

NOTESKARTS

COSMETIC SCIENCE

B.Pharmacy 8th Semester

UNIT 5 — Detailed Study Notes

As per AKTU / PCI Syllabus

Oily Skin | Dry Skin | Skin Moisturization | Comedogenic | Dermatitis
Hair & Scalp Problems: Dandruff | Hair Fall
Skin Problems: Blemishes | Wrinkles | Acne | Prickly Heat | Body Odour
Antiperspirants & Deodorants: Actives and Mechanism of Action

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OILY AND DRY SKIN

Skin type is primarily determined by the amount of sebum produced by sebaceous glands and the skin's ability to retain moisture. The two polar skin types are oily skin (excess sebum) and dry skin (insufficient moisture/lipids).

OILY SKIN (Seborrhoea)

Definition: Oily skin is characterized by excessive sebum production by sebaceous glands, leading to a shiny appearance, enlarged pores, and a tendency toward comedones and acne.

Characteristics of Oily Skin:

Feature	Description
Appearance	Shiny or greasy appearance especially in T-zone (forehead, nose, chin)
Pore Size	Enlarged, visibly open pores due to chronic sebum accumulation and stretching
Texture	Coarse, thick skin texture; prone to uneven surface
Sensitivity	Less susceptible to UV damage and environmental irritants than dry skin
Aging	Ages more slowly — natural sebum acts as moisturizer and UV filter
Acne tendency	High — excess sebum + dead cells block follicles → comedones → acne
Makeup	Foundation slides off; poor makeup longevity; needs oil-free/mattifying products

Causes of Oily Skin:

- **Genetic / Hereditary:** Primary determinant; size and activity of sebaceous glands is genetically programmed
- **Hormonal — Androgens:** Testosterone and DHT (dihydrotestosterone) directly stimulate sebaceous gland size and sebum output; explains oily skin in adolescence, pregnancy, PCOS, and menstruation
- **Diet:** High glycemic index foods, dairy, trans fats increase IGF-1 (Insulin-like Growth Factor) and insulin → stimulate sebaceous glands and keratinocyte proliferation
- **Humidity and heat:** Hot, humid climate (India) increases sebum fluidity and secretion rate
- **Over-cleansing:** Stripping natural oils triggers compensatory sebum overproduction (sebostasis feedback)
- **Comedogenic cosmetics:** Occlusive ingredients block follicles → trigger sebum accumulation → worsen oiliness
- **Stress:** Cortisol → stimulates adrenal androgens → increases sebum production

Sebum Composition:

Sebum Component	Percentage	Role
Triglycerides	41%	Emollient; substrate for Cutibacterium acnes (Propionibacterium acnes)
Wax esters	26%	Unique to humans; skin lubrication; waterproofing
Squalene	12%	Antioxidant; emollient; precursor for cholesterol
Free fatty acids	16%	Antimicrobial; maintain acidic pH; formed from bacterial hydrolysis of triglycerides
Cholesterol esters	3%	Membrane integrity; barrier lipids
Cholesterol	2%	Barrier lipid; skin protection

Management of Oily Skin — Cosmetic Approach:

- **Cleansing:** Gentle foaming face wash (SLS/SLES + CAPB); pH 5-6.5; twice daily; avoid harsh cleansers that over-strip
- **Toners:** Alcohol-free toners with niacinamide, zinc, salicylic acid; minimize pores; reduce sebum
- **Moisturizer:** Oil-free, non-comedogenic; gel or water-based formulation; hyaluronic acid; niacinamide 5-10%
- **Mattifying agents:** Silica microspheres, kaolin clay, tapioca starch — absorb excess sebum and reduce shine
- **Actives:** Niacinamide 5% (reduces sebum); Salicylic acid 0.5-2% (keratolytic, unclogs pores); Zinc PCA

DRY SKIN (Xerosis)

Definition: Dry skin (xerosis) is a condition of abnormally low moisture content in the stratum corneum, leading to tightness, flakiness, rough texture, itching, and compromised skin barrier function.

Characteristics of Dry Skin:

Feature	Description
Appearance	Dull, lack of radiance; ashy or flaky appearance; fine scales visible
Texture	Rough, tight, and rough to touch; may show fine surface cracks

Sensitivity	Highly sensitive; prone to irritation, redness, and contact dermatitis
Aging	Ages faster — dehydration accentuates fine lines and wrinkles
TEWL	Elevated Transepidermal Water Loss due to compromised stratum corneum barrier
Itch	Common symptom — pruritus from dry SC; reduced NMF; nerve fiber exposure
Seasonal variation	Worse in winter (low humidity, cold air) and air-conditioned environments

Causes Leading to Dry Skin — Detailed

CAUSES OF DRY SKIN — CATEGORIES:

1. Intrinsic (Internal) Causes: Genetics, aging, systemic disease
2. Extrinsic (External) Causes: Environment, cleansing, diet, medications
3. Disease-related Causes: Atopic dermatitis, hypothyroidism, diabetes, psoriasis

A. Intrinsic Causes:

Cause	Mechanism and Details
Aging (Senile Xerosis)	With age: sebaceous gland activity declines 23% per decade after age 40; NMF components decrease; ceramide synthesis reduces; SC lipid content falls → poor water retention → dry skin
Genetics / Filaggrin Mutation	Filaggrin (FLG) gene mutation → deficient filaggrin protein → reduced NMF (amino acids, PCA, urocanic acid) in SC → compromised barrier → water loss → dry skin; also predisposes to atopic dermatitis
Hormonal Changes	Oestrogen decline (menopause) reduces skin thickness, sebum, and hyaluronic acid content → post-menopausal women experience increased skin dryness
Low sebum production	Naturally low sebaceous gland activity (opposite of oily skin) → insufficient surface lipids → poor water-retaining film on skin
Reduced NMF	Natural Moisturizing Factor (amino acids 40%, PCA 12%, urea 7%, lactate 12%) decreases → SC cannot hold water effectively

B. Extrinsic (Environmental) Causes:

Cause	Mechanism and Details
Low Humidity	Dry air (winter, air conditioning, desert) draws moisture from SC via evaporation; humidity below 30% dramatically increases TEWL; indoor heating lowers relative humidity to <20%

Cold Weather	Cold reduces sebaceous activity and constricts blood vessels → less lipid secretion; dry cold wind increases evaporation → cracked, dry skin in winter
Hot Water Bathing	Hot water removes skin surface lipids (NMF, ceramides); long hot baths most damaging; shower water temperature should be lukewarm not hot
Harsh Cleansing Agents	SLS, soap bars (pH 9-10, alkaline): disrupt SC lipid bilayers; remove ceramides and NMF; raise SC pH above 5.5 → enzyme activity and barrier disruption → dry skin
Sun / UV Exposure	Chronic UV damage degrades collagen and elastin; oxidizes SC lipids; reduces hyaluronic acid → photoaged, dry skin
Occupational exposure	Frequent hand-washing (healthcare workers); chemical/solvent contact; wet work → repeated barrier disruption → occupational dry skin and contact dermatitis
Swimming in chlorinated pools	Chlorine oxidizes and removes SC lipids and NMF → post-swim dryness; exacerbated by repeated exposure

C. Disease / Drug-related Causes:

Cause	Details
Atopic Dermatitis	FLG mutation + Th2-skewed immune response + Malassezia colonization → severely compromised barrier; chronic itch-scratch cycle; requires intensive moisturization
Hypothyroidism	Reduced thyroid hormone → decreased sebaceous gland activity, reduced sweating, and reduced skin cell turnover → coarse, dry, cold skin
Diabetes Mellitus	Autonomic neuropathy → reduced sweat gland function → anhidrosis → skin dryness; also peripheral neuropathy-related dry feet
Psoriasis	Abnormally rapid keratinocyte turnover (6-7 days instead of 30) → immature SC cells; silver scales; barrier dysfunction
Ichthyosis	Genetic disorders of keratinization; severely dry, scaly skin; severe form of FLG or transglutaminase deficiency
Medications	Diuretics → dehydration; retinoids (acitretin, isotretinoin) → severe xerosis; antihistamines → reduced sweating; cholesterol-lowering drugs → SC lipid reduction

SKIN MOISTURIZATION —

Skin moisturization is the process of maintaining and restoring adequate water content in the stratum corneum (SC), ensuring its proper function as a barrier, preventing TEWL, and keeping skin soft, supple, and healthy.

