Line Derma 1
Medical training
(Derma Product)

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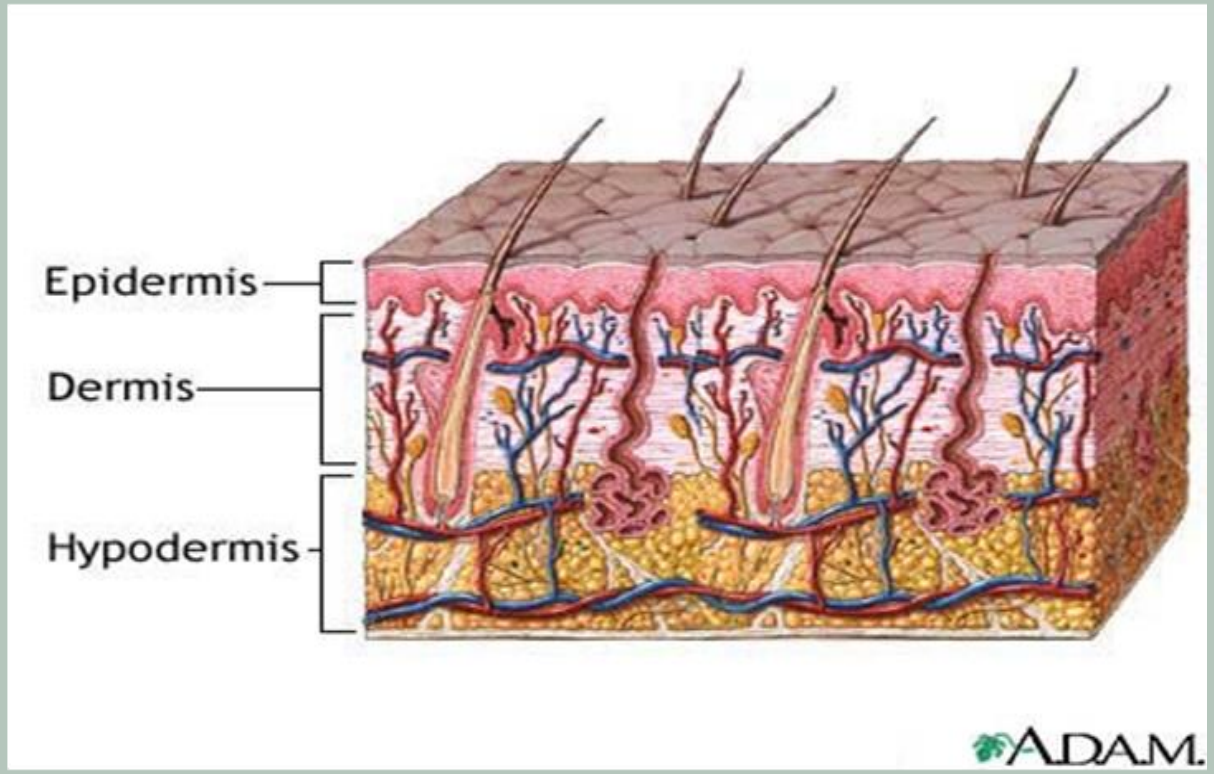
Medical background of derma portfolio

(Allerzone- Perfectodil- Flacort- Trozal)

Skin Structure

1-Epidermis
2-Dermis
3-Subcutaneous layer



Epidermis

- **Formed from 5 layers.**

1.Stratum cornium

2.Stratum lucidum

3.Stratum granulosum

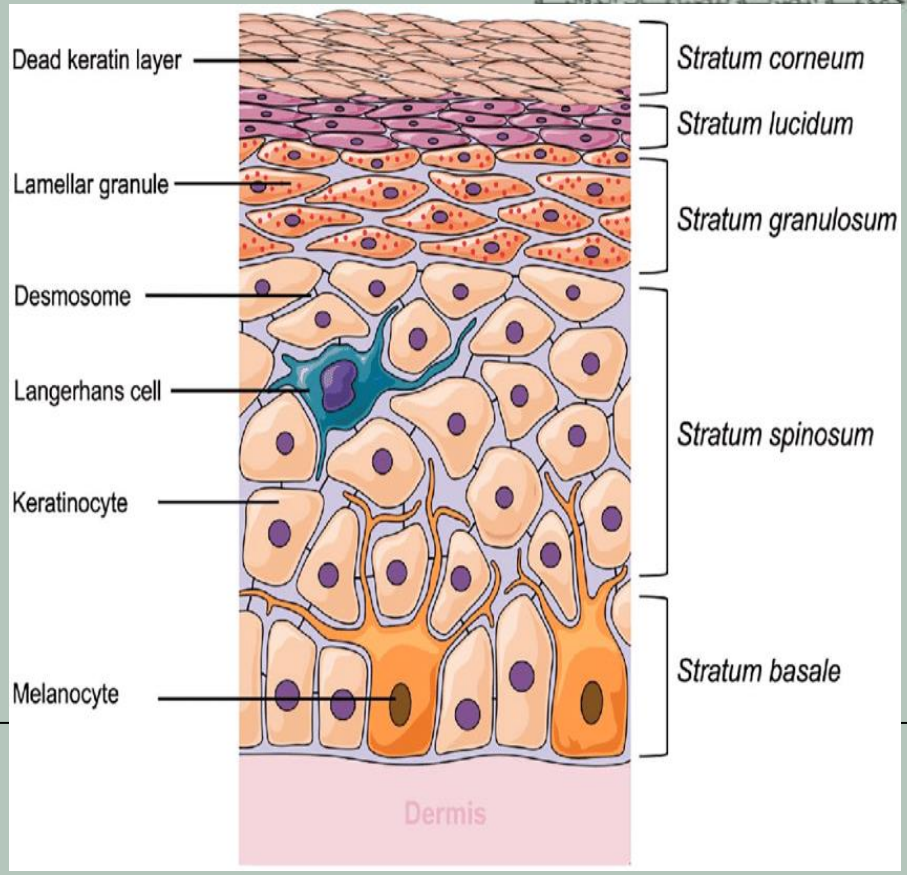
4.Stratum spinosum

5.Stratum basale

- acts as a protective barrier

-The epidermis also contains **melanocytes**,

which are cells that produce **melanin** (skin pigment).

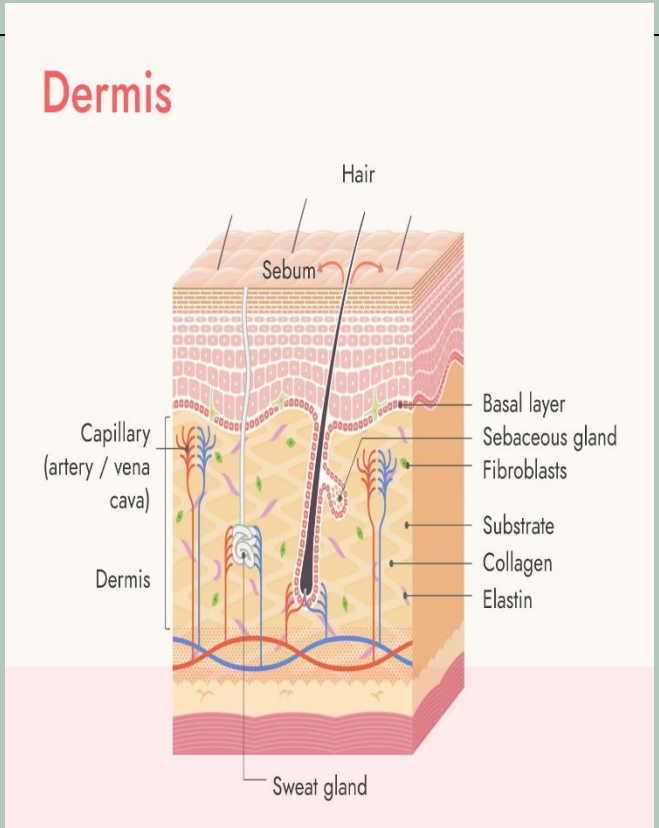


Dermis

The **dermis** is the middle layer of the skin. The dermis is made up of the following:

- blood vessels
- lymph vessels
- **hair follicles**
- sweat glands

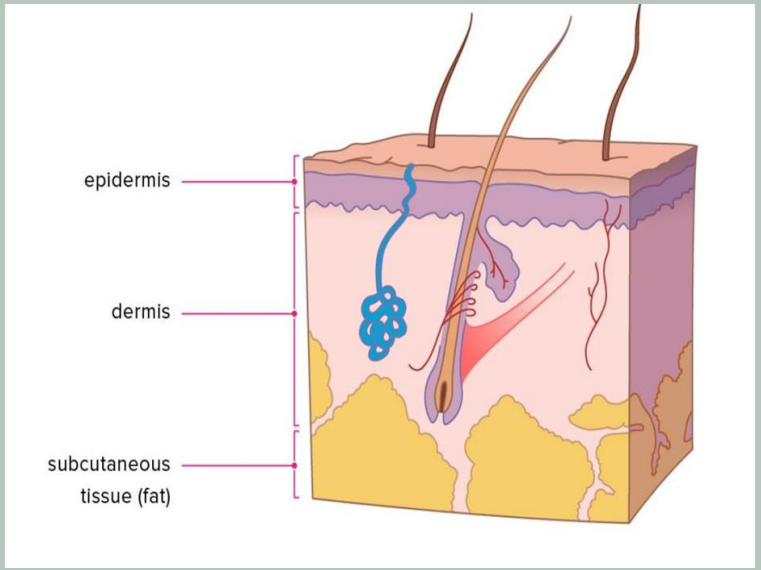
The dermis is held together by a protein called collagen, made by fibroblasts (skin cells that give the skin its strength and elasticity). This layer also contains pain and touch receptors.



Hypodermis

Is the deepest layer of skin and is also known as the subcutaneous layer.

consisting of a network of fat cells that helps conserve the body's heat



Barrier function

- What it is: **The skin's barrier function** is its role as a protective shield against the outside world, preventing excessive water loss and keeping harmful substances like microorganisms, chemicals, and UV radiation out, and it is maintained through hydration and sebum regulation.
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The skin's barrier function

- **Hydration** is crucial for barrier function, as the skin needs to prevent excessive water loss, and the stratum corneum is composed of layers of lipids that form a permeability barrier.

- Sebum, the oily substance produced by sebaceous glands, moisturizes the skin, lubricates hair, and its slightly acidic nature helps protect against pathogens.

Hydration

Role: Proper hydration is critical for maintaining the skin's barrier function.
Mechanism: The intercellular lipid layer in the stratum corneum forms a tight, effective barrier that prevents excessive water loss from within the body.
Sebum regulation
What it is: Sebaceous glands produce sebum, an oily substance that lubricates and protects the skin and hair.

Moisturization

Moisture: Sebum helps prevent excessive water loss from the skin. Protection: Its slightly acidic pH (4.5-6.0) helps inhibit the growth of harmful bacteria and other pathogens.

Thermoregulation: Sebaceous secretions can emulsify sweat to delay dehydration during hot conditions and repel water in cold conditions.

Hydration vs. moisturization

Hydration: Focuses on increasing the water content in the skin, often using humectant ingredients like hyaluronic acid and glycerin.

Moisturization: Focuses on locking in that water using emollient ingredients like ceramides and fatty acids, which create a protective barrier to prevent water loss.

The relationship: Hydration and moisturization are both essential. You need to hydrate your skin with water before you can seal it in with a moisturizer. Products that provide both are often the most effective.

Urea usage

- Urea in skincare acts as a powerful humectant (drawing in moisture) and keratolytic (exfoliating) agent, deeply hydrating, softening rough skin, supporting the skin's natural barrier, and improving texture by breaking down dead skin cells,
- making it ideal for treating dry conditions like eczema, psoriasis, and calluses, and even boosting penetration of other topical treatments.

- Key Functions of Urea for Skin:
- **Hydration (Humectant):** Urea attracts water from the environment and binds it to the skin, keeping it moisturized, plump, and soft.
- **Exfoliation (Keratolytic):** At higher concentrations, it dissolves the "glue" holding dead skin cells together, allowing them to shed, which smooths rough skin and improves texture.

Common Uses

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- **Key Functions of Urea for Skin:**
- **Skin Penetration: It helps other active ingredients penetrate deeper into the skin for better effectiveness.**
- **Soothing & Healing: It has anti-inflammatory, antibacterial, and antifungal properties, helping to calm irritation and support wound healing.**

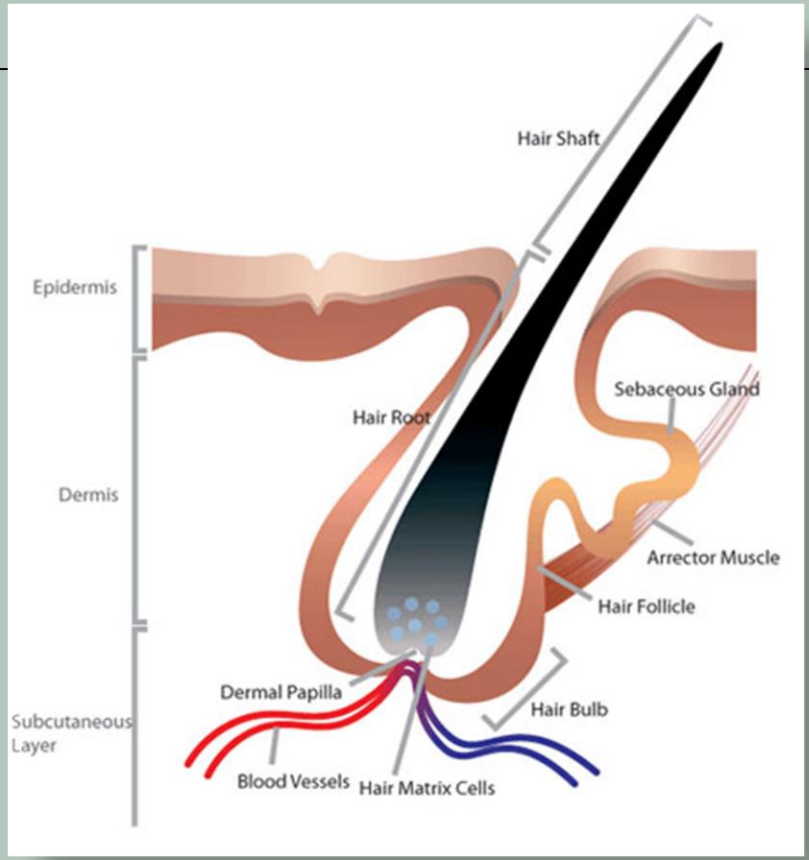
- **Treating severely dry skin (xerosis).**
- **Managing symptoms of eczema, psoriasis, and ichthyosis.**
- **Softening thick, callused skin on feet, elbows, and knees.**
- **Debridement (removal) of thick nails.**

Hair Structure

- Hair is composed of strong structural protein called keratin.
- Keratin is the same kind of protein that makes up the nails and the outer layer of skin.

