

The Integumentary System 5

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Would you be enticed by an advertisement for a coat that is waterproof, stretchable, washable, and permanent press, that automatically repairs small rips and burns, and that is guaranteed to last a lifetime with reasonable care? It sounds too good to be true, but you already have such a coat—your skin. The skin and its appendages (sweat glands, oil glands, hair, and nails) serve a number of functions. Together, these organs make up the **integumentary system** (in-teg’u-men’tar-e; “covering”).

Freeze fracture section through the hair follicles in the skin (colored SEM).

THE SKIN AND THE HYPODERMIS

- Name the tissue types that compose the epidermis and the dermis.
- Name the layers of the epidermis and the dermis, and describe the structure and functions of each layer.
- Describe the structure and function of the hypodermis.
- Describe the factors that contribute to skin color.

The **skin (integument)** and its appendages are the first *organs* discussed in this book. Recall from Chapter 1 that an organ consists of tissues working together to perform certain functions. Although the skin is less complex than most other organs, it is still an architectural marvel. It is also the largest of all the organs, accounting for about 7% of total body weight.

The skin varies in thickness from 1.5 to 4 mm or more in different regions of the body. It has two distinct regions (**Figure 5.1** and **Figure 5.2**): The superficial region is a thick

epithelial tissue, the *epidermis*. Deep to the epidermis is the *dermis*, a fibrous connective tissue. Just deep to the skin lies a fatty layer called the *hypodermis*, composed of loose areolar connective tissue and adipose tissue. Although the hypodermis is not part of the integumentary system, it shares some of the skin's functions and is therefore described in this chapter.

The skin performs a variety of functions:

1. **Protection.** Skin cushions and insulates the deeper body organs and protects the body from bumps, scrapes, and cuts. Skin also protects the body from chemicals and invading microorganisms. The epidermis is waterproof, preventing unnecessary loss of water across the body surface, and cells in the epidermal layer produce pigment to protect the skin from the harmful effects of ultraviolet (UV) radiation.
2. **Body temperature regulation.** The skin's rich capillary networks and sweat glands regulate the loss of heat from the body, helping to control body temperature.

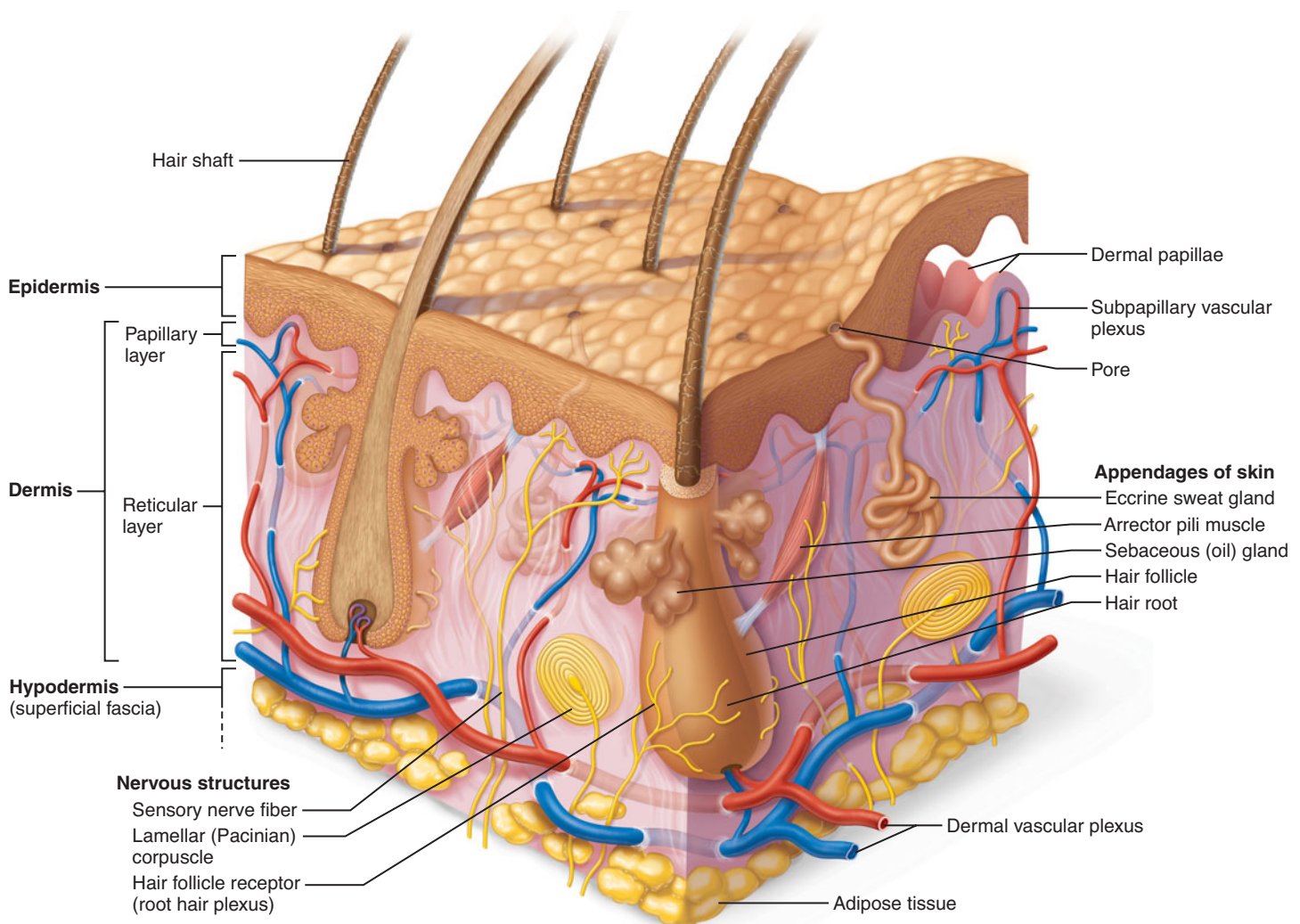


FIGURE 5.1 Skin structure. Generalized three-dimensional diagram of the skin, accessory structures, and the underlying hypodermis. The various nerve endings and sensory receptors are discussed in Chapter 14.

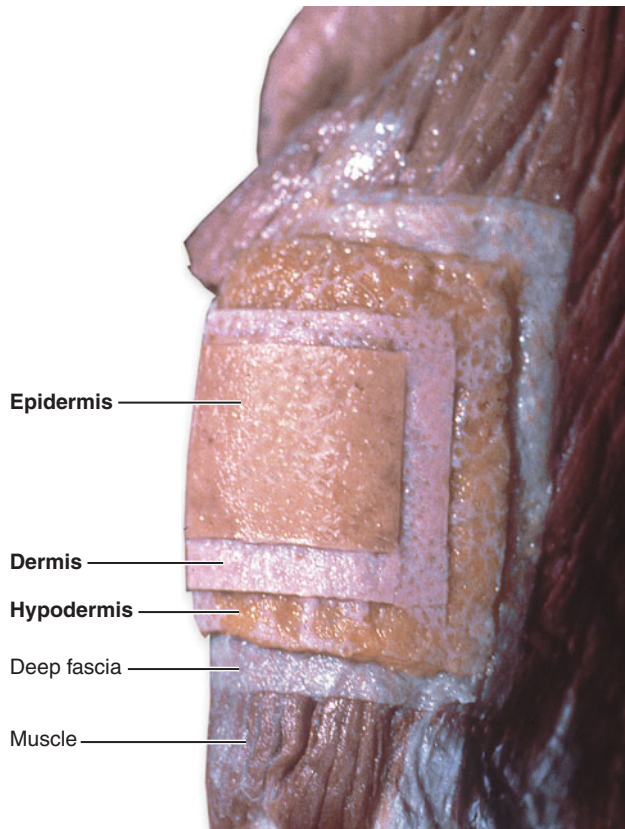


FIGURE 5.2 Gross structure of skin and underlying tissues. Photo of the upper arm of a cadaver.

3. **Excretion.** The skin acts as a miniature excretory system when urea, salts, and water are lost through sweat.
4. **Production of vitamin D.** The epidermal cells use UV radiation to synthesize vitamin D, a molecule necessary for absorbing calcium from the digestive tract.
5. **Sensory reception.** The skin contains sense organs called *sensory receptors* that are associated with nerve endings. By sensing touch, pressure, temperature, and pain, these receptors keep us aware of conditions at the body surface.

You will explore the skin's functions in greater detail as you explore its anatomy.

check your understanding

1. What is the meaning of the word roots *epi-*, *hypo-*, and *derm*?
2. Name the five major functions of skin.

For answers, see Appendix B.

Epidermis

The **epidermis** (ep'i-der'mis; "on the skin") is a keratinized stratified squamous epithelium that contains four distinct types of cells: *keratinocytes*, *melanocytes*, *tactile epithelial cells*, and *dendritic cells* (Figure 5.3).

Keratinocytes (ke-rat'i-no-sīts), the most abundant epidermal cell, produce **keratin**, a tough fibrous protein that gives the epidermis its protective properties. However, keratinocytes do more than just provide physical and mechanical protection. They also produce antibiotics and enzymes that detoxify the harmful chemicals to which our skin is exposed.

Tightly connected to one another by a large number of desmosomes, keratinocytes arise in the deepest part of the epidermis from cells that undergo almost continuous mitosis. As these cells are pushed up by the production of new cells beneath them, they make the keratin that eventually fills their cytoplasm. By the time they approach the skin surface, they are dead, flat sacs completely filled with keratin. Millions of these dead cells rub off every day, giving us an entirely new epidermis every 35 to 45 days—the average time from the birth of a keratinocyte to its final wearing away. Where the skin experiences friction, both cell production and keratin formation are accelerated.

The other epidermal cell types are sparsely distributed among the keratinocytes. We will discuss these cells and their functions as we examine the layers of the epidermis.

Layers of the Epidermis

Variation in the thickness of the epidermis determines whether skin is thick or thin. In **thick skin**, which covers the palms and soles, the epidermis consists of five layers, or *strata* (stra'tah; "bed sheets") (Figure 5.4). In **thin skin**, which covers the rest of the body, only four strata are present.

Stratum Basale (Basal Layer) The **stratum basale** (stra'tum ba-sal'e), the deepest epidermal layer, is firmly attached to the underlying dermis along a wavy borderline. Also called the *stratum germinativum* (jer-mī-na'te'vum; "germinating layer"), it consists of a single row of cells, mostly stem cells representing the youngest keratinocytes (see Figure 5.3). These cells divide rapidly, and many mitotic nuclei are visible. **Tactile epithelial cells**, or Merkel cells, are distributed sparsely among the keratinocytes. Each hemisphere-shaped tactile epithelial cell is intimately associated with a disclike sensory nerve ending and functions as a receptor for touch.