Normal Skin Water Content:

- **Stratum corneum water content:** 10-20% water for normal feel and function
- **Below 10%:** Dry, flaky, rough skin; impaired barrier; TEWL increases sharply
- **Dermis water content:** 70% water — this provides skin volume and turgor (not felt as surface moisture)

The Three-Component Moisturization System:

Component	Function	Examples	Mechanism
HUMECTANTS	Attract water into SC from atmosphere and dermis	Glycerin, Hyaluronic acid, Urea, Sodium PCA, Panthenol, Sorbitol	Hygroscopic; form H-bonds with water molecules; draw moisture from environment (>70% RH) or from deeper skin layers
EMOLLIENTS	Smooth and soften skin by filling intercellular spaces; improve skin texture	Fatty alcohols (cetyl, stearyl), plant oils, silicones, petrolatum, squalane	Fill gaps between corneocytes; lubricate skin surface; restore intercellular lipid bilayer
OCCLUSIVES	Form a physical barrier film on skin surface to prevent TEWL	Petrolatum, mineral oil, beeswax, dimethicone, lanolin, zinc oxide	Hydrophobic film formation; prevents water evaporation from SC surface; most effective moisture sealers

Natural Moisturizing Factor (NMF) — Detailed:

NMF is a collection of water-soluble compounds in the corneocytes of the stratum corneum that maintain SC hydration by attracting and binding water molecules.

NMF Component	Percentage (%)	Source and Role
Amino acids (free)	40%	Filaggrin breakdown products; major water-binding molecules in SC
Pyrrolidone Carboxylic Acid (PCA)	12%	Derived from glutamic acid (filaggrin); most hygroscopic NMF component

Lactate	12%	From eccrine sweat; buffers SC pH; water-binding
Urea	7%	Keratolytic at high conc.; humectant at low conc.; reduces scaling; from protein metabolism
Inorganic ions (Na+, K+, Ca2+, Mg2+, Cl-)	18%	From transepidermal fluid; regulate SC enzyme activity; water binding
Sugars, organic acids, peptides	11%	Minor humectant and buffering roles

Moisturization Strategies in Cosmetic Formulation:

- **For oily skin:** Lightweight O/W gel or lotion; primarily humectant (HA, glycerin, niacinamide); no or minimal occlusive; oil-free
- **For normal skin:** Balanced O/W cream; humectant + mild emollient; SPF optional
- **For dry skin:** Rich O/W or W/O cream; high humectant + strong emollient + occlusive; ceramides; urea 5-10%; petrolatum
- **For very dry / eczema skin:** Medical moisturizer (emollient therapy); ceramide-dominant formulation (ceramides:FA:cholesterol = 1:1:1); petrolatum; urea 10%+
- **For sensitive skin:** Fragrance-free, minimal ingredient list; mild nonionic emulsifiers; no irritating actives; ceramides + glycerin

COMEDOGENIC

Comedogenic: A substance or cosmetic product is described as 'comedogenic' if it has the tendency to clog skin pores (follicle openings), leading to the formation of comedones — the primary lesion of acne.

What is a Comedone?

- **Definition:** A comedone is a clogged hair follicle (skin pore) that develops when excess sebum, dead keratinocytes, and bacteria accumulate within the follicular infundibulum
- **Open comedone (Blackhead):** Follicle opening remains open; contents (sebum + keratin + melanin oxidation) exposed to air → oxidize and turn black; NOT due to dirt
- **Closed comedone (Whitehead):** Follicle opening closed by overlying skin; contents appear white; can progress to inflammatory acne

Comedogenicity Rating Scale:

Rating	Category	Interpretation
0	Non-comedogenic	Will not clog pores; safe for acne-prone skin; e.g., Dimethicone, Glycerin
1	Slightly comedogenic	Very low pore-clogging potential; generally safe; e.g., Isopropyl Palmitate (borderline)

2	Moderately comedogenic	Low to moderate risk; use with caution in acne-prone skin; e.g., Jojoba oil, Shea butter
3	Moderately high	May cause breakouts in sensitive/acne-prone individuals; e.g., Cocoa butter, Linseed oil
4	Highly comedogenic	Likely to cause comedones; avoid in acne-prone skin; e.g., Coconut oil (pore-clogging for some)
5	Severely comedogenic	Very high pore-clogging; avoid; e.g., Wheat germ oil, Isopropyl Isostearate

Common Comedogenic Ingredients to Avoid:

Highly Comedogenic Ingredients (Rating 4-5)	Non-Comedogenic Alternatives
Coconut oil (raw/unrefined)	Squalane, Hemp seed oil, Rosehip oil
Wheat germ oil	Jojoba oil (liquid wax; rating 2)
Linseed/Flaxseed oil	Argan oil, Sunflower oil
Isopropyl Isostearate, Isopropyl Myristate	Ethylhexyl palmitate (lower rating)
Algae/seaweed extract (some forms)	Niacinamide, Hyaluronic acid
Lanolin (anhydrous)	Dimethicone, Cyclomethicone

Testing for Comedogenicity:

- **Rabbit ear assay:** Traditional; apply ingredient inside rabbit ear canal for 14 days; count follicular plugging histologically; not always predictive for human skin
- **Human clinical test:** Apply product on human upper back (comedone-prone area) occlusively for 4-6 weeks; comedone count vs control; more clinically relevant
- **In vitro:** Measure keratinocyte proliferation in follicle-like conditions; newer, animal-free method

Non-Comedogenic Cosmetics — Formulation Principles:

- Use oils with rating 0-2: Squalane, Argan oil, Jojoba, Hemp seed, Marula oil
- Avoid occlusive waxes and thick butters in high concentration
- Use lightweight emulsifiers: Polysorbate 20, Sucrose esters
- Include sebum-regulating actives: Niacinamide 5%, Salicylic acid 0.5-2%, Zinc PCA
- Prefer gel or serum format over thick cream for oily/acne-prone skin

DERMATITIS

Dermatitis: Dermatitis is a broad term for inflammation of the skin, characterized by redness, itching, swelling, blistering, and sometimes scaling or crusting. It is one of the most common skin conditions and has several distinct types relevant to cosmetic science.

Classification of Dermatitis:

Type	Alternative Name	Features
Contact Dermatitis — Irritant (ICD)	Irritant Contact Dermatitis	Most common; 70-80% of contact dermatitis; caused by direct chemical damage to skin barrier; no prior sensitization needed; occurs in anyone with sufficient exposure
Contact Dermatitis — Allergic (ACD)	Allergic Contact Dermatitis	Type IV (delayed) hypersensitivity reaction; requires prior sensitization; reaction occurs 24-72 hours after re-exposure; only in sensitized individuals
Atopic Dermatitis	Eczema	Chronic, relapsing inflammatory skin disease; genetic (FLG mutation); Th2-skewed immunity; severe barrier dysfunction; begins in childhood; linked to food allergy, asthma, rhinitis (atopic triad)
Seborrhoeic Dermatitis	Dandruff (scalp form)	Malassezia yeast-triggered; sebum-rich areas (scalp, face, chest); chronic; greasy yellow scales; treated with antifungals (ketoconazole, ZPT)
Perioral Dermatitis	Rosacea-like dermatitis	Around mouth and nose; caused by topical steroids, fluorinated toothpaste, heavy moisturizers; small pustules and papules
Photodermatitis	Photosensitivity dermatitis	Abnormal skin reaction to UV; caused by photosensitizing ingredients (certain fragrances like bergapten) + sun exposure; phototoxic or photoallergic

A. Irritant Contact Dermatitis (ICD) — Most Relevant to Cosmetics:

Aspect	Details
Mechanism	Direct chemical insult to skin barrier; disrupts SC lipids and tight junctions; releases pro-inflammatory cytokines (IL-1alpha, IL-8, TNF-alpha) from keratinocytes
Common cosmetic culprits	SLS (Sodium Lauryl Sulfate) — most common; preservatives (formaldehyde releasers, MIT/CMIT); fragrances; propylene glycol; alpha hydroxy acids at high conc.

