Chapter 5: The Integumentary System

Organ - an aggregate of tissues that perform a specific function. 

Epidermis – stratified squamous. 
Connective tissue - Loose/areolar, adipose & vascular tissue.

System - a group of organs operating together to perform specialized functions.

Integumentary System - the skin and its derivatives constitute this system. eg. hair, glands, nails, receptors to touch & temp.

I. SKIN - is an organ because it consists of tissues performing specific functions. 
* largest organ of the body (~2 sq. meters) 
A. Dermatology – diagnosis & treatment of skin 
B. Physiology of integumentary:
1. Regulation of body temperature - vasodilation and vasoconstriction 
2. Protection – from microbes, UV light, pollutants. 
3. Reception of stimuli – temp, touch, pain. 
4. Excretion – sweat, oil, wax.  
5. Promotes Vitamin D Synthesis- ie. the liver makes vit. D when skin is exposed to UV rays 

C. Structure - 2 parts: (epidermis & dermis) 
   * See fig 5.1, page 146 
   1. Epidermis - outer, thinner portion (epithelium) cemented to connective tissue (dermis). **Can be thick or thin depending on the area of the body. 
   2. Dermis - thick connective tissue; vascularized. 
      i. Subcutaneous layer(sub -Q), - a layer beneath the dermis; aka. Hypodermis (the basement membrane for skin) 
         * consists of loose-areolar and adipose tissue   
         * anchors the skin to the underlying muscle or bone. 
         * acts as a shock absorber. 
         * Blisters – epidermis is torn away from the dermis 

NOTE: you must know the anatomy of the skin!
II. Epidermis - stratified squamous- keratinized

A. Epidermis contains 4 types of cells:
1. Keratinocyte – most abundant in the epi.; produce the protein keratin which helps to waterproof the skin.
2. Melanocyte - produces melanin which is skin pigment.
3. Langerhan's cells – involved in immunity. WBC's that help in an immune response. (eg, respond to poison oak or bug bites)
4. Merkel cells – in the deepest part of the epi.; detect touch.

B. 5 layers of epidermis from deep to most superficial:
NOTE: 4 to 5 layers of epidermis depending on location, eg. soles of feet vs. the eye lids

1. Stratum Basale (aka. Stratum germinativum) -
   *germinates new cells that push up towards the surface which eventually become part of the top layers and shed.
   *cells contain desmosomes for strength/flexibility.
   *cells undergo continual mitotic cell division.
   *this layer gives rise to the other strata.
   *other cells from this layer migrate to the dermis and become sweat glands, oil glands, or hair follicles.
   *contains melanocytes

2. Stratum Spinosum - spine-like projections on cells that help to join cells together; desmosomes are found here too.

3. Stratum Granulosum - usually the cells in this layer are at various stages of degeneration due to lack of nutrients, and are preparing to be shed. *Cells are starting to keratinize here.
4. Stratum Lucidum - usually found only in the thick skin of palms & soles; not always present.
5. Stratum Corneum “Horny layer” – 3/4 of skin’s thickness. Contains dead cells that are completely keratinized and ready to be shed. This layer serves as a protective barrier (glycolipids) against light, heat waves, bacteria, and many chemicals. Callus – abnormally thickened stratum corneum; friction.
C. Keratinization and Growth of Epidermis:

*Keratinization* – a process by which cells of the *stratum basale* slowly migrate towards the surface and fill with keratin. Eventually, the dead cells slough off the body. ~ 4 weeks for cells to migrate from *stratum basale* & shed.

*this process speeds up when epidermis scuffed/scraped, and is regulated by hormones (EGH).

**Clinical application:** *Psoriasis* or “dandruff” on the scalp.

*Rapid keratinization.* An excessive amount of dead skin cells sloughing-off; as little as 7-10 days. Can occur all over the body.

III. Dermis - largest part of the skin; fat soluble tissue – rapid absorption of fat soluble substances into the circulatory system such as solvents like paint thinner. This can lead to organ failure (kidneys/liver).