Hair Structure

- The part of the hair strand above the epidermis is called the *hair shaft*
- Below the surface of the skin is the *hair root*, which is enclosed within a hair follicle
- A sheath of connective tissue surrounds the whole hair root and together these form a *hair follicle*



Hair Structure

Hair shaft consists of three layers:

1. **Medulla:** The innermost layer , which is present only in large thick hairs.

2. **Cortex:** The middle layer, which provides strength, color and texture to the hair.

3. **Cuticle:** The outermost layer is a thin and colorless layer, which serves as a protector of the cortex.

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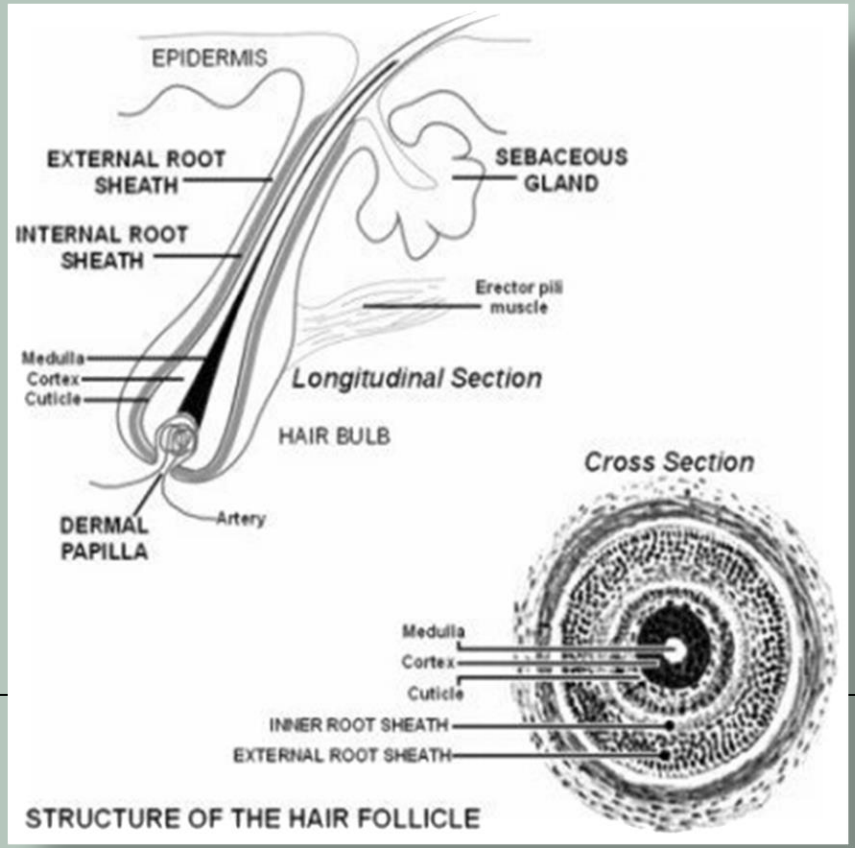

Hair Growth Cycle

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Hair follicles grow in repeated cycles, while each cycle is divided into three phases

Hair growth cycle

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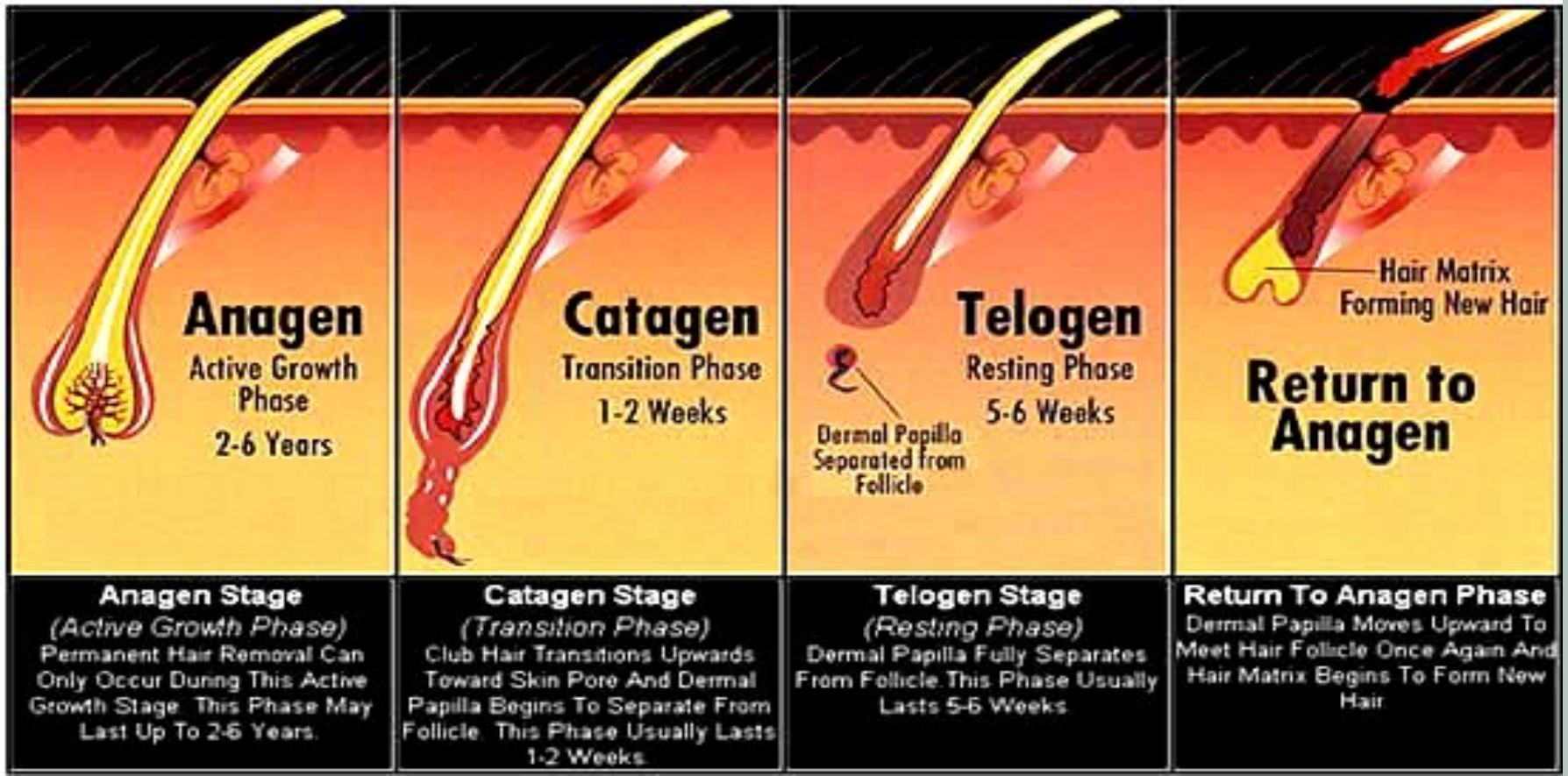
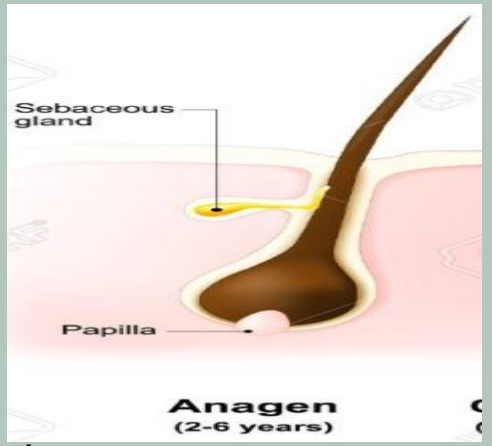


Hair Growth Cycle

- ***ANAGEN* Phase** (Growth Phase)

At any one time, 80 - 90 percent of your hair follicles on your scalp are in the ANAGEN phase



- During this period
- Your hair grows continuously
- The growing will continue for 2 to 6 years
- Grows at the rate of half an inch a month
- The hair bulb produces your hair pigment
- Blood supply provides nutrients and minerals to your hair
- Looks thick and nourished

Hair Growth Cycle

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- ***CATAGEN* Phase** (transitional Phase)

During this phase; hair will turn into a transitional phase before going to rest.

This short phase is last for 2 to 4 weeks.

- During this period

- Hair detaches from the blood supply
- The detached follicle will slowly shrink to about 1/6 its size
- The hair bulb stops producing the color pigment
- The bulb will be pushed upwards towards the surface when the new hair is formed
- Approximately 2 - 3 % of hair will be in this phase in your scalp

Hair Growth Cycle

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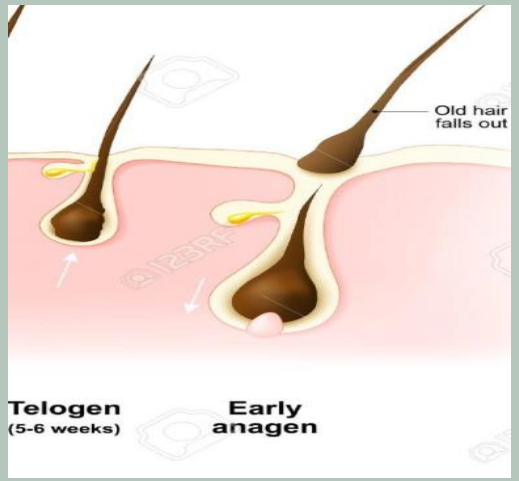
- ***TELOGEN* Phase** (Resting Phase)

During this phase; hair follicles will slowly fall off and replaced by a new hair.

Around 10 - 15 % of the hair in your scalp will be in TELOGEN phase



- During this period
- 50 - 100 hairs from this phase will shed daily
- This period lasts for 3 months before the hair falls out
- The hair follicles become weak and thin and you can easily pull them out - new hair follicle will emerge once the hair falls.

Skin Functions

Protection	Heat Regulation	Sensation	Identification
• Toxins • Injuries • Sun • Temperature	Through sweat	Through nerves	• Finger • Sole prints

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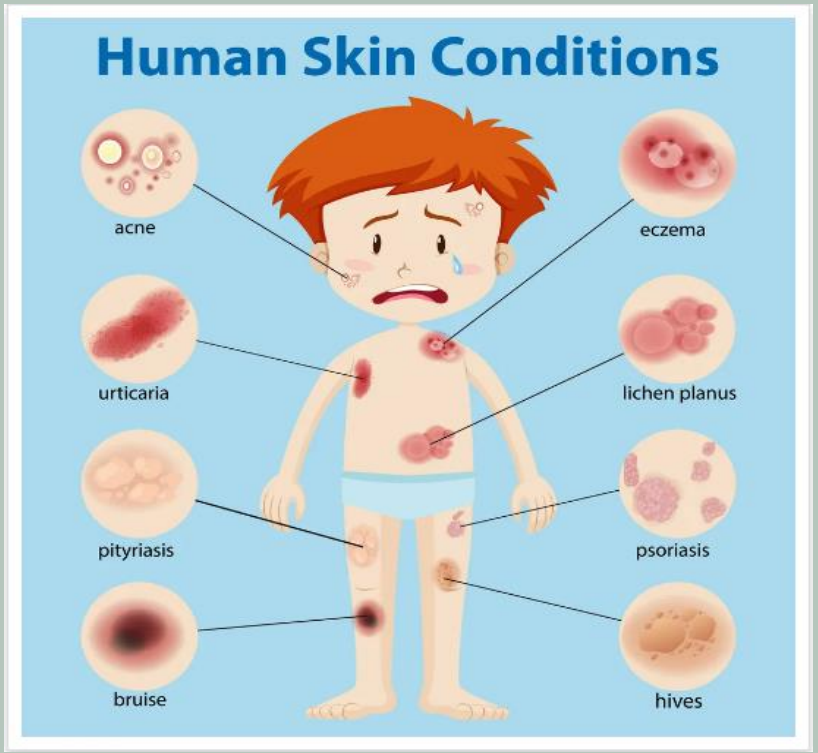
1-Hair loss
2- Contact Dermatitis
3- Diaper Dermatitis
4- Atopic Dermatitis
5- Seborrheic Dermatitis
6-Lichen Planus
7- Lichen Simplex
8-PITYRIASIS ROSEA
9-DERMATOMYCOSIS



Causes of Hair Loss

- **Improper hair care**
  - **Chemical treatments including dyes, tints, bleaches, straighteners & permanent waves.**
  - **Hairstyles like ponytails .**
- **Inadequate protein in diet : in this case; the body will save protein by shifting growing hair into resting phase.**
- **Hereditary thinning or balding : Androgenetic Alopecia.**

- **Autoimmune factors**: Alopecia Areata.
- **Medications:** drugs for gout, depression, heart problems, and high blood pressure.
- **Chemotherapy**
- **Low serum iron**: low iron content in iron-deficient or heavy menstrual women
- **Thyroid diseases**: over- and underactive

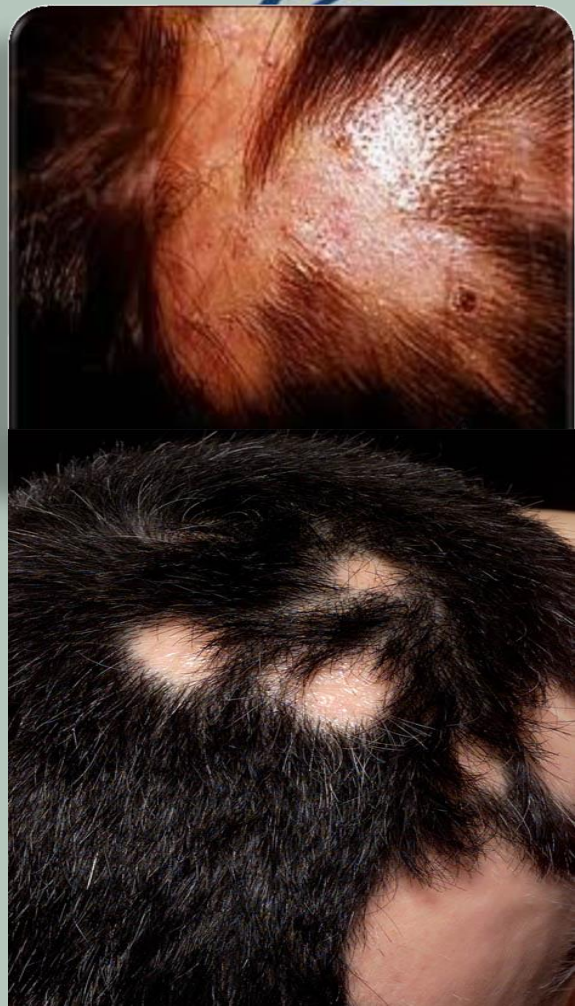
Types of Hair Loss

- **Scarring Alopecia**

- ***Rare disorders*** **that destroy the hair follicle, replace it with scar tissue, and cause permanent hair loss.**

- ***Seen as*** **localized areas of scarring alopecia of the scalp.**

- ***Causes*** **: bacterial, viral and fungal infections.**



Types of Hair Loss

➢ Non Scarring Alopecia

1. Due To Abnormal Breakage

- **Traction Alopecia**: Due to excessive traction of hair.