Clinical features	Burning, stinging, tightness at site of contact; redness; dryness; blistering at high concentrations; appears within minutes to hours
Diagnosis	Clinical history; patch test to confirm (ICD shows reaction at 24h, ACD at 48-72h); absence of sensitization history distinguishes from ACD
Prevention	Use milder surfactant blends; avoid SLS in sensitive skin products; include barrier-repairing actives (ceramides, niacinamide, glycerin)

B. Allergic Contact Dermatitis (ACD) — Most Relevant to Cosmetics:

Aspect	Details
Mechanism	Type IV delayed hypersensitivity (T-cell mediated); sensitization phase (1st exposure; hapten binds protein; T-cells primed); elicitation phase (2nd exposure; memory T-cells activated → inflammatory cascade → eczema)
Common cosmetic allergens	Fragrances (top cause; 'fragrance mix' on patch test); Preservatives (methylisothiazolinone MIT, CMIT/MIT); PPD (para-phenylenediamine) in hair dye; Nickel in jewelry; Lanolin; Colophony; Rubber accelerators
Clinical features	Erythema, vesicles, weeping, crusting at contact site; severe itch; delayed onset 24-72 hours; can spread beyond contact site with severe reaction
Diagnosis	Standard European Baseline Patch Test Series (TRUE Test); 48-hour occlusive patch application; read at 48h and 96h; positive = erythema + infiltration + vesicles (+1 to +3 scale)
Common fragrance allergens (IFRA list)	Isoeugenol, Cinnamal (cinnamon), Lyral (HICC), Oakmoss, Geraniol, Linalool — restricted in EU Cosmetics Regulation Annex III
Prevention	Fragrance-free formulation; avoid MIT/CMIT (EU banned in leave-on products); use hypoallergenic formulations; patch test before dyeing hair (PPD)

Cosmetic-Relevant Dermatitis Quick Reference:

Parameter	ICD	ACD	Atopic Dermatitis
Mechanism	Chemical damage	Type IV hypersensitivity	Genetic + immune
Prior sensitization	Not needed	Required	Not applicable
Onset	Minutes to hours	24-72 hours after re-exposure	Chronic; since childhood

Common cosmetic cause	SLS, AHAs, preservatives	Fragrance, MIT, PPD	FLG mutation; multiple triggers
Treatment	Remove irritant; barrier repair	Remove allergen; corticosteroids	Emollient therapy + steroids + biologics

COSMETIC PROBLEMS ASSOCIATED WITH HAIR AND SCALP

DANDRUFF (Pityriasis Capitis)

Dandruff: Dandruff is a chronic, non-inflammatory condition of the scalp characterized by excessive shedding of dead skin cells (corneocytes) as visible white to grayish flakes, often accompanied by itching (pruritus). It is the mildest form of seborrhoeic dermatitis.

Epidemiology:

- Affects 50% of adults worldwide; most common chronic scalp condition
- Peak incidence at puberty (hormonal; sebum increases); males more affected than females
- Can occur at any age; chronic condition with periods of remission and exacerbation

Pathophysiology of Dandruff — The Three-Factor Model:

Factor	Detailed Mechanism
1. Malassezia species (Yeast)	M. globosa and M. restricta are lipophilic yeasts naturally present on scalp; in dandruff, overgrowth occurs; they secrete phospholipase enzyme → hydrolyzes sebum triglycerides → releases oleic acid and arachidonic acid → penetrates SC → triggers skin barrier disruption and inflammation
2. Sebum (Substrate)	Malassezia requires lipids for survival; high sebum scalp provides abundant growth substrate; post-pubescent oily scalp = ideal environment; explains association of dandruff with oily scalp type
3. Host Susceptibility	Immune response variation; some individuals mount excessive inflammatory response to Malassezia oleic acid → rapid keratinocyte turnover (cycle shortened from 30 days to 7-21 days) → immature cells shed as visible clumps = dandruff flakes; genetic predisposition for abnormal immune response

Classification of Dandruff/Seborrhoeic Dermatitis:

Type	Features	Treatment Approach
Dry Dandruff (Pityriasis Capitis Simplex)	Small, dry, white-grey flakes; dry scalp; minimal	Moisturizing anti-dandruff shampoo; ZPT; mild anti-fungal; scalp oil treatment

	inflammation; flakes fall off easily onto shoulders	
Oily Dandruff (Pityriasis Steatoides)	Larger, yellowish, greasy clumps adhering to scalp and hair; inflamed scalp; oily; more severe; true seborrhoeic dermatitis	Ketoconazole 1-2% or selenium sulfide; medicated shampoo; anti-inflammatory agents; may need topical steroids

Symptoms:

- **Visible flaking:** White to yellowish skin flakes on scalp and falling on shoulders/clothing
- **Scalp pruritus:** Itching from oleic acid irritation and inflammatory mediators; itch worsens with stress and heat
- **Scalp redness:** Erythema in seborrhoeic dermatitis grade; minimal in simple dandruff
- **Scalp odor:** Secondary to bacterial overgrowth on oil-rich flakes and skin; unpleasant scalp odor

Triggering and Aggravating Factors:

- **Stress:** Cortisol suppresses immunity → Malassezia proliferates; stress is the most common exacerbating factor
- **Season:** Winter — cold, dry air + indoor heating → scalp dryness; less UV-mediated Malassezia control → dandruff worse in winter
- **Infrequent shampooing:** Sebum and skin cell buildup provides more substrate for Malassezia
- **Neurological disease:** Parkinson's disease, epilepsy — 10x higher seborrhoeic dermatitis incidence (dopamine pathway involvement)
- **Immunosuppression:** HIV/AIDS — severe seborrhoeic dermatitis in up to 85% of AIDS patients; indicates immunocompromise

Anti-Dandruff Actives and Mechanism:

Active	Conc.	Mechanism of Action
Zinc Pyrithione (ZPT)	1-2%	Interferes with Malassezia enzyme transport chain; disrupts membrane permeability; fungistatic + fungicidal; most widely used (Head & Shoulders); also mildly antibacterial
Selenium Sulfide (SeS ₂)	1-2.5%	Cytostatic: slows scalp cell turnover rate (direct antiproliferative on keratinocytes); antifungal against Malassezia; strong smell; not for color-treated hair
Ketoconazole	1-2%	Imidazole antifungal; inhibits ergosterol synthesis in Malassezia cell membrane → membrane disruption → cell death; most potent; 2% = prescription strength; 1% = OTC

Piroctone Olamine	0.5-1%	Newer; inhibits Malassezia metabolism (amino acid uptake inhibition); better cosmetic profile than ZPT; less residue on hair
Ciclopirox Olamine	1-1.5%	Broad-spectrum antifungal; chelates essential metal ions (Fe ³⁺) needed for Malassezia enzyme function; anti-inflammatory properties also
Salicylic Acid	1.8-3%	Keratolytic: breaks down protein bonds holding flakes together → helps remove existing flakes; does NOT kill Malassezia; useful in combination with antifungal
Coal Tar	0.5-5%	Cytostatic + antifungal + anti-inflammatory; reduces keratinocyte turnover; dark color; carcinogenicity concern limits use; effective in severe cases
Climbazole	0.5-2%	Imidazole antifungal like ketoconazole; common in European OTC anti-dandruff shampoos; good cosmetic elegance

HAIR FALL (Alopecia) — Causes

Hair Fall / Alopecia: Hair fall (alopecia) is the excessive loss of hair from the scalp or body beyond the normal physiological shedding of 50-100 hairs per day. It can be temporary or permanent, diffuse or patterned, and has numerous causes.