* made of *loose & adipose connective tissue*

* has numerous glands, nerves & hair follicles

* contains macrophages, WBC’s (immunity)

* very elastic region of skin; has ability to be stretched and returned to original shape, eg. during pregnancy or wt. gain

**2 basic regions of the dermis:**

* upper 1/5 is known as the *papillary region* (Loose CT)

* lower 4/5 is known as the *reticular region* (Dense CT)

* *reticular region* contains a lot elastin & collagen protein fibers

A. Dermal papillae - finger-like projections that project into the epidermis (responsible for finger prints)

a) contain sweat & oil glands on ridges that leave prints

b) contains *Meissner's Corpuscles* (corpuscles of touch) – nerve endings sensitive to touch. Ends of fingers & toes have the highest density (#'s) of receptors than any other part of the body.
B. **Epidermal Ridges and Grooves** - outer surface of skin on hands and feet are marked by ridges & grooves by lines or loops and whorls; a conformity to the *dermal papillae*.  
**Function:** To increase friction for better grip of hands & feet.  
Act as tiny suction cups. The patterns (whorls, arches, loops) are genetically determined, but not the pattern arrangement itself. ie. you don't have the same finger prints as your parents!

C. **Striae** aka. “Stretch Marks” – torn collagen fibers in the dermis due to overstretching of skin, ie. pregnancy, wt. gain, growing too fast & muscle-bound.

D. **Lines of cleavage** “tension lines” – collagen/elastin fibers. Most important to surgeons because if incisions are made **perpendicular** to the lines of cleavage the wound tends to “gape open” and scar. * (see the ventral side of your fingers).

IV. Skin Color - due to pigment cells and blood capillaries.  
a. Pigment cells - 2 types (melanocytes & carotene cells = *plants*)

1. **Melanocytes** - found in the epidermis  
*produce a pigment called melanin.  
*skin color varies from pale yellow to black tinge (ethnicity)  
*the amount of melanocytes is about the same in all races; difference in skin color is due to the amount of melanin produced by the melanocyte.  
**“Coffee stains”** – concentrated areas of melanin, ie. birth marks

**Albanism** - an inherited inability in which the individual cannot produce the enzyme tyrosinase; therefore, cannot convert the amino acid tyrosine into melanin(pigment). It is absent in the hair, skin, and eyes.

**Vitiligo** - partial or complete loss of melanocytes from areas of the skin; causes patchiness. eg. Michael Jackson's claim?

**Freckles** - melanin formed in concentrated areas.
**Tanning** - exposure to UV light allows melanocytes to synthesize melanin with the aide of amino acid (tyrosine), and *melanin-stimulating hormone* (MSH) in the brain. Darkening of the skin “pigmentation” serves to protect the nucleus(DNA) against radiation damage.

Tyrosinase (enzyme)

Tyrosine → Melanin pigment

**Malignant Melanoma** – very invasive cancer of the melanocyte.

**Liver Spots** aka. Sun Spots - usually develop after 55 yrs. of age; clusters of melanocytes; “age spots”

2. **Carotene** – a yellow-orange *plant pigment* (carrots); it accumulates in *stratum corneum*, esp. where thickest, eg. palm/sole. Have you ever seen an orange kid?

3. Blood capillaries (containing Hb) in the dermis account for pinkish color of Caucasians; “blushing” due to vasodilation. “**Strawberry marks**” – concentrated blood capillaries; a strawberry-red birthmark.

4. **Cyanosis** “blue” – poorly oxygenated blood; respiratory disorder.

5. **Jaundice** – a buildup of yellow pigment *bilirubin* in the skin; indicates liver disease, eg. hepatitis

V. **Epidermal Derivatives: Accessory Structures** such as:

*(Hair, Glands & Nails)*

1. **Hair (pili)** - protects the body from injury, sun's rays, and foreign particles.

Composed of 4 parts:

a) **Shaft** – portion above the surface; has a cuticle - outer layer that contain *keratinized anucleated cells* (scale-like surface), keratin helps to waterproof the hair.

b) **Bulb** - an onion shaped structure at the base of each hair follicle. It has sensory nerve endings wrapped around it (root hair plexus).

c) **Hair follicle** - surrounds the root.
d) **Root** - portion below skin that penetrates the dermis

* Has *nucleated cells* (have DNA – used in identification)

**A. Hair Growth:** ~ 2 mm per week on average

- Influenced by hormones and nutrition
- Follicles are active for years then remain inactive, ie. eyebrow follicles are active for 3 – 4 months compared to follicles on the scalp; explains the diff. in hair length

**B. Hair removal:**

a. electrolysis – permanent; destroys the bulb w/ an electrical current.
b. Laser – permanent; destroys the bulb (more common now)
c. waxing or depilatory – “plucks” hair; doesn’t destroy bulb.

