

NOTES SKIN STRUCTURES

SKIN ANATOMY & PHYSIOLOGY

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- Skin is body's largest organ
 Seven percent of total body weight
- Comprises integumentary system, appendages (hair, nails, oil, sweat glands)
 - Protects body (infection, abrasion, dehydration, etc)
 - Regulates body temperature
 - Detects pain, sensation, pressure
 - Essential for vitamin D production

• Three layer division

Epidermis, dermis, hypodermis



Figure 25.1 The three layers of the skin, from superficial to deep, include: the epidermis, dermis, and hypodermis.

EPIDERMIS

- Epidermis
 - Stratified squamous epithelium
 - Thin outermost layer
- Multiple layers of developing keratinocytes (contain keratin)
 - Make, secrete glycolipids; prevent water seeping into/out of body

Stratum basale

- Innermost layer: single columnar stem cell layer; dividing, producing keratinocytes
 - Keratinocytes contain cholesterol precursors activated by UVB light → vitamin D (regulates calcium absorption)
- Also contains melanocytes (secrete melanin, giving skin its color)
 - UVB light stimulates melanin secretion

 → placed into melanosomes, moved up
 by keratinocytes → scatters UVB light
 → natural sunscreen (prevents skin
 cancer from excessive UVB light)

Stratum spinosum

- Second layer: comprises 8–10 keratinocyte cell layers which can no longer divide
 - Proteins on keratinocytes help them adhere together
 - Dendritic cells seek out invading microbes

Stratum granulosum

- Third layer: comprises 3–5 keratinocyte cell layers undergoing keratinization (flatten out, die) → epidermal skin barrier formed
 - Keratohyalin granules in keratinocytes contain keratin precursors which aggregate, cross-link → keratin bundles
 - Lamellar granules in keratinocytes contain glycolipids (secreted to cell surface, glues cells together)

Stratum lucidum

- Fourth layer: comprises 2–3 dead keratinocyte cell layers that have secreted most of their lamellar granules
 - Only found in thick skin (e.g. palms, soles of feet)

Stratum corneum

- Uppermost layer: comprises 20–30 dead keratinocyte cell layers glued together with glycolipids
 - Dead keratinocytes secrete defensins to fight pathogens
 - \circ Cells from stratum lucidum push up \rightarrow cells from this layer shed \rightarrow skin flakes/ dandruff
- Blood vessels dilate when hot (blood moves closer to surface → allows heat loss)/contract when cold (blood moves away from surface → prevents heat loss)
- Sweat glands ↑ secretion when hot (↑ heat to evaporate sweat)/↓ when cold (↓ heat to evaporate sweat)

FREE NERVE

ENDINGS

PAPILLARY LAYER

MEISSNER CORPUSCI E



Figure 25.2 The five layers of the epidermis. Stratum basale is the deepest layer and stratum corneum is the most superficial.

DERMIS

- Dermis
 - Central layer
 - Two layer division (papillary layer; deeper, thicker reticular layer)

Papillary layer

- Fibroblasts (producing collagen) arranged in papillae
- Contains blood vessels, macrophages, nerve endings (e.g. Meissner's corpuscles for fine touch, free nerve endings for pain)
- Responsible for fingerprints († gripping, sensing abilities)

Reticular layer

- Fibroblasts (produces elastin for flexibility)
- Contains oil, sweat glands; lymphatic, blood vessels; hair follicles; macrophages; nerves (e.g. Pacinian corpuscle for pressure, vibration)
- Collagen packed tightly $\rightarrow \uparrow$ support
- Regulates temperature with blood vessels, sweat glands



Figure 25.3 The papillary layer of the dermis contains multiple types of nerve endings.

Figure 25.4 Contents of the reticular layer of the dermis.

HYPODERMIS

- Hypodermis (subcutaneous tissue) inner layer
 - Contains adipocytes (store fat), fibroblasts, macrophages, blood vessels, nerves, lymphatics
 - Insulates deeper tissues; provides padding; anchors skin to underlying muscle with connective tissue (e.g. collagen)

HAIR, SKIN, & NAILS

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- Skin appendages include hair, nails, skin glands (oil/sebaceous, sweat/sudoriferous)
 - Regulate body temperature; environmental protection
 - Originate in dermis
- Hair, nails comprised of long, filamentous protein (keratin)
 - Keratin: produced by keratinocytes during keratinization (cells rapidly replicate, die)
 - Soft keratin (produced by skin); hard keratin (produced by hair, nails)

HAIR

- Includes vellus hairs (short, thin); terminal hairs (more visible, growth starts at puberty)
- Found everywhere
 - Exceptions: palms, soles of feet, lips
- Hair strands sit in follicle; epidermal tissue dips into dermis
 - Associated with sebaceous glands,

arrector pili muscles, apocrine glands, nerve receptors

- Composition: shaft, root, bulb
 - Hair matrix: active hair growth site, found inside bulb; contains keratinocytes, melanocytes; blood supplied by papilla
- Keratinocytes die, flatten out → hard keratin fills up cell → gradually get pushed up follicle forming hair
 - Hair growth: includes growth, resting phases
 - Keratinocytes in bulb replicate set number of times → follicle eventually stops producing hair/produce vellus hairs instead (genetically determined) → baldness
- Melanocytes produce melanin (protein pigments that give hair color)
 - Melanocytes move melanin into melanosomes → taken up by keratinocytes



Figure 25.5 Composition of hair and associated structures.