Normal Hair Physiology Review:

- **Normal hair density:** 100,000 scalp hairs; losing 50-100/day is physiological (telogen shedding)
- **Anagen (growth):** 85-90% of hairs; 2-6 years; 0.3-0.4 mm/day growth
- **Catagen (transition):** 2-3%; 2-3 weeks; regression
- **Telogen (rest/shedding):** 10-15%; 3 months; club hair shed

Classification of Hair Fall — By Pattern:

Type	Pattern	Cause
Diffuse Alopecia	Even hair thinning all over scalp; no pattern	Telogen effluvium, nutritional deficiency, hypothyroidism, systemic illness
Patterned Alopecia (AGA)	Specific patterns: frontal recession, crown thinning (males); diffuse crown thinning (females); Hamilton-Norwood scale males; Ludwig scale females	Androgenetic alopecia — hereditary + DHT

Focal/Patchy Alopecia	Circular or oval patches of complete hair loss	Alopecia areata (autoimmune); tinea capitis (fungal); traction alopecia; trichotillomania
Scarring Alopecia (Cicatricial)	Permanent; follicles destroyed and replaced by fibrosis	Lichen planopilaris, discoid lupus, central centrifugal cicatricial alopecia

Causes of Hair Fall:

Cause	Detailed Mechanism
Androgenetic Alopecia (AGA) — Most Common	5-alpha reductase type II converts testosterone → DHT (dihydrotestosterone) in follicle dermal papilla; DHT binds androgen receptors → shortens anagen phase progressively → miniaturization of follicle over years → vellus hair replaces terminal hair → baldness; genetic predisposition (AR gene polymorphism); affects 50% men by age 50
Telogen Effluvium	Physiological or pathological stressors push anagen follicles prematurely into telogen → 1-3 months later, massive synchronous shedding; triggers: childbirth (postpartum telogen effluvium), major surgery, fever, crash dieting, severe emotional stress, discontinuing OCPs; typically reversible in 3-6 months
Alopecia Areata (AA)	Autoimmune: T-lymphocytes (CD8+ and CD4+) attack hair follicle matrix cells; collapse of immune privilege of the follicle; causes patchy hair loss; can progress to alopecia totalis (entire scalp) or universalis (entire body); associated with thyroid disease, vitiligo, Down syndrome
Nutritional Deficiency	Iron deficiency (ferritin <30 ug/L): reduced oxygen delivery to follicle matrix; Zinc deficiency: impairs keratin synthesis enzymes; Biotin (Vit B7): rare, but supplementation popular; Protein (low calorie diet): hair is 95% keratin protein; Vitamin D deficiency: VDR (Vitamin D Receptor) in follicle regulates anagen; Niacin deficiency: pellagra causes hair loss
Hormonal Causes	Hypothyroidism: reduced T3/T4 → impaired follicle metabolism → diffuse hair loss + coarsening; Hyperthyroidism: accelerated turnover → diffuse loss; PCOS: elevated androgens → female pattern hair loss; Postpartum: estrogen drop after delivery → massive telogen effluvium; Menopause: estrogen decline → androgen relatively dominant → thinning
Scalp Infections	Tinea capitis (ringworm): dermatophyte (Trichophyton, Microsporum) invasion of hair shaft cortex → fragile hair breaks → patchy alopecia with inflammation and scaling; treated with systemic griseofulvin or terbinafine; Folliculitis: bacterial (S. aureus) or fungal infection of follicle → scarring possible
Mechanical / Traction Alopecia	Chronic tension on hair follicles from tight hairstyles (tight braids, weaves, buns, ponytails, hair extensions) → physical pulling on

	follicle papilla → inflammation → follicle miniaturization → hair loss; initially reversible; prolonged = permanent (scarring); common in Indian and African women with tight hairstyles
Drugs / Medications	Chemotherapy (anagen effluvium): cytotoxic drugs arrest rapidly dividing matrix cells → hair breaks at constriction → sudden massive loss; Anti-coagulants (heparin, warfarin), Retinoids (acitretin), Beta-blockers, Lithium, OCP withdrawal, Antithyroid drugs → telogen effluvium
Chemical Damage (Cosmetic)	Over-processing: bleaching (H ₂ O ₂), permanent waving (thioglycolate), relaxers (NaOH) → break disulfide bonds → hair shaft structural damage → breakage mimicking hair loss; not true alopecia but can cause significant cosmetic thinning

Cosmetic Management of Hair Fall:

Approach	Products and Actives
Anti-DHT (5-AR inhibition)	Saw palmetto extract (0.5-2%); Pygeum africanum; Pumpkin seed oil; Green tea (EGCG) — in hair serums and shampoos
Scalp circulation improvement	Minoxidil 2-5% (OTC/Rx); Rosemary oil; Caffeine 0.001% shampoo (blocks DHT in follicle; Froebel et al.); Niacinamide; Adenosine
Nutritional supplementation	Biotin-enriched shampoos; Keratin amino acid supplements; Vitamin D, Zinc, Iron supplementation for deficiency states
Anti-inflammatory scalp care	Scalp serums with niacinamide, panthenol, allantoin; reduce scalp inflammation that impairs follicle cycling
Mechanical damage prevention	Gentle wide-tooth comb; minimize heat styling; deep conditioning; avoid tight hairstyles; silk pillowcase

COSMETIC PROBLEMS ASSOCIATED WITH SKIN

BLEMISHES

Blemishes: Blemishes are any visible marks, discolorations, or imperfections on the skin surface. In cosmetic science, blemishes include hyperpigmentation, dark spots, post-inflammatory hyperpigmentation (PIH), melasma, freckles, and uneven skin tone.

Types of Blemishes:

Type	Cause	Characteristics
Post-Inflammatory Hyperpigmentation (PIH)	UV + skin trauma (acne, cuts, burns) triggers melanin overproduction	Flat dark spots at previous injury sites; more common in darker skin (Fitzpatrick IV-VI); fades slowly in 6-24 months

Melasma	UV + hormonal (estrogen, progesterone from OCP, pregnancy); stimulates melanocyte activity	Symmetrical, brownish-grey patches on cheeks, forehead, upper lip; called 'mask of pregnancy'; chronic and recurrent
Solar Lentigines (Age Spots)	Cumulative UV exposure; increased melanocyte density in sun-exposed areas	Well-defined, flat, brown spots; appear after age 40 on face, hands, forearms; benign but indicate photoaging
Ephelides (Freckles)	Genetic (MC1R gene variant); UV triggers melanin expression	Small, flat, light to medium brown; appear on sun-exposed areas; lighten in winter; common in fair skin Fitzpatrick I-III
Drug-induced Pigmentation	Amiodarone, tetracyclines, antimalarials, chemotherapy agents	Grey-blue discoloration; often in light-exposed areas; reversible with drug discontinuation

Mechanism of Hyperpigmentation:

Normal melanin synthesis pathway:

- Tyrosine → (Tyrosinase) → DOPA → (Tyrosinase) → Dopaquinone → → Eumelanin (brown/black) or Pheomelanin (red/yellow)
- **Tyrosinase:** Rate-limiting enzyme; copper-containing oxidase; activated by UV, hormones (MSH, estrogen), inflammation (arachidonic acid metabolites)
- **MSH (Melanocyte Stimulating Hormone):** Released by pituitary after UV damage; binds MC1R on melanocytes → activates cAMP → MITF transcription factor → tyrosinase synthesis → increased melanin