About 10% to 25% of the cells in the stratum basale are spider-shaped **melanocytes** (mel'ah-no-sīts; "melanin cells"), which make the dark skin pigment **melanin** (mel'ah-nin; "black"). Melanin is made in membrane-walled granules and then transferred through the cell processes (the "spider-legs") to nearby keratinocytes. Consequently, the basal keratinocytes contain more melanin than do the melanocytes themselves. This melanin clusters on the superficial side of keratinocytes between the incoming radiation and the cell nuclei, thus shielding the cell nuclei from UV rays, which can damage DNA and cause cancer (see p. 116). In light-skinned people, the melanin is digested by lysosomes in cells a short distance above the basal layer. In dark-skinned people, no such digestion occurs, so melanin occupies keratinocytes throughout the epidermis. Although dark-skinned people have a darker melanin, more granules, and more pigment in

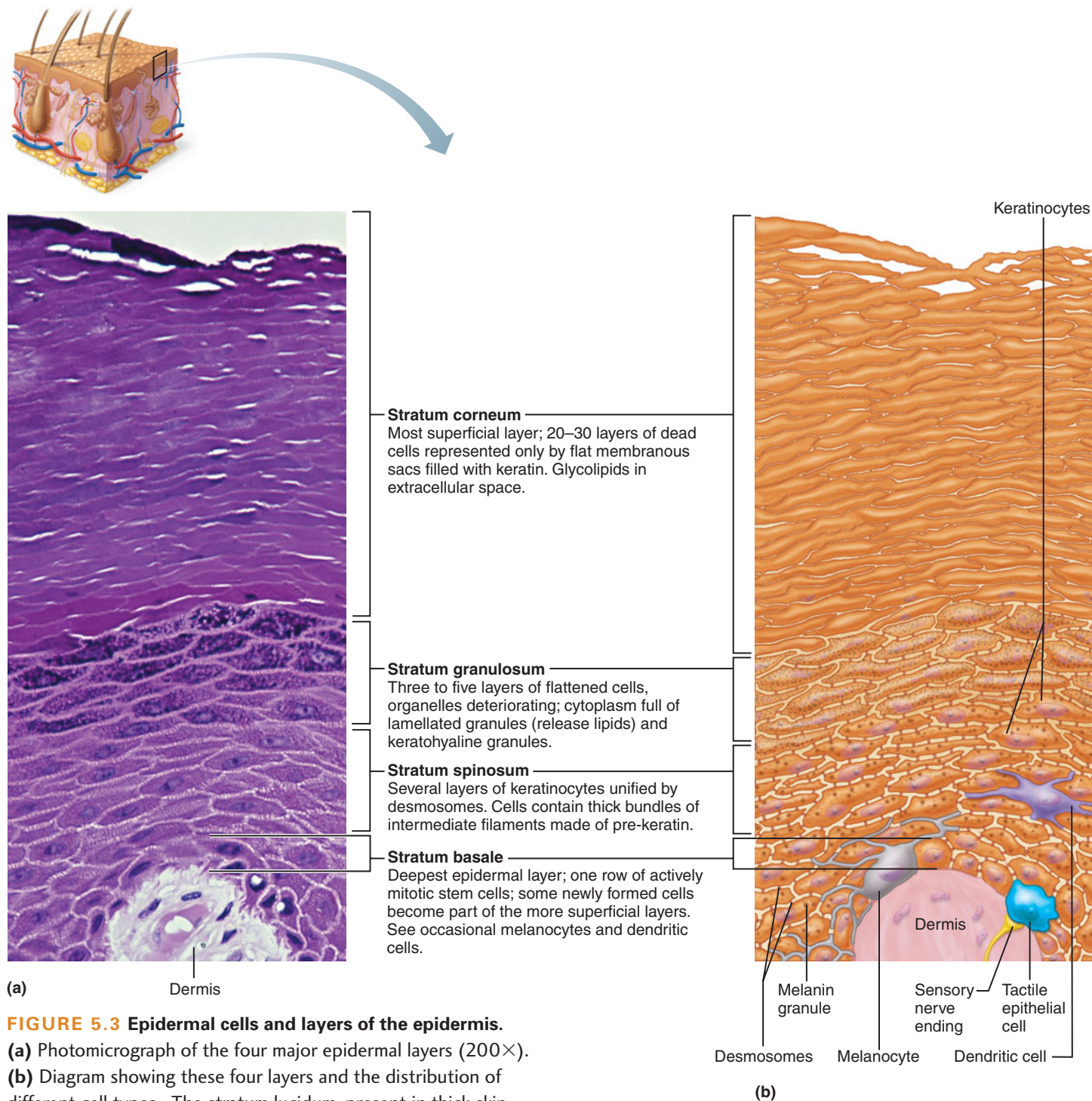


FIGURE 5.3 Epidermal cells and layers of the epidermis.

(a) Photomicrograph of the four major epidermal layers (200X).

(b) Diagram showing these four layers and the distribution of different cell types. The stratum lucidum, present in thick skin, is not illustrated here.

each melanocyte, they do *not* have more melanocytes in their skin. In all but the darkest people, melanocytes respond to ultraviolet radiation (UVR) by increasing the production of melanin and increasing its transfer to keratinocytes, the protective response we know as suntanning.

The role of melanocytes in skin pigmentation has long been recognized. More recently it has been found that these

cells also secrete a variety of signaling molecules in response to ultraviolet radiation that act to modulate the immune response in the skin. These molecules influence the inflammatory response and may have other regulatory functions.

Stratum Spinosum (Spiny Layer) The **stratum spinosum** (spi-no'sum) is several cell layers thick (see Figure 5.3). Mitosis occurs here, but less often than in the basal layer. This

layer gets its name from the many spinelike extensions of its keratinocytes, as seen in typical histological slides. However, these spines do not exist in living cells: They are artifacts created during tissue preparation, when the cells shrink while holding tight at their many desmosomes. Cells of the stratum spinosum contain thick bundles of intermediate filaments, which consist of a tension-resisting protein pre-keratin.

Scattered among the keratinocytes of the stratum spinosum are **dendritic cells**. These star-shaped cells are part of the immune system (see Chapter 21). Dendritic cells police the outer body surface, using receptor-mediated endocytosis (p. 28) to take up foreign proteins (antigens) that have invaded the epidermis. Then they leave the skin and travel to a nearby lymph node and initiate an immune response to all foreign cells that carry the antigen (see the discussion of lymphocyte activation in Chapter 21).

Stratum Granulosum (Granular Layer) The thin **stratum granulosum** (gran'u-lo"sum) consists of one to five layers of flattened keratinocytes. Along with abundant pre-keratin intermediate filaments, these cells also contain *keratohyalin granules* and *lamellated granules*, thus its name—granular layer. The keratohyalin (ker'ah-to-hi"ah-lin) granules help form keratin in the more superficial layers, as described shortly. The lamellated granules (lam"i-la-ted; "plated") contain a waterproofing glycolipid that is secreted into the extracellular space and plays a major role in slowing water loss across the epidermis. Furthermore, the plasma membranes of the cells thicken so that they become more resistant to destruction. It is as though the keratinocytes "toughen up" to make the outer layer the strongest skin region.

Like all epithelia, the epidermis relies on capillaries in the underlying connective tissue (the dermis) for its nutrients. Above the stratum granulosum, the epidermal cells are too far from the dermal capillaries to receive nourishment, so they die, a completely normal occurrence.

Stratum Lucidum (Clear Layer) The **stratum lucidum** (lu'si-dum) occurs in thick skin (see Figure 5.4) but not in thin skin. It is also named the *transition zone*. Appearing through the light microscope as a thin translucent band, it consists of a few rows of flat, dead keratinocytes. Electron microscopy reveals that its cells are identical to those at the bottom of the next layer, the stratum corneum.

Stratum Corneum (Horny Layer) The most external part of the epidermis, the **stratum corneum** (kor'ne-um; *cornu* = horn), is many cells thick. It is much thicker in thick skin than in thin skin (see Figure 5.4). Its dead keratinocytes are flat sacs completely filled with keratin because their nuclei and organelles disintegrated upon cell death. Keratin consists of the pre-keratin intermediate filaments embedded in a "glue" from the keratohyalin granules. Both the keratin and the thickened plasma membranes of cells in the stratum corneum protect the skin against abrasion and penetration. Additionally, the glycolipid between its cells keeps this layer waterproof. It is amazing that a layer of dead cells can still perform so many functions!

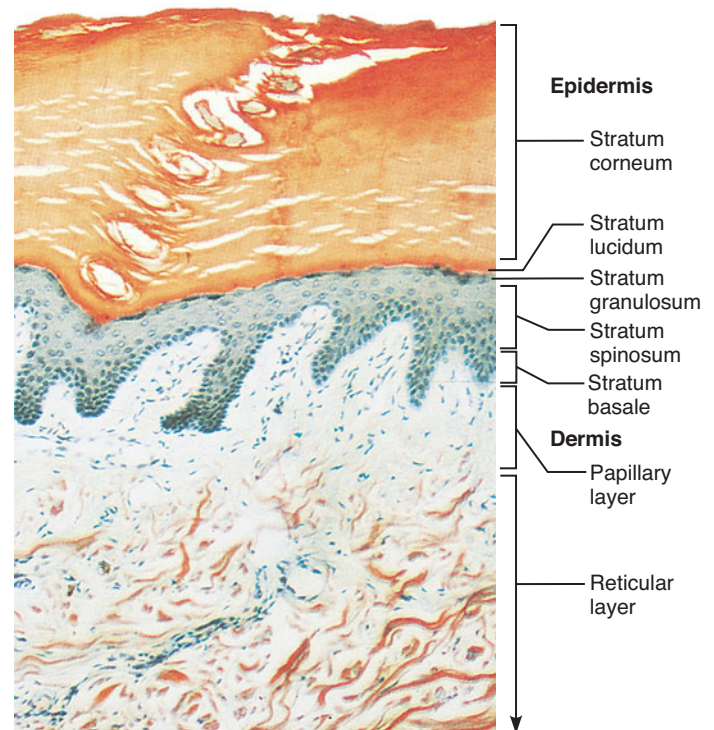


FIGURE 5.4 Thick skin. Photomicrograph of the epidermis and dermis of thick skin (145 \times). Notice the thicker stratum corneum and the additional epidermal layer, the stratum lucidum. (See *A Brief Atlas of the Human Body*, Second Edition, Plate 37.)

The cells of the stratum corneum are shed regularly. These cells are the dandruff shed from the scalp and the flakes that come off dry skin. The average person sheds 18 kg (40 pounds) of these skin flakes in a lifetime. These shed cells are replaced by cells from the deeper layers. The next time you hear the saying "Beauty is only skin deep," consider that nearly everything you see when you look at someone is dead!

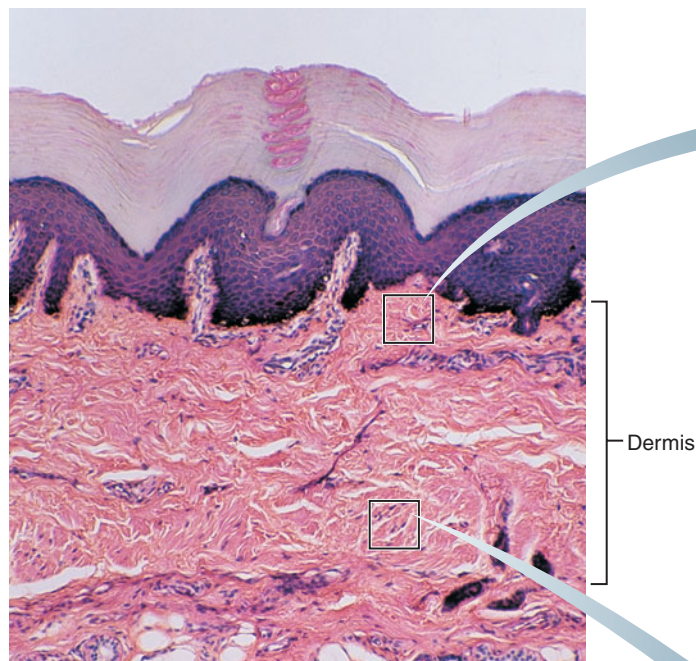
SKIN'S RESPONSE TO FRICTION Persistent friction (from a poorly fitting shoe, for example) causes a thickening of the epidermis called a **callus**. Short-term but severe friction (from using a hoe, for example) can cause a **blister**, the separation of the epidermis from the dermis by a fluid-filled pocket. A large blister is called a **bulla** (bul'ah; "bubble").



check your understanding

- Which layers of the epidermis contain living cells?
- Which functions of skin are performed by cells located in the epidermis?
- How does thick skin differ from thin skin in structure?

For answers, see Appendix B.



