part of the monocyte–macrophage system. They engulf pathogens, cell debris, and damaged blood cells. Kupffer cells also store iron, some lipids, and heavy metals (such as tin or mercury) that are absorbed by the digestive tract.

Blood enters the liver sinusoids from small branches of the hepatic portal vein and hepatic artery proper. A typical liver lobule has a hexagonal shape in cross section (**Figure 24–20a**). There are six **portal areas,** or *portal triads*, one at each corner of the lobule. A portal area contains three structures: (1) a branch of the hepatic portal vein, (2) a branch of the hepatic artery proper, and (3) a small branch of the bile duct (**Figure 24–20a–c**).

Branches from the arteries and veins deliver blood to the sinusoids of adjacent liver lobules (**Figure 24–20a,b**). As blood flows through the sinusoids, hepatocytes absorb solutes from the plasma and secrete materials such as plasma proteins. Blood then leaves the sinusoids and enters the central vein of the lobule. The central veins ultimately merge to form the hepatic veins, which then empty into the inferior vena cava. Liver diseases, such as the various forms of *hepatitis*, and conditions such as alcoholism, can lead to degenerative changes in the liver tissue and constriction of the circulatory supply.

Pressures in the hepatic portal system are usually low, averaging 10 mm Hg or less. This pressure can increase markedly, however, if blood flow through the liver is restricted by a blood clot or damage to the organ. Such a rise in portal pressure is called *portal hypertension*. As pressures rise, small peripheral veins and capillaries in the portal system become

Figure 24–20 Liver Histology.

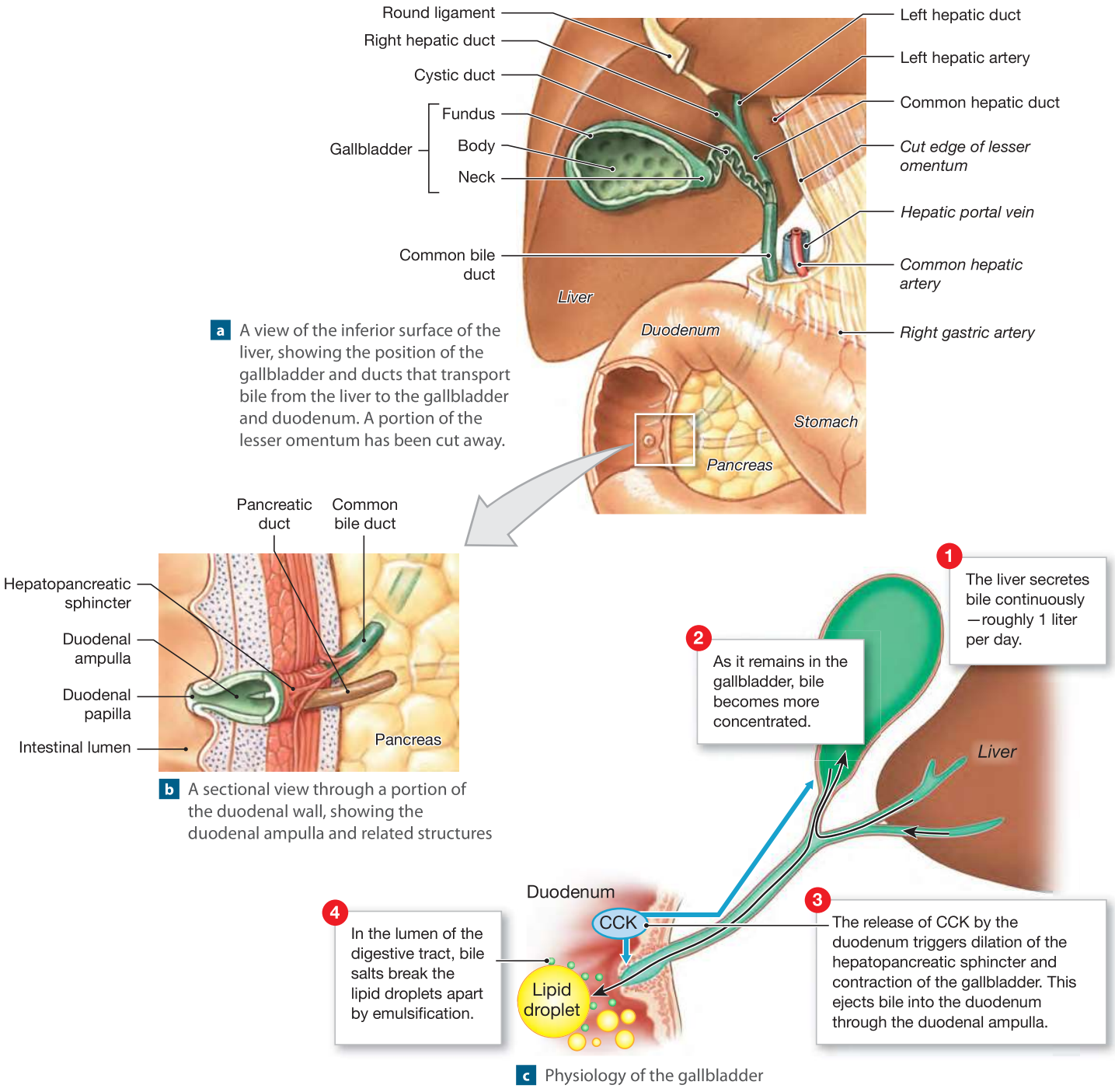


distended. If they rupture, extensive bleeding can take place. Portal hypertension can also force fluid into the peritoneal cavity across the serosal surfaces of the liver and viscera, producing ascites (p. 864).

The Bile Duct System

The liver secretes a fluid called **bile** into a network of narrow channels between the opposing membranes of adjacent liver cells. These passageways, called **bile canaliculi,** extend outward, away from the central vein (**Figure 24–20b**). Eventually, they connect with fine **bile ductules** (DUK-tūlz), which carry bile to bile ducts in the nearest portal area (**Figure 24–20a**). The **right** and **left hepatic ducts** (**Figure 24–21a**) collect bile from all the bile ducts of the liver lobes. These ducts unite to form the **common hepatic duct,** which leaves the liver. The bile in the common hepatic duct either flows into the *common bile duct,* which empties into the duodenal ampulla, or enters the *cystic duct,* which leads to the gallbladder.

Figure 24–21 The Anatomy and Physiology of the Gallbladder and Bile Ducts. ATLAS: Plates 49c,e; 51a; 54b–d



The **common bile duct** is formed by the union of the **cystic duct** and the common hepatic duct. The common bile duct passes within the lesser omentum toward the stomach, turns, and penetrates the wall of the duodenum to meet the pancreatic duct at the duodenal ampulla (**Figure 24–21b**).

The Physiology of the Liver

The liver carries out more than 200 functions. They fall into three general categories: (1) *metabolic regulation*, (2) *hematological regulation*, and (3) *bile production*. In this discussion we provide a general overview.

Clinical Note

Cirrhosis Any condition that severely damages the liver represents a serious threat to life. The liver has a limited ability to regenerate itself after injury, but liver function does not fully recover unless the normal vascular pattern is restored. Examples of important types of liver disease include **cirrhosis,** which is characterized by the replacement of lobules by fibrous tissue, and various forms of *hepatitis* caused by viral infections. In some cases, liver transplants are used to treat liver failure, but the supply of suitable donor tissue is limited. The success rate is highest in young, otherwise healthy individuals. Clinical trials are now under way to test an artificial liver known as *ELAD* (*e*xtracorporeal *l*iver *a*ssist *d*evice) that may prove suitable for the long-term support of individuals with chronic liver disease.

Metabolic Regulation. The liver is the primary organ involved in regulating the composition of circulating blood. All blood leaving the absorptive surfaces of the digestive tract enters the hepatic portal system and flows into the liver. Liver cells extract nutrients or toxins from the blood before it reaches the systemic circulation through the hepatic veins. The liver removes and stores excess nutrients. It corrects nutrient deficiencies by mobilizing stored reserves or performing synthetic activities. The liver's regulatory activities affect the following:

- *Carbohydrate Metabolism.* The liver stabilizes blood glucose levels at about 90 mg/dL. If blood glucose levels drop, hepatocytes break down glycogen reserves and release glucose into the bloodstream. They also synthesize glucose from other carbohydrates or from available amino acids. The synthesis of glucose from other compounds is called *gluconeogenesis*. If blood glucose levels climb, hepatocytes remove glucose from the bloodstream. They either store it as glycogen or use it to synthesize lipids that can be stored in the liver or other tissues. Circulating hormones, such as insulin and glucagon, regulate these metabolic activities. ↩ pp. 620–622
- *Lipid Metabolism.* The liver regulates circulating levels of triglycerides, fatty acids, and cholesterol. When those levels decline, the liver breaks down its lipid reserves and releases the breakdown products into the bloodstream. When the levels are high, the lipids are removed for storage. However, this regulation takes place only after lipid levels have risen within the general circulation, because most lipids absorbed by the digestive tract bypass the hepatic portal circulation.
- *Amino Acid Metabolism.* The liver removes excess amino acids from the bloodstream. These amino acids can be used to synthesize proteins or can be converted to lipids or glucose for energy storage.
- *Waste Product Removal.* When converting amino acids to lipids or carbohydrates, or when breaking down amino acids to get energy, the liver strips off the amino groups. This process is called *deamination*. Ammonia, a toxic waste product, is formed. The liver neutralizes ammonia by converting it to *urea*, a fairly harmless compound excreted by the kidneys. The liver also removes other waste products, circulating toxins, and drugs from the blood for inactivation, storage, or excretion.
- *Vitamin Storage.* Fat-soluble vitamins (A, D, E, and K) and vitamin B_{12} are absorbed from the blood and stored in the liver. These reserves are used when your diet contains inadequate amounts of those vitamins.
- *Mineral Storage.* The liver converts iron reserves to ferritin and stores this protein–iron complex. ↩ p. 648
- *Drug Inactivation.* The liver removes and breaks down circulating drugs, limiting the duration of their effects. When physicians prescribe a particular drug, they must take into account the rate at which the liver removes that drug from the bloodstream. For example, a drug that is absorbed relatively quickly must be administered every few hours to keep plasma concentrations at therapeutic levels.

Hematological Regulation. The liver, the largest blood reservoir in your body, receives about 25 percent of cardiac output. As blood passes through it, the liver performs the following functions:

- *Phagocytosis and Antigen Presentation.* Kupffer cells in the liver sinusoids engulf old or damaged red blood cells, cellular debris, and pathogens, removing them from the bloodstream. Kupffer cells are antigen-presenting cells (APCs) that can stimulate an immune response. ↩ p. 780
- *Synthesis of Plasma Proteins.* Hepatocytes synthesize and release most of the plasma proteins. These proteins include the albumins (which contribute to the osmotic concentration of the blood), the various types of transport proteins, clotting proteins, and complement proteins.
- *Removal of Circulating Hormones.* The liver is the primary site for the absorption and recycling of epinephrine, norepinephrine, insulin, thyroid hormones, and steroid hormones, such as the sex hormones (estrogens and androgens) and corticosteroids. The liver also absorbs cholecalciferol (vitamin D_3) from the blood. Liver cells then convert the cholecalciferol, which may be synthesized in the skin or absorbed in the diet, into an intermediary product, 25-hydroxy-D_3, that is released back into the bloodstream. The kidneys absorb this intermediary and use it to generate calcitriol, a hormone important to Ca^{2+} metabolism. ↩ p. 624
- *Removal of Antibodies.* The liver absorbs and breaks down antibodies, releasing amino acids for recycling.

- *Removal or Storage of Toxins.* The liver absorbs lipid-soluble toxins in the diet, such as the insecticide DDT, and stores them in lipid deposits, where they do not disrupt cellular functions. The liver removes other toxins from the bloodstream and either breaks them down or excretes them in the bile.
- *The Synthesis and Secretion of Bile.* The liver synthesizes bile and excretes it into the lumen of the duodenum. Hormonal and neural mechanisms regulate bile secretion. Bile consists mostly of water, with minor amounts of ions, *bilirubin* (a pigment derived from hemoglobin), cholesterol, and an assortment of lipids collectively known as **bile salts.** (Bile salts play a role in the digestion of lipids, as we discuss in the next section.) The water and ions help dilute and buffer acids in chyme as it enters the small intestine. Bile salts are synthesized from cholesterol in the liver. Several related compounds are involved. The most abundant are derivatives of the steroids *cholate* and *chenodeoxycholate*.

The Functions of Bile. Most dietary lipids are not water soluble. Mechanical processing in the stomach creates large drops containing a variety of lipids. Pancreatic lipase is not lipid soluble, so the enzymes can interact with lipids only at the surface of a lipid droplet. The larger the droplet, the more lipids are inside, isolated and protected from these enzymes. Bile salts break the droplets apart in a process called **emulsification** (ē-mul-si-fi-KĀ-shun), which dramatically increases the surface area accessible to enzymes.

Emulsification creates tiny *emulsion droplets* with a superficial coating of bile salts. The formation of tiny droplets increases the surface area available for enzymatic attack. In addition, the layer of bile salts facilitates interaction between the lipids and lipid-digesting enzymes from the pancreas.

After lipid digestion has been completed, bile salts promote the absorption of lipids by the intestinal epithelium. More than 90 percent of the bile salts are themselves reabsorbed, primarily in the ileum, as lipid digestion is completed. The reabsorbed bile salts enter the hepatic portal circulation. The liver then collects and recycles them. The cycling of bile salts from the liver to the small intestine and back is called the **enterohepatic circulation of bile.**

Tips & Tricks

Oil and water ordinarily don't mix. The emulsification process helps mix oils and water-soluble enzymes so that fats can be broken down.

