The Integumentary System

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Integumentary System – Skin Development

Overview

- Largest organ of the body.
- Dual origin:
    1. Epidermis → from surface ectoderm.
    2. Dermis → from underlying mesenchyme (mesoderm + neural crest).

Epidermis Development

1. Initial stage:
    - Embryo initially covered by single-layered ectoderm.
2. 2nd Month:
    - Ectoderm divides → forms periderm (epitrichium) = superficial flattened layer.
3. With basal layer proliferation:

- Intermediate zone forms.

4. By 4th Month → definitive 4 layers:

    - Basal layer (germinative layer) → stem cell layer; forms ridges & hollows → fingerprints.
    - Spinous layer → large polyhedral cells with tonofibrils.
    - Granular layer → keratohyalin granules.
    - Horny layer (stratum corneum) → dead keratinized cells → protective barrier.

5. Periderm fate:

    - Shed in 2nd half of intrauterine life → cells found in amniotic fluid.

6. Melanocytes:

    - Derived from neural crest → migrate into epidermis by 3rd month.
    - Contain melanosomes → transfer melanin to keratinocytes.
    - Responsible for skin & hair pigmentation.

Dermis Development

- Derived from mesenchyme with 3 sources:

1. Paraxial mesoderm → dermis of back.

2. Lateral plate mesoderm → dermis of limbs & body wall.

3. Neural crest cells → dermis of face & neck.

- 3rd–4th months:

    - Dermis (corium) forms dermal papillae → project into epidermis.
    - Each papilla usually contains a capillary loop or sensory nerve ending.

- Deeper dermis (subcorium) → rich in fatty tissue.

Special Features at Birth

- Vernix caseosa:

    - Whitish, greasy covering over newborn skin.
    - Formed by:
        - Sebaceous gland secretions.
        - Degenerated epidermal cells.
        - Fine hairs.
    - Function → protects skin against maceration by amniotic fluid.

Clinical Correlates

1. Pigmentary Disorders

- Piebaldism → patchy absence of melanocytes → areas without hair/skin pigment.
- Waardenburg Syndrome (WS):
  - Features: white forelock, heterochromia iridis, depigmented patches, deafness.
  - Cause: defective neural crest migration → absence of melanocytes in stria vascularis.
  - Gene: PAX3 mutations → WS1 & WS3.
- Albinism (oculocutaneous albinism – OCA):
  - Defect in melanin synthesis/processing → little/no pigmentation in skin, hair, eyes.
- Vitiligo:
  - Autoimmune destruction of melanocytes.
  - Patchy depigmentation in skin, hair, oral mucosa.
  - Associated with other autoimmune diseases (esp. thyroid disease).

2. Fingerprints (Dermatoglyphics)

- Formed by epidermal ridges (from basal layer).
- Appear on fingertips, palms, soles.
- Genetically determined → unique to each person.
- Clinical importance:
    - Used in forensics & genetic studies.
    - Abnormal patterns seen in chromosomal disorders (e.g., Down syndrome).

3. Keratinization Disorders

- Ichthyosis:
    - Excessive keratinization → scaly, dry skin.
    - Mostly autosomal recessive, some X-linked.
- Severe form – Harlequin fetus:
    - Thick, cracked, armor-like skin.
    - Often fatal shortly after birth.

Hair Development

Origin

- From epidermis (ectoderm) → solid proliferations from germinative (basal) layer.
- These proliferations grow downward into dermis.

Stages of Development

1. Hair Bud Formation
    - Solid epidermal outgrowth penetrates dermis.
2. Hair Papilla Formation
    - Terminal end of hair bud invaginates, forming hair papilla.
    - Papilla filled with mesodermal tissue → blood vessels & nerve endings.
3. Differentiation
    - Central cells of hair bud → spindle-shaped → keratinized → hair shaft.
    - Peripheral cells → cuboidal → epithelial hair sheath.
4. Surrounding Mesenchyme
    - Forms dermal root sheath.
    - Also gives rise to arrector pili muscle (smooth

muscle).

5. Growth

    - Continuous proliferation of epithelial cells at base pushes hair shaft upward.
    - By end of 3rd month → first hairs appear (eyebrows & upper lip).
    - First hair = lanugo hair: fine, soft, temporary → shed around birth and replaced by coarser terminal hair.

Associated Structures

- Sebaceous Glands:

    - Develop as buds from epithelial wall of hair follicle.
    - Central gland cells degenerate → form sebum (oily secretion).
    - Sebum passes into hair follicle → reaches skin surface.

Clinical Correlates

Abnormalities of Hair Distribution

1. Hypertrichosis (excessive hairiness)

- Cause: increased number of hair follicles.
- Types:
  - Localized → e.g., lumbosacral region over spina bifida occulta.
  - Generalized → entire body covered.

2. Atrichia (congenital absence of hair)

- Usually associated with other ectodermal defects (e.g., teeth & nail anomalies).

Sweat Glands & Mammary Glands Development

Sweat Glands

Types

1. Eccrine Sweat Glands

- Origin: Buds from germinative layer of epidermis.
- Growth: Buds extend into dermis, coiling at the ends → form secretory portion.
- Associated smooth muscle cells also derive from epidermal buds.
- Mode of secretion: Merocrine (exocytosis).
- Function: Temperature regulation.
- Distribution: Present all over body (except a few regions like lips, external genitalia).

2. Apocrine Sweat Glands

    - Origin: From same epidermal buds as hair follicles.
    - Location: Axillae, pubic region, face, areola, perianal region.
    - Development: Begin functioning only at puberty (hormonal influence).
    - Opening: Into hair follicles (not directly onto skin).
    - Secretion: Contains lipids, proteins, pheromones.
    - Odor: Due to bacterial breakdown of secretions.
    - Mode of secretion: Classified as apocrine (part of cytoplasm lost with secretion).

Mammary Glands

- Modified sweat glands.

- Initial appearance:

    - As bilateral mammary lines (ridges) → thickened epidermis.
    - Extend from axilla to inguinal region (base of forelimb → hindlimb).

- Most of mammary line disappears, except a small portion in thoracic region → persists & penetrates underlying mesenchyme.

Stages

1. Sprouting stage
    - 16–24 solid epithelial sprouts form.
    - Later canalize → lactiferous ducts.
2. Nipple formation
    - Initially → ducts open into epithelial pit.
    - After birth → mesenchymal proliferation everts the pit → nipple.
3. At birth
    - Only duct system present.
    - No alveoli or secretory units.
4. At puberty (female)
    - Under influence of estrogen & progesterone:
        - Branching of ducts.
        - Formation of alveoli & secretory cells.

Clinical Correlates

Sweat Glands

- Not many embryological anomalies described. Important mainly for function & secretion type.

Mammary Glands

1. Polythelia
    - Accessory nipples due to persistence of mammary line fragments.
    - Usually in axillary region.
    - Most common mammary abnormality.
2. Polymastia
    - Extra breast develops along mammary line.
3. Inverted Nipple
    - Failure of epithelial pit to evert.
    - Clinical importance: May mimic or predispose to pathological nipple inversion in adults.

✅ Exam Points

- Eccrine = all over body, merocrine, temp regulation.
- Apocrine = puberty, hair follicle association, apocrine secretion.

- Mammary gland = modified sweat gland, ridge origin, ducts form prenatally, alveoli only after puberty.

- Abnormalities: Polythelia > Polymastia > Inverted nipple.