(a) Light micrograph of thick skin identifying the extent of the dermis, (100 \times)

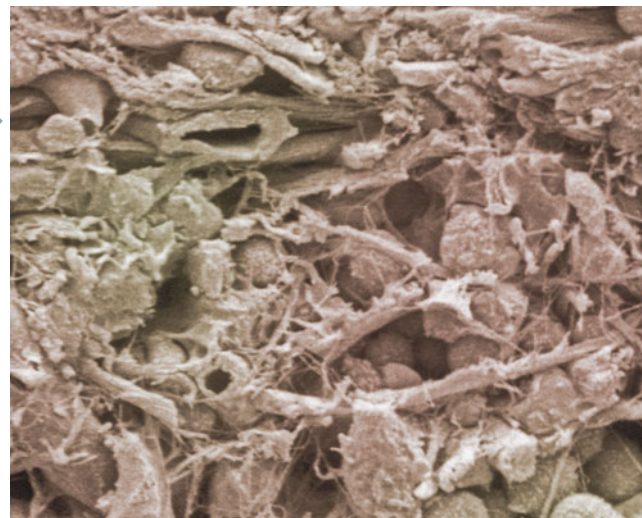
FIGURE 5.5 The two regions of the dermis. The superficial papillary layer consists of areolar connective tissue; the deeper reticular layer is dense irregular connective tissue.

Source: Kessel and Kardon/Visuals Unlimited.

Dermis

The **dermis**, the second major region of the skin, is a strong, flexible connective tissue. The cells of the dermis are typical of any connective tissue proper: fibroblasts, macrophages, mast cells, and scattered white blood cells (see pp. 78–79). The fiber types—collagen, elastic, and reticular—also are typical. The dermis binds the entire body together like a body stocking. It is your “hide” and corresponds to animal hides used to make leather products.

The dermis is richly supplied with nerve fibers and blood vessels. The nerve supply of the dermis (and epidermis) is discussed in Chapter 14. The dermal blood vessels consist of two vascular plexuses (a plexus is a network of converging and diverging vessels) (Figure 5.1). The deep **dermal plexus** is located between the hypodermis and the dermis. It nourishes the hypodermis and the structures located within the deeper portions of the dermis. The more superficial **subpapillary plexus**, located just below the dermal papillae, supplies the more superficial dermal structures, the dermal papillae (described below), and the epidermis. Dermal blood vessels do more than just nourish the dermis and overlying epidermis; they also perform a critical role in temperature regulation. These vessels are so extensive that they can hold 5% of all blood in the body. When internal organs need more blood or more heat, nerves stimulate the dermal vessels to constrict, shunting more blood into the general circulation and making it available to the internal



(b) Papillary layer of dermis, SEM (570 \times)



(c) Reticular layer of dermis, SEM (430 \times)

organs. By contrast, on hot days the dermal vessels engorge with warm blood, cooling the body by radiating heat away from it.

THE PATCH DRUG DELIVERY SYSTEM The transdermal patch is designed so that drug molecules diffuse through the epidermis to the blood vessels in the dermal layer. A typical patch works well for small lipid-soluble molecules (for example, estrogen, nitroglycerin, and nicotine) that can make their way between epidermal cells. Patches are being developed to deliver larger molecules or molecules that are water soluble, such as insulin and vaccines. In one new patch design, thin, hollow needles extend from the reservoir of the drug, through the epidermis, and down to the dermis, deep enough to deliver drugs to the subpapillary plexus but not so deep as to reach the nerve endings.



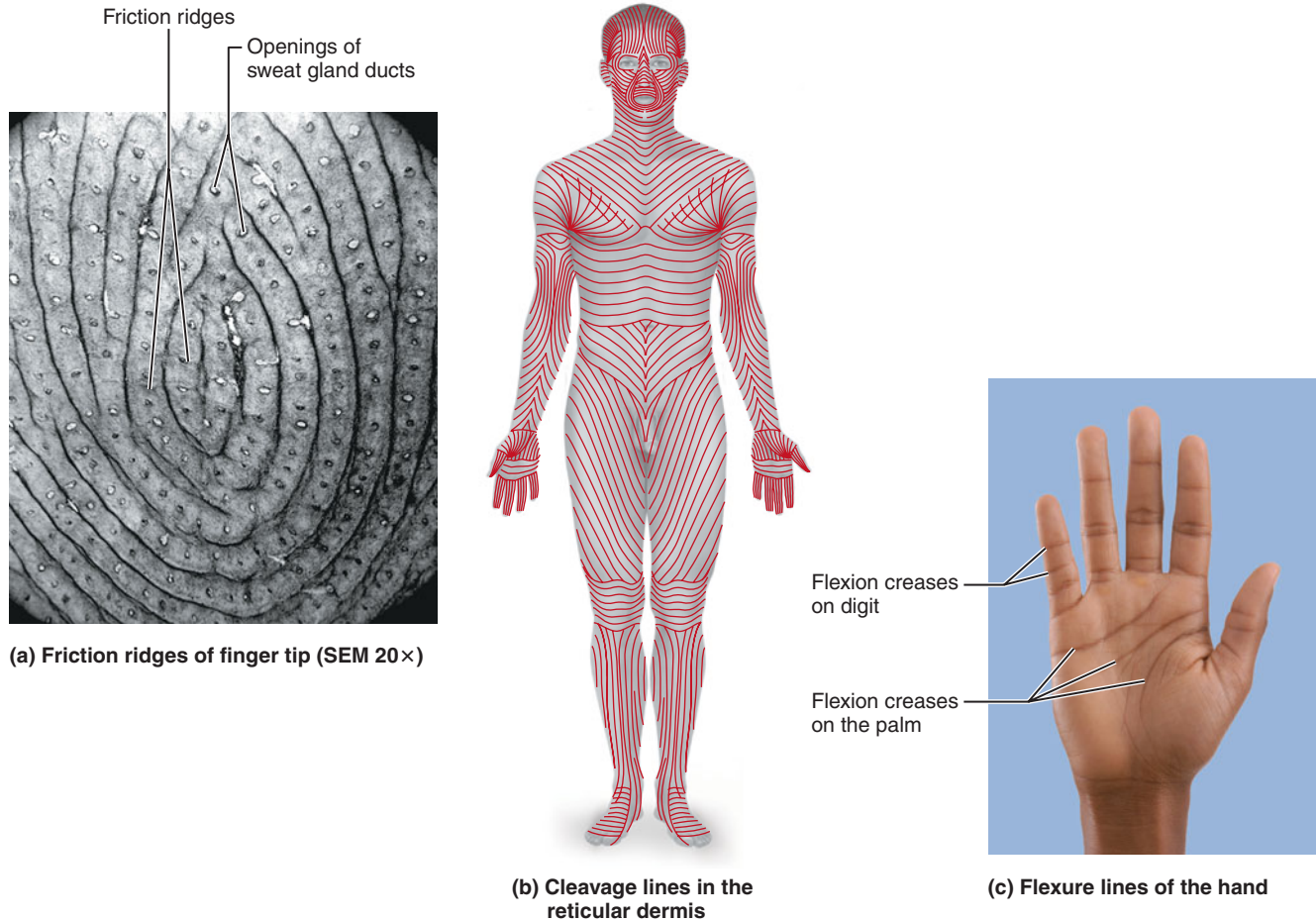


FIGURE 5.6 Dermal modifications. (a) Epidermal ridges superficial to the dermal papillary ridges form the friction ridges. Sweat ducts opening along the crests of the ridges are responsible for fingerprints. (b) Cleavage lines represent the separations between bundles of collagen fibers in the reticular layer of the dermis. (c) Flexure lines form where dermis is closely attached to the underlying fascia.

Source: Kessel and Kardon/Visuals Unlimited.

The dermis has two layers: papillary and reticular (Figure 5.4 and Figure 5.5). The **papillary** (pap'y-lar-e) **layer**, the superficial 20% of the dermis, is areolar connective tissue containing very thin collagen and elastic fibers. It includes the **dermal papillae** (pah-pil'le; “nipples”), fingerlike projections that extend into the overlying epidermis. These projections of the dermal papillae into the epidermis increase the surface area for exchange of gases, nutrients, and waste products between these layers. Recall that the epidermis is avascular and depends on the diffusion of these materials from the underlying dermis. The interdigitation of these layers also strengthens the dermal-epidermal junction and thus reduces blister formation.

On the palms of the hands and the soles of the feet, the dermal papillae lie atop larger mounds called *dermal ridges*. These elevate the overlying epidermis into *epidermal ridges* or *friction ridges*, which create fingerprints, palmprints, and footprints (Figure 5.6a). Epidermal ridges increase friction and enhance the gripping ability of the hands and feet.

Patterns of these ridges are genetically determined and unique to each person. Because *sweat pores* open along the crests of the friction ridges, they leave distinct fingerprints on almost anything they touch. Thus, fingerprints are “sweat films.”

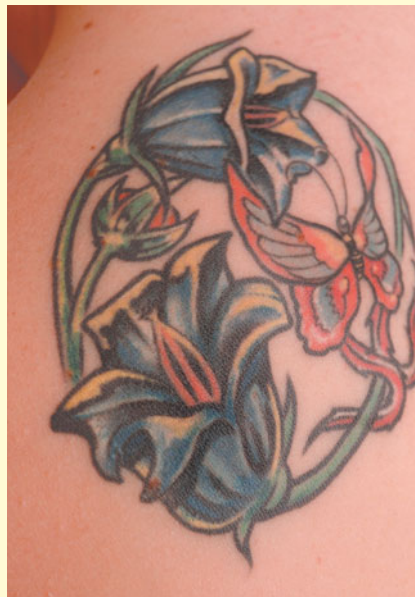
The deeper **reticular layer**, which accounts for about 80% of the thickness of the dermis, is dense irregular connective tissue. Its extracellular matrix contains thick bundles of interlacing collagen and elastic fibers that run in many different planes. However, most run parallel to the skin surface. The reticular layer is named for its networks of collagen fibers (*reticulum* = network); the name does not imply any special abundance of reticular fibers. Separations or less dense regions between the collagen bundles form the *cleavage lines* or *tension lines* of the skin (Figure 5.6b). These invisible lines occur over the entire body: They run longitudinally in the skin of the limbs and head and in circular patterns around the neck and trunk. A knowledge of cleavage lines is important to surgeons. Incisions made *parallel* to

a closer look

Tattoos

Tattooing—using a needle to deposit pigment in the skin dermis—was first practiced around 8000 B.C. Today, some men use tattoos to signal membership in a group such as the military or a street gang, and others use tattoos to express their individuality. In recent years, women have increasingly used tattoos not only for individual expression but also for cosmetic purposes. Application of permanent eyeliner and liplines account for more than 125,000 tattoos a year.

But what if the tattoo becomes unfashionable or the pigment migrates? Until very recently, once you had a tattoo, you were essentially stuck with it because attempts at removal—dermabrasion, cryosurgery (freezing the tissue), or applying caustic chemicals—left nasty scars, some worse than the original tattoo. The lasers of today can usually remove “older” tattoos, that is, the blue and black designs applied a generation ago. The newer, colored tattoos are another matter entirely. They require the use of several different



lasers that emit light at different frequencies to remove each of the different pigments. Tattoo removal requires seven to nine treatments spaced about one month apart, and it can hurt as much as obtaining the tattoo in the first place. Even then,

tattoos can't always be eradicated completely: Green and yellow pigments are the most difficult to vaporize.

Tattoos present some other risks. The U.S. Food and Drug Administration does exert some control over the composition of the pigments used, but they are approved for application to the skin surface only—the safety of injecting them under the skin is not well established. Furthermore, state laws vary widely. Some states forbid the practice entirely, and others have no regulations at all. Tattooing involves needles and bleeding, training requirements for practitioners are minimal, and there is a risk of allergic reactions and bloodborne infectious diseases such as hepatitis. (Even if the practitioner uses sterile needles, they may be dipped into pigments that are contaminated with the blood of previous customers.) Even with the availability of laser removal techniques, it makes sense to consider the risks of getting a tattoo.

these lines tend to gape less and heal more readily than incisions made *across* cleavage lines.

