

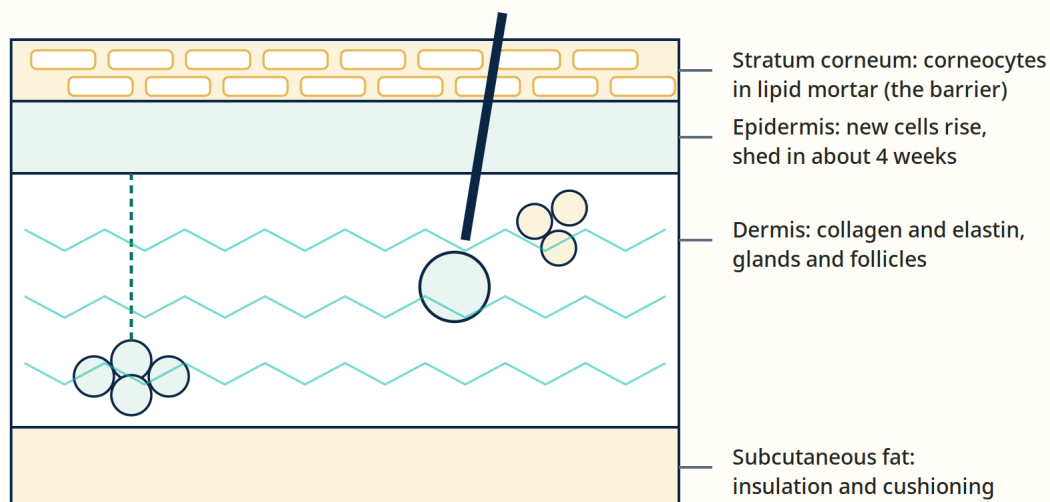
Unit 2: Cosmetics, Biological Aspects

Free notes at pharmacolearn.fyi

2.1 Structure of skin: the surface every cosmetic meets

Skin is the largest organ of the body and the substrate of almost every product in this subject. Its three layers are the epidermis, the dermis and the subcutaneous fat. The **epidermis** is a stratified squamous epithelium whose surface layer, the **stratum corneum**, is the working barrier: roughly 10 to 20 micrometres of flattened, dead corneocytes embedded in a lipid mortar of ceramides, cholesterol and free fatty acids, the classic bricks and mortar model. Corneocytes are continuously made at the basal layer and shed from the surface, a turnover of about four weeks in young skin. On the surface sits the acid mantle, a thin film of sweat, sebum and degraded corneocyte contents giving a surface pH around 4.5 to 5.5, and the natural moisturizing factor (NMF) inside corneocytes, a hygroscopic mixture of amino acids, urea, lactic acid and salts derived largely from the protein filaggrin, which holds water in the stratum corneum. The **dermis** beneath is connective tissue: collagen for tensile strength, elastin for recoil, and a gel of glycosaminoglycans such as hyaluronic acid holding water, with blood vessels, nerves, sweat glands and the pilosebaceous units (hair follicle plus sebaceous gland) passing through it. The **subcutaneous layer** insulates and cushions. Two barrier ideas run through the whole subject: water is lost through the barrier as transepidermal water loss (TEWL), the quantitative measure of barrier health, and almost nothing applied to intact skin penetrates far past the stratum corneum, which is why cosmetics act at the surface and why their claims are framed there.

Structure of Skin: The Cosmetic Substrate



Acid mantle pH about 4.5 to 5.5; NMF holds water in corneocytes

© 2026 PharmacoLearn, pharmacolearn.fyi

Figure: The layers of skin. Almost every cosmetic acts at or above the stratum corneum, the barrier layer at the top.