The Gallbladder

The **gallbladder** is a hollow, pear-shaped organ that stores and concentrates bile prior to its excretion into the small intestine. This muscular sac is located in a fossa, or recess, in the posterior surface of the liver's right lobe (**Figure 24–21a**). The gallbladder is divided into three regions: (1) the **fundus,** (2) the **body,** and (3) the **neck.** The cystic duct extends from the gallbladder to the point where it unites with the common hepatic duct to form the common bile duct. At the duodenum, the common bile duct meets the pancreatic duct before emptying into a chamber called the **duodenal ampulla** (am-PUL-luh) (**Figure 24–21b**), which receives buffers and enzymes from the pancreas and bile from the liver and gallbladder. The duodenal ampulla opens into the duodenum at the **duodenal papilla,** a small mound.

The muscular **hepatopancreatic sphincter** *(sphincter of Oddi)* encircles the lumen of the common bile duct and, generally, the pancreatic duct and duodenal ampulla as well.

Physiology of the Gallbladder

A major function of the gallbladder is *bile storage*, but it is released into the duodenum only under the stimulation of the intestinal hormone CCK (**Figure 24–21c**). Without CCK, the hepatopancreatic sphincter remains closed, so bile exiting the liver in the common hepatic duct cannot flow through the common bile duct and into the duodenum. Instead, it enters the cystic duct and is stored within the expandable gallbladder. Whenever chyme enters the duodenum, CCK is released, relaxing the hepatopancreatic sphincter and stimulating contractions of the gallbladder that push bile into the small intestine. The amount of CCK secreted increases markedly when the chyme contains large amounts of lipids.

The gallbladder also functions in *bile modification*. When full, the gallbladder contains 40–70 mL of bile. The composition of bile gradually changes as it remains in the gallbladder: Much of the water is absorbed, and the bile salts and other components of bile become increasingly concentrated.

If bile becomes too concentrated, crystals of insoluble minerals and salts begin to form. These deposits are called *gallstones*. Small gallstones are not a problem so long as they can be flushed down the bile duct and excreted. In *cholecystitis* (kō-lē-sis-TĪ-tis; *chole*, bile + *kystis*, bladder + *itis*, inflammation), the gallstones are so large that they can damage the wall of the gallbladder or block the cystic duct or common bile duct. In that case, the gallbladder may need to be surgically removed. This does not seriously impair digestion, because bile production continues at normal levels. However, the bile is more dilute, and its entry into the small intestine is not as closely tied to the arrival of food in the duodenum.

The Coordination of Secretion and Absorption

A combination of neural and hormonal mechanisms coordinates the activities of the digestive glands. These regulatory mechanisms are centered on the duodenum, where acids must be neutralized and appropriate enzymes added.

Neural mechanisms involving the CNS prepare the digestive tract for activity (through parasympathetic innervation) or inhibit its activity (through sympathetic innervation). Neural mechanisms also coordinate the movement of materials along the length of the digestive tract (through the enterogastric, gastroenteric, and gastroileal reflexes).

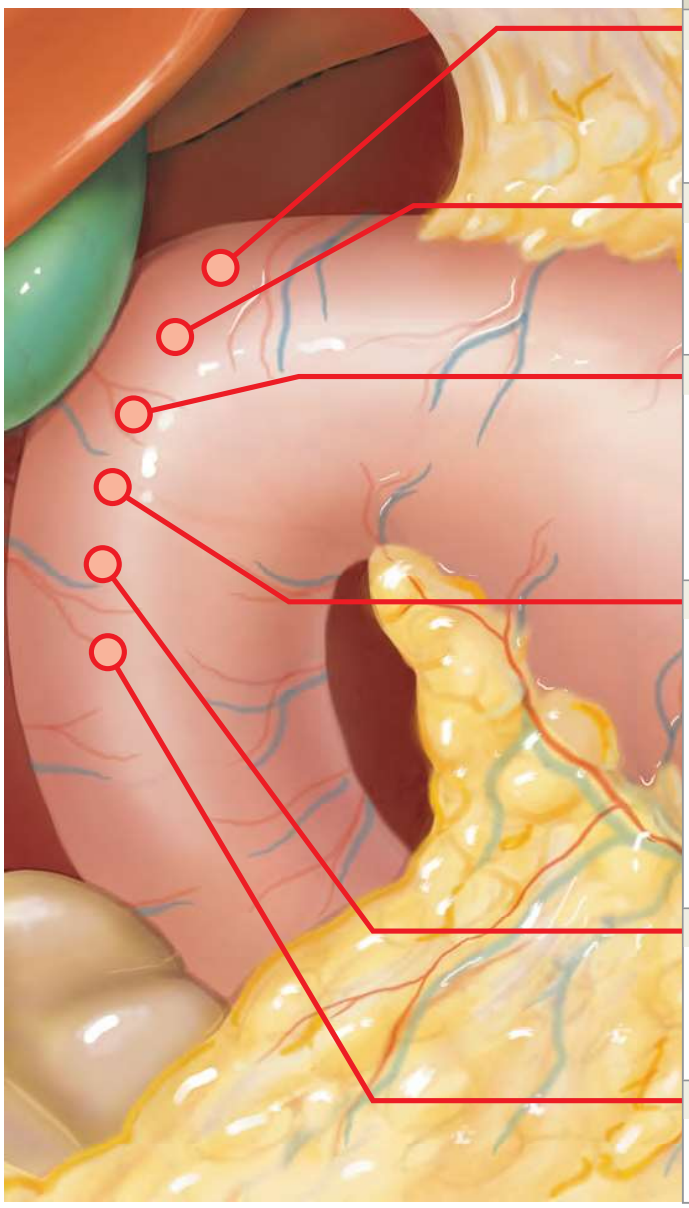
In addition, motor neurons synapsing in the digestive tract release a variety of neurotransmitters. Many of these chemicals are also released in the CNS. In general, their functions are poorly understood. Examples of neurotransmitters that may be important include substance P, enkephalins, and endorphins.

We will now summarize the information presented thus far on the regulation of intestinal and glandular function. We will also consider some additional details about the regulatory mechanisms involved.

Intestinal Hormones

The intestinal tract secretes a variety of peptide hormones with similar chemical structures. Many of these hormones have multiple effects in several regions of the digestive tract, and in the accessory glandular organs as well. The origins and primary effects of these important digestive hormones are shown in **Figure 24–22**.

Figure 24–22 Major Duodenal Hormones.



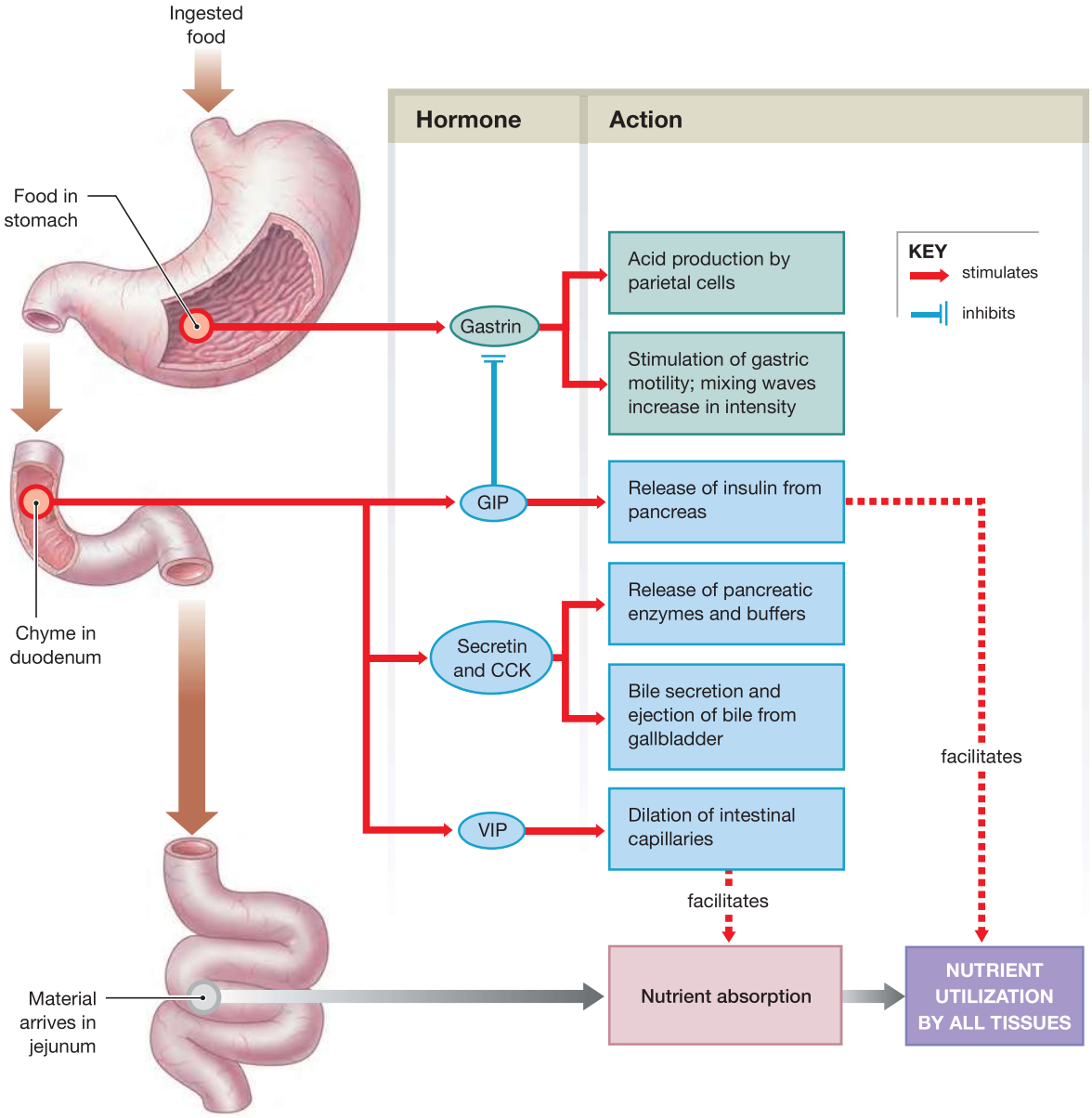
Major Duodenal Hormones
Gastrin
Gastrin is secreted by G cells in the duodenum when they are exposed to large quantities of incompletely digested proteins. The functions of gastrin include promoting increased stomach motility and stimulating the production of gastric acids and enzymes. (Gastrin is also produced by the stomach.)
Secretin
Secretin is released when chyme arrives in the duodenum. Secretin's primary effect is an increase in the secretion of bile (by the liver) and buffers (by the pancreas), which in turn act to increase the pH of the chyme. Among its secondary effects, secretin reduces gastric motility and secretory rates.
Gastric Inhibitory Peptide (GIP)
Gastric inhibitory peptide is secreted when fats and carbohydrates—especially glucose—enter the small intestine. The inhibition of gastric activity is accompanied by the stimulation of insulin release at the pancreatic islets. GIP has several secondary effects, including stimulating duodenal gland activity, stimulating lipid synthesis in adipose tissue, and increasing glucose use by skeletal muscles.
Cholecystokinin (CCK)
Cholecystokinin is secreted when chyme arrives in the duodenum, especially when the chyme contains lipids and partially digested proteins. In the pancreas, CCK accelerates the production and secretion of all types of digestive enzymes. It also causes a relaxation of the hepatopancreatic sphincter and contraction of the gallbladder, resulting in the ejection of bile and pancreatic juice into the duodenum. Thus, the net effects of CCK are to increase the secretion of pancreatic enzymes and to push pancreatic secretions and bile into the duodenum. The presence of CCK in high concentrations has two additional effects: It inhibits gastric activity, and it appears to have CNS effects that reduce the sensation of hunger.
Vasoactive Intestinal Peptide (VIP)
Vasoactive intestinal peptide stimulates the secretion of intestinal glands, dilates regional capillaries, and inhibits acid production in the stomach. By dilating capillaries in active areas of the intestinal tract, VIP provides an efficient mechanism for removing absorbed nutrients.
Enterocrinin
Enterocrinin is released when chyme enters the duodenum. It stimulates mucin production by the submucosal glands.

Other intestinal hormones are produced in relatively small quantities. Examples include *motilin*, which stimulates intestinal contractions; *villikinin*, which promotes the movement of villi and the associated lymph flow; and *somatostatin*, which inhibits gastric secretion. Functional interactions among gastrin, GIP, secretin, CCK, and VIP are diagrammed in **Figure 24–23**.

Intestinal Absorption

On average, it takes about five hours for materials to pass from the duodenum to the end of the ileum. The first of the materials to enter the duodenum after you eat breakfast may leave the small intestine at lunchtime. Along the way, the organ's absorptive effectiveness is enhanced by the fact that so much of the mucosa is movable. The microvilli can be moved by their supporting microfilaments, the individual villi by smooth muscle cells, groups of villi by the muscularis mucosae, and the plicae circulares by the muscularis mucosae and the muscularis externa. These movements stir and mix the intestinal contents, changing the environment around each epithelial cell from moment to moment.

Figure 24–23 The Activities of Major Digestive Tract Hormones. The primary actions of gastrin, GIP, secretin, CCK, and VIP are depicted.



Checkpoint

21. Name the three regions of the small intestine from proximal to distal.
22. How is the small intestine adapted for the absorption of nutrients?
23. Does a high-fat meal raise or lower the level of cholecystokinin in the blood?
24. How would the pH of the intestinal contents be affected if the small intestine did not produce secretin?
25. The digestion of which nutrient would be most impaired by damage to the exocrine pancreas?

See the blue Answers tab at the back of the book.