The collagen fibers of the dermis give skin its strength and resilience. Thus, many jabs and scrapes do not penetrate this tough layer. Furthermore, elastic fibers in the dermis provide the skin with stretch-recoil properties. Extreme stretching of the skin, as occurs in obesity and pregnancy, can tear the collagen in the dermis. Such dermal tearing results in silvery white scars called *striae* (stri'e; “streaks”), which we know as “stretch marks.” The dermis is also the receptive site for the pigments used in tattoos (see [A Closer Look](#)).

From the deep part of the dermis arise the skin surface markings called *flexure lines*. Observe, for example, the deep skin creases on your palm (Figure 5.6c). These result from a continual folding of the skin, often over joints, where the dermis attaches tightly to underlying structures. Flexure lines are also visible on the wrists, soles, fingers, and toes.

DECUBITUS ULCER Bedsores, or **decubitus ulcers**, are a constant concern for patients who have impaired mobility. Bedridden elderly people are especially susceptible, as are wheelchair-bound individuals such as those with spinal cord injuries. A decubitus ulcer usually occurs over a bony prominence, such as the hip, sacrum, or heel. Constant pressure from body weight causes a localized breakdown of the skin due to reduction in blood supply. Tissue death can occur in 2–3 hours. Over time, the epidermis and papillary layer of the dermis are lost, and the damaged area shows an increase in collagen fibers. Without the protective covering of the epidermis, infectious agents can easily enter the body and cause serious and fatal complications.



Hypodermis

Just deep to the skin is the fatty **hypodermis** (hi"po-der'mis), from Greek words meaning "below the skin" (see Figures 5.1 and 5.2). This layer is also called the **superficial fascia** and the *subcutaneous layer* (sub"ku-ta'ne-us; from Latin words meaning "below the skin"). The hypodermis consists of both areolar and adipose connective tissue, but adipose tissue normally predominates. Besides storing fat, the hypodermis anchors the skin to the underlying structures (mostly to muscles), but loosely enough that skin can slide relatively freely over those structures. Such sliding ensures that many blows just glance off our bodies. The hypodermis is also an insulator: Because fat is a poor conductor of heat, it helps prevent heat loss from the body. The hypodermis thickens markedly with weight gain, but this thickening occurs in different body areas in the two sexes. In women, subcutaneous fat accumulates first in the thighs and breasts, whereas in men it first accumulates in the anterior abdomen as a "beer belly."

check your understanding

- How do the dermal blood vessels regulate body temperature?
- What type of tissue makes up (a) the papillary layer of the dermis, (b) the reticular layer of the dermis, and (c) the hypodermis?
- What types of cells are found in the dermis, and how does each contribute to the functions of skin?

For answers, see Appendix B.

Skin Color

Three pigments contribute to skin color: melanin, carotene, and hemoglobin. **Melanin**, the most important, is made from an amino acid called tyrosine. Present in several varieties, melanin ranges from yellow to reddish to brown to black. As noted earlier, melanin passes from melanocytes to keratinocytes in the stratum basale of the epidermis. The variations in skin color in humans result from differences in both the amount and type of melanin produced.

FRECKLES AND MOLES Both freckles and moles (nevi) are localized accumulations of melanin in the skin. In freckles, the increase in melanin is restricted to the basal layer of the epidermis. Freckles form as a result of repeated exposure to sunlight. Individuals with light complexions are more prone to freckle formation. Moles form when clusters of melanocytes are transformed into melanin-containing cells (nevus cells). These clusters are located in the basal layer of the epidermis and in the top layers of the dermis. Moles form shortly after birth and can appear anytime through young adulthood. They do not form as a result of ultraviolet exposure.



Carotene (kar'o-tēn) is a yellow-orange pigment that the body obtains from vegetable sources such as carrots and tomatoes. It tends to accumulate in the stratum corneum of the epidermis and in the fat of the hypodermis.

The pink hue of Caucasian skin reflects the crimson color of oxygenated **hemoglobin** (he'mo-glo"bin) in the capillaries of the dermis. Because Caucasian skin contains little melanin, the epidermis is nearly transparent and allows the color of blood to show through. Black-and-blue marks represent discolored blood that is visible through the skin. Such bruises, usually caused by blows, reveal sites where blood has escaped from the circulation and clotted below the skin. The general term for a clotted mass of escaped blood anywhere in the body is a **hematoma** (he"mah-to'mah; "blood swelling").

CYANOSIS When hemoglobin is poorly oxygenated, both the blood and the skin of Caucasians appear blue, a condition called **cyanosis** (si"ah-no'sis; "dark blue condition"). Skin often becomes cyanotic in people with heart failure or severe respiratory disorders. In dark-skinned people, the skin may be too dark to reveal the color of the underlying vessels, but cyanosis can be detected in the mucous membranes and nail beds.



The reasons for variations in the skin colors of different human populations are not fully understood. Theories explaining this variation reflect the fact that the sun's UV rays are both dangerous and essential. One hypothesis proposes that dark skin coloration evolved to eliminate the danger of skin cancer caused by UV radiation. An alternative hypothesis argues that sunlight's effects on levels of folic acid in the blood was the selective pressure for the evolution of dark skin in tropical areas. In light-skinned people, high levels of sunlight decrease levels of folic acid in the blood. Decreased folic acid levels in pregnant women increase the risk of neural tube defects in the developing fetus. The evolution of dark skin in the tropics protects blood folic acid levels and promotes healthy offspring.

The other factor influencing skin color is vitamin D production. UV rays stimulate the deep epidermis to produce vitamin D, a vital hormone required for the uptake of calcium from the diet and essential for healthy bones. Calcium, and thus adequate vitamin D, is also critical to the developing fetus.

Producing vitamin D poses no problem in the sunny tropics, but Caucasians in northern Europe receive little sunlight during the long, dark winter. Therefore, their colorless epidermis ensures that enough UV rays will penetrate for vitamin D production. Natural exposure to UV rays in these temperate regions is not sufficient to diminish folic acid levels. Most human skin color types evolved at intermediate latitudes (China, the Middle East, and so on) and are characterized by moderately brown skin tones that are neither "white" nor "black." Such skin is dark enough to provide some protection from sunlight's negative effects in the summer, especially

when it tans, yet light enough to allow vitamin D production in the moderate winters of intermediate latitudes. The trade-off between protection from the harmful effects of UV radiation and production of vitamin D may have been the selective pressure behind the evolution of skin coloration as early humans moved away from the tropics.

check your understanding

9. Which pigment causes the large variation in human skin color?
10. Should a dark-skinned person living in the northern latitudes have concerns about vitamin D production? Why or why not?

For answers, see Appendix B.

APPENDAGES OF THE SKIN

- Describe the structure of nails.
- List the parts of a hair and a hair follicle, and explain the function of each part.
- Compare the structure and location of oil and sweat glands.
- Compare eccrine and apocrine sweat glands.

Along with the skin itself, the integumentary system includes several derivatives of the epidermis. These **skin appendages** include nails, hair and hair follicles, sebaceous (oil) glands, and sweat glands. Although they derive from the epithelial cells of the epidermis, they all extend into the dermis.

Nails

A **nail** (Figure 5.7) is a scalelike modification of the epidermis that corresponds to the hoof or claw of other mammals. Fingernails are built-in tools that enable us to pick up small objects and scratch the skin when it itches. Nails are made of dead, keratinized cells. The **hard keratin** that predominates in hairs and nails differs from the **soft keratin** that is found in typical epidermal cells in two ways: (1) It is tougher and more durable, and (2) the cells of hard keratin do not flake off. Each nail has a distal **free edge**, a **body** (the visible attached part), and a **root** (the proximal part embedded in the skin). The nail rests on a bed of epidermis called the **nail bed**. This bed contains only the deeper layers of the epidermis, because the nail itself corresponds to the superficial keratinized layers.

Nails look pink because of the rich network of capillaries in the underlying dermis. At the root and the proximal end of the nail body, the bed thickens to form the **nail matrix**, the actively growing part of the nail. The matrix is so thick that the pink dermis cannot show through it. Instead, we see a white crescent, the **lunule** (lōō'nōōl'; "little moon"), under the nail's proximal region. The lateral and proximal borders of the nail are overlapped by skin folds called **nail folds**. The

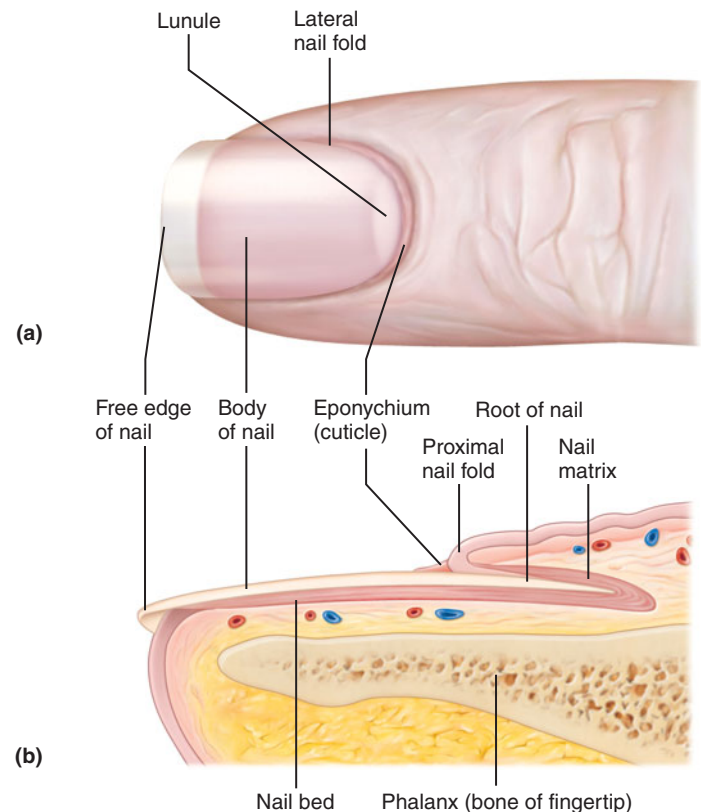


FIGURE 5.7 Structure of a nail. (a) Surface view of the distal part of a finger. (b) Sagittal section through a fingertip.

proximal nail fold projects onto the nail body as the cuticle or **eponychium** (ep"o-nik'e-um; "on the nail").

An *ingrown toenail* is just what its name implies, a nail whose growth pushes it painfully into the lateral nail fold. This happens when the nail grows crookedly, most commonly from the pressure of an ill-fitting shoe.

check your understanding

11. How does the keratin in hair and nails differ from the keratin in the epidermis?

For the answer, see Appendix B.

Hair and Hair Follicles

Together, hairs and their follicles form complex structural units (Figure 5.8). In these units, the *hairs* are the long filaments, and the *hair follicles* are tubular invaginations of the epidermis from which the hairs grow.