**C. Two Accessory Structures of Hair:**

1. **papilla** of the hair which provides nourishment (blood supply) to growing hair
2. **arrector pili muscle** - contracts under stress, fear, coldness (goose bumps)

*Gray Hair..... Never!*  
As people grow older the melanocytes are unable to produce tyrosinase; w/out this enzyme the melanocytes can’t convert tyrosine to melanin; the hair shaft is filled w/ air & stiff.

**Question?**
How are graying hair and albinism the same?

2. **Skin Glands** - 3 types of *exocrine glands*
   a) sebaceous glands - oil glands that secrete sebum.
   * connected to hair follicles; head & neck region
   * prevents *excessive water evaporation* from skin
   * helps keep hair and skin from drying
   * sebum inhibits growth of some bacteria

*Blackhead* - clogged oil gland w/ oxidized oil (black-head)  
*Pimple* - clogged and infected (bacterial) oil gland
*Seborrhea* – “cradle-cap”; hyperactive sebaceous glands
Vernix – fetal covering; waterproofs the skin from amniotic fluid
Lanugo – fetal hair; shed within two weeks of birth
Mammary glands- modified oil glands

b) sudoriferous gland - sweat gland
*secretes water, salts, urea, sugars, amino acids, & lactic acid
*functions to help regulate body temperature by evaporation of water in sweat which carries off large quantities of heat from surface of body.

c) ceruminous glands - secrete wax in external auditory canal; prevents entrance of foreign particles
*impacted cerumen - abnormal amount of ear wax built up in the ear and can prevent normal hearing.

3. Nails - hard keratinized cells of the epidermis
*grows out from nail root ~1mm/week
*the longer the digit the faster the growth
*provide protection to ends of digits and allow for better grasp

VI. Homeostasis of Body Temperature:
(see fig. 25.19, pages 977-978)
A Negative Feedback System!
**The output(cooling), is the opposite of the original condition(overheating) and vise versa.

A. Temperature Regulation Mechanisms in homeotherms:
1. Perspiration – increase or decrease activity.
2. Adjusting blood flow to the skin – vasodilation or vasoconstriction
3. Regulating metabolic rate – increase or decrease.
4. Regulating involuntary skeletal muscle contraction – “shivering” or not.

VII. Skin Wound Healing: (2 types); Figure 5.6, page 158-159
1. Superficial wound – trauma to epidermis;
24- 48 hours to heel, eg. abrasions; 1st & 2nd degree burns.
a) *basale cells* (stratum germinativum) migrate & advance towards one another until they encounter each other, ie. *contact inhibition.*
b) other basal cells divide to replace migrated cells.
c) once coverage of new cells beneath the scab is sufficient, the scab sloughs off.
d) the new surface becomes keratinized.

(Deep wound healing -4 phases); ~8 months to heel; eg. surgical cuts & accidental lacerations.

a) **Inflammatory** - a vascular(blood) & immune response.
   • serves to remove microbes, foreign material & dead tissue.
   • blood clot forms to unite edges of wound.

   ↑ blood flow(swelling- *adema* & redness/heat)
   due to vasodilation

   ↑ permeability of blood vessels to carry phagocytic cells(WBC’s)
   due to an immune response

b) **Migratory** - migration of epithelial cells beneath the scab; bridging the wound.
   • connective tissue(fibroblasts) synthesize *scar tissue* - *collagen fibers*
   • blood vessels regrow (*vaso-genesis*)

c) **Proliferative** - extensive growth of epi cells beneath scab.
   • continued deposition of scar tissue
   • continued growth of blood vessels

d) **Maturation** - scab sloughs off to restored epithelium.
   • collagenous fibers become more organized, ie. less scar tissue

*Scar tissue* – irregular, densely packed collagen fibers; no epidermis & may not have hair glands, or sensory neurons.
Skin may be raised and ugly in this region.
VIII. Effects of Aging:
- By late forties collagen fibers decrease in number, stiffen, break apart and form matted tangles.
- Elastic fibers lose elasticity, fray & clump; as a result the skin forms crevices “wrinkles”.
- *Fibroblasts*, which produce collagen & elastin, become fewer, ie. less collagen & elastin production.
- *Macrophages* (immune cells) become less efficient phagocytes, which increases susceptibility to skin infection; skin heals poorly.
- Sebaceous glands reduce in size and oil production which causes dryness and broken skin.
- Production of sweat diminishes w/ age; elderly have a higher incidence of heat stroke.
- Decreased number and size of functioning melanocytes resulting in gray hair & *atypical* skin pigmentation, ie. liver spots and grayish colored skin.