24-7 The large intestine is divided into three parts with regional specialization

The horseshoe-shaped **large intestine** begins at the end of the ileum and ends at the anus. The large intestine lies inferior to the stomach and liver and almost completely frames the small intestine (**Figure 24–1**). The large intestine stores digestive wastes and reabsorbs water. Bacteria in the large intestine are an important source of vitamins, especially vitamin K, biotin, and vitamin B_5.

The large intestine, also known as the *large bowel*, has an average length of about 1.5 meters (4.9 ft) and a width of 7.5 cm (3 in.). We can divide it into three parts: (1) the pouchlike *cecum*, the first portion of the large intestine; (2) the *colon*, the largest portion; and (3) the *rectum*, the last 15 cm (6 in.) of the large intestine and the end of the digestive tract (**Figure 24–24a**).

The Cecum

Material arriving from the ileum first enters an expanded pouch called the **cecum** (SĒ-kum). The ileum attaches to the medial surface of the cecum and opens into the cecum at the *ileocecal valve* (**Figure 24–24a,b**). The cecum collects and stores materials from the ileum and begins the process of compaction.

The slender, hollow **appendix,** or *vermiform appendix* (*vermis*, worm), is attached to the posteromedial surface of the cecum (**Figure 24–24a,b**). The appendix is normally about 9 cm (3.6 in.) long, but its size and shape are quite variable. A small mesentery called the **mesoappendix** connects the appendix to the ileum and cecum. Lymphoid nodules dominate the mucosa and submucosa of the appendix. The primary function of the appendix is as an organ of the lymphoid system. Inflammation of the appendix is known as *appendicitis*.

The Colon

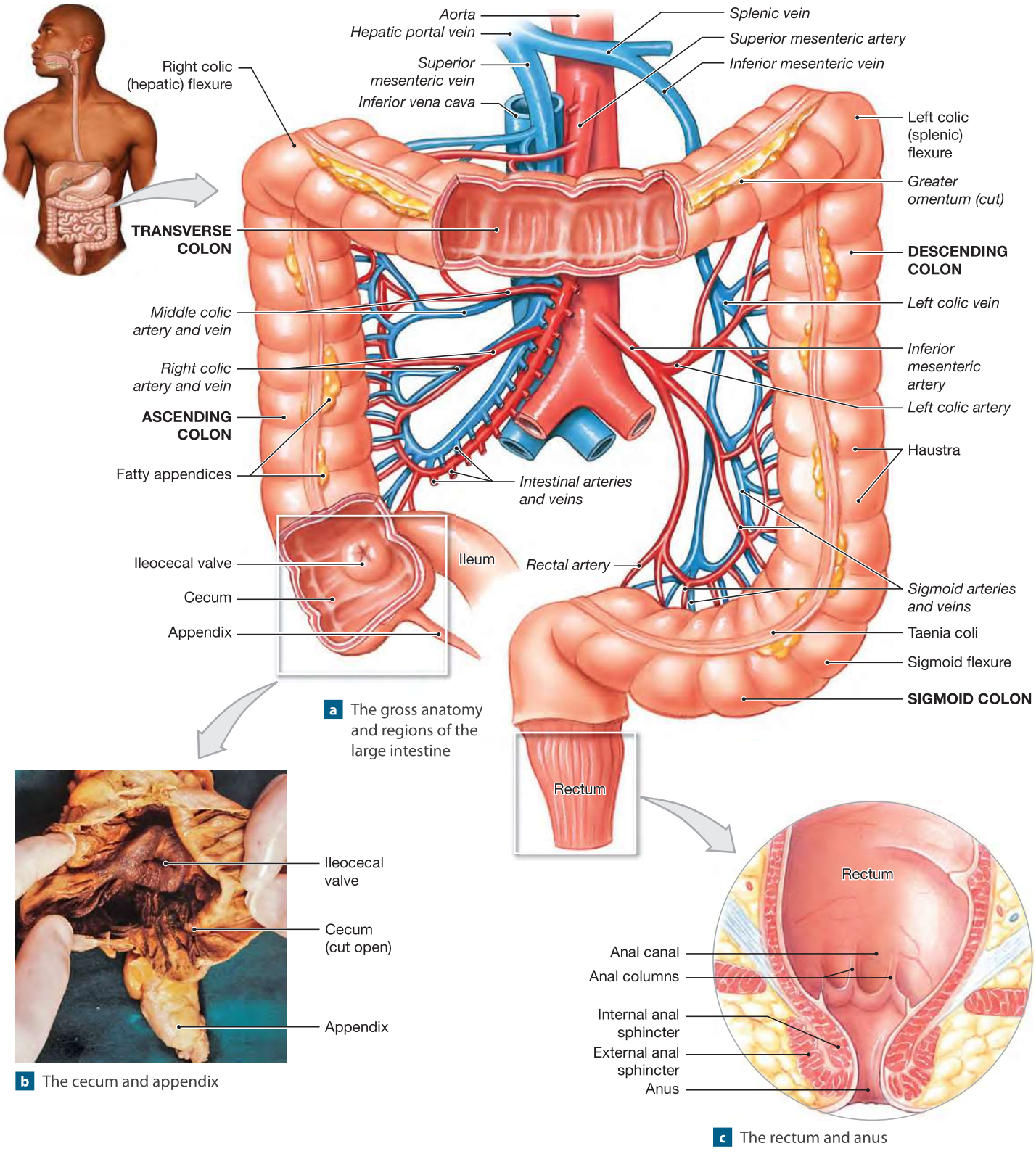
The **colon** has a larger diameter and a thinner wall than the small intestine. Distinctive features of the colon include the following (**Figure 24–24a**):

- The wall of the colon forms a series of pouches, or **haustra** (HAWS-truh; singular, *haustrum*). The creases between the haustra affect the mucosal lining as well, producing a series of internal folds. Haustra permit the colon to expand and elongate.
- Three separate longitudinal bands of smooth muscle—called the **taeniae coli** (TĒ-nē-ē KŌ-lē)—run along the outer surfaces of the colon just deep to the serosa. These bands correspond to the outer layer of the muscularis externa in other portions of the digestive tract. Muscle tone within the taeniae coli is what creates the haustra.
- The serosa of the colon contains numerous teardrop-shaped sacs of fat called **fatty appendices,** or *epiploic* (ep-i-PLŌ-ik; *epiploon*, omentum) *appendages*.

We can subdivide the colon into four regions: the ascending colon, transverse colon, descending colon, and sigmoid colon (**Figure 24–24a**).

1. The **ascending colon** begins at the superior border of the cecum and ascends along the right lateral and posterior wall of the peritoneal cavity to the inferior surface of the liver. There, the colon bends sharply to the left at the **right colic flexure,** or *hepatic flexure*. This bend marks the end of the ascending colon and the beginning of the transverse colon.
2. The **transverse colon** curves anteriorly from the right colic flexure and crosses the abdomen from right to left. The transverse colon is supported by the transverse mesocolon. It is separated from the anterior abdominal wall by the layers of the greater omentum. At the left side of the body, the transverse colon passes inferior to the greater curvature of the stomach. Near the spleen, the colon makes a 90° turn at the **left colic flexure,** or *splenic flexure*, and becomes the descending colon.
3. The **descending colon** proceeds inferiorly along the left side until reaching the iliac fossa formed by the inner surface of the left ilium. The descending colon is retroperitoneal and firmly attached to the abdominal wall. At the iliac fossa, the descending colon curves at the **sigmoid flexure** and becomes the sigmoid colon.
4. The sigmoid flexure is the start of the **sigmoid** (SIG-moyd) **colon** (*sigmeidos*, the Greek letter *S*), an S-shaped segment that is only about 15 cm (6 in.) long. The sigmoid colon lies posterior to the urinary bladder, suspended from the sigmoid mesocolon. The sigmoid colon empties into the *rectum*.

Figure 24–24 The Large Intestine. ATLAS: Plates 49a–c; 58a–c; 59; 64; 65



Clinical Note

Colorectal Cancer Colorectal cancer is relatively common in the United States. Aside from skin cancers, colorectal cancer is the third most common cancer in the United States, affecting both men and women. The National Cancer Institute estimates that in 2010 there will be 142,579 new cases of colon cancer and 51,370 people will die from the disease. Colorectal cancer is the second leading cause of cancer-related deaths. However, the death rate has declined over the past 15 years. The best defense appears to be early detection and prompt treatment. Standard screening involves checking the feces for blood. This simple procedure can be performed easily on a stool (fecal) sample as part of a routine physical. For those individuals at increased risk because of family history, associated disease, or older age, visual inspection of the lumen by fiberoptic colonoscopy to discover polyps before they develop into cancers is prudent. The 5-year survival rate for people whose cancer is found at an early stage and treated immediately is greater than 90%.

The large intestine receives blood from branches of the superior mesenteric and inferior mesenteric arteries. The superior mesenteric and inferior mesenteric veins collect venous blood from the large intestine. ↩ p. 755

The Rectum

The **rectum** (REK-tum) forms the last 15 cm (6 in.) of the digestive tract (**Figure 24–24a,c**). It is an expandable organ for the temporary storage of feces. The movement of fecal material into the rectum triggers the urge to defecate (expel feces).

The last portion of the rectum, the **anal canal,** contains small longitudinal folds called **anal columns.** The distal margins of these columns are joined by transverse folds that mark the boundary between the columnar epithelium of the proximal rectum and a stratified squamous epithelium like that in the oral cavity. The **anus,** or *anal orifice,* is the exit of the anal canal. There, the epidermis becomes keratinized and identical to the surface of the skin.

The circular muscle layer of the muscularis externa in this region forms the **internal anal sphincter** (**Figure 24–24c**). The smooth muscle cells of this sphincter are not under voluntary control. The **external anal sphincter,** which guards the anus, consists of a ring of skeletal muscle fibers that encircles the distal portion of the anal canal. This sphincter consists of skeletal muscle and is under voluntary control.

The lamina propria and submucosa of the anal canal contain a network of veins. If venous pressures there rise too high due to straining during defecation, the veins can become distended, producing *hemorrhoids*.

Histology of the Large Intestine

The diameter of the colon is about three times that of the small intestine, but its wall is much thinner. The major characteristics of the colon are the lack of villi, the abundance of mucous cells, and the presence of distinctive intestinal glands (**Figure 24–25**). The glands in the large intestine are deeper than those of the small intestine and are dominated by mucous cells. The mucosa of the large intestine does not produce enzymes. Any digestion that occurs results from enzymes introduced in the small intestine or from bacterial action. The mucus provides lubrication as the fecal material becomes drier and more compact. Mucus is secreted as local stimuli, such as friction or exposure to harsh chemicals, trigger short reflexes involving local nerve plexuses. Large lymphoid nodules are scattered throughout the lamina propria and submucosa.

The muscularis externa of the large intestine is unusual, because the longitudinal layer has been reduced to the muscular bands of the taeniae coli. However, the mixing and propulsive contractions of the colon resemble those of the small intestine.

Physiology of the Large Intestine

Less than 10 percent of the nutrient absorption under way in the digestive tract occurs in the large intestine. Nevertheless, the absorptive operations in this segment of the digestive tract are important. The large intestine also prepares fecal material for ejection from the body.

Absorption in the Large Intestine

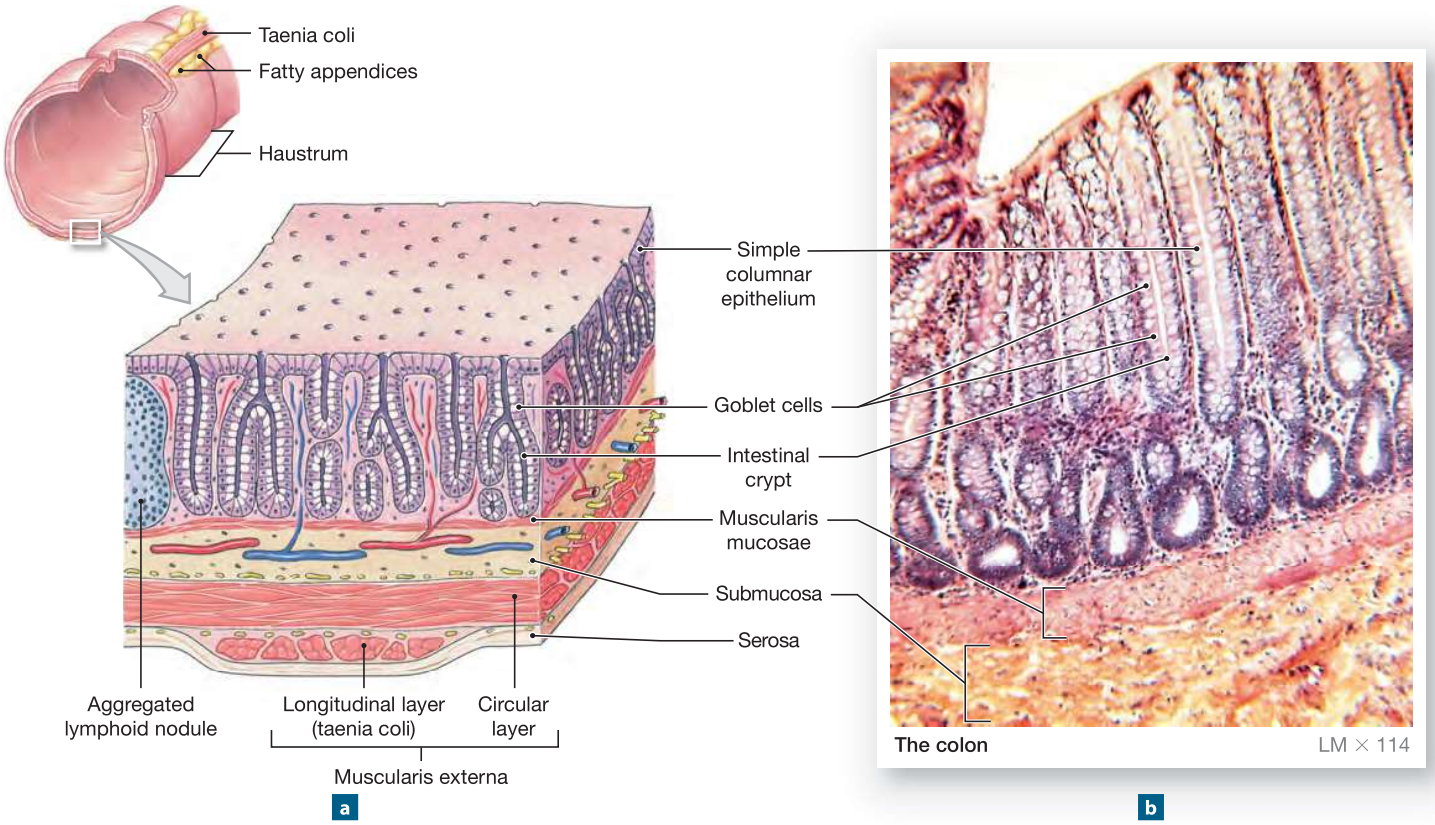
The reabsorption of water is an important function of the large intestine. Roughly 1500 mL of material enters the colon each day, but only about 200 mL of feces is ejected. To appreciate how efficient our digestion is, consider the average composition of feces: 75 percent water, 5 percent bacteria, and the rest a mixture of indigestible materials, small quantities of inorganic matter, and the remains of epithelial cells.