- **Cosmetic Breakage**: Due to excessive use of hair cosmetics as straighteners and shampoos.

- **Acquired** or **Congenital** Hair Shaft Abnormalities

Types of Hair Loss

➢ Non Scarring Alopecia

2. Due To Abnormal Cycling

- **Telogen effluvium**:

Results from increasing number of hair entering into resting phase.

Causes: Pregnancy, Iron deficiency, rapid weight loss

- **Anagen Arrest**:

Most common cause is Chemotherapy or radiation

It is not permanent and once treatment is stopped hair will grow back.

Types of Hair Loss

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➢ Non Scarring Alopecia

2. Due To Abnormal Cycling

- **Androgenetic Alopecia**:

  (Male / Female Pattern Hair Loss)
  The most common type of hair loss.

- **Causes** :

Presence of Dihydrotestosterone hormone (DHT) triggers Androgenetic Alopecia in genetically susceptible persons.

DHT formed from conversion of Testosterone Hormone by the action of 5 alpha reductase enzyme

Types of Hair Loss

➢ Non Scarring Alopecia

2. Due To Abnormal Cycling

- **Alopecia Areata:**

  - **Auto-Immune disorder resulted from attacking hair follicles by T-Lymphocytes what causes hair stop growing and enter into Telogen Phase.**

  - **Seen as smooth round bare patches.**

Several Ways For Management of Hair Loss

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1. **Lifestyle Modification**

   - Aerobic Exercise (stress & free, Testosterone levels)

   - Diet containing adequate amounts of Protein , Carbohydrates & Fat

2. **Medications**

3. **Hair Transplantation**

4. **Laser Treatment**

5. **Alternative Medicine**

   - Using Essential oils in the massaging of scalp

DHT Inhibitors

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- Synthetic molecules inhibit the formation of DHT through inhibition of 5 alpha reductase enzyme that is responsible for converting Testosterone to (Dihydrotestosterone) DHT
- **Example :** Finasteride
- **Side Effects :**

  Impotence , Ejaculatory abnormalities , Erectile dysfunction , Testicular pain and Depression.
- **Contraindications :**

  - contraindicated for use by women as it cause congenital disorders & birth defects.

  - crushed tablets shouldn`t be handled by pregnant women as it absorbed through skin.

Anti-Androgens

- This medication act by stopping the Dihydrotestosterone (DHT) from binding to the androgen receptors.
- Example : Topical Spironolactone
- Spironolactone is a Pot. Sparing diuretic, its oral form used mainly in treatment of hypertension but as many side effects when used in the treatment of hair loss.
- Side Effects for oral therapy:
- Erectile Dysfunction, Ataxia, Nausea & Vomiting.
- Topical Spironolactone is more preferable form to avoid these side effects , but its results are seen after 1-2 years

Anti-Inflammatory & Immune-Suppressive Medications

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- **Anti- Inflammatory**
- act by reducing inflammation, itching and redness caused by the hormonal and immune system reactions going on in your scalp.

- **Immune-Suppressive** act by inhibiting T-Lymphocytes from attacking hair follicles so allow hair re-growth.

- Example : **Corticosteroids**

➢ These medications used in treatment of Alopecia Areata , and can be also used in combination with other medications for hair loss treatment.

- Side Effects :

Edema , general immune-suppression & flaring of infections.

Growth Stimulants

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These medications are shown to be effective in slowing or stop hair loss and stimulates new hair growth.

Example : Minoxidil

Minoxidil can be used alone or in combination with DHT inhibitors for treatment of hair loss.

It remains a fact that only two hair loss treatments have been approved by the FDA and both can be classed as strong drug-based medications

Minoxidil & Finasteride


BREAK
TIME !!

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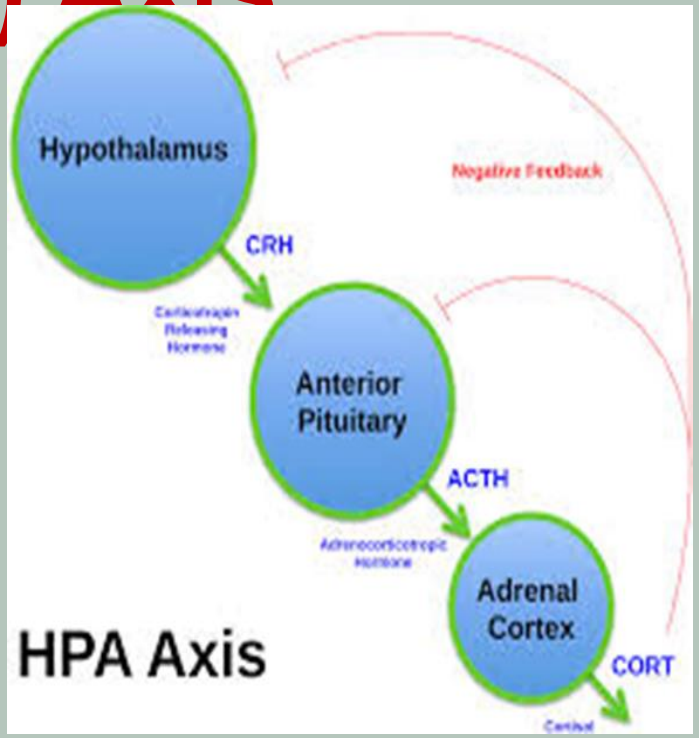
Hypothalamic-Pituitary-Adrenal (HPA) Axis

- **The hypothalamic-pituitary-adrenal (HPA) axis** is your body's main way of responding to stress.

- It consists of 3 organs that each release hormones to raise cortisol levels in your body eventually.



- The **HPA axis** consists of your:

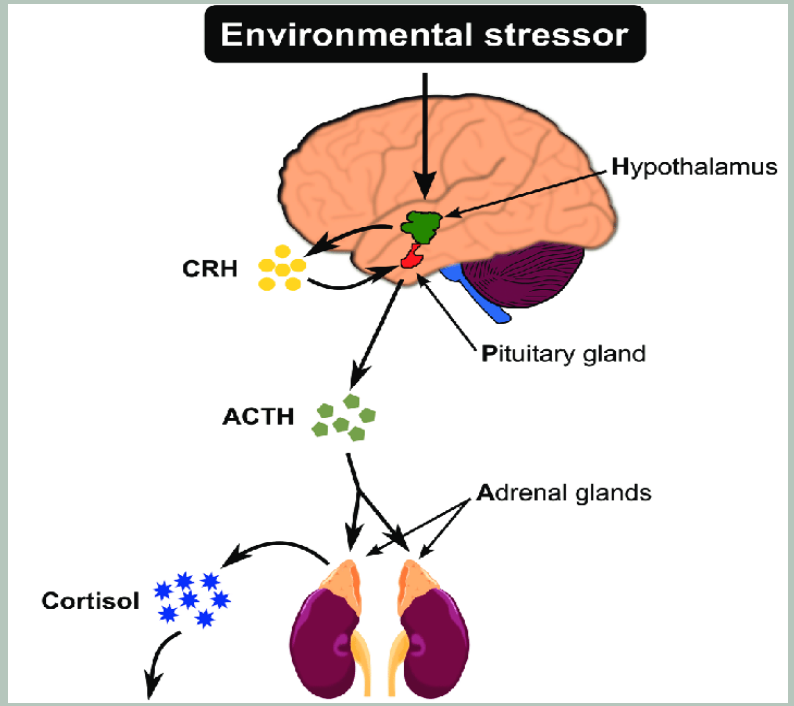
- **Hypothalamus**: The hypothalamus is a structure deep within your brain. Your hypothalamus keeps your body in a balanced state concerning body temperature, hunger, mood and many other functions. It does this by directly influencing your autonomic nervous system or managing hormones.

- **Pituitary gland**: The pituitary gland is a small, pea-sized gland located at the base of your brain below your hypothalamus. It makes several essential hormones and regulates other endocrine glands. The anterior pituitary, specifically, is part of the HPA axis.

- **Adrenal glands**: Adrenal glands are small, triangle-shaped glands on top of each of your kidneys. They produce certain hormones that help regulate several important bodily functions. The adrenal cortex, specifically, is part of the HPA axis.

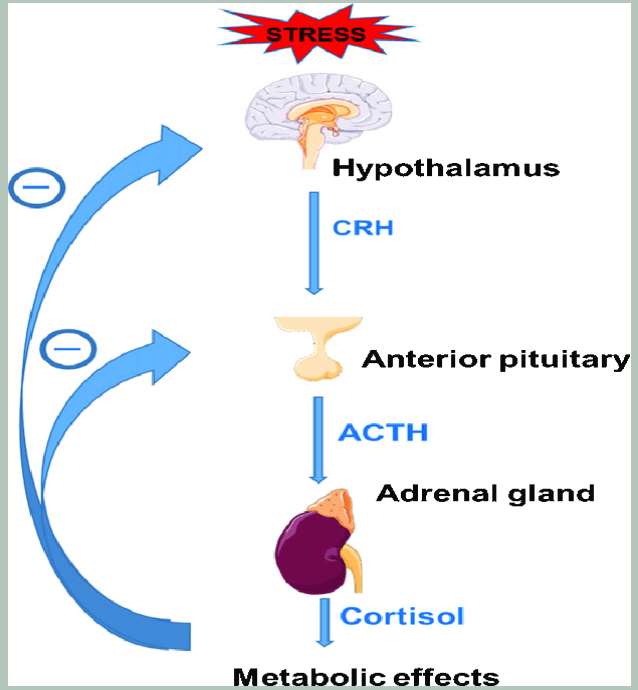
Hypothalamic-Pituitary-Adrenal (HPA) Axis

- The main function of your HPA axis is to release **cortisol** (a glucocorticoid, or steroid hormone).
- **How does the HPA axis work?**
- 1- In response to a stressful situation, your autonomic nervous system triggers your hypothalamus to release **corticotrophin-releasing hormone (CRH).**
- 2- CRH triggers your anterior pituitary to release **adrenocorticotropic hormone (ACTH).**
- 3- **ACTH** then triggers your adrenal glands, specifically your adrenal cortex, to release **cortisol.**



Hypothalamic-Pituitary-Adrenal (HPA) Axis

- The HPA axis is meant to have a fine-tuned negative feedback loop: the cortisol in your body triggers your hypothalamus to stop making CRH,
- ending the stress response.



Natural Synthesis of corticosteroids

Adrenal cortex

Corticosteroids

Sex hormons

Glucocorticoids

Mineral corticoids

Androgens

Cortisol

Aldosterone

Controlling of inflammatory responses

Corticosteroids

- **The main corticosteroids produced by the adrenal cortex are cortisol and aldosterone.**[1]
- **Corticosteroids** are synthetic (human-made) drugs that are similar to cortisol, a hormone your body naturally produces.
- Corticosteroids have a similar anti-inflammatory effect throughout your body.
- **Corticosteroids** are involved in a wide range of physiologic systems such as:

-stress response
- immune response
- regulation of inflammation
-carbohydrate metabolism
-protein catabolism
-blood electrolyte levels
- behavior.