IX. Skin Cancer – 3 types: (see photo plates 5.8, page 164)
- all are *malignant tumors* of the skin that metastasize (spread to other parts of the body) at various rates.

1. Basal cell carcinoma – most common (75% of skin cancers); 99% cure.
2. Squamous cell carcinoma – tends to metastasize complete cure is good.
   - Most invasive & deadly

**Causes:**
Chronic Overexposure of the skin to UV light; most dangerous!
- See the ABCD(E) Rule on page 164.

**Treatment:** radiation therapy, excision and cryosurgery.

A. Causes of Skin Cancer:
*Chronic Overexposure of the skin to UV rays!
Ultraviolet rays; (2 types)
1. *UVA* - most of the UV that reaches the earth’s surface; they penetrate deep into the skin to activate the melanocytes; they cause tanning of the skin. Thought to cause skin cancer because sunscreens DO NOT block UVA.
2. *UVB* - cause burns; more likely to accelerate the aging process. Sunscreens DO block UVB rays.
X. **Burns** – tissue damage caused by heat, electrical, wind, friction, UV rays or chemicals; all denature the proteins in the skin cells then they die.

- **Types of burns**: (see pages 164 - 165)
  - 1\(^{st}\) degree – only the epidermis; no scar
  - 2\(^{nd}\) degree – all of epidermis & upper dermis; can scar
  - 3\(^{rd}\) degree “full-thickness burn” – all of the skin; Requires a skin graf.

  - 3\(^{rd}\) degree burns are Life-threatening due to:
    1. Severe fluid and electrolyte loss leading to dehydration and circulatory shock (lowered blood volume).
    2. Increased risk of infection because of the lack of protection by the skin. Also, lowered immune system.

**Rule of Nines** – method for determining the amount of body surface burned. Divides the body into 11 areas accounting for 9% each. (9 x 11 = 99%)

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**The Poison Oak Scenario:**

**A Cell-Mediated Immune Response**

An allergic contact dermititis caused by the shrub *Toxicodendron diversilobum* which contains a resin (urushiol) w/in resin canals of stems, roots, leaves & fruits.

**Phase I: Induction phase -**

1. Urushiol (allergen) penetrates the statified squamous cells and binds to Langerhan cells or is engulfed by the L-cells. The allergen is “displayed” on the cell membrane of the L-cell.

2. The L-cell, presenting the allergen, migrates to a nearby lymph node where special immune cells (*effector T-cells*) are programmed to recognize urushiol. [This is a cell-mediated immune response!] The *effector T-cells* (with their glycoprotein receptors) roam the blood & lymphatic system searching for urushiol.
Phase II: (Elicitation phase) - if you get urushiol allergen absorbed into the skin during a subsequent encounter with poison oak; the L-cell will bind to it.
1. A patrolling effector T-cell may encounter the L-cell and bind to it; T-cells have memory!
2. The effector T-cell makes more clones of itself and releases special proteins called lymphokines that attract a host of other WBC’s (macrophages, and “killer T-cells”).
3. These WBC’s release toxic enzymes that destroy the urushiol and skin cell junctions (tight junctions); makes the skin leaky.
4. Blistering rashes, lymphatic ooze, redness and itching occur.
Note:
* no evidence of racial immunity
* poison oak does not spare sex, age, or color of skin

**Why are some people not sensitive to Poison Oak?**

**Four scenarios:**

1) They may **not have** effector T-cells w/receptors for poison oak (urushiol), or ....
2) They may have **few** effector T-cells w/receptors for poison oak, and the allergen (urushiol) may be absorbed or degraded before **effector T-cells** find it.
3) Immunity to poison oak w/age & exposure or use of homeopathic remedies may involve **suppressor T-cells** that inhibit or block the action & reproduction of other T-cells (immune cells).
4) AIDS patients may have reduced sensitivity to poison oak.

**WHY SO?**

Note: These individuals may be able to roll in poison oak with impunity!

**The Poison Oak Scenario Diagrammed:**
Langerhan's phagocitize Urushiol → L-cell

Langerhan's travel to nearby lymph node & present urushiol to the immune system

Lymph Node

Immune system produces Effector T-cells with receptors for recognizing Urushiol

This complex of Effector T-cells & Langerhan's w/urushiol release a chemical called "lymphokines." This chemical attracts other immune cells (killer T-cells & macrophages) which release toxic, destructive enzymes that destroy your skin.

"lymphokines"

Effector T-Cell

Effector T-cells Roam the Blood Stream & Lymphatic vessels looking for urushiol to bind with

Destructive enzymes to skin!