In addition to reabsorbing water, the large intestine absorbs a number of other substances that remain in the feces or were secreted into the digestive tract along its length. Examples include useful compounds such as bile salts and vitamins; organic waste products such as urobilinogen; and various toxins generated by bacterial action. Most of the bile salts entering the large intestine are promptly reabsorbed in the cecum and transported in blood to the liver for secretion into bile.

Vitamins. Vitamins are organic molecules that are important as cofactors or coenzymes in many metabolic pathways. The normal bacterial residents of the colon generate three vitamins that supplement our diets:

1. *Vitamin K,* a fat-soluble vitamin the liver requires for synthesizing four clotting factors, including prothrombin. Intestinal bacteria produce about half of your daily vitamin K requirements.

Figure 24–25 The Mucosa and Glands of the Colon.



2. *Biotin*, a water-soluble vitamin important in various reactions, notably those of glucose metabolism.
3. *Vitamin* B_5 (pantothenic acid), a water-soluble vitamin required in the manufacture of steroid hormones and some neurotransmitters.

Vitamin K deficiencies lead to impaired blood clotting. They result from either: (1) not enough lipids in the diet, which impairs the absorption of all fat-soluble vitamins; or (2) problems affecting lipid processing and absorption, such as inadequate bile production or chronic diarrhea (frequent, watery bowel movements). Disorders due to deficiencies of biotin or vitamin B_5 are extremely rare after infancy. The intestinal bacteria generally produce sufficient amounts to supplement any dietary shortage.

Organic Wastes. We discussed the fate of bilirubin, a breakdown product of heme, in Chapter 19. ↩ p. 646 In the large intestine, bacteria convert bilirubin to *urobilinogens* and *stercobilinogens*. Some urobilinogens are absorbed into the bloodstream and then excreted in urine. The urobilinogens and stercobilinogens remaining within the colon are converted to **urobilins** and **stercobilins** by exposure to oxygen. These pigments in various proportions give feces a yellow-brown or brown color.

Bacterial action breaks down peptides that remain in the feces. This action generates (1) ammonia, in the form of soluble *ammonium ions* (NH_4^+); (2) *indole* and *skatole*, two nitrogen-containing compounds that are primarily responsible for the odor of feces; and (3) hydrogen sulfide (H_2S), a gas with a "rotten egg" odor. Significant amounts of ammonia and smaller amounts of other toxins cross the colonic epithelium and enter the hepatic portal circulation. The liver removes these toxins and converts them to relatively nontoxic compounds that can be released into the blood and excreted at the kidneys.

Intestinal enzymes do not alter indigestible carbohydrates. These materials arrive in the colon virtually intact. These complex polysaccharides provide a reliable nutrient source for bacteria in the colon. The metabolic activities of these bacteria create small amounts of **flatus,** or intestinal gas. Foods with large amounts of indigestible carbohydrates (such as beans) stimulate bacterial gas production. Distension of the colon, cramps, and the frequent discharge of intestinal gases can result.

Movements of the Large Intestine

The gastroileal and gastroenteric reflexes move materials into the cecum while you eat. Movement from the cecum to the transverse colon is very slow, allowing hours for water absorption to convert the already thick material into a sludgy paste. Peristaltic waves move material along the length of the colon. Segmentation movements, called *haustral churning*, mix the contents of adjacent haustra. Powerful peristaltic contractions called **mass movements** occur a few times each day. They move

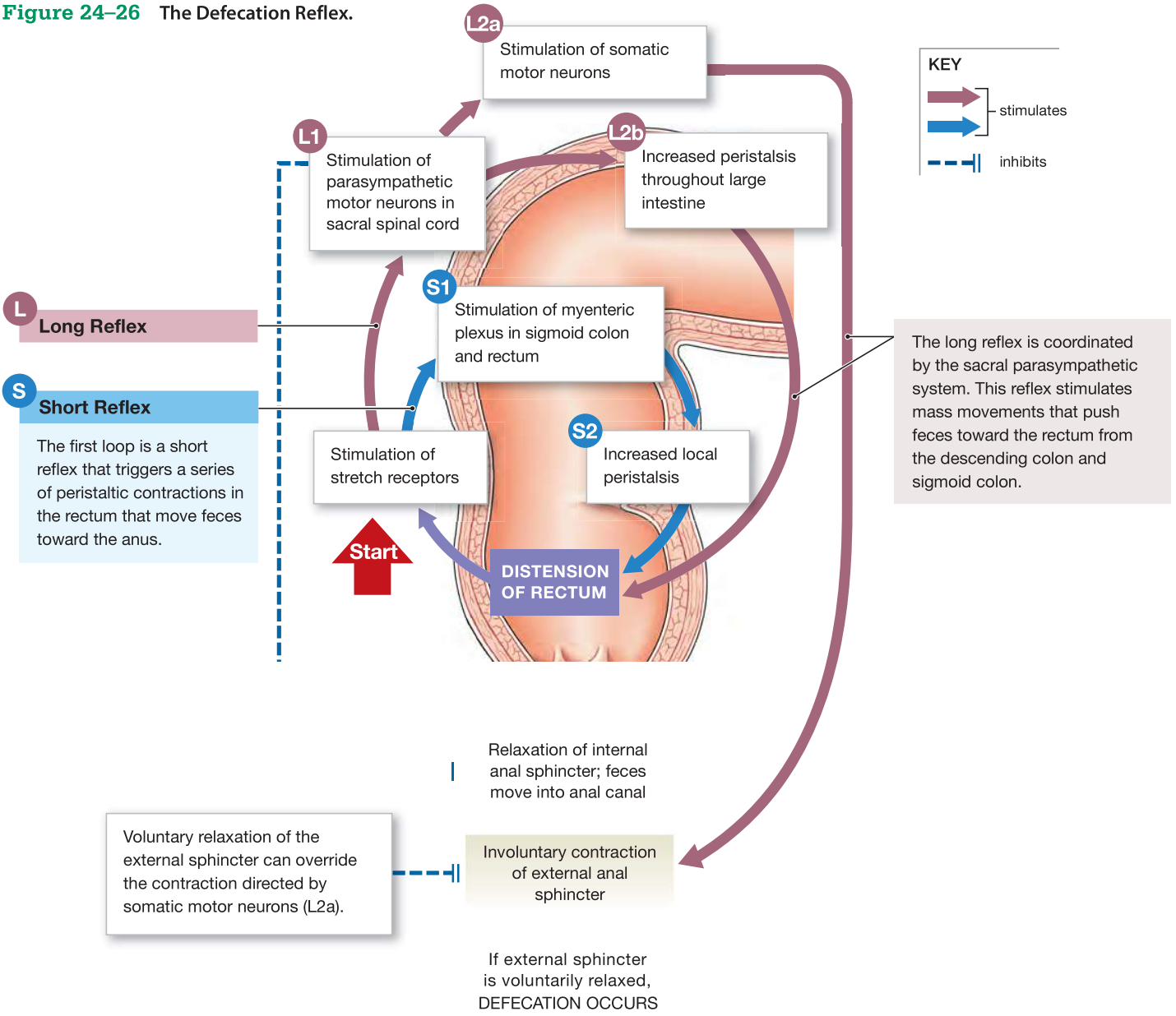
material from the transverse colon through the rest of the large intestine. The stimulus is distension of the stomach and duodenum. The commands are relayed over the intestinal nerve plexuses. The contractions force feces into the rectum and produce the conscious urge to defecate.

The rectal chamber is usually empty, except when a powerful peristaltic contraction forces feces out of the sigmoid colon. Distension of the rectal wall then triggers the **defecation reflex.** This reflex involves two positive feedback loops (**Figure 24–26**). Both loops are triggered by the stimulation of stretch receptors in the rectum.

Rectal stretch receptors also trigger two reflexes important to the *voluntary* control of defecation. One is a long reflex mediated by parasympathetic innervation within the pelvic nerves. This reflex causes the internal anal sphincter to relax. This smooth muscle sphincter controls the movement of feces into the anal canal. The second is a somatic reflex that stimulates the immediate contraction of the external anal sphincter, a skeletal muscle. pp. 345–347 The pudendal nerves carry the motor commands.

Both the internal and external anal sphincters must relax for feces to be eliminated. However, the two reflexes just mentioned open the internal sphincter but close the external sphincter. The actual release of feces requires a conscious effort to open the external sphincter. In addition, other consciously directed activities can raise intra-abdominal pressures and help force fecal material out of the rectum. These activities include

Figure 24–26 The Defecation Reflex.



tensing the abdominal muscles or elevating intra-abdominal pressures by attempting to exhale forcibly with a closed glottis.

If the external anal sphincter remains constricted, the peristaltic contractions cease. However, additional rectal expansion triggers further defecation reflexes. The urge to defecate usually develops when rectal pressure reaches about 15 mm Hg. If this pressure is more than 55 mm Hg, the external anal sphincter involuntarily relaxes and defecation takes place. This mechanism brings about defecation in infants, and in adults with severe spinal cord injuries.

Checkpoint

26. **Identify the four regions of the colon.**
27. **What are some major histological differences between the large intestine and the small intestine?**
28. **Differentiate between haustral churning and mass movements.**

See the blue Answers tab at the back of the book.

24-8 Digestion is the mechanical and chemical alteration of food that allows the absorption and use of nutrients

A balanced diet contains all the ingredients needed to maintain homeostasis. These ingredients include six nutrients: carbohydrates, lipids, proteins, vitamins, minerals, and water. This section describes the chemical events involved in the processing and absorbing of these nutrients.

The Processing and Absorption of Nutrients

Food contains large organic molecules, many of them insoluble. The digestive system first breaks down the physical structure of the ingested material and then disassembles the component molecules into smaller fragments. This disassembly eliminates any antigenic properties, so that the fragments do not trigger an immune response after absorption. Cells absorb the molecules released into the bloodstream and either (1) break them down to provide energy for the synthesis of ATP or (2) use these molecules to synthesize carbohydrates, proteins, and lipids. In this section we focus on the mechanics of digestion and absorption. The fates of the compounds inside cells are the focus in Chapter 25.

Most ingested organic materials are complex chains of simpler molecules. In a typical dietary carbohydrate, the basic molecules are simple sugars. In a protein, the building blocks are amino acids. In lipids, they are generally fatty acids. And in nucleic acids, they are nucleotides. Digestive enzymes break the bonds between the component molecules of carbohydrates, proteins, lipids, and nucleic acids in a process called *hydrolysis*. ↩ p. 36

The classes of digestive enzymes differ with respect to their targets. *Carbohydrases* break the bonds between simple sugars, *proteases* split the linkages between amino acids, and *lipases* separate fatty acids from glycerides. Some enzymes in each class are even more selective, breaking bonds between specific molecules. For example, a particular carbohydrase might break the bond between two glucose molecules, but not those between glucose and another simple sugar.

Digestive enzymes secreted by the salivary glands, tongue, stomach, and pancreas are mixed into the ingested material as it passes along the digestive tract. These enzymes break down large carbohydrates, proteins, lipids, and nucleic acids into smaller fragments. Typically these fragments in turn must be broken down even further before absorption can occur. The final enzymatic steps involve brush border enzymes, which are attached to the exposed surfaces of intestinal microvilli.

Nucleic acids are broken down into their component nucleotides. Brush border enzymes digest these nucleotides into sugars, phosphates, and nitrogenous bases that are absorbed by active transport. However, nucleic acids represent only a small fraction of all the nutrients absorbed each day. The digestive fates of carbohydrates, lipids, and proteins, the major dietary components, are shown in **Spotlight Figure 24–27**. **Table 24–1** summarizes the major digestive enzymes and their functions. Next we take a closer look at the digestion and absorption of carbohydrates, lipids, and proteins.

Carbohydrate Digestion and Absorption

The digestion of complex carbohydrates (simple polysaccharides and starches) proceeds in two steps. One step involves carbohydrases produced by the salivary glands and pancreas. The other step uses brush border enzymes.

The Actions of Salivary and Pancreatic Enzymes

The digestion of complex carbohydrates involves two enzymes—salivary amylase and pancreatic alpha-amylase (**Spotlight Figure 24–27**). Both function effectively at a pH of 6.7–7.5. Carbohydrate digestion begins in the mouth during mastication, through the action of salivary amylase from the parotid and submandibular salivary glands. Salivary amylase breaks down starches (complex carbohydrates) into a mixture composed mostly of *disaccharides* (two simple sugars) and *trisaccharides* (three simple sugars). Salivary amylase continues to digest the starches and glycogen in the food for 1–2 hours before stomach acids render the enzyme inactive. Only a small amount of digestion takes place over this period because the enzymatic content of saliva is not high.