Hair

Although hair serves to keep other mammals warm, human body hair is far less luxuriant and less useful. Even so, hair is distributed over all our skin surface, except on the palms, soles, nipples, and parts of the external genitalia (the head of the penis, for example). The main function of this sparse body hair is to sense things that lightly touch the skin. The hair on the scalp protects the head against direct sunlight in summer and against

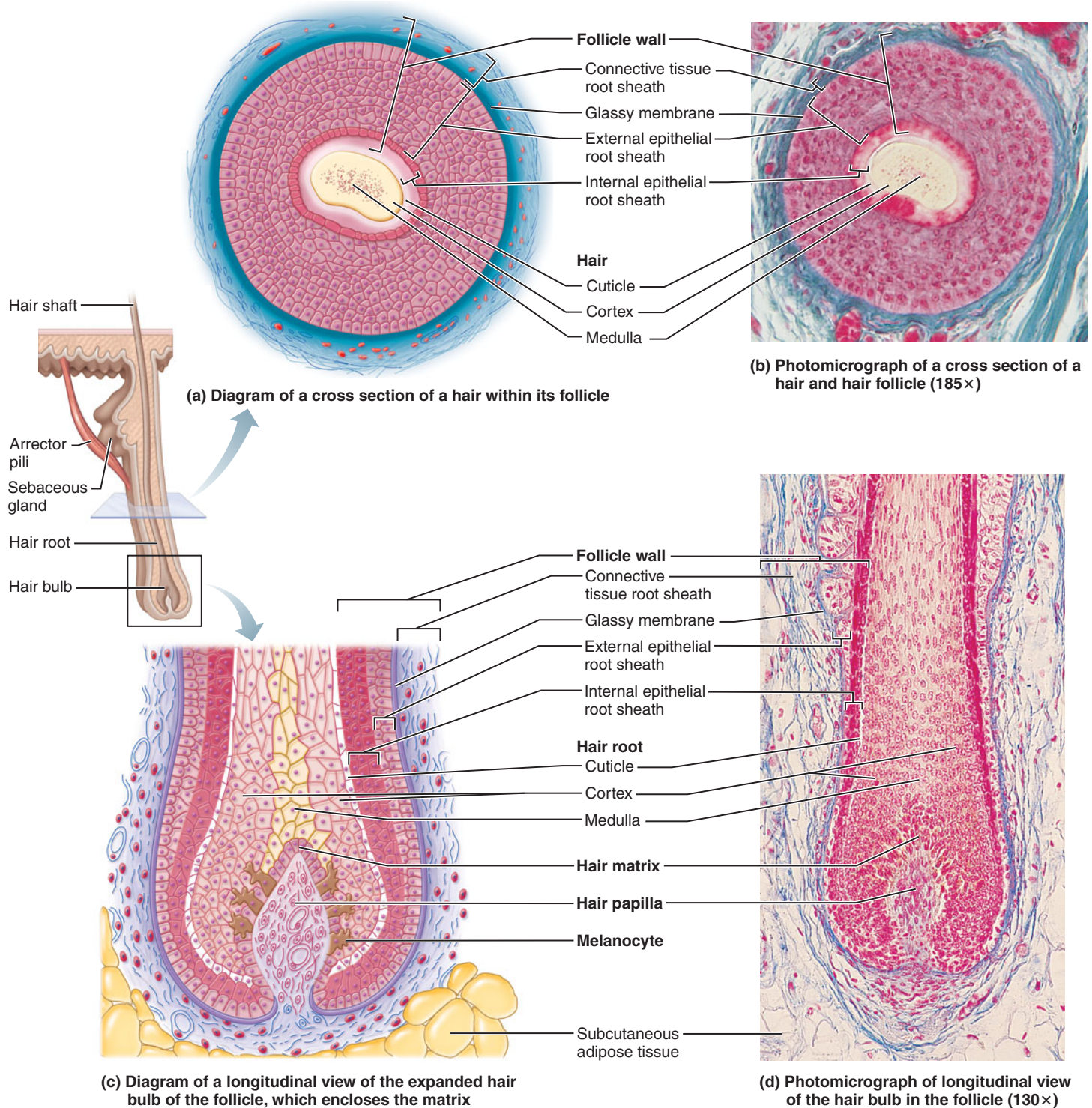


FIGURE 5.8 Structure of a hair and hair follicle.

heat loss on cold days. Eyelashes shield the eyes, and nose hairs filter large particles such as insects and lint from inhaled air.

A **hair** is a flexible strand made of dead cells filled with hard keratin. The chief parts of a hair are the **root**, the part embedded in the skin, and the **shaft**, the part that projects above the skin surface (Figure 5.8). If its shaft is flat and ribbon like in cross section, the hair is kinky; if oval in section, the hair is wavy; if perfectly round, the hair is straight.

A hair consists of three concentric layers of keratinized cells (Figure 5.8a and b). Its central core, the **medulla** (mĕ-dul'ah; "middle"), consists of large cells and air spaces. The medulla is absent in fine hairs. The **cortex**, surrounding the medulla, consists of several layers of flattened cells. The outermost **cuticle** is a single layer of cells that overlap one another from below like shingles on a roof. This shingle

pattern helps to keep neighboring hairs apart so that the hair does not mat. Hair conditioners smooth out the rough surface of the cuticle to make hair look shinier. The cuticle is the most heavily keratinized part of the hair, providing strength and keeping the inner layers tightly compacted. Because it is subjected to the most abrasion, the oldest part of the hair cuticle tends to wear away at the tip of the hair shaft. The abrasion causes the keratin fibrils in the cortex and medulla to frizz out, creating “split ends.”

Hair pigment is made by melanocytes at the base of the hair follicle (shown in Figure 5.8c) and is transferred to the cells of the hair root. Different proportions of two types of melanin (black-brown and yellow-rust in color) combine to produce all the common hair colors—black, brown, red, and blond. Graying or whitening of hair results from a decrease in the production of melanin and from the replacement of melanin by colorless air bubbles in the hair shaft.

Hair Follicles

Hair follicles extend from the epidermal surface into the dermis. The deep end of the follicle is expanded, forming a **hair bulb** (Figure 5.8c). A knot of sensory nerve endings wraps around each hair bulb to form a **hair follicle receptor** or **root hair plexus** (see Figure 5.1). Bending the hair shaft stimulates these nerve endings. Hairs are thus exquisite touch receptors. (Test this by lightly running your hand over the hairs on your forearm.)

A nipplelike bit of the dermis, the **connective tissue papilla (hair papilla)**, protrudes into each hair bulb. This papilla contains a knot of capillaries that deliver substances that stimulate hair growth and supply nutrients to the growing hair. If the hair papilla is destroyed by trauma, the follicle permanently stops producing hair.

The epithelial cells in the hair bulb just above the papilla make up the **hair matrix**. These matrix cells proliferate to form the hair shaft.

The wall of a hair follicle is actually a compound structure composed of an outer **connective tissue root sheath**, derived from the dermis, and an inner **epithelial root sheath**, derived from the epidermis (Figure 5.8a–d). The epithelial root sheath has two parts: Of these, the **internal root sheath** derives from the matrix cells, which it surrounds. The **external root sheath**, by contrast, is a direct continuation of the epidermis. The **glassy membrane** is where the epithelial root sheath meets the connective tissue root sheath; in essence it is the basement membrane of the epithelium of the follicle.

Epidermal stem cells are located in a bulge in the superficial region of the external root sheath. These stem cells give rise to the hair matrix cells, which form the hair shaft as well as new epidermal cells. Stem cells from this region have been cultured and grown to produce patches of epidermis derived from a patient’s own cells that are used to treat chronic wounds.

Associated with each hair follicle is a bundle of smooth muscle cells called an **arrector pili** muscle (ah-rek’tor pi’li; “raiser of the hair”) (see Figures 5.1 and 5.8). Each arrector pili runs from the most superficial part of the dermis to a

deep-lying hair follicle. When their arrector pili is relaxed, most hairs lie flat because most follicles lie at an oblique angle to the skin surface. Then, when this muscle contracts in response to cold or fear, the hair stands erect and the skin surface dimples, producing goose bumps. Even though such a “hair-raising” response is not very useful to humans, with our relatively sparse hair, it does keep fur-bearing animals warmer by trapping a layer of insulating air in their fur. Moreover, a frightened animal with its hair on end looks more formidable to an enemy.

Types and Growth of Hair

Hairs come in various sizes and shapes, but as a rule they can be classified as either **vellus** (vel’us; *vell* = wool, fleece) or **terminal**. The body hair of children and women is of the fine, short, vellus variety. The longer, coarser hair of everyone’s scalp is terminal hair. Terminal hairs also appear at puberty in the axillary (armpit) and pubic regions in both sexes and on the face, chest, arms, and legs of men. These terminal hairs grow under the influence of male sex hormones called **androgens**, of which *testosterone* is the most important type.

Hairs grow an average of 2 mm per week, although this rate varies widely among body regions and with sex and age. Each follicle goes through growth cycles. In each cycle, an active growth phase is followed by a resting phase, in which the hair matrix is inactive and the follicle atrophies somewhat. At the start of each active phase, the newly growing hair pushes out the old hair, causing it to be shed. The life span of hairs varies: On the scalp, the follicles stay active for an average of 4 years, so individual hairs grow quite long before being shed. On the eyebrows, by contrast, the follicles are active for only a few months, so the eyebrows never grow very long. Fortunately, the cycles of adjacent hair follicles on the scalp are not in synchrony; thus, humans shed only a small percentage of the hairs on the head at any one time.

Hair Thinning and Baldness

Given ideal conditions, hair grows fastest from the teen years to the 40s. When hairs are no longer replaced as quickly as they are shed, the hair thins. By age 60 to 65, both sexes usually experience some degree of balding. Coarse terminal hairs are replaced by vellus hairs, and the hair becomes increasingly wispy.

True baldness is different. The most common type, **male pattern baldness**, is a genetically determined, gender-influenced condition. It is thought to be caused by a gene that is not expressed until adulthood, at which time it changes the response of hair follicles to androgens. The hairs respond to androgens by increasingly shortening their growth cycles. The cycles become so short that many hairs never emerge from their follicles before being shed, and those that do emerge are fine vellus hairs that look like peach fuzz in the “bald” area. The drugs used to treat male pattern baldness either inhibit the production of androgens or increase blood flow to the skin and hair follicles. These treatments are only partly successful.

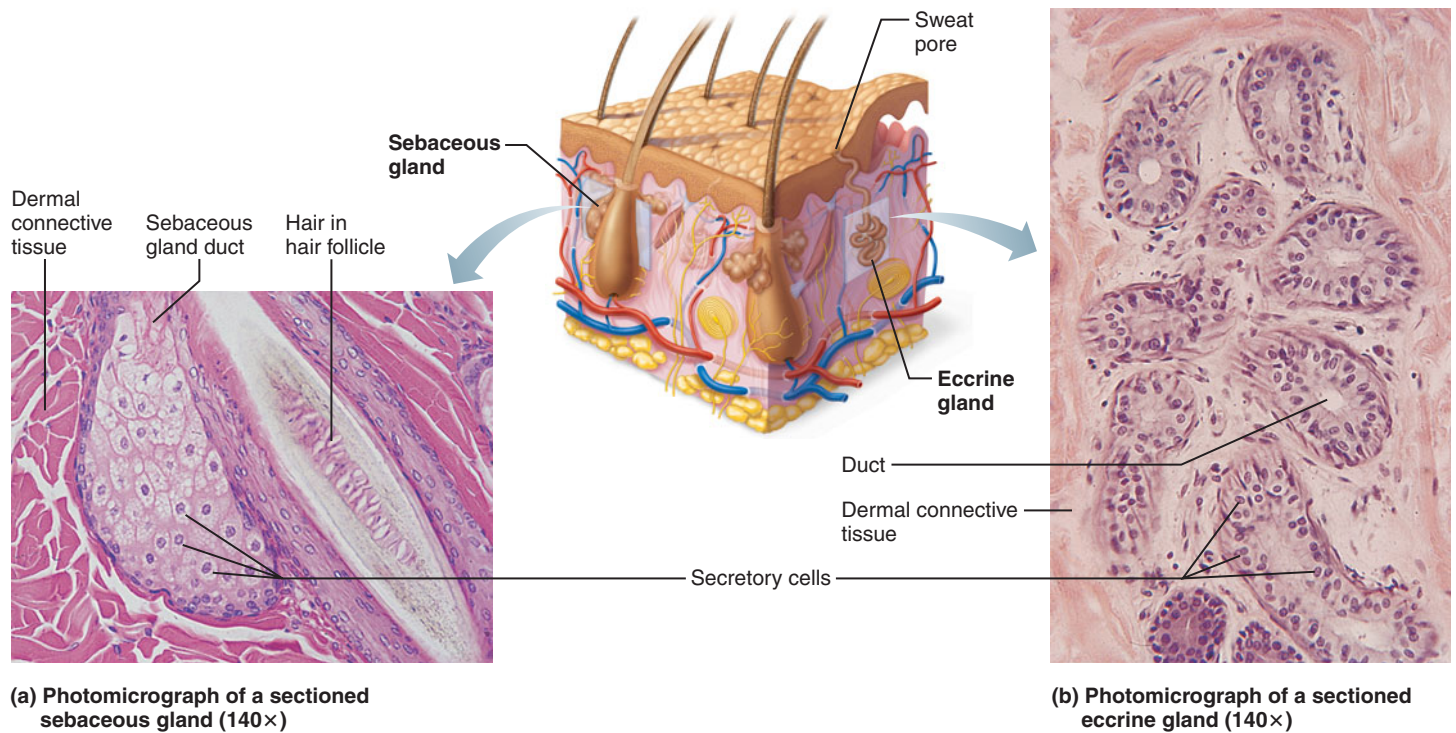


FIGURE 5.9 Skin glands.