Cosmetic Treatments for Blemishes — Anti-Pigmentation Actives:

Active Ingredient	Conc.	Mechanism Against Hyperpigmentation
Hydroquinone (HQ)	2-4%	Most effective tyrosinase inhibitor; also inhibits melanocyte metabolism and causes selective melanocyte toxicity; Gold standard but restricted in EU/India; Rx needed at 4%; risk of ochronosis with long-term use
Kojic Acid	0.5-2%	Chelates copper in tyrosinase active site → enzyme inactivation; also antioxidant; produced from <i>Aspergillus oryzae</i> fermentation; mild sensitizer
Arbutin (Alpha/Beta)	1-3%	Hydroquinone glycoside; hydrolyzes to release HQ at follicle level; gentler and safer than HQ; alpha-arbutin preferred (more stable, more potent)
Vitamin C (L-Ascorbic acid)	10-20%	Reduces dopaquinone back to DOPA → blocks melanin synthesis; antioxidant; stimulates collagen

		synthesis; unstable — must be formulated at pH 2.5-3.5; sodium ascorbyl phosphate is stable alternative
Niacinamide	4-5%	Inhibits melanosome transfer from melanocytes to keratinocytes (blocks Pak1/protease-activated receptor PAR2 pathway) → skin brightening without inhibiting melanin synthesis; anti-inflammatory; sebum-reducing; well tolerated
Azelaic Acid	10-20%	Selective cytotoxicity against hyperactive melanocytes; also anti-acne (inhibits P. acnes); antikeratinizing; Rx at 20% but 10% OTC in some countries
Tranexamic Acid	2-5%	Inhibits plasmin → reduces prostaglandin synthesis → reduces UV-mediated MSH release → less melanocyte stimulation; very effective for melasma; oral or topical
Retinoids	0.025-0.1%	Accelerate epidermal turnover → disperse melanin granules faster; downregulate tyrosinase; prevent UVB-induced tyrosinase activation; normalize pigmentation distribution

WRINKLES

Wrinkles: Wrinkles (rhytides) are creases, folds, or ridges that develop in the skin as a result of intrinsic aging (chronological aging) and extrinsic aging (photoaging from UV, pollution, smoking). They represent visible deterioration of skin structure due to loss of collagen, elastin, and hyaluronic acid.

Classification of Wrinkles:

Type	Origin	Characteristics
Dynamic / Expression Wrinkles	Repeated muscle contractions (facial expressions)	Appear on movement; crow's feet (orbicularis oculi); frown lines (corrugator); forehead lines; become permanent over time
Static / Structural Wrinkles	Loss of collagen, elastin, HA; dermal atrophy	Visible at rest; nasolabial folds; marionette lines; cheek wrinkles; result of aging and photoaging
Gravitational Wrinkles / Folds	Gravity + loss of skin elasticity and subcutaneous fat	Jowls; sagging; ptosis; hooding; skin redistribution downward
Sleep Lines	Mechanical compression during sleep on pillow	Appear on cheeks and chin; may become permanent; sleeping on back or silk pillowcase can prevent

Pathophysiology of Wrinkle Formation:

A. Intrinsic Aging:

- **Collagen reduction:** Collagen synthesis decreases ~1% per year after age 30; collagen cross-linking increases → stiffer, less pliable skin; total collagen decreases 30% by age 70
- **Elastin degradation:** Elastin fibers become fragmented and disorganized → skin loses ability to return to shape after deformation → permanent wrinkle lines
- **Hyaluronic acid loss:** HA content in dermis decreases with age → reduced water binding → skin volume loss → hollows and wrinkles
- **Subcutaneous fat loss:** Volume loss in cheeks, temples → sagging and folding of overlying skin
- **Cell turnover slowdown:** Epidermal renewal slows from 21 days (young) to 40 days (old) → dull, thickened, less radiant skin

B. Extrinsic Aging (Photoaging — UV Damage):

- **UVA-induced MMP activation:** UVA generates ROS → activates AP-1 transcription factor → upregulates matrix metalloproteinases (MMP-1 collagenase, MMP-3 stromelysin, MMP-9 gelatinase) → degrade collagen I and III → wrinkle formation; 10x more MMP activity in sun-exposed vs sun-protected skin
- **Elastotic material:** Chronic UV → abnormal elastin accumulation (solar elastosis) → degraded, tangled elastic fibers → loss of skin elasticity → deep wrinkles and leathery texture
- **Cross-linking by AGEs:** Advanced Glycation End-products (AGEs) from high blood glucose + UV → collagen cross-linking → stiff, yellow-tinted, wrinkled skin (diabetic photoaging)
- **Smoking:** Nicotine → vasoconstriction → reduced skin oxygenation and nutrient delivery; also generates free radicals → MMP activation → prominent perioral and periorbital wrinkles in smokers

Anti-Wrinkle Cosmetic Actives:

Active	Conc.	Mechanism
Retinoids (Retinol, Retinaldehyde)	0.025-0.3%	Bind nuclear retinoic acid receptors → stimulate fibroblast collagen I and III production; inhibit MMP-1; accelerate cell turnover; increase epidermal thickness; reduce fine lines; Gold standard anti-aging cosmeceutical
Peptides (Signal peptides)	0.01-0.1%	Argireline (Acetyl hexapeptide-3): inhibits acetylcholine release → relaxes expression lines; Matrixyl (Palmitoyl pentapeptide): signals fibroblasts to produce collagen; Leuphasyl: muscle relaxing peptide
Vitamin C (L-Ascorbic acid)	10-20%	Essential cofactor for prolyl and lysyl hydroxylase → collagen synthesis; antioxidant (neutralizes ROS);

		inhibits MMP activation; stimulates collagen gene expression
Hyaluronic Acid (HA)	0.1-2%	Fills dermal HA deficit → plumps fine lines; multi-MW: low MW (5-50 kDa) penetrates skin + high MW (1500 kDa) surface film; immediately plumps skin appearance
Niacinamide	5%	Stimulates ceramide and collagen production; inhibits MMPs; anti-glycation; reduces yellowing of skin from AGEs
AHAs (Glycolic, Lactic acid)	5-20%	Chemical exfoliants; dissolve desmosomes between corneocytes → accelerate cell turnover; at higher concentration stimulate GAG (glycosaminoglycan) and collagen synthesis in dermis
Resveratrol	0.1-1%	Polyphenol from grapes; activates SIRT1 (sirtuin-1) gene → anti-aging pathway; antioxidant; anti-inflammatory; inhibits collagen degradation

ACNE VULGARIS

Acne Vulgaris: Acne vulgaris is a chronic inflammatory skin disease involving the pilosebaceous unit (hair follicle + sebaceous gland), characterized by comedones, papules, pustules, nodules, and cysts, occurring primarily on the face, chest, and back.