In the duodenum, pancreatic alpha-amylase breaks down the remaining complex carbohydrates. Any disaccharides or trisaccharides produced, and any present in the food, are not broken down further until they contact the intestinal mucosa.

Spotlight

Figure 24–27
Chemical Events in Digestion

A typical meal contains carbohydrates, proteins, lipids, water, minerals (electrolytes), and vitamins. The digestive system handles each component differently. Large organic molecules must be broken down by digestion before they can be absorbed. Water, minerals, and vitamins can be absorbed without processing, but they may require special transport mechanisms.

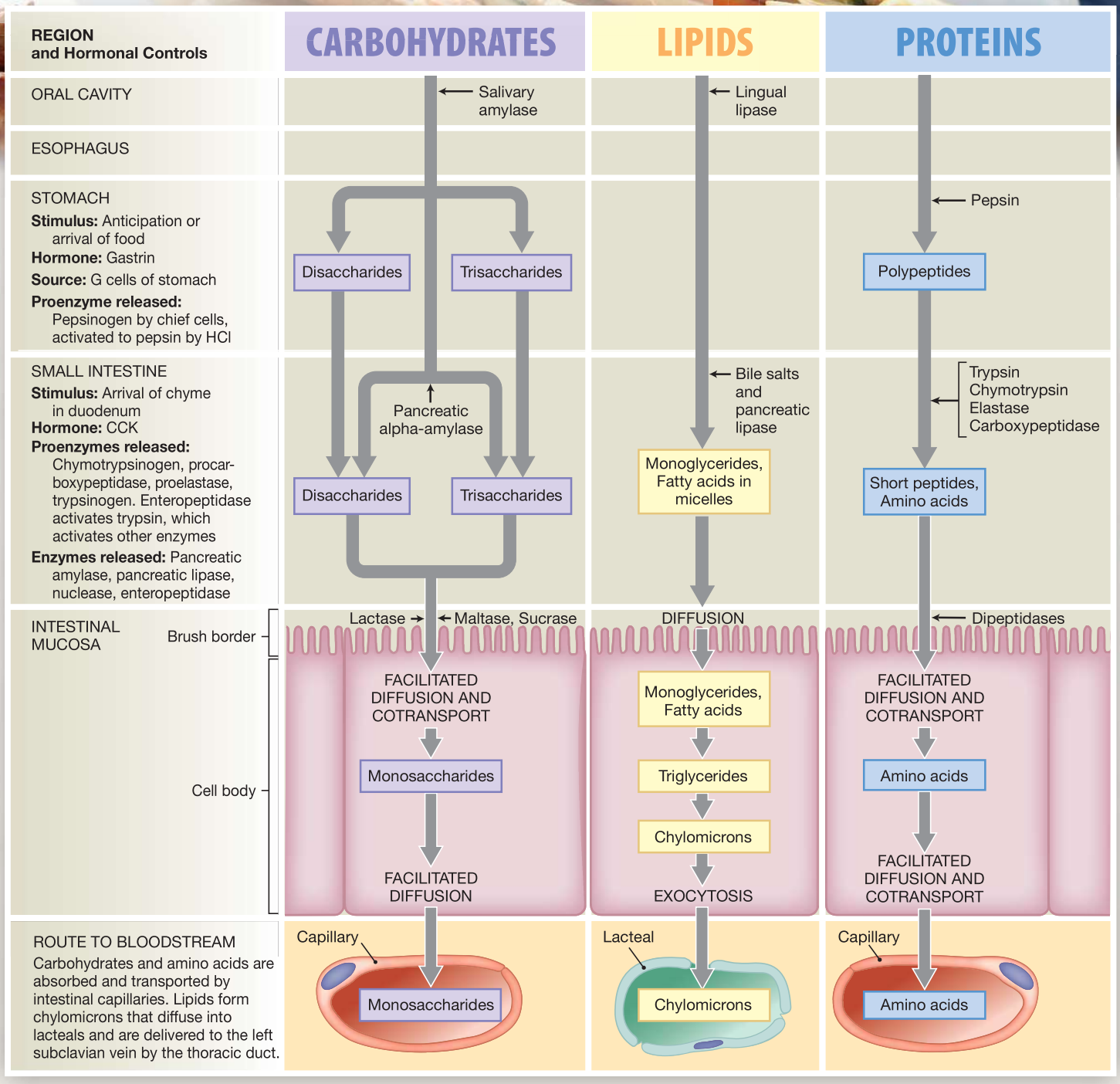


Table 24–1 Digestive Enzymes and Their Functions

Enzyme (proenzyme)	Source	Optimal pH	Target	Products	Remarks
CARBOHYDRASES					
Maltase, sucrase, lactase	Brush border of small intestine	7–8	Maltose, sucrose, lactose	Monosaccharides	Found in membrane surface of microvilli
Pancreatic alpha-amylase	Pancreas	6.7–7.5	Complex carbohydrates	Disaccharides and trisaccharides	Breaks bonds between simple sugars
Salivary amylase	Salivary glands	6.7–7.5	Complex carbohydrates	Disaccharides and trisaccharides	Breaks bonds between simple sugars
PROTEASES					
Carboxypeptidase (procarboxypeptidase)	Pancreas	7–8	Proteins, polypeptides, amino acids	Short-chain peptides	Activated by trypsin
Chymotrypsin (chymotrypsinogen)	Pancreas	7–8	Proteins, polypeptides	Short-chain peptides	Activated by trypsin
Dipeptidases, peptidases	Brush border of small intestine	7–8	Dipeptides, tripeptides	Amino acids	Found in membrane surface of brush border
Elastase (proelastase)	Pancreas	7–8	Elastin	Short-chain peptides	Activated by trypsin
Enteropeptidase	Brush border and lumen of small intestine	7–8	Trypsinogen	Trypsin	Reaches lumen through disintegration of shed epithelial cells
Pepsin (pepsinogen)	Chief cells of stomach	1.5–2.0	Proteins, polypeptides	Short-chain polypeptides	Secreted as proenzyme pepsinogen; activated by H^+ in stomach acid
Rennin	Stomach	3.5–4.0	Milk proteins		Secreted only in infants; causes protein coagulation
Trypsin (trypsinogen)	Pancreas	7–8	Proteins, polypeptides	Short-chain peptides	Proenzyme activated by enteropeptidase; activates other pancreatic proteases
LIPASES					
Lingual lipase	Glands of tongue	3.0–6.0	Triglycerides	Fatty acids and monoglycerides	Begins lipid digestion
Pancreatic lipase	Pancreas	7–8	Triglycerides	Fatty acids and monoglycerides	Bile salts must be present for efficient action
NUCLEASES					
	Pancreas	7–8	Nucleic acids	Nitrogenous bases and simple sugars	Includes ribonuclease for RNA and deoxyribonuclease for DNA

Actions of Brush Border Enzymes

Brush border enzymes of the intestinal microvilli break disaccharides and trisaccharides into *monosaccharides* (simple sugars) prior to absorption. The enzyme **maltase** splits bonds between the two glucose molecules of the disaccharide **maltose. Sucrase** breaks the disaccharide **sucrose** into glucose and *fructose*, another six-carbon sugar. **Lactase** hydrolyzes the disaccharide **lactose** into a molecule of glucose and one of *galactose*. Lactose is the main carbohydrate in milk, so lactase provides an essential function in infancy and early childhood by breaking down lactose. If the intestinal mucosa stops producing lactase, the individual becomes **lactose intolerant.** After ingesting milk and other dairy products, lactose-intolerant individuals can experience a variety of unpleasant digestive problems, including lower abdominal pain, gas, diarrhea, and vomiting.

Absorption of Monosaccharides

The intestinal epithelium then absorbs the monosaccharides by facilitated diffusion and cotransport mechanisms (see **Figure 3–18,** p. 91). Both methods involve a carrier protein. Facilitated diffusion and cotransport differ in three major ways:

1. *Facilitated diffusion moves only one molecule or ion through the plasma membrane, whereas cotransport moves more than one molecule or ion through the membrane at the same time.* In cotransport, the transported substances move in the same direction: down the concentration gradient for at least one of them.

2. *Facilitated diffusion does not require ATP.* Cotransport by itself does not consume ATP, but the cell must often expend ATP to preserve homeostasis. For example, the process may bring in sodium ions that must later be pumped out of the cell.
3. *Facilitated diffusion does not take place if there is an opposing concentration gradient for the particular molecule or ion.* By contrast, cotransport can take place despite an opposing concentration gradient for one of the transported substances. For example, cells lining the small intestine continue to absorb glucose when glucose concentrations inside the cells are much higher than they are in the intestinal contents.

The cotransport system that takes up glucose also brings sodium ions into the cell. This passive process resembles facilitated diffusion, except that both a sodium ion and a glucose molecule must bind to the carrier protein before they can move into the cell. Glucose cotransport is an example of sodium-linked cotransport. ↩ p. 93 Comparable cotransport mechanisms exist for other simple sugars and for some amino acids. These mechanisms deliver valuable nutrients to the cytoplasm, but they also bring in sodium ions that must be ejected by the sodium–potassium exchange pump.

The simple sugars that are transported into the cell at its apical surface diffuse through the cytoplasm. They then reach the interstitial fluid by facilitated diffusion across the basolateral surfaces. These monosaccharides diffuse into the capillaries of the villus for eventual transport to the liver in the hepatic portal vein.

Lipid Digestion and Absorption

Lipid digestion involves lingual lipase from glands of the tongue, and pancreatic lipase from the pancreas (**Figure 24–27**). The most important and abundant dietary lipids are triglycerides. They consist of three fatty acids attached to a single molecule of glycerol (see **Figure 2–16,** p. 47). The lingual and pancreatic lipases break off two of the fatty acids, leaving monoglycerides.

Lipases are water-soluble enzymes, and lipids tend to form large drops that exclude water molecules. As a result, lipases can attack only the exposed surfaces of the lipid drops. Lingual lipase begins breaking down triglycerides in the mouth and continues for a variable time within the stomach. The lipid drops are so large, however, and the available time so short, that only about 20 percent of the lipids have been digested by the time the chyme enters the duodenum.

Bile salts improve chemical digestion by emulsifying the lipid drops into tiny emulsion droplets, thereby providing better access for pancreatic lipase. The emulsification takes place only after the chyme has been mixed with bile in the duodenum. Pancreatic lipase then breaks apart the triglycerides to form a mixture of fatty acids and monoglycerides. As these molecules are released, they interact with bile salts in the surrounding chyme to form small lipid–bile salt complexes called **micelles** (mī-SELZ). A micelle is only about 2.5 nm (0.0025 μm) in diameter.

When a micelle contacts the intestinal epithelium, the lipids diffuse across the plasma membrane and enter the cytoplasm. The intestinal cells synthesize new triglycerides from the monoglycerides and fatty acids. These triglycerides, in company with absorbed steroids, phospholipids, and fat-soluble vitamins, are then coated with proteins. The resulting complexes are known as **chylomicrons** (kī-lō-MĪ-kronz; *chylos*, juice + *mikros*, small).

The intestinal cells then secrete the chylomicrons into interstitial fluid by exocytosis. The protein coating keeps the chylomicrons suspended in the interstitial fluid, but they are generally too large to diffuse into capillaries. Most of the chylomicrons diffuse into the intestinal lacteals, which lack basement membranes and have large gaps between adjacent endothelial cells. From the lacteals, the chylomicrons proceed along the lymphatic vessels and through the thoracic duct. They finally enter the bloodstream at the left subclavian vein.

Most of the bile salts within micelles are reabsorbed by sodium-linked cotransport. Only about 5 percent of the bile

Clinical Note

Inflammatory and Infectious Disorders of the Digestive System

Digestive system disorders are both very diverse and relatively common because the system has so many parts, and those parts have so many functions.

The largest category of digestive disorders includes those resulting from inflammation or infection of the digestive tract. In part, this is because the epithelium that lines most of the digestive tract has two properties that are difficult to reconcile: (1) It must be thin enough to absorb nutrients rapidly and efficiently; and (2) it must resist damage from ingested materials and enzymes.

The delicacy of the epithelium makes it susceptible to damage from chemical attack or abrasion. For example, *peptic ulcers* develop if acids and enzymes contact and erode the gastric or duodenal lining. Pathogens in food, including bacteria, viruses, and multicellular parasites, may also get through the epithelial barriers and cause infections. Small battles are continually being fought; the fact that 80 percent of the body's plasma cells are normally located within the lamina propria of the digestive tract indicates how often antigens of one kind or another somehow cross the epithelial barriers.

High rates of cell division and exposure to strong chemical agents are both correlated with an increased risk of cancer. As a result, cancers of the digestive tract are relatively common. Predictably, most of these are epithelial cancers that develop in the stem cell populations responsible for epithelial cell renewal.

salts secreted by the liver enters the colon. Only about 1 percent is lost in feces.

Protein Digestion and Absorption

Proteins have very complex structures, so protein digestion is both complex and time-consuming. The first task is to disrupt the three-dimensional organization of the food. This step allows proteolytic enzymes to attack individual proteins. This step involves mechanical processing in the oral cavity, through mastication, and chemical processing in the stomach, through the action of hydrochloric acid. The strong acid in the stomach kills pathogens and breaks down plant cell walls and animal connective tissues. Acid also disrupts tertiary and secondary protein structure, exposing peptide bonds to enzymatic attack.

The acidic content of the stomach also provides the proper environment for the activity of pepsin, the proteolytic enzyme secreted in an inactive form by chief cells of the stomach (**Figure 24–27**). Pepsin works effectively at a pH of 1.5–2.0. It breaks the peptide bonds within a polypeptide chain.