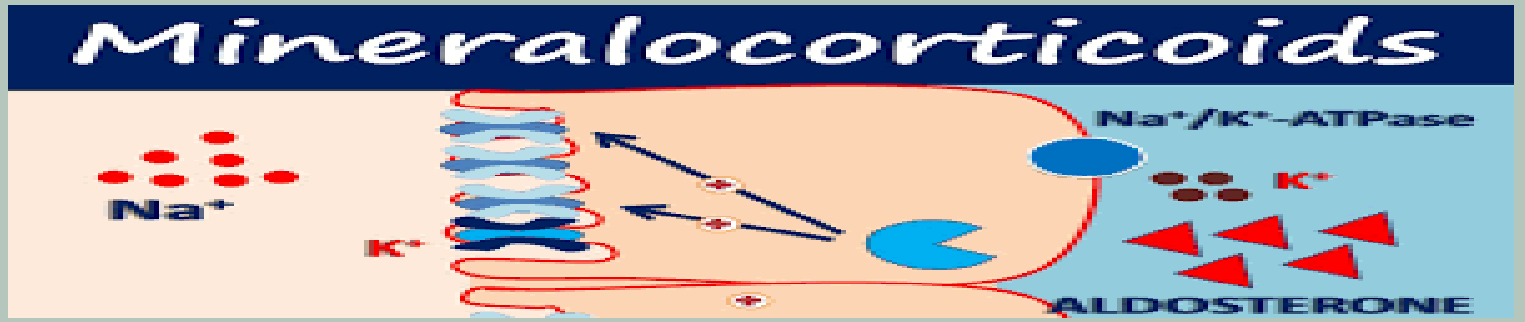
Effect of Cortisol

- **Glucocorticoids** such as cortisol affect : [2]
- carbohydrate
- fat
- protein metabolism
- anti-inflammatory
- immunosuppressive
- anti-proliferative
- and vasoconstrictive effects

Anti-inflammatory effects	are mediated by blocking the action of inflammatory mediators (transrepression) and inducing anti-inflammatory mediators (transactivation
Immunosuppressive effects	are mediated by suppressing delayed hypersensitivity reactions by direct action on T-lymphocytes.
Anti-proliferative effects	are mediated by inhibiting DNA synthesis and epidermal cell turnover.
Vasoconstrictive effects	are mediated by inhibiting the action of inflammatory mediators such as histamine.

Mineralocorticoids

- **Mineralocorticoids** such as aldosterone are primarily involved in:
- regulating electrolyte and water balance by modulating ion transport in the epithelial cells of the renal tubules of the kidney. (2)



CLASSIFICATION OF ADRENOCORTICOSTEROIDS

Natural	Synthetic
Glucocorticoids	
Hydrocortisone (cortisol) Cortisone	Prednisone, Prednisolone Methyl-prednisolone Triamcinolone Betamethasone, Dexamethasone
Mineralocorticoids	
Aldosterone Desoxycorticosterone (DOC)	Fludrocortisone

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• Acc To chemical structure(3)

- **Group A – Hydrocortisone type**

Hydrocortisone, hydrocortisone acetate, cortisone acetate, tixocortol pivalate, prednisolone, methylprednisolone, and prednisone.

- **Group B – Acetonides (and related substances)**

Amcinonide, budesonide, desonide, fluocinolone acetonide, fluocinonide, halcinonide, triamcinolone acetonide, and **Deflazacort (O-isopropylidene derivative)**

- **Group C – Betamethasone type**

Beclometasone, betamethasone, dexamethasone, fluocortolone, halometasone, and mometasone.

- **Group D – Esters.**
- **Group D1 – Halogenated (less labile)**

Alclometasone dipropionate, betamethasone dipropionate, betamethasone valerate, clobetasol propionate, clobetasone butyrate, fluprednidene acetate, and mometasone furoate.

- **Group D2 – Labile prodrug esters**

Ciclesonide, cortisone acetate, hydrocortisone aceponate, hydrocortisone acetate, hydrocortisone buteprate, hydrocortisone butyrate, hydrocortisone valerate, prednicarbate, tixocortol pivalate.

Classification of Corticosteroids

- **ACC To route of administration(4):**
- **Topical steroids :**

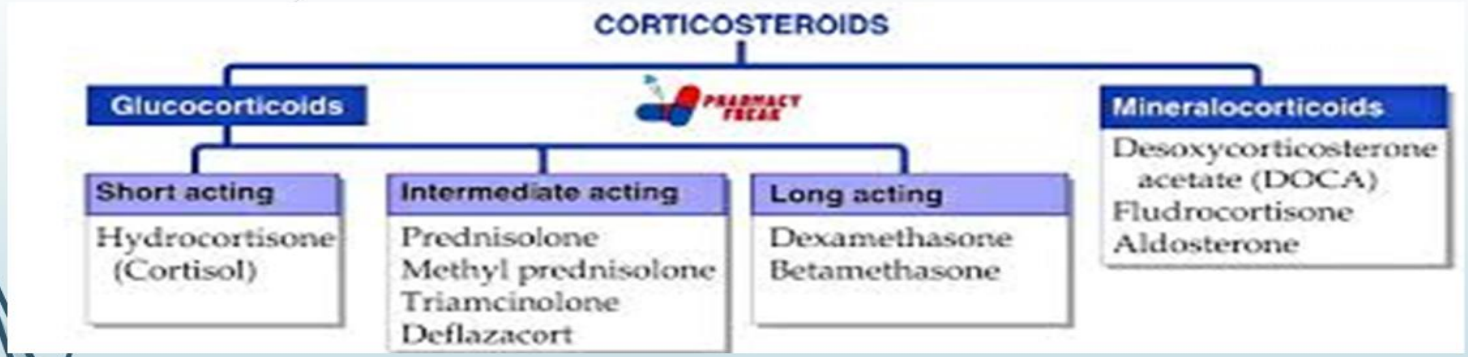
  For use topically on the skin,

  eye, and mucous membranes.
- **Inhaled steroids**

  For nasal mucosa, sinuses, bronchi, and lungs.

- **This group includes:**
- Beclomethasone dipropionate
- Budesonide
- Mometasone furoate
- Ciclesonide

Classification of Corticosteroids



History OF Corticosteroid(5)

Corticosteroid	Introduced
Cortisone	1948
Hydrocortisone	1951
Fludrocortisone acetate	1954
Prednisolone	1955
Prednisone	1955
Methylprednisolone	1956
Triamcinolone	1956
Dexamethasone	1958
Betamethasone	1958
Triamcinolone acetonide	1958
Fluorometholone	1959
Deflazacort	1969

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- **Corticosteroids** produce their effect through multiple pathways.

- **In general**, they produce anti-inflammatory and immunosuppressive effects, protein and carbohydrate metabolic effects, water and electrolyte effects, central nervous system effects, and blood cell effects.(6)
- They have both **genomic** and **nongenomic** mechanisms of action.

- The genomic mechanism of action is mediated through the glucocorticoid receptor, leading to most anti-inflammatory and immunosuppressive effects.(7)

- The glucocorticoid receptor is located intracellularly within the cytoplasm and, upon binding, trans-locates rapidly into the nucleus, where it affects gene transcription and causes inhibition of gene expression and translation for inflammatory leukocytes and structural cells such as epithelium.[6]

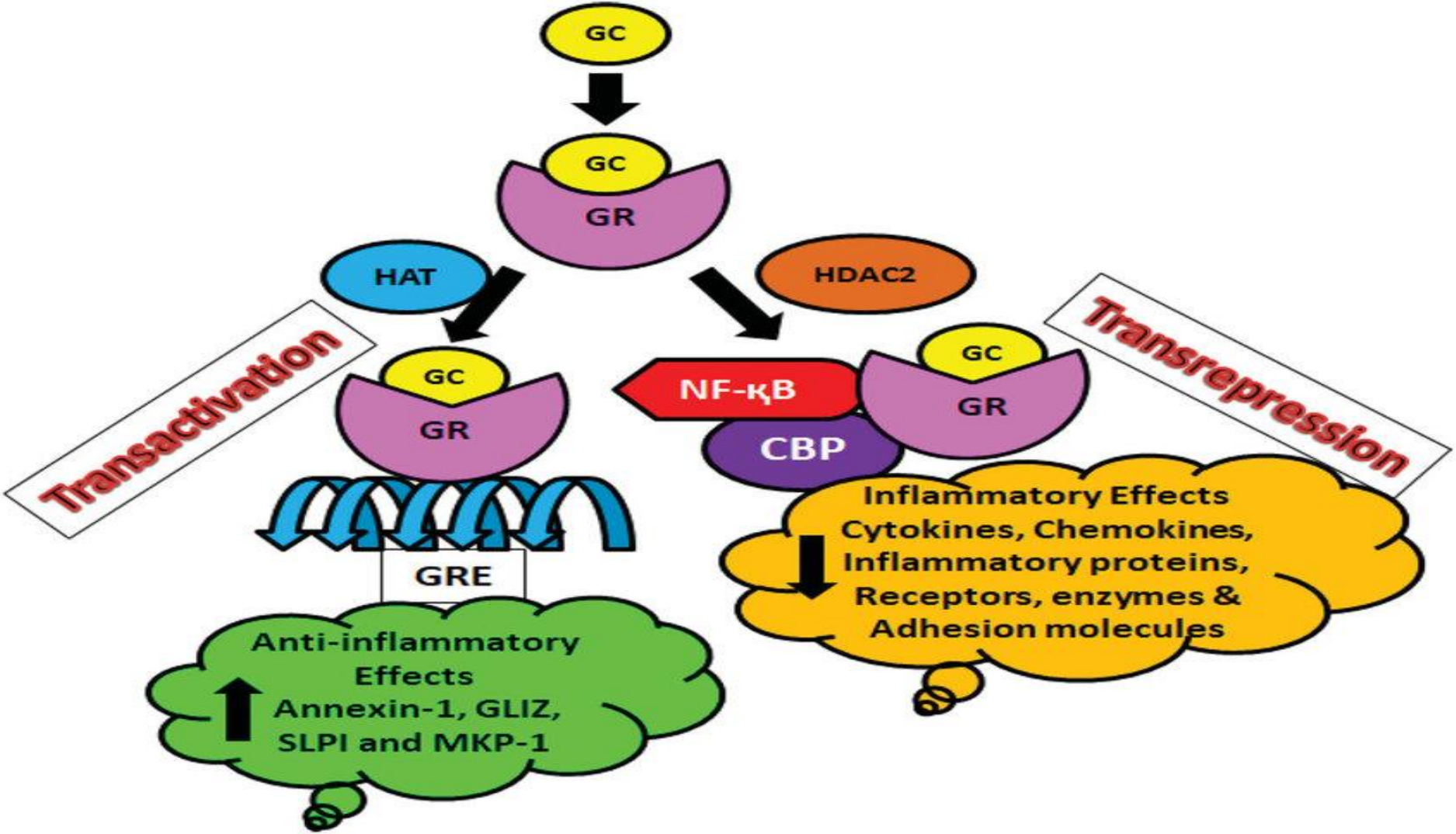
- This action leads to a reduction in proinflammatory cytokines, chemokines, cell adhesion molecules, and other enzymes involved in the inflammatory response