Epidemiology:

- Affects 85% of teenagers; most common skin disease globally
- Persists as adult acne in 50% cases; increasingly common in women over 25
- Grading: Grade I (comedones) → Grade II (papules/pustules) → Grade III (nodules) → Grade IV (cysts/conglobate)

Pathophysiology — Four Factors (CASH Model):

Factor (CASH)	Detailed Mechanism
C — Sebum overproduction (Comedogenesis)	Androgen (DHT) → sebaceous gland hypertrophy → excess sebum → creates anaerobic environment in follicle → promotes <i>C. acnes</i> colonization
A — Abnormal desquamation of follicular epithelium	Hyperkeratinization of follicular infundibulum → corneocytes do not desquamate normally → plug of dead cells + sebum = microcomedone → comedone
S — Cutibacterium acnes (formerly Propionibacterium acnes)	Anaerobic gram-positive bacillus; colonizes sebum-rich follicle; hydrolyzes triglycerides → free fatty acids → chemotactic for neutrophils; activates TLR2 → NF-κB → IL-1, IL-8, TNF-alpha → inflammation; forms biofilm in follicle

H — Host immune response / Inflammation	Neutrophils invade follicle → release proteases and ROS → follicle wall rupture → sebum and bacteria spill into dermis → intense inflammation → nodule/cyst → scarring if severe
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Acne Lesion Progression:

Microcomedone → Open comedone (blackhead) or Closed comedone (whitehead) → Papule (inflamed) → Pustule (pus-filled) → Nodule → Cyst → Scar / PIH

Acne Triggering Factors:

- **Hormones:** Androgens (testosterone → DHT via 5-AR) stimulate sebum production; explains teenage acne, PCOS-related acne, and pre-menstrual flares
- **Diet:** High GI foods + dairy → elevated IGF-1 and insulin → increased androgen signaling → sebum; chocolate, whey protein also implicated
- **Stress:** Cortisol → adrenal androgens → sebum; neuropeptides from stress also activate skin inflammatory pathways
- **Comedogenic cosmetics:** Heavy creams, waxes, silicone occlusive ingredients → cosmetic acne (acne cosmetica) in adults; non-comedogenic selection critical
- **Occlusion and friction:** Helmet straps, chin strap, mask (maskne) → mechanical acne; friction + sweat + occlusion → follicle irritation → inflammation

Anti-Acne Cosmetic and OTC Actives:

Active	Conc.	Mechanism
Salicylic Acid (SA)	0.5-2%	Beta-hydroxy acid; lipophilic → penetrates sebum-filled follicle; keratolytic: dissolves intercorneocyte bonds → unclogs pore; anti-inflammatory: inhibits arachidonic acid pathway; reduces comedones and PIH
Benzoyl Peroxide (BPO)	2.5-5%	Oxidizing agent: generates reactive oxygen species → directly kills C. acnes (bactericidal); no resistance develops; also keratolytic; first-line OTC acne treatment; available 2.5-10%
Niacinamide	5%	Inhibits neutrophil chemotaxis → reduces inflammatory lesions; also reduces sebum and PIH; excellent tolerability; ideal in acne-prone skin cream
Tea Tree Oil	5%	Terpinen-4-ol: disrupts C. acnes cell membrane; anti-inflammatory; studies show comparable efficacy to BPO 5% with fewer side effects
Sulfur	3-8%	Keratolytic; antimicrobial; reduces sebum oxidation products; mild; can combine with SA; causes characteristic sulfur odor
Azelaic Acid	10-20%	Inhibits C. acnes growth; anti-comedogenic (normalizes keratinization); selective melanotoxic

		(reduces PIH); anti-inflammatory (reduces neutrophil ROS); Rx at 15-20%
Zinc (Zinc gluconate, Zinc PCA)	1-2%	Reduces sebum production; inhibits C. acnes lipases; anti-inflammatory; oral zinc is also effective for moderate acne
Retinoids (Retinol, Adapalene OTC)	0.1-0.3%	Normalize follicular desquamation → prevent microcomedone formation; anti-inflammatory; anti-PIH; Adapalene 0.1% gel is now OTC in India and USA

PRICKLY HEAT (Miliaria)

Prickly Heat / Miliaria: Prickly heat (miliaria) is a skin condition caused by blockage of eccrine sweat ducts, leading to sweat retention, inflammation, and a characteristic itchy, prickling rash. It is common in hot, humid climates like India.

Pathophysiology:

- **Primary event:** Sweat duct (eccrine duct) becomes obstructed by sweat and keratin — maceration of stratum corneum in hot, humid conditions creates a hydrated keratin plug
- **Sweat retention:** Eccrine sweat cannot reach surface → builds up pressure in duct → duct ruptures at different levels depending on type
- **Inflammatory response:** Sweat (foreign to dermis) leaks into surrounding tissue → triggers inflammation → mast cell degranulation → histamine → itch and erythema
- **Bacteria:** Staphylococcus epidermidis produces toxins that damage sweat duct cells and contribute to duct occlusion

Classification of Miliaria (by level of duct rupture):

Type	Level of Rupture	Clinical Features	Severity
Miliaria Crystallina	SC (most superficial)	Tiny, clear, crystal-like vesicles; no inflammation; no itch; 1-2 mm; burst easily; asymptomatic	Mildest; newborns + febrile patients
Miliaria Rubra (Prickly Heat)	Epidermis / spinous layer	Small red papules and vesicles; severe itch and prickling sensation; inflamed halo; most common form	Most common; hot climates
Miliaria Pustulosa	Epidermis (infected)	Pustules (bacterial superinfection of Miliaria rubra); S. aureus or S. epidermidis; tender	Moderate; requires antibacterial

Miliaria Profunda	Dermoepidermal junction	Flesh-colored papules; no vesicles; no itch; widespread anhidrosis (can't sweat); severe; impairs thermoregulation	Severe; tropical climates
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Causes and Triggering Factors:

- **Hot, humid environment:** Primary cause; tropical and subtropical climate (India); summer; excessive sweating
- **Tight, non-breathable clothing:** Synthetic fabrics (polyester, nylon) trap heat and sweat → maceration
- **Occlusive cosmetics:** Heavy moisturizers, creams, oils on skin in hot weather → block sweat duct openings
- **Physical activity:** Exercise → profuse sweating → duct overload → miliaria rubra; common in athletes in summer
- **Fever / Immobility:** Febrile patients, bedridden, in areas of pressure → miliaria crystallina
- **Infants:** Underdeveloped sweat ducts; overdressing; common in newborns and young children (diaper area)

Cosmetic Management and Prevention:

Approach	Details
Cooling and ventilation	Stay in cool, air-conditioned environments; avoid heat and direct sun during peak hours (10am-4pm)
Light, breathable clothing	Cotton, linen, bamboo fabrics; loose fit; avoids sweat trapping; moisture-wicking sportswear for exercise
Avoid occlusive products	No heavy creams or oily products in hot weather; use light, non-comedogenic, water-based formulations
Prickly heat powder	Talcum powder (magnesium silicate) or zinc stearate-based powder; absorbs sweat; keeps skin dry; reduces friction; calamine 8% + zinc oxide
Calamine lotion	Calamine 8% + zinc oxide 0.5% in aqueous suspension; soothing, antipruritic, mild astringent; dries weeping lesions
Anti-itch ingredients	Menthol 0.5-1% (cooling; counter-irritant); Camphor 0.5%; Calamine; Diphenhydramine (OTC antihistamine cream)
Antibacterial (for pustular type)	Mupirocin (Rx) for S. aureus; antiseptic washes (chlorhexidine 0.5%); prevent secondary infection

BODY ODOUR

Body Odour (Bromhidrosis): Body odour is the unpleasant smell produced when apocrine sweat is decomposed by resident skin bacteria, particularly in the axillary, groin, and foot regions. The smell arises from volatile sulfur compounds and short-chain fatty acids produced by bacterial metabolism of sweat substrates.