When chyme enters the duodenum, enteropeptidase from the small intestine triggers the conversion of trypsinogen to trypsin. Buffers increase the pH to 7–8. Pancreatic proteases can now begin working. Trypsin, chymotrypsin, and elastase are like pepsin in that they break specific peptide bonds within a polypeptide. For example, trypsin breaks peptide bonds involving the amino acids *arginine* or *lysine*. Chymotrypsin targets peptide bonds involving *tyrosine* or *phenylalanine*.

Carboxypeptidase also acts in the small intestine. This enzyme chops off the last amino acid of a polypeptide chain, no matter which amino acids are involved. Thus, while the other peptidases generate a variety of short peptides, carboxypeptidase produces free amino acids.

The epithelial surfaces of the small intestine contain several peptidases, notably **dipeptidases.** These enzymes break short peptide chains into individual amino acids. (Dipeptidases break apart *dipeptides*.) These amino acids, as well as those produced by the pancreatic enzymes, are absorbed through both facilitated diffusion and cotransport mechanisms.

The amino acids diffuse through the cell to its basolateral surface. There they are released into interstitial fluid by facilitated diffusion and cotransport. Once in the interstitial fluid, the amino acids diffuse into intestinal capillaries for transport to the liver by means of the hepatic portal vein.

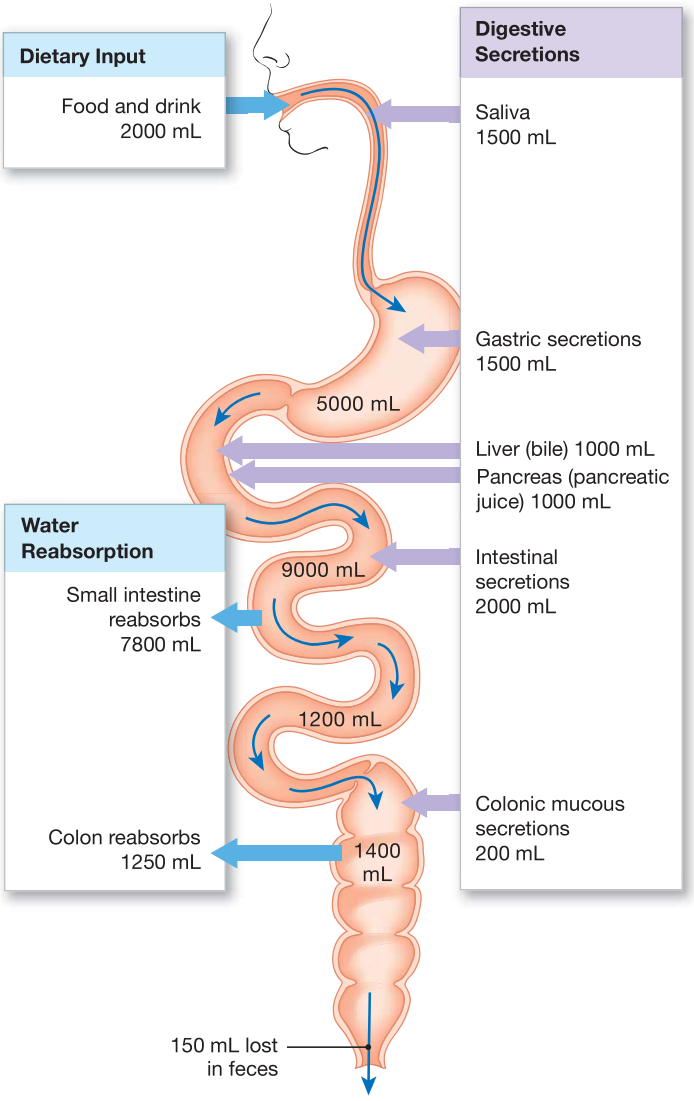
Water Absorption

Cells cannot actively absorb or secrete water. All movement of water across the lining of the digestive tract involves passive water flow down osmotic gradients. The production of glandular secretions also involves passive water flow down osmotic gradients. Recall that when two solutions are separated by a selectively permeable membrane, water tends to flow into the solution that has the higher concentration of solutes. p. 88 Osmotic movements are rapid, so interstitial fluid and the fluids in the intestinal lumen always have the same osmotic concentration of solutes, or osmolarity.

Intestinal epithelial cells continuously absorb nutrients and ions. These activities gradually lower the solute concentration in the lumen. As the solute concentration drops, water moves into the surrounding tissues, maintaining osmotic equilibrium.

Each day, about 2000 mL of water enters the digestive tract in the form of food or drink. The salivary, gastric, intestinal, colonic, pancreatic, and bile secretions provide an additional 7200 mL. Of that total, only about 150 mL is lost in feces. The sites of secretion and absorption of water are shown in **Figure 24–28**.

Figure 24–28 **Digestive Secretion and Absorption of Water.**
The purple arrows indicate secretion, the blue arrows show water reabsorption.



Ion Absorption

Osmosis does not distinguish among solutes. All that matters is the total concentration of solutes. To maintain homeostasis, however, the concentrations of specific ions must be closely regulated. Thus, each ion must be handled individually, and the rate of intestinal absorption of each must be tightly controlled (Table 24–2). Many of the regulatory mechanisms controlling the rates of absorption are poorly understood.

Sodium ions (Na^+) are usually the most abundant cations in food. They may enter intestinal cells by diffusion, by cotransport with another nutrient, or by active transport. These ions are then pumped into interstitial fluid across the base of the cell.

The rate of Na^+ uptake from the lumen is generally proportional to the concentration of Na^+ in the intestinal contents. As a result, eating heavily salted foods leads to increased sodium ion absorption and an associated gain of water through osmosis. The rate of sodium ion absorption by the digestive tract is increased by aldosterone, a steroid hormone from the adrenal cortex. ↩ p. 616

Calcium ion (Ca^{2+}) absorption involves active transport at the epithelial surface. Calcitriol speeds up the rate of transport. ↩ p. 615

As other solutes move out of the lumen, the concentration of potassium ions (K^+) increases. These ions can diffuse into the epithelial cells, driven by the concentration gradient. The absorption of magnesium (Mg^{2+}), iron (Fe^{2+}), and other cations involves specific carrier proteins. The cell must use ATP to obtain and transport these ions to interstitial fluid. Regulatory factors controlling their absorption are poorly understood.

The anions chloride (Cl^-), iodide (I^-), bicarbonate (HCO_3^-), and nitrate (NO_3^-) are absorbed by diffusion or carrier-mediated transport. Phosphate (PO_4^{3-}) and sulfate (SO_4^{2-}) ions enter epithelial cells only by active transport.

Vitamin Absorption

Vitamins are organic compounds required in very small quantities. There are two major groups of vitamins: fat-soluble vitamins and water-soluble vitamins. Vitamins A, D, E, and K are **fat-soluble vitamins.** Their structure allows them to dissolve in lipids. The nine **water-soluble vitamins** include the B vitamins, common in milk and meats, and vitamin C, found in citrus fruits. We consider the functions of vitamins and associated nutritional problems in Chapter 25.

All but one of the water-soluble vitamins are easily absorbed by diffusion across the digestive epithelium. By itself vitamin B_{12} cannot be absorbed by the intestinal mucosa in normal amounts. This vitamin must be bound to *intrinsic factor*, a glycoprotein secreted by the parietal cells of the stomach (p. 881). The combination is then absorbed through active transport.

Fat-soluble vitamins in the diet enter the duodenum in fat droplets, mixed with triglycerides. The vitamins remain in association with these lipids as they form emulsion droplets and, after further digestion, micelles. The fat-soluble vitamins are then absorbed from the micelles along with the fatty acids and monoglycerides. Vitamin K produced in the colon is absorbed with other lipids released through bacterial action. Taking supplements of fat-soluble vitamins while you have an empty stomach, are fasting, or are on a low-fat diet is relatively ineffective. The reason is that proper absorption of these vitamins requires the presence of other lipids.

Table 24–2 The Absorption of Ions and Vitamins

Ion or Vitamin	Transport Mechanism	Regulatory Factors
Na^+	Channel-mediated diffusion, cotransport, or active transport	Increased when sodium-linked cotransport is under way; stimulated by aldosterone
Ca^{2+}	Active transport	Stimulated by calcitriol and PTH
K^+	Channel-mediated diffusion	Follows concentration gradient
Mg^{2+}	Active transport	
Fe^{2+}	Active transport	
Cl^-	Channel-mediated diffusion or carrier-mediated transport	
I^-	Channel-mediated diffusion or carrier-mediated transport	
HCO_3^-	Channel-mediated diffusion or carrier-mediated transport	
NO_3^-	Channel-mediated diffusion or carrier-mediated transport	
PO_4^{3-}	Active transport	
SO_4^{2-}	Active transport	
Water-soluble vitamins (except B_{12})	Channel-mediated diffusion	Follows concentration gradient
Vitamin B_{12}	Active transport	Must be bound to intrinsic factor prior to absorption
Fat-soluble vitamins	Diffusion	Absorbed from micelles along with dietary lipids

Checkpoint

29. What kinds of nutrients does the body require?
30. What component of food would increase the number of chylomicrons in the lacteals?
31. The absorption of which vitamin would be impaired by the removal of the stomach?
32. Why is it that diarrhea is potentially life threatening, but constipation (infrequent defecation) is not?

See the blue Answers tab at the back of the book.

24-9 Many age-related changes affect digestion and absorption

Normal digestion and absorption take place in elderly individuals. However, many changes in the digestive system parallel age-related changes we have already discussed in connection with other systems:

- *The division rate of epithelial stem cells declines.* The digestive epithelium becomes more susceptible to damage by abrasion, acids, or enzymes. Peptic ulcers therefore become more likely. Stem cells in the epithelium divide less frequently with age, so tissue repair is less efficient. In the mouth, esophagus, and anus, the stratified epithelium becomes thinner and more fragile.
- *Smooth muscle tone decreases.* General motility decreases, and peristaltic contractions are weaker as a result of a decrease in smooth muscle tone. These changes slow the rate of fecal movement and promote constipation. Sagging and inflammation of the haustra in the colon can occur. Straining to eliminate compacted feces can stress the less resilient walls of blood vessels, producing hemorrhoids. Problems are not restricted to the lower digestive tract. For example, weakening of muscular sphincters can lead to esophageal reflux and frequent bouts of "heartburn."
- *The effects of cumulative damage become apparent.* A familiar example is the gradual loss of teeth due to *dental caries* (cavities) or gingivitis. Cumulative damage can involve internal organs as well. Toxins such as alcohol and other injurious chemicals that are absorbed by the digestive tract are transported to the liver for processing. The cells of the liver are not immune to these toxic compounds, and chronic exposure can lead to cirrhosis or other types of liver disease.
- *Cancer rates increase.* Cancers are most common in organs in which stem cells divide to maintain epithelial cell populations. Rates of colon cancer and stomach cancer rise with age. Oral, esophageal, and pharyngeal cancers are particularly common among elderly smokers.
- *Dehydration is common among the elderly.* One reason is that osmoreceptor sensitivity declines with age.
- *Changes in other systems have direct or indirect effects on the digestive system.* For example, reduction in bone mass and calcium content in the skeleton is associated with erosion of the tooth sockets and eventual tooth loss. The decline in olfactory and gustatory sensitivities with age can lead to dietary changes that affect the entire body.

Checkpoint

33. Identify general digestive system changes that occur with aging.

See the blue Answers tab at the back of the book.

24-10 The digestive system is extensively integrated with other body systems

Figure 24–29 summarizes the physiological relationships between the digestive system and other organ systems we have studied so far. The digestive system has particularly extensive anatomical and physiological connections to the nervous, cardiovascular, endocrine, and lymphatic systems. As we have seen, the digestive tract is also an endocrine organ that produces a variety of hormones. Many of these hormones, and some of the neurotransmitters produced by the digestive system, can enter the circulation, cross the blood–brain barrier, and alter CNS activity. In this way, a continual exchange of chemical information takes place among these systems.

Checkpoint

34. Identify the functional relationships between the digestive system and other body systems.
35. What body systems may be affected by inadequate calcium absorption?

See the blue Answers tab at the back of the book.