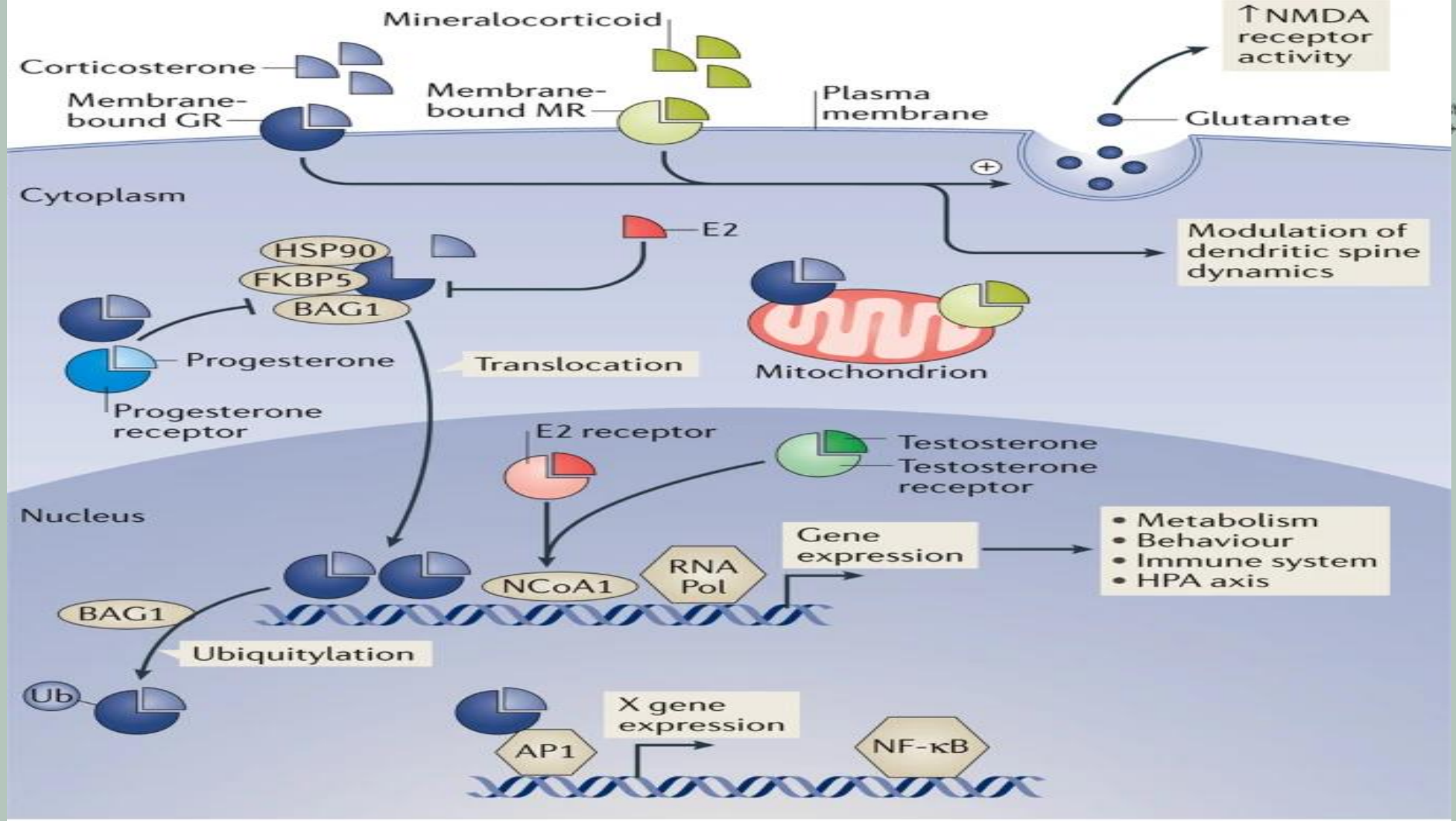
MOA

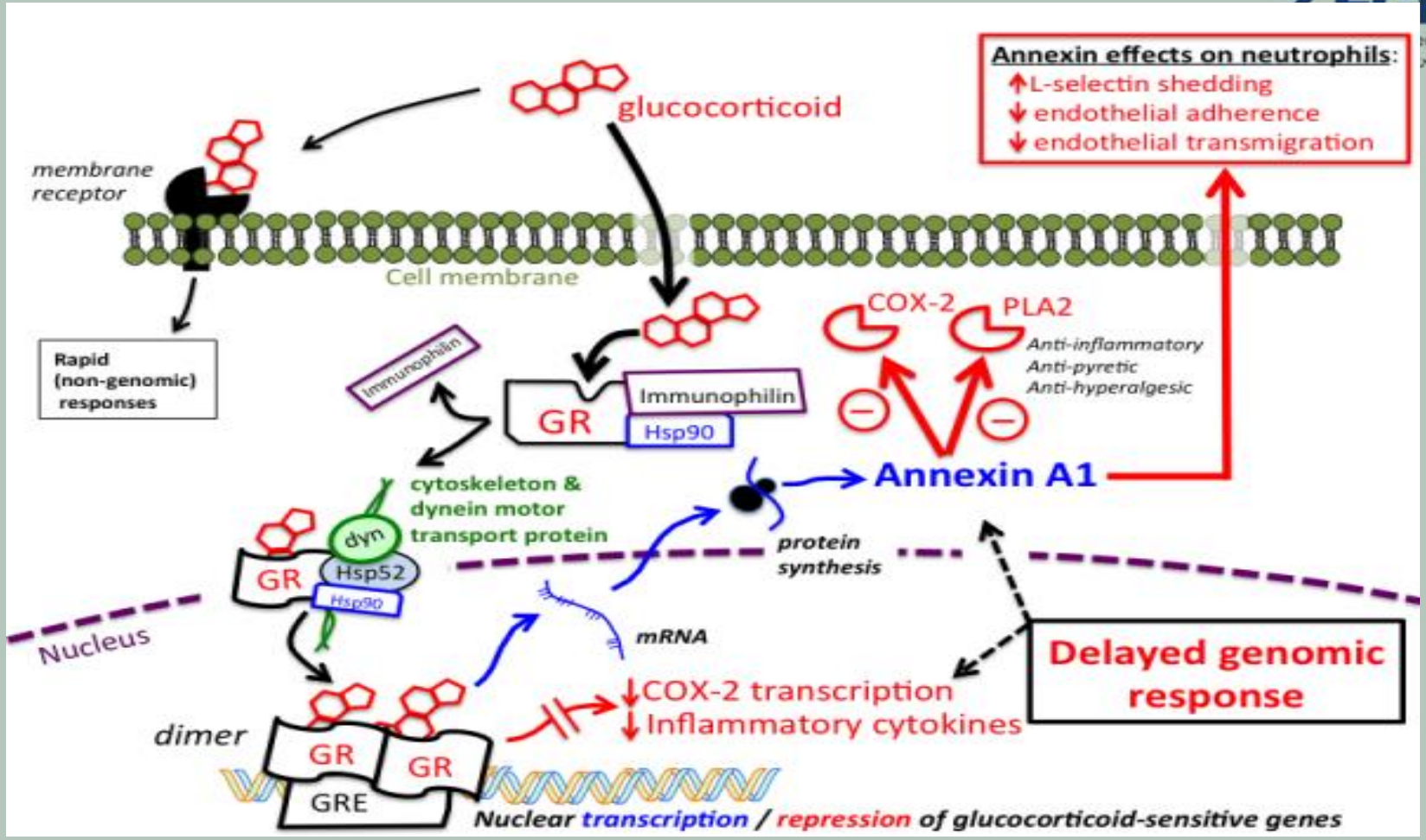
- **The non-genomic** mechanism occurs more rapidly and is mediated through interactions between the intracellular glucocorticoid receptor or a membrane-bound glucocorticoid receptor.

- Within seconds to minutes of receptor activation, a cascade of effects is set off, including inhibition of phospholipase A2, which is critical for producing inflammatory cytokines, impairing the release of arachidonic acid, and regulating of apoptosis in thymocytes.

- Corticosteroids at high concentrations will also inhibit the production of B cells and T cells.(8)(9)


GC
GC
GR
HAT
HDAC2
Transactivation
Transrepression
GC
GR
NF-κB
GC
GR
CBP
GRE
Inflammatory Effects
Cytokines, Chemokines,
Inflammatory proteins,
Receptors, enzymes &
Adhesion molecules
Anti-inflammatory
Effects
Annexin-1, GLIZ,
SLPI and MKP-1


Mineralocorticoid
Corticosterone
Membrane-bound GR
Membrane-bound MR
Plasma membrane
↑NMDA receptor activity
Glutamate
Cytoplasm
E2
HSP90
FKBP5
BAG1
Modulation of dendritic spine dynamics
Progesterone
Progesterone receptor
Translocation
Mitochondrion
E2 receptor
Testosterone
Testosterone receptor
Nucleus
Gene expression
• Metabolism
• Behaviour
• Immune system
• HPA axis
NCoA1
RNA Pol
BAG1
Ubiquitylation
Ub
X gene expression
AP1
NF-κB


Annexin effects on neutrophils:
↑L-selectin shedding
↓endothelial adherence
↓endothelial transmigration
glucocorticoid
membrane receptor
Cell membrane
Rapid (non-genomic) responses
Immunophilin
GR
Immunophilin
Hsp90
COX-2
PLA2
Anti-inflammatory
Anti-pyretic
Anti-hyperalgesic
Annexin A1
cytoskeleton & dynein motor transport protein
dyn
GR
Hsp52
Hsp90
protein synthesis
Nucleus
mRNA
Delayed genomic response
↓COX-2 transcription
↓Inflammatory cytokines
dimer
GR
GR
GRE
Nuclear transcription / repression of glucocorticoid-sensitive genes

➢Potancy ranking of topical corticosteroid

Potency	Corticosteroid
Super potent	Clobetasol propionate
Very potent	Fluocinonide , fluocinolone acetonide
Potent	Fluticasone propionate, betamethasone dipropionate ,
Moderately potent	betamethasone valerate , mometasone furoate , Clobetasone butyrate
Mildly potent	hydrocortisone acetate, methyl-prednisolone acetate

potency group*	Corticosteroid	Vehicle type/form	Brand names
Super-high potency	Clobetasol propionate	Cream , Ointment , lotion	Dermovate cr , oin Clobutra cr Clovacort 0.05%
High potency	Betamethasone dipropionate	Ointment , Cream, augmented formulation (AF)	
	Desoximetasone	Cream , ointment ,spray	
High potency	Betamethasone dipropionate	Cream (hydrophilic emollient)	
	Betamethasone valerate	Ointment , foam	Betaderm ointment Betnovate
	Desoximetasone	Cream , ointment	
	Mometasone furoate	Ointment	
Medium potency	Betamethasone dipropionate		
	Mometasone furoate	Cream , lotion	

potency group*	Corticosteroid	Vehicle type/form	Brand names
Lower-mid potency	Hydrocortisone butyrate	Cream , ointment	Texacort cr
	Betamethasone valerate	cream	Betaderm cr Betnovate
	Hydrocortisone valerate	cream	
Low potency	Betamethasone valerate	lotion	
Least potent	Hydrocortisone	Cream , ointment , lotion	

Adverse effect(10)

- An increased appetite.
- Unexpected weight gain.
- Skin changes, including bruising more easily than usual and increased acne.
- Retaining water, which makes your skin and face look swollen or puffy.
- Stomach irritation.
- Muscle weakness.
- Mood swings, including increased anxiety, restlessness or trouble sleeping.
- Increased body hair.

- Corticosteroids can increase your risk of developing some conditions (or worsen them if you already have them), including:

- Cushing syndrome.
- Diabetes.
- High blood pressure.
- Osteoporosis.
- Infections.

- **Allergy and respiratory medicine**
- Asthma (severe exacerbations)
- Chronic obstructive pulmonary disease (COPD)
- Allergic rhinitis
- Atopic dermatitis
- Angioedema
- Anaphylaxis
- Food allergies
- Drug allergies
- Nasal polyps
- Hypersensitivity pneumonitis
- Sarcoidosis
- Eosinophilic pneumonia
- Interstitial lung disease

- **Dermatology**
- Pemphigus Vulgaris
- Contact dermatitis
- **Rheumatology/Immunology**
- Rheumatoid arthritis
- Systemic lupus erythematosus
- Polymyalgia rheumatica
- Polymyositis
- Dermatomyositis
- Polyarteritis
- Vasculitis

Skin Inflammation (Dermatitis)

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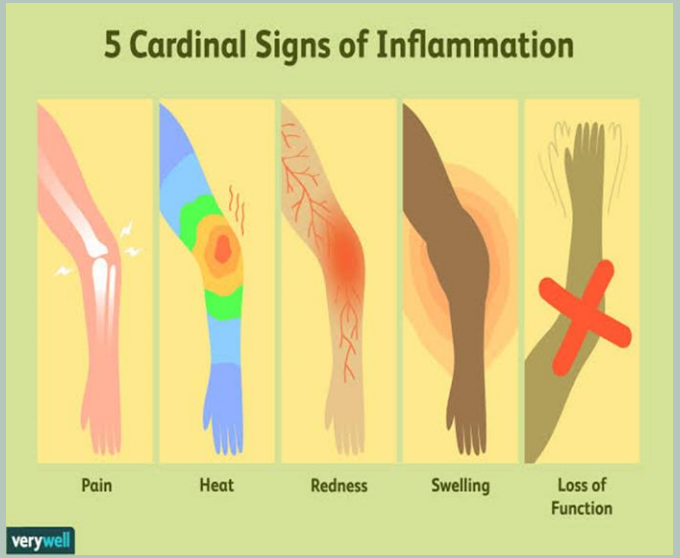
A basic way in which the body reacts to infection, irritation or other injury, the key feature being **redness**, **warmth**, **swelling** and **pain**.



Types of Dermatitis

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➢ According to Duration:

1- Acute Dermatitis
2- Chronic Dermatitis

➢ According to Cause:

1- Contact Dermatitis
2- Diaper Dermatitis
3- Atopic Dermatitis
4- Seborrheic Dermatitis

The difference between Atopic and Contact dermatitis:

- Atopic dermatitis and contact dermatitis are both types of skin inflammation but differ in their causes, mechanisms, and clinical presentations. Here's a breakdown:
- **Cause:**

•**Atopic Dermatitis**: is a chronic, inflammatory skin condition linked to genetics, immune dysfunction, and environmental triggers.

o including asthma, hay fever, or other allergic conditions.

oTriggered by allergens, irritants, stress, temperature changes, or infections.

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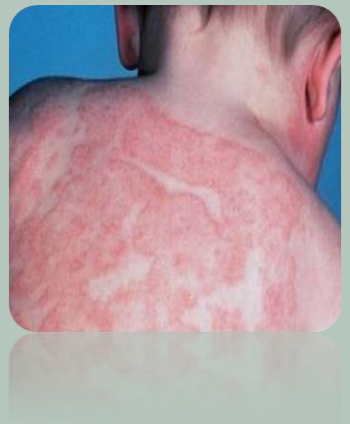
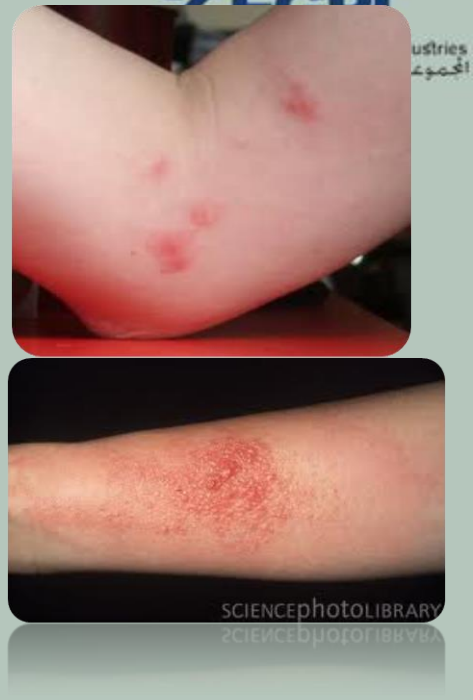


Caused by direct contact with a substance that irritates the skin or triggers an allergic reaction.

o**Two types:**

- **Irritant Contact Dermatitis**: Caused by exposure to irritants (e.g., soaps, detergents, acids).
- **Allergic Contact Dermatitis**: A delayed hypersensitivity reaction to allergens (e.g., nickel, fragrances, latex).



Symptoms

- **Atopic Dermatitis**:
  - **Chronic and relapsing** condition.
  - Symptoms include:
    - **Itchy, red, and inflamed skin.**
    - Dry, scaly, or cracked patches (common on flexural areas like elbows and knees).
    - Oozing and crusting in severe cases.
- **Contact Dermatitis**:
  - **Localized** reaction at the site of contact.
  - Symptoms include:
    - **Redness, swelling, and itching.**
    - Blisters or weeping lesions in severe cases.
    - Skin thickening and scaling with chronic exposure.
  - Symptoms resolve when exposure to the irritant or allergen is eliminated.

Treatment

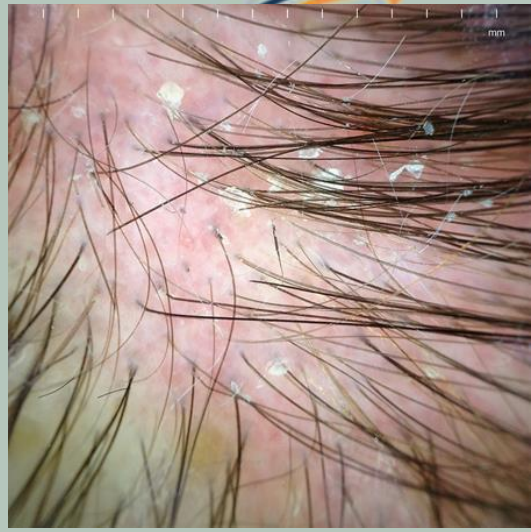
- **Atopic Dermatitis**:
  - Emollients and moisturizers to repair the skin barrier.
  - Topical corticosteroids or calcineurin inhibitors for inflammation.
  - Antihistamines for itching.
  - Avoiding triggers.
- **Contact Dermatitis**:
  - Avoidance of the irritant or allergen.
  - Topical corticosteroids to reduce inflammation.
  - Protective measures (e.g., gloves, barrier creams).