CHEMOTHERAPY AND HAIR LOSS Chemotherapy drugs used to treat cancer target the most rapidly dividing cells in the body, thereby destroying many hair stem cells and causing hair loss. After chemotherapy stops, the hair can recover and regrow. However, hair loss due to severe burns, excessive radiation, or other factors that destroy the follicles is permanent.



check your understanding

- From what region of skin is the hair and hair follicle derived?
- Place the three layers of hair cells in order from deepest to most superficial.
- Why is hair loss due to chemotherapy temporary, but hair loss resulting from a severe burn permanent?

For answers, see Appendix B.

Sebaceous Glands

The **sebaceous glands** (se-ba'shush; "greasy") are the skin's oil glands (Figure 5.1 and **Figure 5.9**). They occur over the entire body, except on the palms and soles. They are simple alveolar glands with several alveoli opening into a single duct (see Figure 4.5 p. 74 for their basic structure), but the alveoli are actually filled with cells, so there is no lumen (central

cavity). Their oily product, called **sebum** (se'būm; "animal fat"), is secreted in a most unusual way: The central cells in the alveoli accumulate oily lipids until they become engorged and burst apart. This process is called **holocrine secretion** (hol'o-krin; *holos* = whole), because *whole* cells break up to form the product. Most sebaceous glands are associated with hair follicles, emptying their sebum into the upper third of the follicle. From there, the sebum flows superficially to cover the skin. In addition to making our skin and hair oily, sebum collects dirt, softens and lubricates the hair and skin, prevents hair from becoming brittle, and keeps the epidermis from cracking. It also helps to slow water loss across the skin and to kill bacteria.

The secretion of sebum is stimulated by hormones, especially androgens. The sebaceous glands are relatively inactive during childhood but are activated in both sexes during puberty, when the production of androgens begins to rise.

ACNE In some teenagers, so much sebum is produced that it cannot be ducted from the glands quickly enough. When a sebaceous gland becomes blocked by sebum, a whitehead appears on the skin surface. If the material oxidizes and dries, it darkens to form a blackhead (the dark color of a blackhead is *not* due to dirt). Blocked sebaceous glands are likely to be infected by bacteria, producing a pimple. The bacteria break down the sebum into irritating fatty acids. These acids, along with the



ACNE, (continued)

bacterial products themselves, induce inflammation, especially when the entire infected mass bursts out of the follicle into the surrounding dermis. The *acne* that results can be mild or extremely severe, leading to permanent scarring.

The treatment of acne addresses the different causal factors: increased sebum production and formation of whiteheads, inflammation, and bacterial infection. Topical medications such as benzoyl peroxide treat inflammatory acne; topical retinoids, derivatives from vitamin A, act to prevent the formation of whiteheads; and antibiotics destroy acne-causing bacteria. Often combinations of treatments are used for maximal results.



Sweat Glands

Sweating prevents overheating of the body, because sweat cools the skin as it evaporates. Only mammals have **sweat glands (sudoriferous glands)**. Humans have more than 2.5 million sweat glands distributed over the entire skin surface, except on the nipples and parts of the external genitalia. Humans normally produce about 500 ml of sweat per day, but this amount can increase to 12 liters (over 3 gallons) on hot days during vigorous exercise. Hair interferes with the evaporation of sweat and the ability to cool the body, so the need for increased temperature regulation through sweating led to a reduction of hairiness in humans.

Sweat is an unusual secretory product in that it is primarily a filtrate of blood that passes through the secretory cells of the sweat glands and is released by exocytosis. Sweat is 99% water, with some salts (mostly sodium chloride) and traces of metabolic wastes (urea, ammonia, uric acid). It is acidic, so it retards the growth of bacteria on the skin.

There are two types of sweat glands, both of which increase their secretion in response to stress as well as to heat: eccrine and apocrine glands. **Eccrine glands** (ek'rin; "secreting") are by far the more numerous type and produce true sweat (Figure 5.9b). They are most abundant on the palms, soles, and forehead. Each is a coiled version of a simple tubular gland (see Figure 4.5 p. 74). The coiled, secretory base lies in the deep dermis and hypodermis, and the duct runs superficially to open at the skin surface through a funnel-shaped **pore**. (Although most pores on the skin surface are sweat pores, the "pores" seen on the face are openings of hair follicles.)

Apocrine (ap'o-krin) **glands** are mostly confined to the axillary, anal, and genital areas. They are larger than eccrine glands, and their ducts open into hair follicles. Apocrine glands produce a special kind of sweat consisting of fatty substances and proteins, in addition to the components of true sweat. For this reason, apocrine sweat is viscous and sometimes has a milky or yellow color. This product is odorless when first secreted, but as its organic molecules are decomposed by bacteria on the skin, it takes on a musky smell. This is the source of body odor.

Apocrine glands start to function at puberty under the influence of androgens. Their activity is increased by sexual foreplay, and they enlarge and recede with the phases of a woman's menstrual cycle. The secretions from the apocrine glands were identified as true human pheromones (chemical signals that convey information to a member of the same species) in the late 1990s when it was shown that they are responsible for the synchrony of the menstrual cycle that occurs in females who live together.

These glands are involved with sexual signaling and appear to function in attractiveness and mate selection. Apocrine secretions signal information about a person's immune system. The genes that encode for the immune system, the major histocompatibility complex (MHC), also influence body odor secretions. Each person has a unique set of these genes. In experiments, women selected body odor scents as "sexy" or "attractive" from men who had immune system genes most different from their own. Mates with complementary immune system genes provide their offspring with greater disease protection and decreased likelihood of recessive disorders.

The skin forms several types of modified sweat glands. *Ceruminous glands* (sě-roo'mĭ-nus; "waxy") are modified apocrine glands in the lining of the external ear canal. Their product is a component of earwax. The *mammary glands* are specialized sweat glands highly modified to secrete milk. Although they are part of the integumentary system, they are discussed along with the female reproductive system in Chapter 25.

check your understanding

- Apocrine glands become active in adolescence. Are these also the glands that cause acne?
- Look at the micrograph of the eccrine gland in Figure 5.9b. What type of tissue forms these glands?
- Which functions of skin are performed by eccrine glands?

For answers, see Appendix B.

DISORDERS OF THE INTEGUMENTARY SYSTEM

- Describe the layers involved in and the symptoms of first-, second-, and third-degree burns, and explain why serious burns are life-threatening.
- Identify the cell type involved, the characteristic appearance, and the degree of malignancy in the three types of skin cancer.

Exposed directly to the dangers and germs of the outside world, skin can develop more than a thousand different conditions and ailments. The most common skin disorders are bacterial, viral, or yeast infections, some of which are described in the Related Clinical Terms at the end of this chapter. The most severe threats to the skin are burns and skin cancer.



(a) Skin bearing partial thickness burn (first- and second-degree burns)



(b) Skin bearing full thickness burn (third-degree burn)

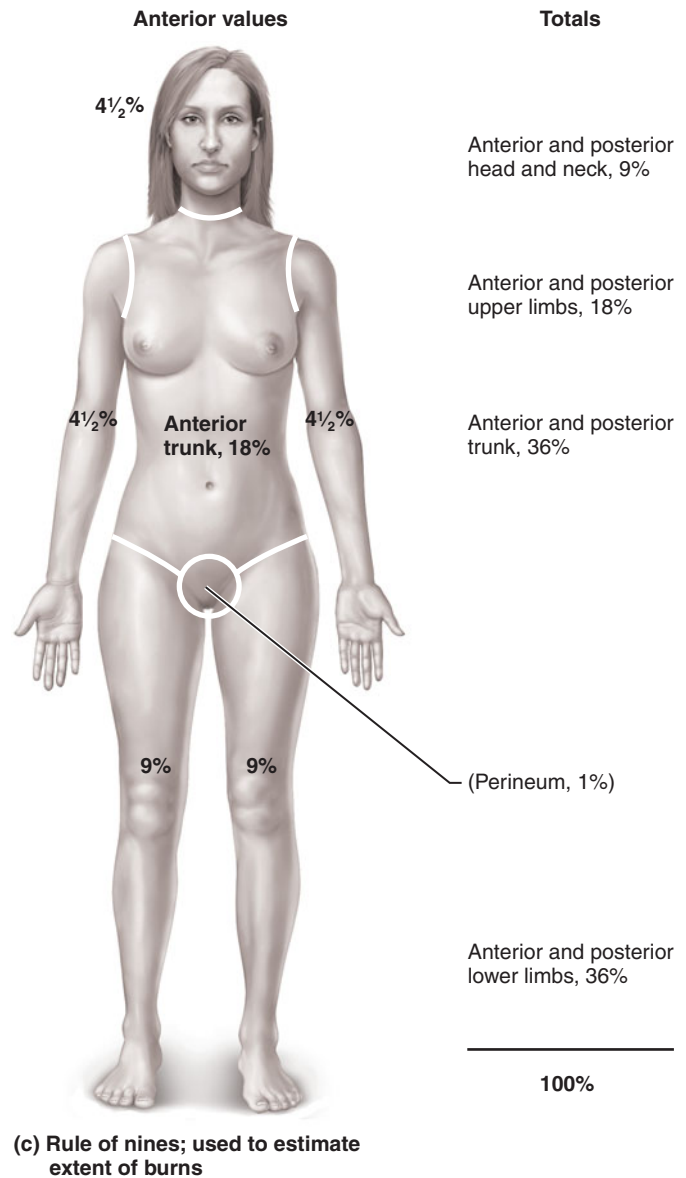


FIGURE 5.10 Burns.

Burns

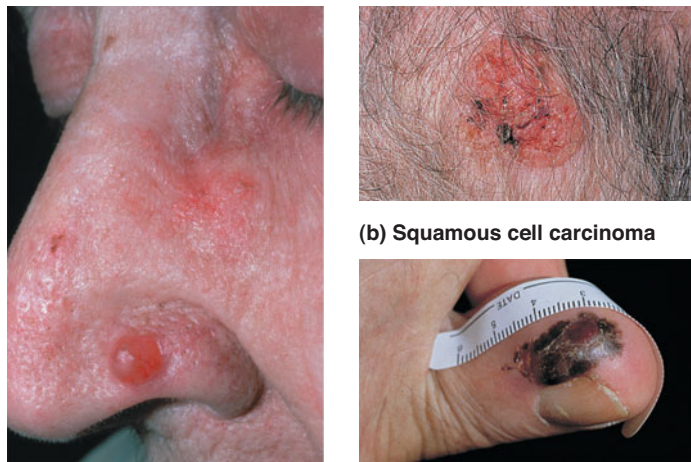
Burns are a devastating threat to the body, primarily because of their effects on the skin. A burn is tissue damage inflicted by heat, electricity, radiation, extreme friction, or certain harmful chemicals.