SYSTEM INTEGRATOR

Body System → Digestive System

Digestive System → Body System

Integumentary
- Provides vitamin D_3 needed for the absorption of calcium and phosphorus
- Provides lipids for storage by adipocytes in subcutaneous layer

Integumentary Page 165

Skeletal
- Skull, ribs, vertebrae, and pelvic girdle support and protect parts of digestive tract; teeth are important in mechanical processing of food
- Absorbs calcium and phosphate ions for incorporation into bone matrix; provides lipids for storage in yellow marrow

Skeletal Page 275

Muscular
- Protects and supports digestive organs in abdominal cavity; controls entrances and exits of digestive tract
- Liver regulates blood glucose and fatty acid levels, metabolizes lactic acid from active muscles

Muscular Page 369

Nervous
- ANS regulates movement and secretion; reflexes coordinate passage of materials along tract; control over skeletal muscles regulates ingestion and defecation; hypothalamic centers control hunger, satiation, and feeding
- Provides substrates essential for neurotransmitter synthesis

Nervous Page 543

Endocrine
- Epinephrine and norepinephrine stimulate constriction of sphincters and depress digestive activity; hormones coordinate activity along digestive tract
- Provides nutrients and substrates to endocrine cells; endocrine cells of pancreas secrete insulin and glucagon; liver produces angiotensinogen

Endocrine Page 632

Cardiovascular
- Distributes hormones of the digestive tract; carries nutrients, water, and ions from sites of absorption; delivers nutrients and toxins to liver
- Absorbs fluid to maintain normal blood volume; absorbs vitamin K; liver excretes heme (as bilirubin), synthesizes coagulation proteins

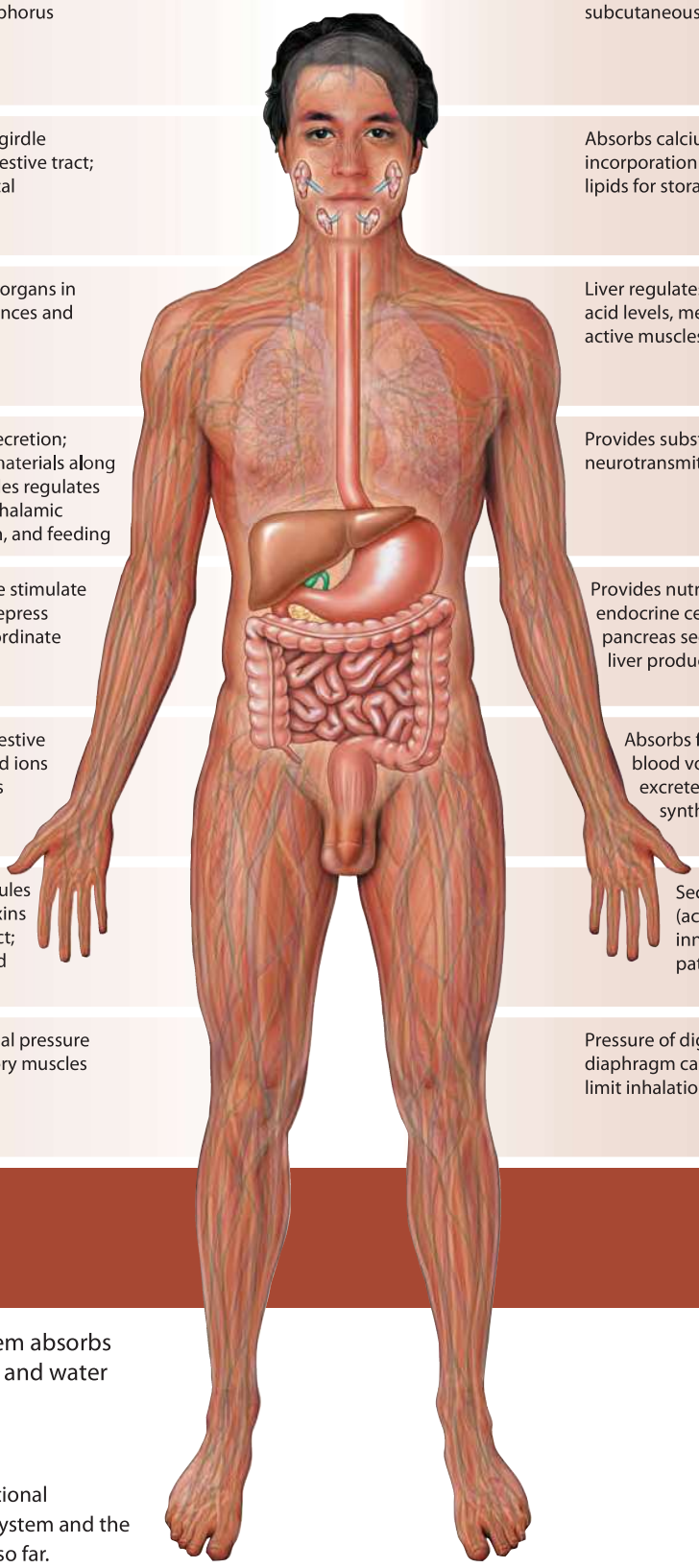
Cardiovascular Page 759

Lymphatic
- Tonsils and other lymphoid nodules defend against infection and toxins absorbed from the digestive tract; lymphatic vessels carry absorbed lipids to venous system
- Secretions of digestive tract (acids and enzymes) provide innate defense against pathogens

Lymphatic Page 807

Respiratory
- Increased thoracic and abdominal pressure through contraction of respiratory muscles can assist in defecation
- Pressure of digestive organs against the diaphragm can assist in exhalation and limit inhalation

Respiratory Page 857



The DIGESTIVE System

For all systems, the digestive system absorbs organic substrates, vitamins, ions, and water required by all cells.

Urinary Page 992

Reproductive Page 1072

Figure 24–29 diagrams the functional relationships between the digestive system and the other body systems we have studied so far.

Related Clinical Terms

borborygmus: A rumbling or gurgling sound made by the movement of fluids and gases in the intestines.
cathartics: Drugs that promote defecation.
cholelithiasis: The presence of gallstones in the gallbladder.
cholera: A bacterial infection of the digestive tract that causes massive fluid losses through diarrhea.
colitis: A general term for a condition characterized by inflammation of the colon.
Crohn's disease: An incurable chronic inflammatory bowel disease that can affect any part of the digestive tract, from the mouth to the anus. The presence of strictures, fistulas, and fissures is common.
diverticulitis: An infection and inflammation of mucosal pockets of the large intestine (diverticula).
diverticulosis: The formation of diverticula, generally along the sigmoid colon.
dysphagia: Difficulty or discomfort in swallowing due to disease.
esophageal varices: Swollen and fragile esophageal veins that result from portal hypertension.
fecal occult blood test: Test to check for hidden blood in feces.
gastrectomy: The surgical removal of the stomach, generally to treat advanced stomach cancer.
gastroesophageal reflux disease (GERD): Chronic condition in which the lower esophageal sphincter allows gastric acids to backflow into the esophagus, causing heartburn, acid indigestion, and possible injury to the esophageal lining.
gastroscope: A fiber-optic instrument inserted into the mouth and directed along the esophagus and into the stomach; used to examine the interior of the stomach and to perform minor surgical procedures.
halitosis: Bad breath that may be due to poor oral hygiene, an infection, diabetes, or other disease.
insoluble fiber: Indigestible plant carbohydrates that do not dissolve in water and pass through the GI tract unchanged. Found in many vegetables and the skins of fruits, insoluble fiber speeds up the passage of material in the GI tract. Individuals consuming diets rich in insoluble fiber decrease their risk for developing diabetes, atherosclerosis, and colorectal cancers, among other diseases.
irritable bowel syndrome (IBS): A common disorder affecting the large intestine, accompanied by cramping, abdominal pain, bloating gas, diarrhea, and constipation.
pancreatic cancer: Malignancy of the pancreas that does not cause symptoms in its early stages, leading to late detection and a survival rate of only 4%.
periodontal disease: A loosening of the teeth within the alveolar sockets caused by erosion of the periodontal ligaments by acids produced through bacterial action.
polyps: Small growths with a stalk protruding from a mucous membrane that is usually benign.
pulpitis: An infection of the pulp of a tooth; treatment may involve a root canal procedure.
pyloric stenosis: Uncommon condition where the muscle of the lower end of the stomach enlarges and prevents food from entering the small intestine.
pylorospasm: Spasm of the pyloric sphincter, accompanied by pain and vomiting.
root canal: Removal of the alveolar nerve in a severely damaged tooth.
soluble fiber: Indigestible plant carbohydrates found in beans, oats, and citrus fruits that dissolve in water when eaten, forming a gel within the digestive tract to slow the passage of material. Diets rich in soluble fiber lower blood cholesterol levels.

Chapter Review

Study Outline

24-1 The digestive system, consisting of the digestive tract and accessory organs, has overlapping food utilization functions p. 863

1. The **digestive system** consists of the muscular **digestive tract** and various **accessory organs.** *(Figure 24–1)*
2. Double sheets of peritoneal membrane called **mesenteries** suspend the digestive tract. The **greater omentum** lies anterior to the abdominal viscera. Its adipose tissue provides padding, protection, insulation, and an energy reserve. *(Figure 24–2)*
3. The *lamina propria* and epithelium form the **mucosa** (mucous membrane) of the digestive tract. Proceeding outward is the **submucosa,** the **muscularis externa,** and a layer of areolar tissue called the *adventitia*. For viscera projecting into the peritoneal cavity, the muscularis externa is covered by a serous membrane called the **serosa.** *(Figure 24–3)*
4. The muscularis externa propels materials through the digestive tract by the contractions of **peristalsis. Segmentation** movements in the small intestine churn digestive materials. *(Figure 24–4)*
5. Neural reflexes, hormones, and local mechanisms control digestive tract activities. *(Figure 24–5)*

24-2 The oral cavity contains the tongue, salivary glands, and teeth, each with specific functions p. 870

6. The functions of the **oral cavity,** or **buccal cavity,** are (1) *sensory analysis* of foods; (2) *mechanical processing* by the teeth, tongue, and palatal surfaces; (3) *lubrication,* by mixing with mucus and salivary gland secretions; and (4) limited *digestion* of carbohydrates and lipids.
7. The oral cavity is lined by the **oral mucosa.** The *hard* and *soft palates* form the roof of the oral cavity, and the *tongue* forms its floor. *(Figure 24–6)*
8. **Intrinsic** and **extrinsic tongue muscles** are controlled by the hypoglossal nerves. *(Figure 24–6)*
9. The **parotid, sublingual,** and **submandibular salivary glands** discharge their secretions into the oral cavity. *(Figure 24–7)*
10. **Mastication** (chewing) of the **bolus** occurs through the contact of the **occlusal** (opposing) **surfaces** of the **teeth.** The

periodontal ligament anchors each tooth in an *alveolus*, or bony socket. **Dentin** forms the basic structure of a tooth. The **crown** is coated with **enamel,** the **root** with **cementum.** *(Figure 24–8)*

11. During childhood and early adulthood, the 20 primary teeth, or **deciduous teeth,** are replaced by the 32 teeth of the **secondary dentition** *(Figure 24–9)*

24-3 The pharynx is a passageway between the oral cavity and esophagus p. 876

12. Propulsion of the bolus through the **pharynx** results from contractions of the *pharyngeal constrictor muscles* and the *palatal muscles*, and from elevation of the larynx.

24-4 The esophagus is a muscular tube that transports solids and liquids from the pharynx to the stomach p. 876

13. The **esophagus** carries solids and liquids from the pharynx to the stomach through the **esophageal hiatus,** an opening in the diaphragm. *(Figure 24–10)*
14. The esophageal mucosa consists of a stratified epithelium. Mucous secretion by esophageal glands of the submucosa reduces friction during the passage of foods. The proportions of skeletal and smooth muscle of the muscularis externa change from the pharynx to the stomach. *(Figure 24–10)*
15. Swallowing, or **deglutition,** can be divided into **buccal, pharyngeal,** and **esophageal phases.** Swallowing begins with the compaction of a bolus and its movement into the pharynx, followed by the elevation of the larynx, reflection of the *epiglottis*, and closure of the *glottis*. After the *upper esophageal sphincter* is opened, peristalsis moves the bolus down the esophagus to the *lower esophageal sphincter*. *(Figure 24–11)*

24-5 The stomach is a J-shaped organ that receives the bolus from the esophagus and aids in chemical and mechanical digestion p. 878

16. The **stomach** has four major functions: (1) storage of ingested food, (2) mechanical breakdown of food, (3) disruption of chemical bonds by acid and enzymes, and (4) production of *intrinsic factor*.
17. The four regions of the stomach are the **cardia, fundus, body,** and **pylorus.** The **pyloric sphincter** guards the exit from the stomach. In a relaxed state, the stomach lining contains numerous **rugae** (ridges and folds). *(Figure 24–12)*
18. Within the **gastric glands,** *parietal cells* secrete *intrinsic factor* and *hydrochloric acid*. **Chief cells** secrete **pepsinogen,** which is converted by acids in the gastric lumen to the enzyme **pepsin. Enteroendocrine** cells of the stomach secrete several compounds, notably the hormone **gastrin.** *(Figures 24–13, 24–14)*
19. Gastric control involves (1) the **cephalic phase,** which prepares the stomach to receive ingested materials; (2) the **gastric phase,** which begins with the arrival of food in the stomach; and (3) the **intestinal phase,** which controls the rate of gastric emptying. Vomiting, or **emesis,** is reverse peristalsis. *(Spotlight Figure 24–15)*

24-6 The small intestine digests and absorbs nutrients, and associated glandular organs assist with the digestive process p. 878

20. Most of the important digestive and absorptive functions occur in the **small intestine.** The pancreas, liver, and gallbladder provide digestive secretions and buffers.
21. The small intestine consists of the **duodenum,** the **jejunum,** and the **ileum.** A sphincter, the **ileocecal valve,** marks the transition between the small and large intestines. *(Figure 24–16)*
22. The intestinal mucosa bears transverse folds called **plicae circulares** and small projections called **intestinal villi.** These folds and projections increase the surface area for absorption. Each villus contains a terminal lymphatic called a **lacteal.** Pockets called **intestinal glands** are lined by enteroendocrine, mucous, and stem cells. *(Figures 24–16, 24–17)*
23. **Intestinal juice** moistens chyme, helps buffer acids, and holds digestive enzymes and digestive products in solution.
24. The **duodenal** (*submucosal* or *Brunner's*) **glands** of the duodenum produce mucus, bicarbonate ions, and the hormone **urogastrone.** The ileum contains masses of lymphoid tissue called *aggregated lymphoid nodules*, or *Peyer's patches*, near the entrance to the large intestine.
25. The **gastroenteric reflex,** initiated by stretch receptors in the stomach, stimulates motility and secretion along the entire small intestine. The **gastroileal reflex** triggers the relaxation of the ileocecal valve.
26. The **pancreatic duct** penetrates the wall of the duodenum. Within each lobule of the **pancreas,** ducts branch repeatedly before ending in the **pancreatic acini** (blind pockets). *(Figure 24–18)*
27. The pancreas has two functions: endocrine (secreting insulin and glucagon into the blood) and exocrine (secreting **pancreatic juice** into the small intestine). Pancreatic enzymes include **carbohydrases, lipases, nucleases,** and **proteolytic enzymes.**
28. The **liver** performs metabolic and hematological regulation and produces **bile.** The bile ducts from all the **liver lobules** unite to form the **common hepatic duct.** That duct meets the **cystic duct** to form the **common bile duct,** which empties into the duodenum. *(Figures 24–19 to 24–21)*
29. The liver lobule is the organ's basic functional unit. **Hepatocytes** form irregular plates arranged in the form of spokes of a wheel. **Bile canaliculi** carry bile to the **bile ductules**, which lead to **portal areas.** *(Figure 24–20)*
30. In **emulsification, bile salts** break apart large drops of lipids, making the lipids accessible to lipases secreted by the pancreas.
31. The liver is the primary organ involved in regulating the composition of circulating blood. All the blood leaving the absorptive surfaces of the digestive tract flows into the liver before entering the systemic circulation. The liver regulates metabolism as it removes and stores excess nutrients, vitamins, and minerals from the blood; mobilizes stored reserves; synthesizes needed nutrients; and removes waste products.
32. The liver's hematological activities include the monitoring of circulating blood by phagocytes and antigen-presenting cells; the synthesis of plasma proteins; the removal of circulating hormones and antibodies; and the removal or storage of toxins.
33. The liver synthesizes bile, which is composed of water, ions, bilirubin, cholesterol, and bile salts (an assortment of lipids).
34. The **gallbladder** stores, modifies, and concentrates bile. *(Figure 24–21)*
35. Neural and hormonal mechanisms coordinate the activities of the digestive glands. Gastrointestinal activity is stimulated by parasympathetic innervation and inhibited by sympathetic innervation. The **enterogastric, gastroenteric,** and **gastroileal reflexes** coordinate movement from the stomach to the large intestine.