Aspect	Atopic Dermatitis	Contact Dermatitis	Eczema
Cause	Genetic, immune dysfunction.	External irritant/allergen exposure.	Varies by eczema type.
Onset	Early childhood.	After exposure to irritant/allergen.	Varies; can occur at any age.
Location	Flexural areas, face, neck.	Contact sites.	Varies widely.
Duration	Chronic and relapsing.	Acute or chronic, resolves with avoidance.	Chronic or acute depending on type.

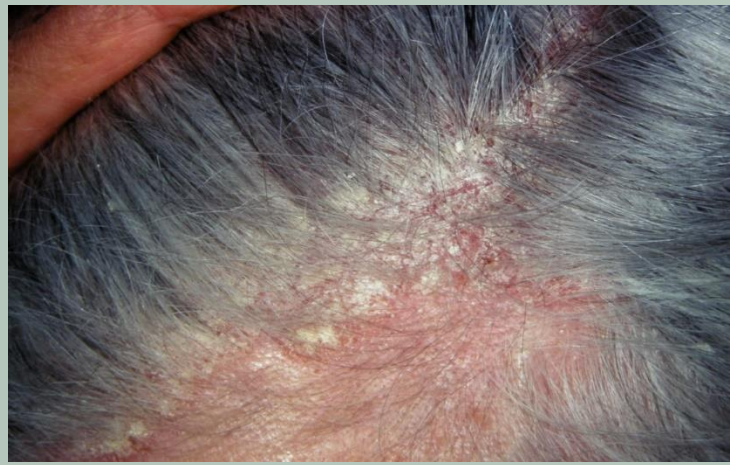
Key Differences

In summary, **eczema** is a broad term that encompasses many skin conditions, including **atopic dermatitis** and **contact dermatitis**, which differ in their causes, mechanisms, and presentations

➢Seborrheic dermatitis

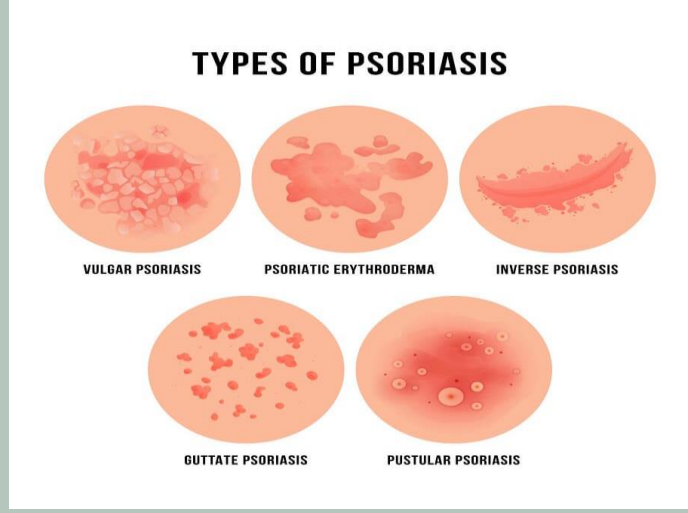


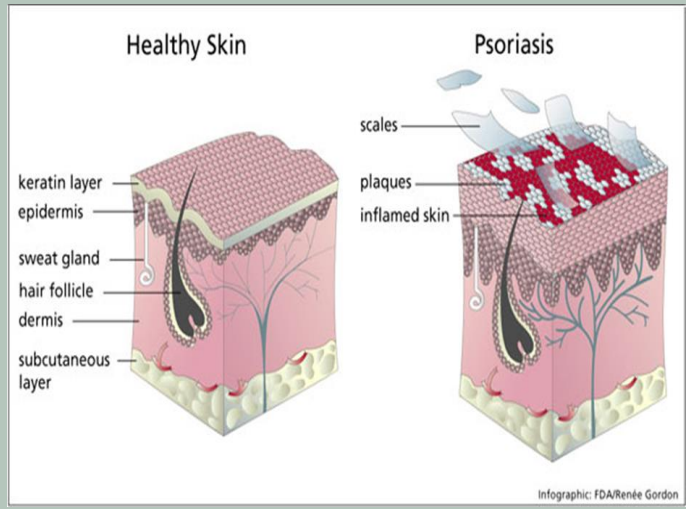
Seborrheic dermatitis is a common, chronic skin condition causing flaky, greasy, yellowish scales and redness on oily areas like the scalp (dandruff), face (eyebrows, sides of nose), ears, chest, and groin, often flaring with stress or cold weather,
and is managed with antifungal shampoos, corticosteroid creams, and lifestyle changes, though it's not contagious and has no cure, only management.
In infants, it's known as cradle cap.



➢ What is … Psoriasis?

- Psoriasis comes from the Greek word psora meaning itch.
- Psoriasis is a chronic non-infectious inflammatory skin disease in which skin cells replicate at rapid rate about eight times faster than normal,
- the rate at which old cells are removed is unchanged. This causes cells to build up on the skin's surface, forming thick patches, or plaques, of red lesions covered with flaky, silvery-white dead skin cells (scales).
- Treatment: Emolient& keratolytic
- Corticosteriod topical& systemic





LICHEN PLANUS

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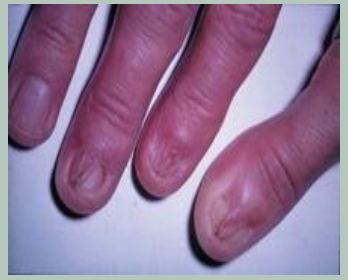
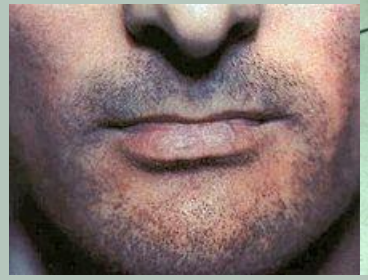
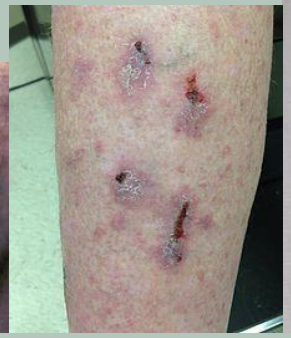
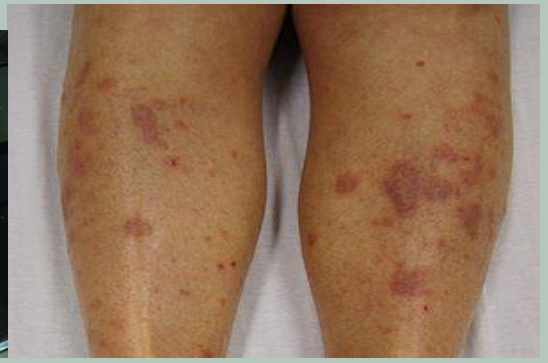
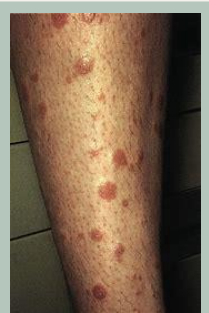
- a **chronic inflammatory and immune-mediated disease**
- **affects the skin, nails, hair, and mucous membranes**
- **commonly affecting dorsal hands, flexural wrists and forearms, trunk, anterior lower legs, and oral mucosa**
- **Polygonal, flat-topped, papules and plaques with overlying, reticulated, fine white scale (Wickham's striae)**

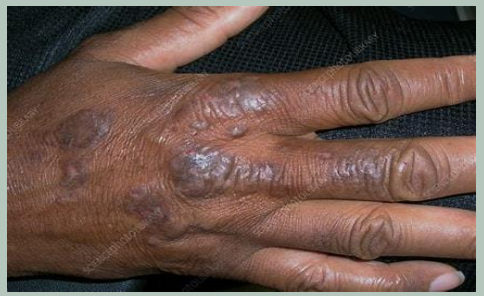





The diffrence betwean LICHEN PLANUS and LICHEN SIMPLEX

- **Definition**
- **Lichen Planus**:
  - A chronic, autoimmune inflammatory condition affecting the skin, mucous membranes, hair, and nails.
- **Lichen Simplex Chronicus**:
  - A localized skin condition caused by **chronic scratching or rubbing** that leads to thickened, hyperpigmented skin with exaggerated skin markings.

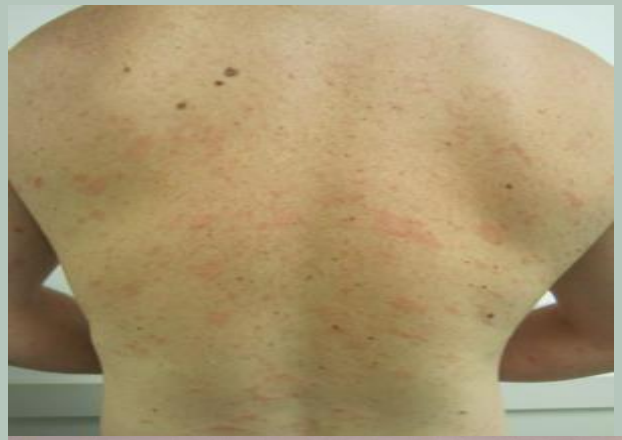
Feature	Lichen Planus	Lichen Simplex
Cause	Autoimmune and inflammatory.	Mechanical irritation from scratching.
Appearance	It is characterized by **purple, flat-topped, polygonal papules and plaques**.	Thickened, hyperpigmented lichenified plaques.
Systemic Involvement	May affect mucosa, nails, and hair.	Limited to the skin.
Histology	Inflammatory infiltrate at dermo-epidermal junction.	Thickened epidermis with minimal inflammation.
Treatment Focus	Immunosuppression and inflammation control.	Breaking the itch-scratch cycle.

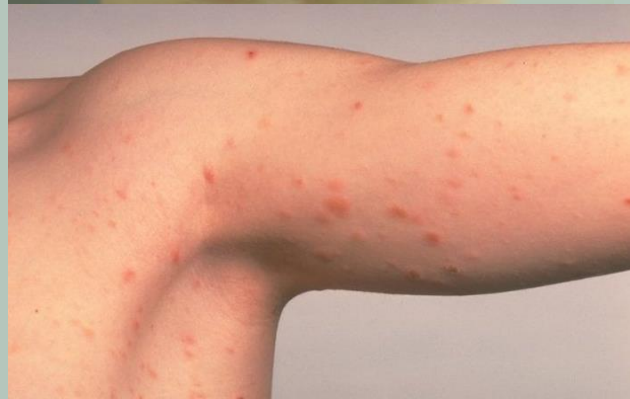




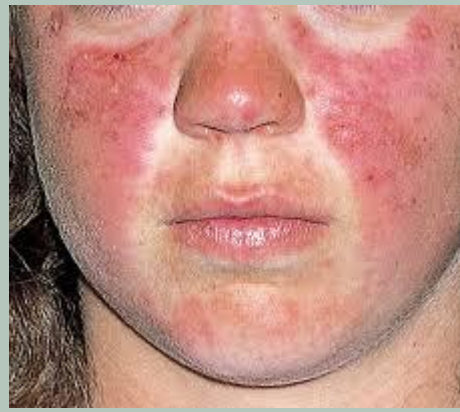
PITYRIASIS ROSEA

- Is a type of skin rash
- begins with a single red and slightly scaly area.
- Days to weeks later, by a pink whole body rash.
- lasts less than three months and goes away without treatment
- it is believed to be related to human herpes virus
- Is often occurs in those between the ages of 10 and 35





Lupus Erythematosus



- a collection of **autoimmune diseases**
- affect different **body systems**
- **including** joints, skin, kidneys, blood cells, heart, and lungs
- most common and **most severe** form is systemic lupus erythematosus
- Almost **everyone with lupus** has joint pain and swelling.
- **chest** pain, **joint** pain, painless **oral** ulcer, **fatigue**, **weight** loss, fever with no other cause, general **discomfort**, uneasiness, or ill feeling (malaise), hair loss, swollen lymph nodes, a "**butterfly**" facial rash, photosensitivity

- **Treatment** consists primarily of immunosuppressive drugs (e.g., **hydroxychloroquine** and **corticosteroids**).
- A second-line drug is **methotrexate** in its low-dose schedule

Nasal/Sinonasal Polyps (NP)

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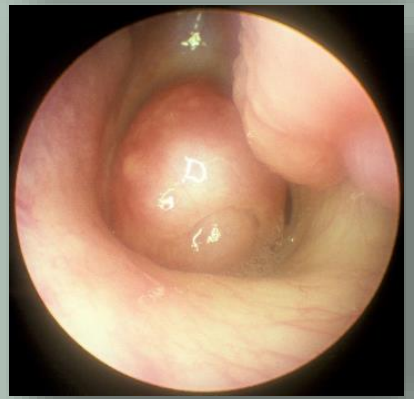
- Are **noncancerous growths** of the **mucous membrane within** the nose or sinuses
- **Symptoms include**
  1) Trouble **breathing**
  2) Loss of **smell**
  3) Decreased **taste**
  4) Post nasal **drip**
  5) **Runny** nose.
- **Growths are**
  1) Sac-like
  2) Movable
  3) Non tender
- **Complications may include**
  1) **Sinusitis**
  2) **Broadening** of the nose
- **The exact cause is unclear**.
  1) Chronic **inflammation**
  2) **Allergies**,
  3) **Infections**

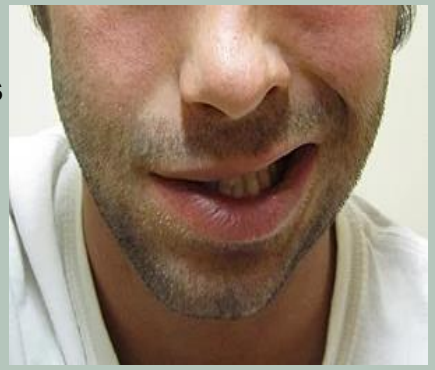


Bell's palsy

A dysfunction of **cranial nerve VII** (the facial nerve)

- **Symptoms**

1. A temporary inability to control the **facial muscles**
2. Drooping of the **eyelid**
3. A change in **taste**
4. Pain around the **ear**
5. **Lacrimation**, **salivation** loose control



- **Etiology**

1. The **cause is unknown**
2. **Risk factors** include diabetes, a recent upper respiratory tract infection

- **Treatment**

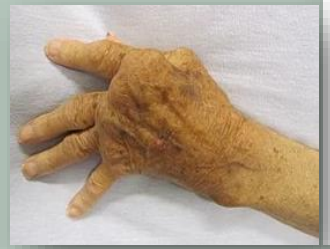
1. **CS:** Improvement begin within 14 days & complete recovery within 6 months.