The immediate threat to life from serious burns is a catastrophic loss of body fluids. Inflammatory edema is severe. As fluid seeps from the burned surfaces, the body quickly loses water and essential salts. This dehydration in turn leads to fatal circulatory shock; that is, inadequate blood circulation caused by the reduction in blood volume. To save the patient, the medical team must replace the lost fluids immediately. After the initial crisis has passed, infection becomes the main threat. Pathogens can easily invade areas where the skin barrier is destroyed.

Burns are classified by their severity (depth) as first-, second-, or third-degree burns (**Figure 5.10**). Third-degree burns are the *most* severe. In **first-degree burns**, only the

epidermis is damaged. Symptoms include redness, swelling, and pain: the typical inflammatory reaction to tissue damage (see p. 93). Generally, first-degree burns heal in a few days without special attention. Sunburn is usually a first-degree burn. **Second-degree burns** involve injury to the epidermis and the upper part of the dermis. Symptoms resemble those of first-degree burns, but blisters also appear as fluid accumulates between the epidermal and dermal layers. The skin regenerates with little or no scarring in 3 to 4 weeks if care is taken to prevent infection. First- and second-degree burns are considered **partial-thickness burns**.

Third-degree burns consume the entire thickness of the skin, and thus are **full-thickness burns**. The burned area appears white, red, or blackened. Although the skin might eventually regenerate, it is usually impossible to wait for this because of fluid loss and infection. Therefore, skin from other parts of the patient's body must be grafted onto the burned area. Such a graft, in which an individual is both donor and recipient, is called an *autograft*.



(a) Basal cell carcinoma

(b) Squamous cell carcinoma

(c) Melanoma

FIGURE 5.11 Photographs of skin cancers.

In general, burns are considered critical if any of the following conditions exists: (1) Over 10% of the body has third-degree burns; (2) 25% of the body has second-degree burns; (3) there are third-degree burns on the face, hands, or feet. A quick way to estimate how much surface is affected by a burn is to use the **rule of nines** (Figure 5.10c). This method divides the body surface into 11 regions, each accounting for 9% (or a multiple of 9%) of total body area. The values shown on the human figure in Figure 5.10c indicate surface area values for the anterior body surfaces; total values for each region are indicated to the right of the illustration. This method is only roughly accurate, so special tables are used when greater accuracy is needed.

For patients whose burns are too large or who otherwise are poor candidates for autografts, good artificial coverings are now available. Artificial skin consists of a “dermis” made from bovine collagen and an “epidermis” made of silicone. The patient’s own dermis gradually replaces and reabsorbs the artificial one, and the silicone sheet is later peeled off and replaced with a network of epidermal cells cultured from the patient’s own skin. The artificial skin is not rejected by the body, saves lives, and results in minimal scarring. However, it is more likely to become infected than is an autograft.

Skin Cancer

Many types of tumors arise in the skin. Most are benign (warts, for example), but some are malignant. Skin cancer is the most common type of cancer, with about a million new cases appearing each year in the United States. As mentioned earlier, the most important risk factor for skin cancer is overexposure to the UV rays in sunlight. Recent data show increased risk of all three types of skin cancer in individuals with a history of indoor tanning.

Basal Cell Carcinoma

Basal cell carcinoma is the least malignant and most common of the skin cancers (Figure 5.11a). Over 30% of all Caucasians get it in their lifetimes! Cells of the stratum

basale proliferate, invading the dermis and hypodermis, and causing tissue erosions there. The most common lesions of this cancer are dome-shaped, shiny nodules on sun-exposed areas of the face. These nodules later develop a central ulcer and a “pearly,” beaded edge. They often bleed. Basal cell carcinoma grows relatively slowly, and metastasis seldom occurs. Full cure by surgical excision or other methods of removal is the rule in 99% of cases.

Squamous Cell Carcinoma

Squamous cell carcinoma (Figure 5.11b) arises from the keratinocytes of the stratum spinosum. The lesion appears as a scaly, irregular, reddened papule (small, rounded elevation) that tends to grow rapidly and metastasize if not removed. If treated early, however, the chance of a complete cure is good, and the overall cure rate is 99%. The carcinoma can be removed by radiation therapy, by surgery, or by skin creams containing anticancer drugs.

Melanoma

Melanoma (mel’ah-no’mah), a cancer of melanocytes, is the most dangerous kind of skin cancer (Figure 5.11c). Melanocytes are derived from neural crest cells, which wander widely during embryonic development. This predisposition for migration may explain the invasive nature of melanoma. Melanoma accounts for only about 1 of every 20 skin cancers, but it is increasing rapidly in countries with light-skinned populations—by 3% to 8% *per year* in the United States. Melanoma can originate wherever there is pigment, but it often arises from existing moles, usually appearing as an expanding dark patch. Because melanoma cells metastasize rapidly into surrounding circulatory vessels, the key to surviving melanoma is early detection. Most people do not survive this cancer if the lesion has grown over 4 mm thick. Melanoma is resistant to chemotherapy and current immunotherapy treatment, although vaccines are being tested.

The American Cancer Society suggests that individuals with frequent sun exposure regularly examine their skin for moles and new pigment spots, applying the **ABCD rule** for recognizing melanoma: **A, Asymmetry**: The two halves of the spot or mole do not match; **B, Border irregularity**: The borders have indentations and notches; **C, Color**: The pigment spot contains several colors, including blacks, browns, tans, and sometimes blues and reds; **D, Diameter**: larger than 6 mm (larger than a pencil eraser). Some experts have found that adding an **E**, for **Elevation** above the skin surface, improves diagnosis, so they use the **ABCD(E) rule**.

check your understanding

18. What type of burn causes blisters?
19. What are the two life-threatening concerns resulting from third-degree burns, and how are they treated?
20. Which type of skin cancer is most common? Which one can be fatal?

For answers, see Appendix B.

THE SKIN THROUGHOUT LIFE

- Identify the primary germ layers that form the skin and its appendages.
- Describe the changes that occur in the skin from birth to old age.

The epidermis develops from embryonic ectoderm, and the dermis and hypodermis develop from mesoderm (see Chapter 3, pp. 56–57). Melanocytes, however, develop from neural crest cells that migrate into the ectoderm during the first 3 months of prenatal development. By the end of the fourth month, the skin is fairly well formed: The epidermis has all its strata, dermal papillae are evident, and the epidermal appendages are present in rudimentary form. During the fifth and sixth months, the fetus is covered with a downy coat of delicate hairs called the *lanugo* (lah-nu’go; “wool or down”). This cloak is shed by the seventh month, and vellus hairs make their appearance.

When a baby is born, its skin is covered with *vernix caseosa* (ver’niks ka’sē-o-sah; “varnish of cheese”), a cheesy-looking substance produced by sebaceous glands. It protects the skin of the fetus from constant contact with amniotic fluid. A newborn’s skin and hypodermis are very thin, but they thicken during infancy and childhood.

With the onset of adolescence, acne may appear (see pp. 113–114). Acne generally subsides in early adulthood, and the skin reaches its optimal appearance in one’s 20s and 30s. Thereafter, the skin starts to show the harmful effects of continued environmental assaults, such as abrasion, wind, sun, and chemicals. Scaling and various kinds of skin inflammation, called **dermatitis** (der’mah-ti’tis), become more common.

In middle age, the lubricating and softening substances produced by the glands start to diminish. As a result, the skin becomes dry and itchy. People with naturally oily skin may avoid this dryness, so their skin often ages well. Therefore, young people who suffer the humiliation of oily skin and acne may be rewarded later in life by looking younger longer.

Recently, it has been discovered that most aspects of skin aging are not intrinsic but are caused by sunlight in a process called “photoaging.” Aged skin that has been protected from the sun has lost some elasticity and is thinner (as is the hypodermis under it), but it remains unwrinkled and unmarked. Sun-exposed skin, by contrast, is wrinkled, loose, inelastic, leathery, and has pigmented spots called “liver spots.” In the dermis of such sun-aged skin, the amount of collagen has declined, and an abnormal, elastin-containing material has accumulated. Much of this change is due to UV-induced activation of enzymes that degrade collagen and other components of the dermis.

By blocking UV rays, large amounts of melanin protect the skin from photoaging. This is why fair-skinned individuals, who have little melanin to begin with, show age-related changes at a younger age than do people with darker skin and hair. For the same reason, dark-skinned people look youthful much longer than Caucasians.

check your understanding

21. From which embryonic germ layer do the appendages of the skin develop? From which embryonic layer does the hypodermis develop?
22. How does UV radiation damage the dermal layer of the skin?

For answers, see Appendix B.

RELATED CLINICAL TERMS

ALOPECIA (al’o-pe’she-ah) Any condition involving absence or loss of hair. Male pattern baldness, the most common type, is technically named *androgenic alopecia*.

ATHLETE’S FOOT Itchy, red, peeling condition of the skin between the toes resulting from fungal infection.

BOILS AND CARBUNCLES (kar’bung’kls; “little glowing embers”) Infection and inflammation of hair follicles and sebaceous glands that has spread into the underlying hypodermis. A boil can be likened to a giant pimple; carbuncles are composite boils. The common cause is bacterial infection.

COLD SORES (FEVER BLISTERS) Small, painful, fluid-filled blisters that usually occur around the lips and in the mucosa in the mouth. They are caused by the herpes simplex virus, which localizes in nerve cells that supply the skin, where it remains dormant until activated by emotional upset, fever, or UV radiation.

IMPETIGO (im’pē-ti’go; “an attack”) Pink, fluid-filled, raised lesions that are common around the mouth and nose. They develop a yellow crust and eventually rupture. This contagious condition, caused by a *Staphylococcus* infection, is common in school-age children.

PSORIASIS (so-ri’ah-sis; “an itching”) A chronic inflammatory condition characterized by reddened epidermal papules covered with dry, silvery scales. The scales result from an overproliferation of the epidermis, and the pink color is due to widened capillaries in the dermis. Relatively common, it affects 2% of Americans. It may be an autoimmune condition triggered by bacterial products. One effective treatment for psoriasis employs a drug that slows epidermal growth and that is activated by exposure to UV light.

VITILIGO (vit’il-i’go; “blemish”) An abnormality of skin pigmentation characterized by light spots surrounded by areas of normally pigmented skin. It can be cosmetically disfiguring, especially in dark-skinned people.

CHAPTER SUMMARY

You can use the following media study tool for additional help when you review specific key topics in Chapter 5.

PAL = Practice Anatomy Lab™

1. The integumentary system consists of the skin and its appendages (hair, sebaceous glands, sweat glands, and nails). The hypodermis, although not part of the integumentary system, is also considered in this chapter.
2. Skin functions to protect our body from bumps and scrapes, chemicals, invading microorganisms, water loss, and ultraviolet (UV) radiation. It also functions in body temperature regulation, excretion, production of vitamin D, and sensory reception.

The Skin and the Hypodermis (pp. 102–110)

Epidermis (pp. 103–105)

3. The epidermis is a keratinized stratified squamous epithelium. Its main cell type is the keratinocyte. From deep to superficial, its strata are the basale, spinosum, granulosum, lucidum, and corneum. The stratum lucidum occurs only in thick skin found on palms and soles.
4. The dividing cells of the stratum basale are the source of new keratinocytes for the growth and renewal of the epidermis.
5. In the stratum spinosum, keratinocytes contain strong pre-keratin intermediate filaments. In the stratum granulosum, keratinocytes contain keratohyalin granules, which combine with the pre-keratin filaments to form tension-resisting keratin. Stratum granulosum cells also secrete an extracellular waterproofing glycolipid. Keratinized cells of the stratum corneum protect the skin and are shed as skin flakes and dandruff.
6. Scattered among the keratinocytes in the deepest layers of the epidermis are pigment-producing melanocytes, tactile epithelial cells, and the dendritic cells of the immune system.