36. Intestinal hormones include **secretin, cholecystokinin (CCK), gastric inhibitory peptide (GIP), vasoactive intestinal peptide (VIP), gastrin,** and **enterocrinin.** (*Figures 24–22 and 24–23*)

24-7 The large intestine is divided into three parts with regional specialization p. 898

37. The main functions of the **large intestine** are to (1) reabsorb water and compact materials into feces, (2) absorb vitamins produced by bacteria, and (3) store fecal material prior to defecation. The large intestine consists of the *cecum*, *colon*, and *rectum*. *(Figure 24–24)*
38. The **cecum** collects and stores material from the ileum and begins the process of compaction. The **appendix** is attached to the cecum. *(Figure 24–24)*
39. The **colon** has a larger diameter and a thinner wall than the small intestine. The colon bears **haustra** (pouches), **taeniae coli** (longitudinal bands of muscle), and sacs of fat **(fatty appendices).** *(Figure 24–24)*
40. The four regions of the colon are the **ascending colon, transverse colon, descending colon,** and **sigmoid colon.** *(Figure 24–24)*
41. The **rectum** terminates in the **anal canal,** leading to the **anus.** *(Figure 24–24)*
42. Histological characteristics of the colon include the absence of villi and the presence of mucous cells and deep intestinal glands. *(Figure 24–25)*
43. The large intestine reabsorbs water and other substances such as vitamins, urobilinogen, bile salts, and toxins. Bacteria are responsible for intestinal gas, or **flatus.**
44. The gastroileal reflex moves materials from the ileum into the cecum while you eat. Distension of the stomach and duodenum stimulates **mass movements** of materials from the transverse colon through the rest of the large intestine and into the rectum. Muscular sphincters control the passage of fecal material to the anus. Distension of the rectal wall triggers the **defecation reflex.** *(Figure 24–26)*

24-8 Digestion is the mechanical and chemical alteration of food that allows the absorption and use of nutrients p. 903

45. The digestive system first breaks down the physical structure of the ingested material and then disassembles the component molecules into smaller fragments by *hydrolysis*. *(Spotlight Figure 24–27; Table 24–1)*
46. Salivary and pancreatic amylases break down complex carbohydrates into *disaccharides* and *trisaccharides*. These in turn are broken down into *monosaccharides* by enzymes at the epithelial surface. The monosaccharides are then absorbed by the intestinal epithelium by facilitated diffusion or cotransport. *(Spotlight Figure 24–27)*
47. *Triglycerides* are emulsified into lipid droplets that interact with bile salts to form **micelles.** The fatty acids and monoglycerides resulting from the action of pancreatic lipase diffuse from the micelles across the intestinal epithelium. Triglycerides are then synthesized and released into the interstitial fluid, for transport to the general circulation by way of the lymphatic system. *(Spotlight Figure 24–27)*
48. Protein digestion involves a low pH (which destroys tertiary and quaternary structure), the gastric enzyme pepsin, and various pancreatic proteases. Peptidases liberate amino acids that are absorbed and exported to interstitial fluid. *(Spotlight Figure 24–27)*
49. About 2000 mL of water is ingested each day, and digestive secretions provide another 7200 mL. Nearly all is reabsorbed by osmosis. *(Figure 24–28)*
50. Various processes, including diffusion, cotransport, and carrier-mediated and active transport, are responsible for the movements of cations (sodium, calcium, potassium, and so on) and anions (chloride, iodide, bicarbonate, and so on) into epithelial cells. *(Table 24–2)*
51. The **water-soluble vitamins** (except B_{12}) diffuse easily across the digestive epithelium. **Fat-soluble vitamins** are enclosed within fat droplets and absorbed with the products of lipid digestion. *(Table 24–2)*

24-9 Many age-related changes affect digestion and absorption p. 909

52. Age-related changes include a thinner and more fragile epithelium due to a reduction in epithelial stem cell divisions, weaker peristaltic contractions as smooth muscle tone decreases, the effects of cumulative damage, increased cancer rates, and increased dehydration.

24-10 The digestive system is extensively integrated with other body systems p. 909

53. The digestive system has extensive anatomical and physiological connections to the nervous, endocrine, cardiovascular, and lymphatic systems. *(Figure 24–29)*

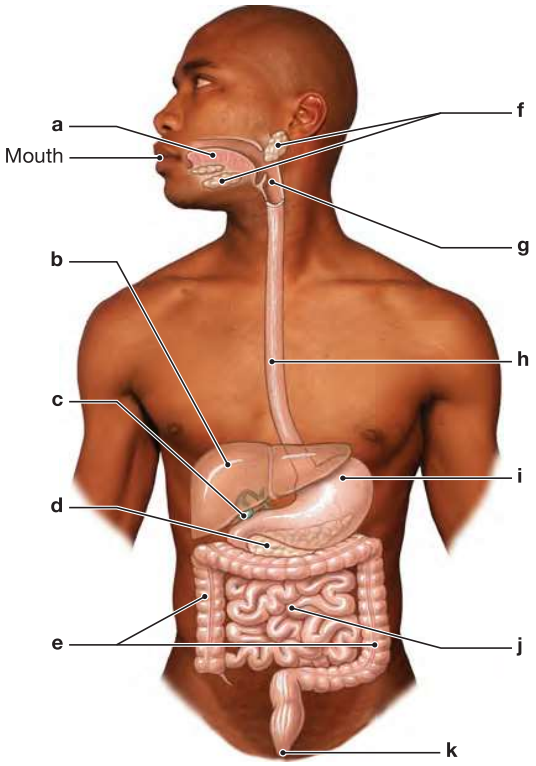
Review Questions

See the blue Answers tab at the back of the book.

LEVEL 1 Reviewing Facts and Terms

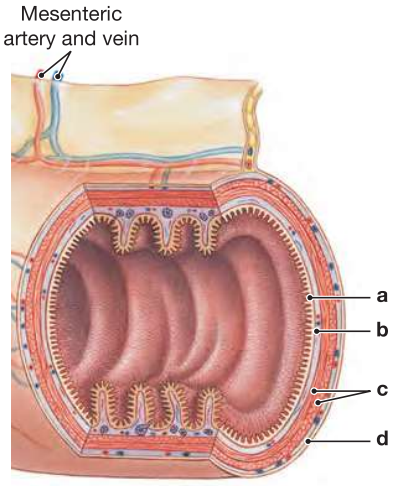
1. The enzymatic breakdown of large molecules into their basic building blocks is called
   (a) absorption. **(b)** secretion.
   (c) mechanical digestion. **(d)** chemical digestion.
2. The outer layer of the digestive tract is known as the
   (a) serosa. **(b)** mucosa.
   (c) submucosa. **(d)** muscularis.
3. Double sheets of peritoneum that provide support and stability for the organs of the peritoneal cavity are the
   (a) mediastina. **(b)** mucous membranes.
   (c) omenta. **(d)** mesenteries.
4. A branch of the portal vein, hepatic artery, and tributary of the bile duct form
   (a) a liver lobule. **(b)** the sinusoids.
   (c) a portal area. **(d)** the hepatic duct.
   (e) the pancreatic duct.

5. Label the digestive system structures in the following figure.



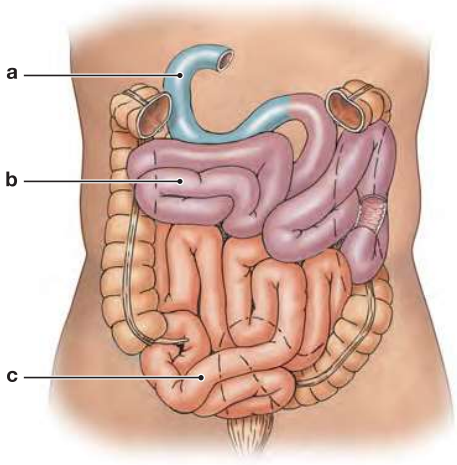
(a) _____ (g) _____
(b) _____ (h) _____
(c) _____ (i) _____
(d) _____ (j) _____
(e) _____ (k) _____
(f) _____

6. Label the four layers of the digestive tract in the following figure.



(a) _____ (c) _____
(b) _____ (d) _____

7. Most of the digestive tract is lined by _____ epithelium.
   (a) pseudostratified ciliated columnar
   (b) cuboidal
   (c) stratified squamous
   (d) simple
   (e) simple columnar
8. Regional movements that occur in the small intestine and function to churn and fragment the digestive material are called
   (a) segmentation.
   (b) pendular movements.
   (c) peristalsis.
   (d) mass movements.
   (e) mastication.
9. Bile release from the gallbladder into the duodenum occurs only under the stimulation of
   (a) cholecystokinin.
   (b) secretin.
   (c) gastrin.
   (d) enteropeptidase.
10. Label the three segments of the small intestine in the following figure.



(a) _____
(b) _____
(c) _____

11. The major function(s) of the large intestine is (are)
    (a) reabsorption of water and compaction of feces.
    (b) absorption of vitamins liberated by bacterial action.
    (c) storage of fecal material prior to defecation.
    (d) a, b, and c.
12. Vitamins generated by bacteria in the colon are
    (a) vitamins A, D, and E.
    (b) B complex vitamins and vitamin C.
    (c) vitamin K, biotin, and pantothenic acid.
    (d) niacin, thiamine, and riboflavin.
13. The final enzymatic steps in the digestive process are accomplished by
    (a) brush border enzymes of the intestinal microvilli.
    (b) enzymes secreted by the stomach.
    (c) enzymes secreted by the pancreas.
    (d) the action of bile from the gallbladder.
14. What are the six steps of digestion?

15. Name and describe the layers of the digestive tract, proceeding from the innermost layer to the outermost layer.
16. What three basic mechanisms regulate the activities of the digestive tract?
17. What are the three phases of swallowing, and how are they controlled?
18. What are the primary digestive functions of the pancreas, liver, and gallbladder?
19. Which hormones produced by duodenal enteroendocrine cells effectively coordinate digestive functions?
20. What are the three primary functions of the large intestine?
21. What two positive feedback loops are involved in the defecation reflex?

LEVEL 2 Reviewing Concepts

22. During defecation,
    (a) stretch receptors in the rectal wall initiate a series of peristaltic contractions in the colon and rectum.
    (b) stretch receptors in the rectal wall activate parasympathetic centers in the sacral region of the spinal cord.
    (c) the internal anal sphincter relaxes while the external anal sphincter contracts.
    (d) all of these occur.
    (e) only a and b occur.
23. *Increased* parasympathetic stimulation of the intestine would result in
    (a) decreased motility.
    (b) decreased secretion.
    (c) decreased sensitivity of local reflexes.
    (d) decreased segmentation.
    (e) none of these.
24. A drop in pH below 4.5 in the duodenum stimulates the secretion of
    (a) secretin.
    (b) cholecystokinin.
    (c) gastrin.
    (d) a, b, and c.
25. Through which layers of a molar would an oral surgeon drill to perform a root canal (removal of the alveolar nerve in a severely damaged tooth)?
26. How is the epithelium of the stomach protected from digestion?
27. How does each of the three phases of gastric secretion promote and facilitate gastric control?
28. Nutritionists have found that after a heavy meal, the pH of blood increases slightly, especially in the veins that carry blood away from the stomach. What causes this increase in blood pH?

LEVEL 3 Critical Thinking and Clinical Applications

29. Some people with gallstones develop pancreatitis. How could this occur?
30. Harry is suffering from an obstruction in his colon. He notices that when he urinates, the color of his urine is much darker than normal, and he wonders if there is any relationship between the color of his urine and his intestinal obstruction. What would you tell him?
31. A condition known as lactose intolerance is characterized by painful abdominal cramping, gas, and diarrhea. The cause of the problem is an inability to digest the milk sugar lactose. How would this cause the observed signs and symptoms?
32. Recently, more people have turned to surgery to help them lose weight. One form of weight control surgery involves stapling a portion of the stomach shut, creating a smaller volume. How would such a surgery result in weight loss?

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- Control of the Digestive System
- Motility
- Secretion
- Digestion and Absorption