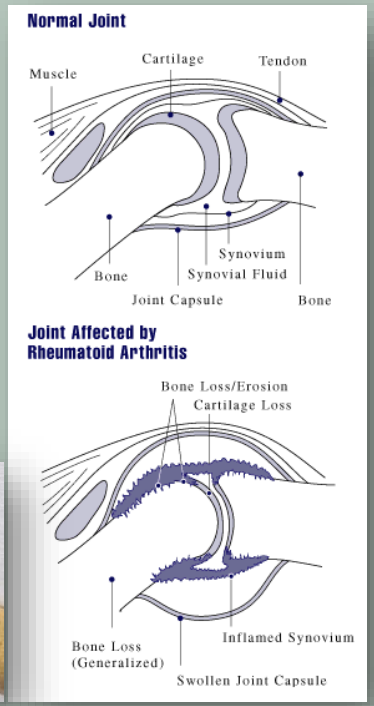
Rheumatoid Arthritis (RA)

- Is a **long-term autoimmune inflammatory** disorder
- Is the **commonest** in clinical practice
- Primarily affects **joints**
- Commonly in the **wrist** and **hands**
- Same joints typically involved on **both sides** of the body
- The **cause is not clear**

  is believed to involve a **genetic** and **environmental** factor
- Results in **warm**, **swollen**, and **painful joints**
- **Pain** and **stiffness** often worsen following rest
- As the pathology progresses the inflammatory activity leads to
  - **Erosion and destruction** of the joint surface
  - Impairs the range of **movement**
  - **Deformity**.

The synovial membrane is a specialized connective tissue lines the inner surface of capsules of synovial joints and tendon sheath. It

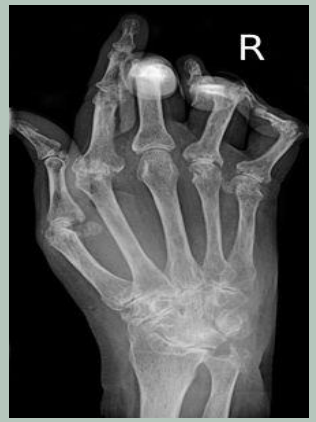




Diagnosis of RA

- **X-ray imaging**

1. **No changes** in the early
2. **Osteopenia** near the joint
3. Soft tissue swelling and a smaller than normal joint space.
4. **Bony erosions**



- **Blood tests**

1. **Rheumatoid factor (RF)**

is the **antibody** that was first found in rheumatoid arthritis.

is defined as an antibody **against** the Fc portion of IgG.

2. **The erythrocyte sedimentation rate (ESR)**

is the rate at which **RBCs** in **anti-coagulated** whole blood **descend** in a standardized tube over a period of **one 1 hr**.

3. **C-reactive protein (CRP)**

- **C-reactive protein**
- protein found in blood **plasma**
- circulating **concentrations** rise in response to **inflammation**
- of **hepatic origin** that increases following interleukin-6 secretion by macrophages and T cells

Capsular-polysaccharide


BREAK
TIME !!

Fungal infection (MYCOSIS)

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A factor can increase the risk of fungal infections

- Misuse of antibiotics (Kills Bacterial Flora)
- Pregnant woman

(changes in hormones, Change the normal PH of the vagina)

- Diabetic Patient

(People with diabetes often have low blood flow to the extremities. With less blood flow, the body is less able to mobilize normal immune defenses and nutrients that promote the body's ability to fight infection and promote healing.

- Immunocompromised patients (chemotherapy, steroid therapy)

FUNGAL Cell structure

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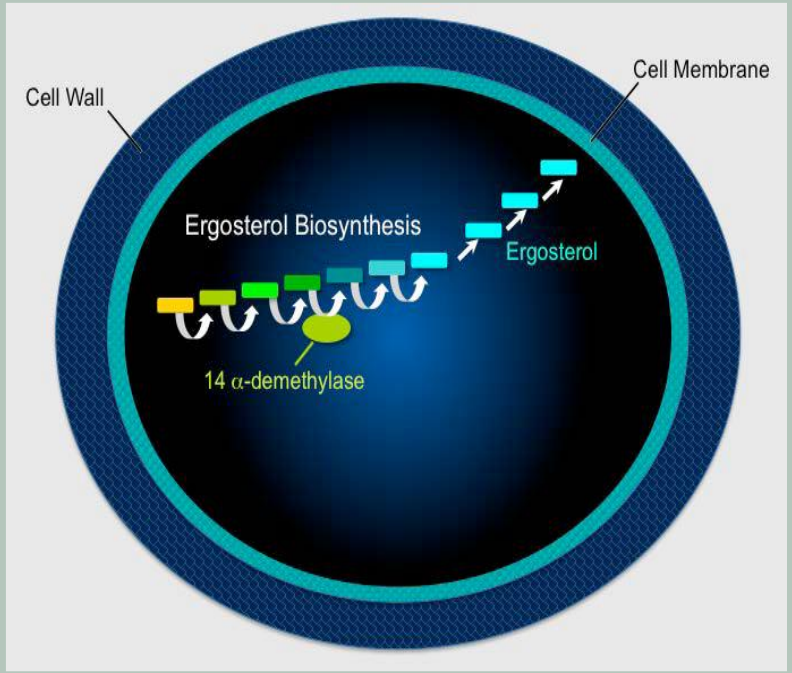
Ergosterol:

- Is a sterol found in fungi
- Ergosterol does not found in plant or animal cells.

It is a component of yeast and fungal cell membranes, serving the same function that cholesterol serves in animal cells.



Types of Infection

Superficial infections(***DERMATOMYCOSIS***)

These fungal infections affect the skin or mucous membranes.

Superficial fungal infections

Systemic infections

These occur when fungi get into the bloodstream and generally cause more serious diseases. Systemic fungal infections may be caused either by an opportunistic organism that attacks a person with a weakened immune system

Fungal Diseases

Dermatophytes infections

keratin substrate (dry)

- Tinea capitis
- Tinea barbae
- Tinea corporis
- Tinea cruris
- Tinea pedis
- Onychomycosis

Non-Dermatophytes infection

lipids substrate(candida species)

- Pityriasis versicolor (PVC)
- Seborrheic dermatitis
- Dandruff
- Cutaneous candidiasis

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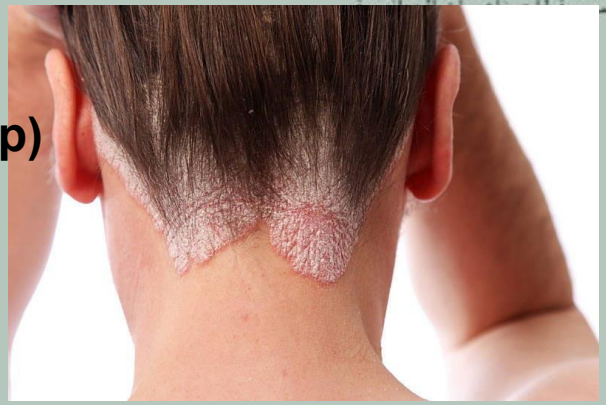
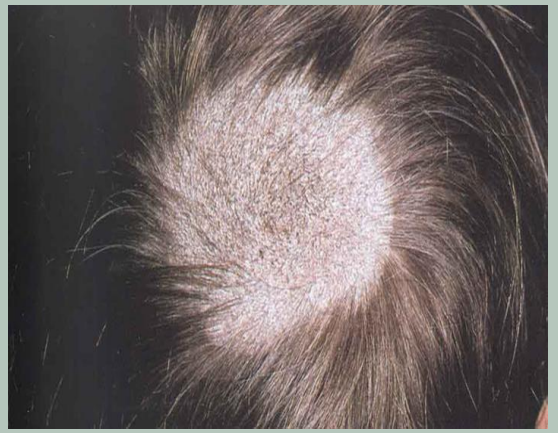
Dermatophytes infections

Dermatophytes infections

Tinea capitis (ringworm)



- Infection of the scalp (itchy bald batches on scalp)
- attacking hair shafts and follicles
- occurs in school-age children rare cases can also affect adults.



- Tinea capitis is very contagious (spreads easily).
- The fungus is usually spread by coming in contact with infected hairs on combs, brushes, hats, or pillow.

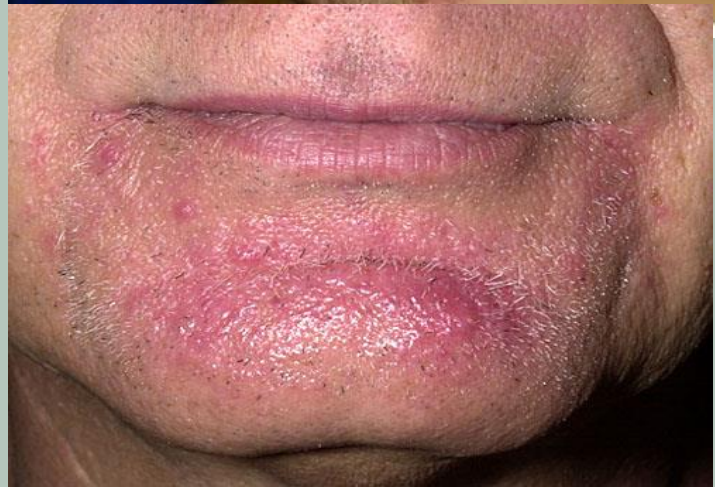
Dermatophytes infections

Tinea barbae

Fungal infection of the bearded areas of the face (post pubertal males)

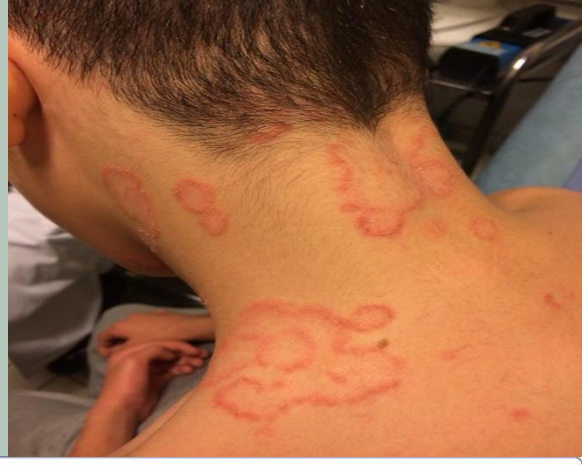
Patients with Tinea barbae present with severe, deep, folliculitis of the beard

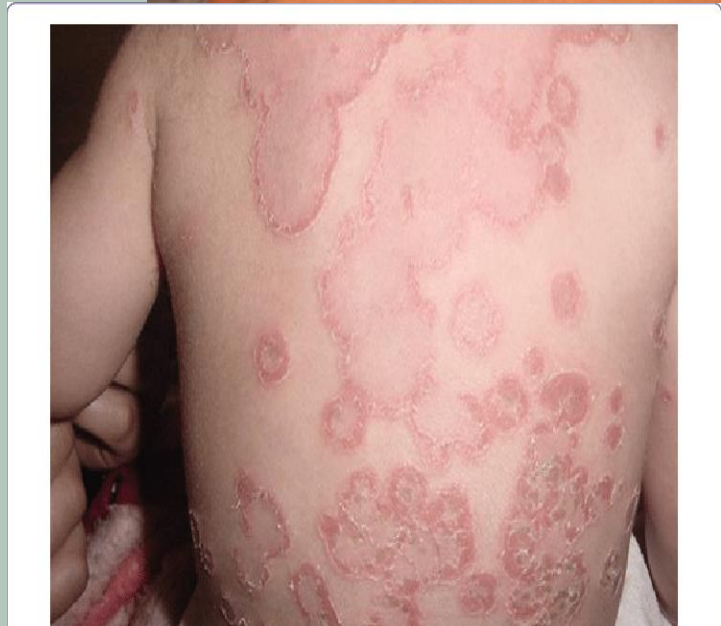
Was commonly acquired from hair-cutting instrument



Tinea Corporis

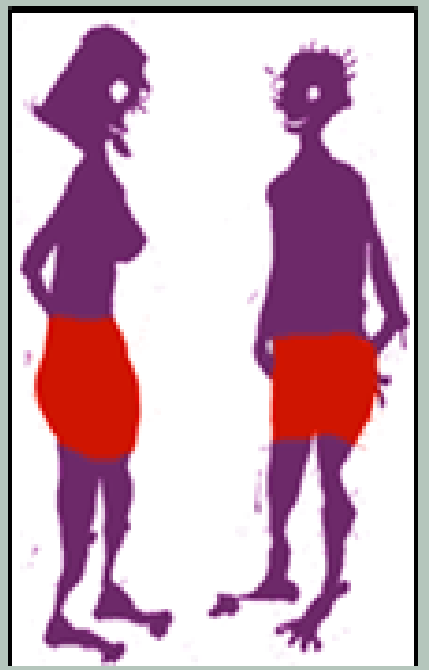
- Dermatophyte infection of the trunk and limbs presents as itchy inflamed red patches and maybe pustular
- Affects all age groups
- Transmitted by direct contact with infected animals, humans or soil