Dermis (pp. 106–108)

7. The leathery dermis, composed of connective tissue proper, is well supplied with vessels and nerves. Also located in the dermis are skin glands and hair follicles.
8. The superficial papillary layer consists of areolar connective tissue, whereas the deep reticular layer is dense irregular connective tissue. The papillary layer abuts the undulating undersurface of the epidermis and is responsible for the epidermal friction ridges that produce fingerprints. In the reticular layer, less dense regions between the collagen bundles produce cleavage lines. Flexure lines occur over joints, where the dermis attaches tightly to underlying structures.

Hypodermis (p. 109)

9. The hypodermis underlies the skin. Composed primarily of adipose tissue, it stores fat, insulates the body against heat loss, and absorbs and deflects blows. During weight gain, this layer thickens most markedly in the thighs and breasts of women and in the anterior abdominal wall of men.

Skin Color (pp. 109–110)

10. Skin color reflects the amounts of pigments (melanin and/or carotene) in the skin and the oxygenation level of hemoglobin in the dermal blood vessels.
11. Melanin, made by melanocytes and transferred to keratinocytes, protects keratinocyte nuclei from the damaging effects of UV radiation.

The epidermis also produces vitamin D under the stimulus of UV radiation.

Appendages of the Skin (pp. 110–114)

12. Skin appendages, which develop from the epidermis but project into the dermis, include hairs and hair follicles, sebaceous glands, sweat glands, and nails.

Nails (p. 110)

13. A nail is a scalelike modification of the epidermis that covers the dorsal tip of a finger or toe. The actively growing region is the nail matrix.

Hair and Hair Follicles (pp. 110–113)

14. A hair is produced by a tube-shaped hair follicle and consists of heavily keratinized cells. Each hair has an outer cuticle, a cortex, and usually a central medulla. Hairs have roots and shafts. Hair color reflects the amount and variety of melanin present.
15. A hair follicle consists of an epithelial root sheath that includes the hair matrix and connective tissue root sheath derived from the dermis. Stem cells, which renew the hair matrix and give rise to new epidermal cells, are in the epithelial root sheath. A knot of capillaries in the hair papilla nourishes the hair, and a nerve knot surrounds the hair bulb. Arrector pili muscles pull the hairs erect and produce goose bumps in response to cold or fear.
16. Fine and coarse hairs are called vellus hairs and terminal hairs, respectively. At puberty, under the influence of androgens, terminal hairs appear in the axillae and pubic regions in both sexes.
17. Hairs grow in cycles that consist of growth and resting phases. Hair thinning results from factors that lengthen follicular resting phases, including natural age-related atrophy of hair follicles and expression of the male pattern baldness gene during adulthood.

Sebaceous Glands (pp. 113–114)

18. Sebaceous glands are simple alveolar glands that usually empty into hair follicles. Their oily holocrine secretion is called sebum.
19. Sebum lubricates the skin and hair, slows water loss across the skin, and acts as a bactericidal agent. Sebaceous glands secrete increased amounts of sebum at puberty under the influence of androgens. Blocked and infected sebaceous glands lead to pimples and acne.

Sweat Glands (p. 114)

20. Eccrine sweat glands are simple coiled tubular glands that secrete sweat, a modified filtrate of the blood. Evaporation of sweat cools the skin and the body.
21. Apocrine sweat glands, which produce pheromones, occur primarily in the axillary, anal, and genital regions. Their secretion contains proteins and fatty acids; bacterial action on this secretion causes body odor.

PAL Histology/Integumentary System

Disorders of the Integumentary System (pp. 114–116)

22. The most common skin disorders result from microbial infections.

Burns (pp. 115–116)

23. In severe burns, the initial threat is loss of body fluids leading to circulatory collapse. The secondary threat is overwhelming infection.

24. The extent of a burn may be estimated by using the rule of nines. The severity of burns is indicated by the terms *first-*, *second-*, and *third-degree*. Third-degree burns harm the full thickness of the skin and require grafting for successful recovery.

Skin Cancer (p. 116)

25. The major risk factor for skin cancer is exposure to UV sunlight.
26. Basal cell carcinoma and squamous cell carcinoma are the most common types of skin cancer and usually are curable if detected early. Melanoma, a cancer of melanocytes, is less common but dangerous. The ABCD or ABCD(E) rule can be used to examine a mole or spot for the possibility of melanoma.

The Skin Throughout Life (p. 117)

27. Epidermis develops from embryonic ectoderm; dermis and hypodermis develop from mesoderm; and melanocytes develop from neural crest cells.
28. At 5–6 months, a fetus has a downy lanugo coat. Fetal sebaceous glands produce vernix caseosa that protects the skin within the amniotic sac.
29. In old age, the skin thins and loses elasticity. Damage from sunlight leads to wrinkles, age spots, and loose and leathery skin; this reflects a loss of collagen and accumulation of elastin-containing material in the dermis.

REVIEW QUESTIONS

Multiple Choice/Matching Questions

For answers, see Appendix B.

- Which epidermal cell type functions in the immune system? (a) keratinocyte, (b) melanocyte, (c) dendritic cell, (d) tactile epithelial cell.
- Match each epidermal layer in column B to its description in column A.

Column A

- ____ (1) Desmosomes and shrinkage artifacts give its cells “prickly” projections.
- ____ (2) Its cells are flat, dead bags of keratin.
- ____ (3) Its cells divide, and it is also called the stratum germinativum.
- ____ (4) It contains keratohyalin and lamellated granules.
- ____ (5) It is present only in thick skin.

Column B

- (a) stratum basale
- (b) stratum corneum
- (c) stratum granulosum
- (d) stratum lucidum
- (e) stratum spinosum

- The ability of the epidermis to resist rubbing and abrasion is largely due to the presence of (a) melanin, (b) carotene, (c) collagen, (d) keratin.
- Skin color is determined by (a) melanin, (b) carotene, (c) oxygenation level of the blood, (d) all of these.
- What is the major factor accounting for the waterproof nature of the skin? (a) desmosomes in stratum corneum, (b) glycolipid between stratum corneum cells, (c) the thick insulating fat of the hypodermis, (d) the leathery nature of the dermis.
- Circle the false statement about vitamin D: (a) Dark-skinned people make no vitamin D. (b) If the production of vitamin D is inadequate, one may develop weak bones. (c) If the skin is not exposed to sunlight, one may develop weak bones. (d) Vitamin D is needed for the uptake of calcium from food in the intestine.
- Use logic to deduce the answer to this question. Given what you know about skin color and skin cancer, the highest rate of skin cancer on earth occurs among (a) blacks in tropical Africa, (b) scientists in research stations in Antarctica, (c) whites in northern Australia, (d) Norwegians in the United States, (e) blacks in the United States.

- An arrector pili muscle (a) is associated with each sweat gland, (b) causes the hair to stand up straight, (c) enables each hair to be stretched when wet, (d) squeezes hair upward so it can grow out.
- Sebum (a) lubricates the skin and hair; (b) consists of dead cells and fatty substances; (c) collects dirt, so hair has to be washed; (d) all of these.
- An eccrine sweat gland is a ____ gland: (a) compound tubular, (b) compound tubuloacinar, (c) simple squamous, (d) simple tubular.
- Identify which embryonic layer listed in column B forms each of the skin structures in column A.

Column A

- ____ (1) hypodermis
- ____ (2) sebaceous gland
- ____ (3) epidermis
- ____ (4) dermis
- ____ (5) melanocytes

Column B

- (a) ectoderm
- (b) mesoderm
- (c) endoderm
- (d) neural crest

- The reticular layer of the dermis (a) provides strength and elasticity to the skin, (b) is composed of loose connective tissue, (c) insulates to prevent heat loss, (d) forms the dermal papilla.
- Match the skin structures in column B with their function listed in column A.

Column A

- ____ (1) protection from ultraviolet radiation
- ____ (2) insulation, energy storage
- ____ (3) waterproofing and prevention of water loss
- ____ (4) temperature regulation
- ____ (5) initiation of an immune response to invading bacteria
- ____ (6) excretion of water, urea, and salts
- ____ (7) help in bonding the epithelium to the dermis

Column B

- (a) dendritic cells
- (b) dermal vascular plexuses
- (c) papillary layer of the dermis
- (d) hypodermis
- (e) melanocytes
- (f) stratum corneum
- (g) eccrine sweat glands
- (h) reticular layer of the dermis

14. Thick skin differs from thin skin in (a) the thickness of the stratum spinosum, (b) the presence of an additional layer, the stratum granulosum, (c) thickness of the stratum corneum, (d) the distribution of sweat glands.
15. From the list of tissues in column B, identify the primary tissue that forms each of the structures in column A.

Column A

- (1) epidermis
- (2) reticular layer of the dermis
- (3) hypodermis
- (4) papillary layer of the dermis
- (5) apocrine sweat glands
- (6) hair follicle
- (7) nails

Column B

- (a) dense irregular connective tissue
- (b) dense regular connective tissue
- (c) areolar connective tissue
- (d) epithelial tissue
- (e) adipose tissue

Short Answer Essay Questions

16. Is a bald man really hairless? Explain.
17. Explain why thin skin is also called hairy skin, and why thick skin is also called hairless skin. (Note their locations.)
18. Distinguish first-, second-, and third-degree burns.
19. Why does skin that is exposed to sunlight age so much more than nonexposed skin?
20. Explain why no skin cancers originate from stratum corneum cells.
21. Explain each of these familiar phenomena in terms of what you learned in this chapter: (a) goose bumps, (b) dandruff, (c) stretch marks from gaining weight, (d) leaving fingerprints, (e) the almost hairless body of humans, (f) getting gray hairs.
22. Eric, an anatomy student, said it was so hot one day that he was “sweating like a pig.” His professor overheard him and remarked, “That is a stupid expression, Eric. No pig ever sweats nearly as much as a person does!” Explain her remark.
23. List three functions of the skin.
24. After studying the skin in anatomy class, Toby grabbed the large “love handles” around his own abdomen and said, “I have a thick hypodermis, but this layer performs some valuable functions!” What are the functions of the hypodermis?
25. What type of tissue(s) form each of the following structures? (a) dermis, (b) epidermis, (c) hypodermis. What embryonic germ layer is the source of each?

CRITICAL REASONING & CLINICAL APPLICATION QUESTIONS

1. What are the two most important clinical problems encountered by severely burned patients? Explain each in terms of the absence of skin.
2. Dean, a 40-year-old beach boy, tells you that his suntan made him popular when he was young. Now his face is wrinkled, and he has several moles that are growing rapidly and are as large as coins. When he shows you the moles, you immediately think “ABCD.” What do these letters mean, and what disease may Dean have?
3. Long-term patients confined to hospital beds are turned every 2 hours. Why? Why is this effective?
4. Carmen slipped on some ice and split her chin on the sidewalk. As the emergency room physician was giving her six stitches, he remarked that the split was right along a cleavage line. How cleanly is her wound likely to heal, and is major scarring likely to occur?
5. A man got his finger caught in a machine at the factory, and his entire nail was torn off his right index finger. The nail parts lost were the body, root, bed, matrix, and eponychium. Is this nail likely to grow back? Why or why not?
6. On a diagram of the human body, mark off various regions according to the rule of nines. What percent of the total body surface is affected if someone is burned (a) over the entire posterior trunk and buttocks? (b) over an entire lower limb? (c) on the entire front of the left upper limb?
7. Why are melanomas the most common type of skin cancer to metastasize? (Recall that melanocytes are formed from neural crest tissue, an embryonic tissue that wanders extensively during early development.)
8. What are the harmful effects of UV radiation, and how is UV radiation beneficial to our well-being?



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