Types of Sweat Glands:

Parameter	Eccrine Glands	Apocrine Glands
Distribution	Entire body; highest density on palms, soles, forehead	Axillae, groin, areola, eyelids, ear canal, perianal region
Secretion	Aqueous: water + NaCl + lactate + urea + ammonia	Viscous, milky/oily; proteins, lipids, carbohydrates, steroids, pheromone precursors
Function	Thermoregulation; electrolyte balance; minor excretory role	Unknown true function in humans; vestigial pheromone gland
Odour	Essentially odorless; ammonia odor only at extremes	Odorless when secreted; odor develops when bacteria metabolize the protein-rich sweat
Innervation	Cholinergic (acetylcholine); stimulated by heat + exercise	Adrenergic; stimulated by emotional stress, fear, pain, sexual arousal
Development	Present from birth	Active from puberty onwards (androgen-dependent)

Mechanism of Body Odour Formation — Detailed:

- **Step 1:** Apocrine glands secrete odorless, protein-rich, oily sweat into hair follicle (not directly onto skin surface)
- **Step 2:** Resident bacteria (*Corynebacterium striatum*, *Staphylococcus epidermidis*, *Anaerococcus* species) on skin surface metabolize sweat substrates
- **Step 3a (Acid pathway):** *Corynebacterium* species hydrolyze Glu-3M3SH (glutamine conjugate) → release 3-methyl-3-sulfanylhexan-1-ol (3M3SH) — primary axillary thioalcohol responsible for characteristic 'onion-like' odor
- **Step 3b (Fatty acid pathway):** *S. epidermidis* and *Corynebacterium* degrade branched-chain amino acids (leucine) → produce 3-methylbut-2-enoic acid (3M2H) and 3-methyl-2-hexenoic acid (3MH) → 'cheesy/sweaty' odor of axilla
- **Step 3c (Steroid pathway):** 5- α -androsthenol and androsthenone (steroids in apocrine sweat) contribute to 'musky' aspect of body odor
- **Result:** Complex mixture of volatile organic compounds → perceived as axillary body odour; individual variation due to HLA (human leukocyte antigen) genes and microbiome composition

Malodorous Compounds from Axillary Region:

Compound	Producing Bacteria	Odour Description
3-Methyl-3-sulfanylhexan-1-ol (3M3SH)	Corynebacterium striatum	Onion-like, sulfury — most potent axillary odorant
3-Methylbut-2-enoic acid (3M2H)	Staphylococcus hominis	Sweaty, cheesy — major axillary odorant
3-Methyl-2-hexenoic acid (3MH)	Corynebacterium spp.	Sweaty, rancid
4-Ethyl-octanoic acid	Apocrine sweat, bacteria	Goat-like odour
Androstenol, androstenone	Apocrine steroid precursors	Musky, urine-like
Isovaleric acid	Staphylococcus epidermidis	Sweaty feet, cheesy odour (foot odour)

Other Forms of Body Odour:

- **Foot odour (Bromodosis):** Eccrine sweat on feet → bacterial (Brevibacterium linens, Staphylococcus epidermidis) decomposition → isovaleric acid (cheese odor); worsened by occlusive footwear
- **Halitosis:** Oral bacteria on tongue dorsum → VSCs (H₂S, methyl mercaptan) → bad breath (see Unit 3 mouthwash notes)
- **Trimethylaminuria (Fish Odor Syndrome):** Rare genetic disorder; cannot metabolize trimethylamine → fish-like odor excreted in sweat, urine, breath

ANTIPERSPIRANTS AND DEODORANTS — ACTIVES AND MECHANISM

Deodorants and antiperspirants are the primary cosmetic solutions for managing body odour and sweat. They are the most commercially important personal care category after skin care, with the global market exceeding USD 25 billion.

Difference — Deodorant vs Antiperspirant

Parameter	DEODORANT	ANTIPERSPIRANT
Primary Action	Reduces ODOUR by inhibiting odor-causing bacteria or masking odor	Reduces SWEATING by blocking eccrine sweat ducts
Active	Antibacterials, fragrance, zinc salts, triclosan	Aluminium salts (ACH, AlCl, AZAG)
Target Gland	Apocrine gland (bacteria on apocrine sweat)	Eccrine sweat glands (sweat duct blockage)

Regulatory Status	Cosmetic (India, EU, most markets)	OTC drug (USA FDA); Cosmetic in EU/India
Duration of action	12-24 hours; requires reapplication	1-2 days; some 48-hour products available
Examples	Axe Bodyspray, Nivea Deodorant, roll-ons	Clinical Strength products, Dove Antiperspirant, Sure

ANTIPERSPIRANT ACTIVES

Classification and Comparison of Aluminium Salts:

Active Ingredient	Max Conc.	Efficacy	Properties and Notes
Aluminium Chlorohydrate (ACH)	25%	Moderate	Most widely used OTC antiperspirant; good cosmetic profile; minimal irritation; $Al_6(OH)_{15}Cl_3$ complex; roll-ons, sticks, sprays
Aluminium Chloride Hexahydrate ($AlCl_3$)	15-20%	Highest	Most potent; used for hyperhidrosis (excessive sweating); irritating to skin; must apply to completely dry skin; Drysol, Driclor brands; Rx at >20%
Aluminium Sesquichlorohydrate (ASH)	25%	Moderate-high	Between ACH and $AlCl_3$ in efficacy and irritation; $Al_2(OH)_3Cl_3$ formula; less irritating than $AlCl_3$
Aluminium Zirconium Tetrachlorohydrate-Gly (AZAG)	20%	Highest (OTC)	Most effective OTC antiperspirant; glycine complex; penetrates deeper into eccrine duct; 48-hour products; 'clinical strength'; Dove, Degree brands
Aluminium Zirconium Trichlorohydrate-Gly (AZTG)	20%	High	Slightly less efficacious than AZAG; otherwise similar properties
Potassium Alum (Natural Crystal)	N/A	Low	Natural mineral alum deodorant crystal; $KAl(SO_4)_2 \cdot 12H_2O$; mild astringent + antibacterial; minimal true antiperspirant effect; popular in 'natural' deodorant category; leaves invisible film

Mechanism of Antiperspirant Action — Step by Step:

HOW ALUMINIUM SALTS BLOCK SWEATING:

- **Step 1:** Aluminium salt solution applied to axillary skin; enters eccrine sweat duct opening
- **Step 2:** Al³⁺ ions contact eccrine duct lumen; interact with duct wall glycoprotein matrix (mucopolysaccharides)
- **Step 3:** Al³⁺ reacts with glycoproteins → forms insoluble aluminium-protein complex → creates a gelatinous, colloidal plug within the duct lumen → Al(OH)₃ / aluminium glycoprotein gel forms
- **Step 4:** Gel acts as mechanical occlusion of sweat duct → physically blocks sweat flow from eccrine gland to skin surface
- **Step 5:** Increased back-pressure from perspiration → compresses gel further → enhanced sealing effect during activity
- **Step 6 (Optional — deep penetration):** AZAG (with glycine) has higher oil-phase compatibility → penetrates further into acrosyringium (upper portion of eccrine duct) → more profound and lasting blockage → 48-hour clinical strength products
- **Duration and reversal:** Plug dissolves over 1-2 days with normal skin desquamation and sweat → effect is temporary; NOT permanent; duct regenerates normally; daily application maintains effect
- **Efficacy measurement:** Gravimetric method: weigh absorbent pads before and after sweating episode; compare treated vs untreated axilla; efficacy expressed as % reduction in sweat output

Does Aluminium Block Lymph Nodes? — Addressing the Controversy:

- **Claim:** Social media concern that aluminium antiperspirant blocks lymph node drainage in axilla → accumulates → causes breast cancer
- **Scientific evidence:** NO credible scientific evidence of a causal link between aluminium antiperspirants and breast cancer; WHO, FDA, Cancer Research UK, IARC all confirm no established link
- **Absorption:** Aluminium skin penetration from antiperspirant is extremely low (<0.01% absorbed); orders of magnitude less than dietary aluminium from food

DEODORANT ACTIVES

Classification and Mechanism of Deodorant Actives:

Active Ingredient	Conc.	Mechanism of Action
Triclosan (being phased out)	0.1-0.3%	Broad-spectrum antibacterial; inhibits enoyl-ACP reductase (FabI enzyme) in bacterial fatty acid synthesis → bacteriostatic/bactericidal; FDA Safety Review 2016 → phasing out from consumer products
Benzalkonium Chloride (BAK)	0.1-0.2%	Quaternary ammonium compound (QUAT); positively charged → binds negatively charged bacterial membrane → disrupts membrane integrity