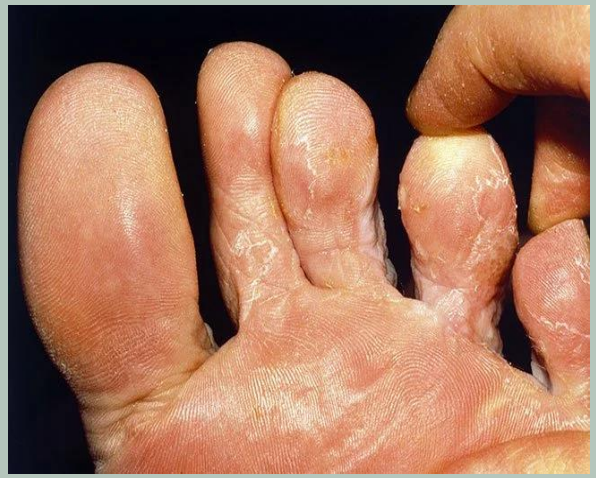
Tinea Cruris

- Localized form of T. corporis, affecting the groin mainly in adult men.
- The infection is bilateral and itchy
- Common on hot humid countries
- May be associated with poor personal hygiene



Tinea pedis

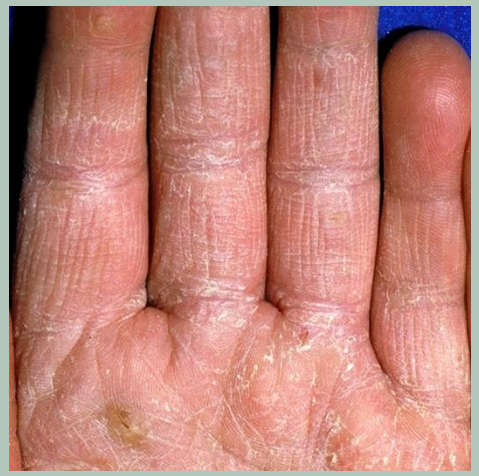
- "Athlete's foot" is the most common type of dermatophyte infection
- Warmth and dampness are ideal for fungal growth
- Red or white lesions, areas of cracking and fissuring between the toes



Tinea Manum

Infection of the hand

- frequently associated with Tinea pedis
- The most common organism is T.rubrum
- presents as diffuse scaling of the palms



Tinea Unguium *(Onychomycosis)*

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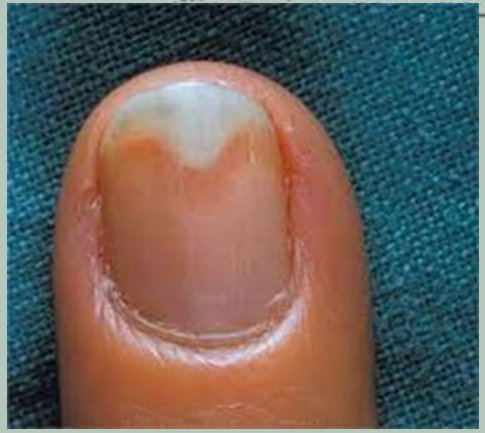
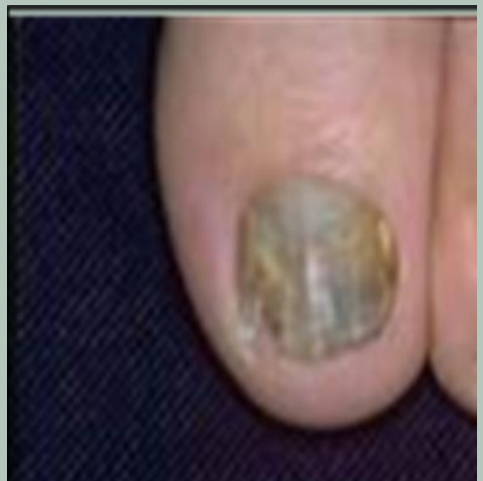
Dermatophytic infections of the nail caused by any fungus including non-dermatophytes.

- caused by

T. rubrum and T. mentagrophytes

- Some cases are associated with Tinea pedis or Tinea manuum





Tinea Unguium *(Onychomycosis)*

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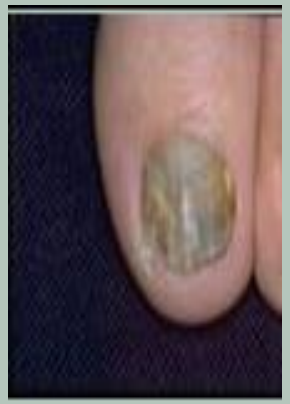
Infection of the toenails is more common than infection of the fingernails, reflecting the higher prevalence of Tinea pedis

▪One or more nails show yellowish white or yellowish gray focal thickening of the nail



- Important Non-Dermatophytosis Producing Species
- Candida species (most common)

Candida albicans

- (sometimes referred to as monilia)
- is a fungus that is normally present on the skin and in mucous membranes such as the vagina and mouth. The fungus also can travel through the bloodstream and affect the throat, intestines,

Candida species may become opportunistic pathogens under a variety of circumstances including diabetes mellitus, antibiotic therapy, or immunosuppression

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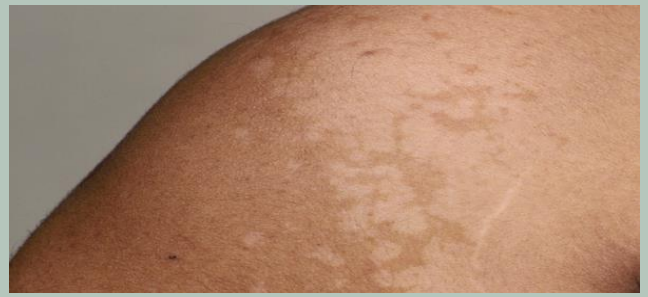
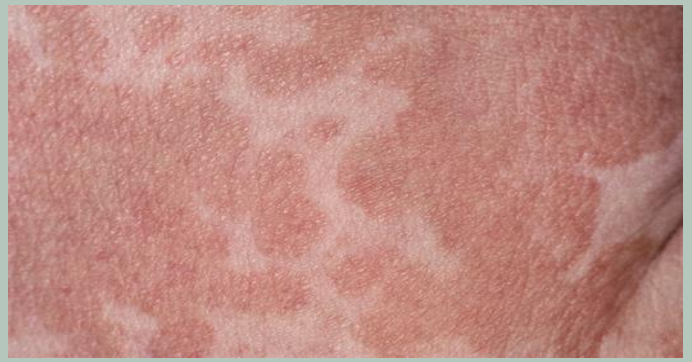
Clinical Manifestations of Candidosis

- Cutaneous Disease
- Mucosal Disease
- Vaginal Candida Vulvovaginitis
- Systemic Disease

Pityriasis Versicolor, tinea versicolor (PVC)



On brown or black skin, the patches tend to be paler than the surrounding skin.

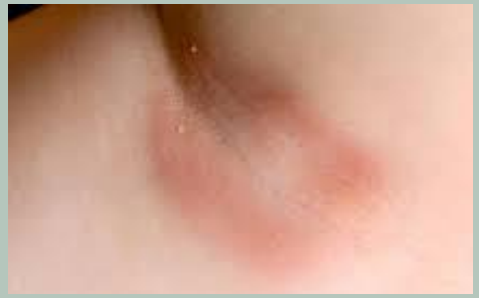


On white skin, the patches are usually pink, red, or pale brown.

- occurs most frequently in teens and young adults.
- The fungus interferes with the normal pigmentation of the skin, resulting in small, discolored patches. These patches may be lighter or darker in color than the surrounding skin and most commonly affect the trunk and shoulders.

cutaneous candidiasis

- The skin is infected with candida fungi. This type of infection is fairly common. It can involve almost any skin on the body, but most often it occurs in warm, moist, creased areas such as the armpits and groin.



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Antifungal Therapy

Topical Antifungal
Systemic Antifungal

Systemic Antifungal Therapy

I. Griseofulvin

II. Azoles:

- Ketoconazole
- Fluconazole
- Itraconazole

III. Allylamines :

- Terbinafine (Lamifen)

Griseofulvin Tablets

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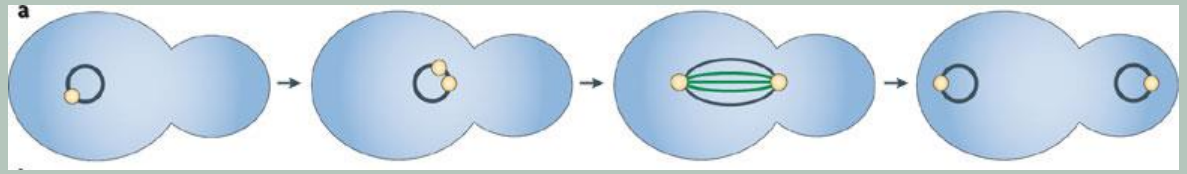
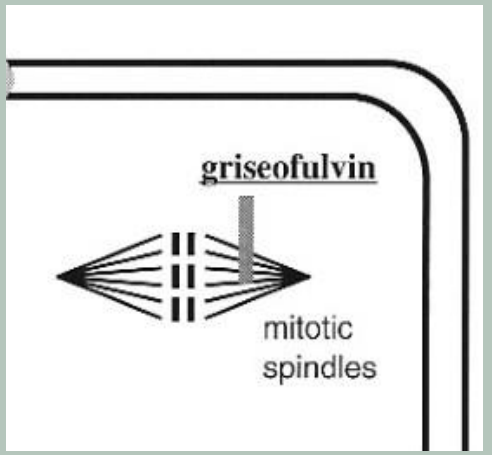
Mechanism of action:

Inhibits fungal mitosis by disrupting the mitotic spindle

Antifungal spectrum:

Narrow spectrum fungistatic with high MICs against dermatophytes only





Griseofulvin Tablets

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Indications:

Superficial fungal infections

Weaknesses:

Narrow spectrum and fungistatic

High relapse rate

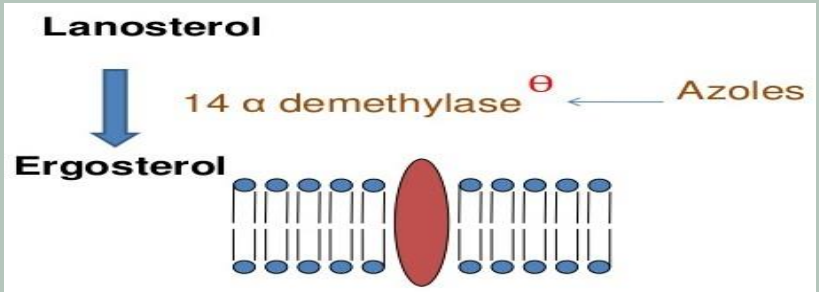
Long treatment courses with a high incidence of adverse effects

(hepatotoxicity)

Examples :- Ultragriseofulvin

Azole antifungals

- The azole antifungals inhibit the cytochrome P450 dependent enzyme.
- which converts lanosterol to ergosterol, the main sterol in the fungal cell membrane. Depletion of ergosterol causing increased membrane permeability and inhibition of fungal growth.
- damages the cell membrane resulting in cell death.
- azoles antifungals are particularly susceptible to clinically-significant drug drug interactions with other medications metabolized through the P450 pathway.



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Lamifen (Terbinafine)

Sequalene

✗ **Squalene Epoxidase***

2,3-Oxidosequalene

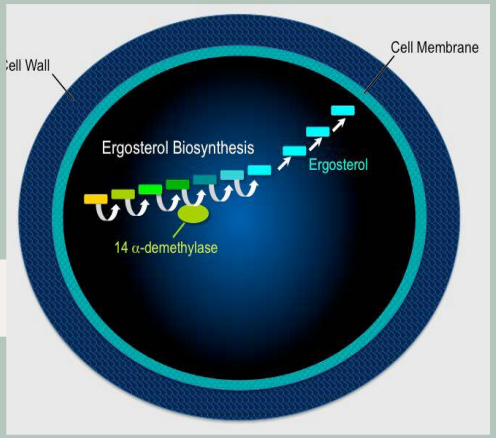
Lanosterol

Azoles

✗ **14 – Alpha demethylase†**

Ergosterol

Cell membrane



Azoles can also inhibit many mammalian cytochrome P450-dependent enzymes.

*Squalene epoxidase: non-cytochrome P-450 enzyme.
†14-demethylase: cytochrome P-450 enzyme.

- **Mechanism of action:**

inhibition of {14a-demethylase enzyme}that leads to ergosterol (sterol biosynthesis) .

- Antifungal spectrum:

Covers mainly non-dermatophytes

- Indications :

Superficial fungal infections

- Weaknesses :

- The oldest and least effective of azole group High incidence of drug interactions and adverse effects (hepatotoxicity ....etc) High relapse rate

- Examples: Kizol, Ketozol.

Itraconazole

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➢ Mechanism of action:

Inhibition of{ 14a-demethylase }This enzyme is in the sterol biosynthesis pathway that leads to ergosterol

➢ Antifungal spectrum:

Broad spectrum involving dermatophytes & candida even fluconazole resistant candida

➢ Indications :

Superficial fungal infections (onychomycosis ,PV,...)

➢ Weaknesses :

Appearance of resistant strains

High incidence of hepatotoxicity

incidence drug interactions

➢ Examples :

Sporanox 100 mg, Itranox, Itrapex.

Tioconazole

- **Mechanism of action:**
- **How it Works**
- **Tioconazole works by killing the fungus or yeast or preventing its growth. It does this by interfering with the synthesis of ergosterol, a vital component of the fungal cell membrane, which increases the cell membrane's permeability and leads to fungal cell death.**

Tioconazole is an **antifungal medication** of the imidazole class used to treat infections caused by fungus and yeast.

It is available in various topical forms, including vaginal ointment, dermal cream, and nail solution.

Uses

Tioconazole is used to treat a variety of fungal and yeast infections:

Tioconazole

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Skin fungal infections, including:

Athlete's foot (tinea pedis).

Jock itch (tinea cruris).

Ringworm (tinea corporis).

Tinea versicolor (sometimes called "sun fungus").

Yeast infections of the skin (cutaneous candidiasis).

Nail infections (onychomycosis) caused by susceptible fungi and bacteria (in a special nail lacquer formulation).


Thank
you