- $^{\circ} \uparrow \text{age} \rightarrow \downarrow \text{melanin} \rightarrow \text{faded},$ white hair
- Nerve receptors around bulb stimulated when hair shaft moves
- Arrector pili muscle contracts, pulls hair (e.g. cold weather/frightened) → goosebumps

NAILS

- Grow from proximal to distal fingertips/toes
 Surrounded on either side by nail folds
 - Closed off proximally by eponychium → forms cuticle (dead skin keratinocytes that cover junction between nail, skin)
- Nail matrix composition: lunula, nail plate
 - Lunula: white, crescent-shaped part of nail near eponychium
 - Free edge: nail plate portion hanging over skin
- Modified keratinocytes in matrix form plate by keratinization (similar to hair)
- Nails grow continually through life (unlike hair)



Figure 25.6 Superior view and cross section of a finger illustrating components of the nail.

SEBACEOUS GLANDS

- Secrete sebum (softens hair shaft, prevents moisture-loss, deters pathogens) onto hair follicles/through pores → skin surface
- During puberty: ↑ androgen hormones → ↑ sebum production → blocks pores, plugs hair follicles → enclosures allow infection development (e.g. acne, folliculitis)

SUDORIFEROUS GLANDS

AKA sweat glands

Eccrine (merocrine) glands

- Found everywhere
 - Exceptions: lips, ear canal, clitoris, glans of penis
- Coil-shaped structure; in dermis; duct opens into pore on skin surface
- Sweat: hypotonic (mostly water, electrolytes); dermcidin (destroys bacteria); cools body (evaporation)
- Sympathetic nervous system activation during
 cardiovascular activity, fight-orflight response, fear/anxiety

Apocrine glands

- Found in armpits, genitals
- Become active during puberty
- Similar to eccrine glands
 - Bigger, fewer; produce secretions with lipids, proteins
 - \circ Secretions metabolized by bacteria \rightarrow body odor
- Several modified apocrine gland types
 - Ceruminous glands: in ear; produce cerumen; protects eardrum (with ear canal hairs)
 - Mammary glands: in breasts; produce milk



Figure 25.7 The two types of sweat glands (sudoriferous glands).

WOUND HEALING

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- Damaged tissue repair process
 - Acute wounds heal quickly (daysweeks)
 - Chronic wounds heal slowly (months)

Regenerative tissue capacity

- Classification: labile, stable, permanent
- Labile tissues (e.g. skin, connective tissue, intestines)
 - \circ Heal well: stem cells constantly divide \rightarrow rapid, effective healing
- Stable tissues (e.g. liver, endocrine glands, proximal kidney tubules)
 - Heal slowly: mature differentiated cells divide/regenerate by hyperplasia
- Permanent tissues (e.g. skeletal muscle, cartilage, neurons)
 - Heal poorly: lack of stem cells, no hyperplasia → replaced by scar tissue (fibrosis) → function loss

Open wounds

- Open wounds healed by primary, secondary, tertiary intention
- Primary intention (most surgical wounds)
 - Wound edges fuse (e.g. stitching/ gluing) → stem cells (e.g. epidermis) approximate, regenerate damaged tissue (minimal scarring)
- Secondary intention
 - Wound edges too far apart (e.g. pressure ulcers, tooth extraction, severe burns) → stem cells do not approximate → wound replaced by connective tissue growing from base upwards (slower healing; more scar tissue)
- Tertiary intention (delayed closure)
 - Wound cleaned, debrided → purposefully left open (↓ bacterial contamination likelihood) → closed by primary intention/left open for secondary intention

Penetrating trauma wound healing

- Penetrating trauma wound healing steps (e.g. cutting finger → damaged epidermis, dermis, interstitial space)
- Hemostasis (first step)
 - Blood vessels constrict → platelets adhere to site → forms platelet plug → fibrin mesh reinforces platelet plug → forms blood clot
- Inflammation (second step)
 - Damaged cells release chemokines, cytokines → neutrophils, macrophages recruited; blood vessels dilate → immune cells clear debris, digest dead/ damaged cells, destroy microbes → blood clot, dead macrophages combine, form scab
- Epithelization/migration (third step)
 - Basal cells (epidermal stem cells) proliferate, replace lost/damaged cells → rejuvenated epidermal layer (approx. 48 hours)
- Fibroplasia (fourth step)
 - Fibroblasts in dermis proliferate, secrete collagen (assemble → form collagen fibrils → collaged bundles) → blood vessel growth stimulated (angiogenesis); fibroblasts also produce glycoproteins, sugars → create granulation tissue in dermal layer
- Maturation (fifth step)
 - Collagen cross-linking: covalent bonds form between collagen bundles, improving tensile strength
 - Collagen remodeling: fibroblasts degrade subpar collagen
 - Contraction: myofibroblasts produce contractile proteins, pulling wound's edges together
 - Repigmentation: melanocytes proliferating, restoring color to damaged skin

Chronic wounds

- Healing prevention factors \rightarrow chronic wounds
 - Narrowed capillaries: prolonged compression/disease (e.g. diabetes, atherosclerosis) → ↓ blood flow → damaged tissue cannot be reached by immune cells, insufficient oxygen/ nutrients → tissue necrosis
 - Infection: pathogens compete for oxygen; cause ongoing damage, inflammation
 - Edema: disrupts fibroblast activity, collagen deposition, collage cross linking

WOUND HEALING



Figure 25.8 The five steps of penetrating trauma wound healing.