		→ leakage → bactericidal; broad-spectrum including <i>Corynebacterium</i> and <i>Staph</i>
Chlorhexidine	0.05-0.5%	Bisbiguanide; disrupts bacterial outer and inner membrane; forms complex with DNA; substantive (binds to skin for prolonged release); broad-spectrum against odor-causing <i>Corynebacterium</i> and <i>Staphylococcus</i> ; also effective against <i>Candida</i>
Ethanol (SD Alcohol 40)	40-60%	Primary mechanism in alcohol-based sprays; bactericidal → denatures bacterial proteins and dissolves membrane lipids; evaporates rapidly (skin drying effect); also effective solvent for fragrance and other actives
Zinc Ricinoleate	0.5-2%	Unique mechanism: NOT antibacterial; zinc ricinoleate (castor oil-derived zinc salt) traps and chelates malodorous volatile molecules (VSCs, fatty acids) → forms non-volatile, odorless zinc-molecule complex; odor molecules removed from air
Zinc Oxide	1-5%	Mild antibacterial (Zn^{2+} ions disrupt bacterial membrane); astringent (reduces sweat secretion slightly); absorbs moisture; anti-inflammatory; common in natural deodorant formulas
Magnesium Hydroxide $Mg(OH)_2$	2-5%	Raises axillary pH (from slightly acidic to pH 8-9) → unfavorable environment for odor-causing bacteria (<i>Corynebacterium</i> prefer pH 6-7) → bacteriostatic by alkalization; widely used in natural/crystal deodorant products
Sodium Bicarbonate (Baking Soda)	5-20%	Alkaline pH buffering → bacteriostatic; absorbs moisture; odor neutralization via pH change; common in natural deodorants; can irritate sensitive skin with long-term use
Activated Charcoal	1-5%	Adsorbs malodorous volatile compounds onto its large surface area (physical adsorption, not chemical chelation); fashion trend in natural deodorants; also absorbs excess moisture
Fragrance / Essential Oils	0.5-3%	Masking: pleasant fragrance overrides body odor (temporary); certain EOs (tea tree, lavender, eucalyptus) also have antibacterial activity against axillary bacteria

Formulation Types for Antiperspirants and Deodorants

Dosage Form	Formulation Features	Advantages and Disadvantages
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Roll-on Liquid	15-25% Al salt in water + ethanol; glycerin; fragrance; carbomer thickener	+ Even coverage; controlled application; conditioning additives possible - Slower drying; may feel wet initially
Solid Stick	Anhydrous; AZAG + silicone (cyclopentasiloxane); sodium stearate gellant; fatty alcohol	+ Dry feel immediately; most popular; convenient - Leaves white marks on dark clothing; waxy residue
Invisible Solid Stick	Translucent; AZAG + castor wax + silicone; no sodium stearate	+ No white marks; clear; elegant - Less conditioning than regular stick
Cream	O/W emulsion; Al salt + emollients + emulsifiers	+ Very moisturizing; good for sensitive dry skin - Less popular; messy application; limited shelf life
Aerosol Spray	Al salt or deodorant active in ethanol; propellants (butane, propane, nitrogen)	+ Fast drying; very popular globally (India); even application - Propellant environmental concern; inhalation risk; less Al active deposited per application
Pump Spray (no propellant)	Al salt in ethanol-water; no pressure propellant	+ Eco-friendly; recyclable; no inhalation of propellant - Less mist than aerosol; dries slower
Gel	Clear carbomer gel; ethanol; Al salt; fragrance	+ Transparent; fresh feel; no white residue - May feel wet longer; needs shaking or mixing before use
Wipe/Pad	Al salt or deodorant in moistened cloth wipe	+ Convenient for travel and gym; portable - Single-use; creates waste; higher cost per use

Mechanism Comparison — How Different Deodorant Forms Work

Product Category	Mechanism Chain
Traditional antiperspirant (Al salt)	Al salt → eccrine duct penetration → Al-glycoprotein gel → physical duct blockage → sweat reduction → less sweat substrate for bacteria → less bacterial metabolism → less odor + less wetness
Antibacterial deodorant (BAK, chlorhexidine)	BAK/CHX → kills Corynebacterium + Staphylococcus → no bacterial metabolism of apocrine sweat → no volatile acids/thioalcohols produced → no odor; sweating continues
Odor-trapping deodorant (Zinc ricinoleate)	Zinc ricinoleate → chelates VSCs and volatile fatty acids in axillary air → odor molecules become non-volatile zinc complexes → physical odor capture; bacteria still active; sweat continues

Natural pH-raising deodorant (MgOH ₂ , baking soda)	Alkaline agent → raises axillary pH from ~6 to ~8-9 → unfavorable pH for <i>Corynebacterium</i> (optimal pH 6-7) → bacterial growth inhibited → less odor production; sweating continues
Fragrance-masking deodorant	Pleasant volatile fragrance molecules → compete with malodorous VSCs for olfactory receptors → brain perceives fragrance not odor; bacteria not affected; sweat not affected; temporary masking only

Regulatory Status of Antiperspirants Globally

Region	Classification	Regulations
USA (FDA)	OTC Drug — Antiperspirant Drug Products	FDA 21 CFR Part 350; monograph system; approved actives: ACH, AlCl, AZAG, AZTG; efficacy testing required (gravimetric, Lanman-Daman method); SPF/claim regulations
European Union	Cosmetic product (EU Cosmetics Reg. 1223/2009)	Safety assessment (CPSR) required; aluminium compounds restricted (Annex III); max 10.6% Al in sprays, 6.25% in other forms as Al; SCCS opinions on Al safety reviewed periodically
India	Cosmetic (D&C Act 1940)	Regulated under cosmetics; Schedule M GMP; CDSCO oversight; no specific OTC drug classification for antiperspirants; BIS standards apply for labeling
Japan	Quasi-drug	Antiperspirants classified as quasi-drugs; intermediate regulation; manufacturing approval needed; specific actives and concentrations listed in positive list

★ EXAM IMPORTANT POINTS

Q: What is the primary cause of oily skin? Excess androgen (DHT) stimulation of sebaceous glands; genetic

Q: What is comedogenic? Substance that blocks pores and causes comedone formation; rated 0-5 scale

Q: Difference between open and closed comedone? Open = blackhead (follicle open; melanin oxidizes black); Closed = whitehead (follicle sealed; white)

Q: What is ICD vs ACD? ICD = direct chemical damage, no sensitization needed; ACD = Type IV hypersensitivity, prior sensitization required

Q: Primary cause of dandruff? *Malassezia globosa/restricta* yeast + sebum + host immune response

Q: Mechanism of ZPT in anti-dandruff? Inhibits *Malassezia* electron transport chain; disrupts membrane permeability; fungistatic

Q: Most common cause of hair fall in men? Androgenetic alopecia — DHT via 5-alpha reductase type II

Q: CASH model of acne? C=Comedogenesis (sebum), A=Abnormal desquamation, S=Staph/*C.acnes*, H=Host inflammation

Q: What is miliaria? Sweat duct blockage → sweat retention; Miliaria rubra = prickly heat (most common)

Q: Mechanism of AZAG as antiperspirant? Al^{3+} + glycine → deep duct penetration → Al-glycoprotein gel → physical eccrine duct blockage

Q: How does zinc ricinoleate work in deodorant? Chelates/traps malodorous molecules → forms non-volatile complexes; NOT antibacterial

Q: Primary odor compound in axillary BO? 3M3SH (3-methyl-3-sulfanylhexan-1-ol) — 'onion-like'; produced by *Corynebacterium*

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