2.2 Dry skin: where the barrier leaks

Dry skin (xerosis) is a failure of stratum corneum water content, which normally stays above about 10 percent. The causes combine: reduced NMF from filaggrin deficiency or ageing, loss or disorganisation of the intercellular lipids that seal the corneocytes, low environmental humidity, and stripping of surface lipids

and NMF by harsh surfactants and hot water. The result is a stiff, cracked surface film: corneocytes shrink and separate, fine scale appears, the barrier leaks so TEWL rises, and fissures admit irritants, producing tightness, itching and secondary inflammation. The biology dictates the three intervention classes that Unit 4 builds into moisturizers: occlusives that lay a water proof film over the surface and cut TEWL, humectants that bind water in the stratum corneum, and emollients that fill the gaps between corneocytes and restore flexibility. Dry skin is also the model for sensitive skin generally, because a defective barrier admits preservatives, fragrances and other small molecules that intact skin would exclude.

2.3 Acne: the pilosebaceous unit under pressure

Acne vulgaris is a disorder of the pilosebaceous unit, the hair follicle and its sebaceous gland, most active on the face, chest and back. Four factors combine. Sebaceous glands enlarge and secrete more sebum under androgen stimulation, typically at puberty. The follicle lining hyperkeratinizes, so shed cells stick together instead of flowing out, plugging the follicle outlet as a comedo (open, a blackhead, when the plug darkens at the surface; closed, a whitehead, when it does not). The anaerobic bacterium *Cutibacterium acnes* (formerly *Propionibacterium acnes*) proliferates in the sebum rich, oxygen poor plug and its enzymes and metabolites provoke inflammation, converting a comedo into papules, pustules and, in severe disease, nodules and cysts that scar. Cosmetic relevance is threefold: comedogenic ingredients can add to follicle plugging; cleansing and sebum control are legitimate cosmetic aims; and the boundary with medicine is close, because established inflammatory acne is a disease whose treatment (retinoids, benzoyl peroxide as a drug, antibiotics) sits on the drug side in most classifications, so a cosmetic product supports care and must not claim cure.

2.4 Pigmentation: melanin and its distribution

Skin colour and its disorders come from melanin, made by melanocytes in the basal epidermis. Each melanocyte supplies pigment to a fixed neighbourhood of keratinocytes (the epidermal melanin unit), synthesising melanin in organelles called melanosomes from tyrosine through the rate limiting enzyme tyrosinase: tyrosine is oxidised to dopaquinone, which proceeds to the brown black eumelanin or, with cysteine, the yellow red pheomelanin. Ultraviolet radiation stimulates melanogenesis, the tanning response, and hormones and inflammation modulate it. Hyperpigmentation disorders include melasma (symmetrical facial patches, driven by sun, hormones and visible light), post inflammatory hyperpigmentation after acne or injury, and solar lentigines (age spots) from chronic sun. Hypopigmentation, loss of pigment as in vitiligo, is a medical condition outside cosmetic claims. Cosmetically, pigmentation is addressed by limiting melanin production at tyrosinase, by interrupting melanosome transfer to keratinocytes, by accelerating shedding of pigmented surface cells, and above all by photoprotection, because every lightening strategy fails under continued ultraviolet exposure. Unit 4 develops each of these into formulations.

2.5 Prickly heat: a sweat duct problem

Prickly heat (miliaria) is a disorder of the eccrine sweat duct in hot, humid conditions. Sweat glands deliver sweat to the surface through a coiled duct; when the duct is blocked by swollen, hydrated stratum corneum, sweat is retained and leaks into the surrounding skin at the level of the block. Blockage within the stratum corneum gives tiny clear vesicles (miliaria crystallina); blockage deeper, in the epidermis, gives the itchy red papules of miliaria rubra, the familiar prickly heat rash, common in infants and in adults newly exposed to tropical heat. The cosmetic logic follows the mechanism: keep the skin cool and dry, avoid heavy occlusive products that hydrate and swell the surface further in hot weather, use light, absorbent preparations and powders that reduce surface wetness and friction, and include soothing agents for the itch. Prickly heat is also the reason heavy cold creams of Unit 3 are winter products in the Indian market and light lotions and powders dominate summer shelves.

2.6 Wrinkles: ageing written in the dermis

Wrinkles are the visible surface record of dermal change. Intrinsic ageing slowly thins the dermis: fibroblasts make less collagen and elastin, the collagen that remains is fragmented and disorganised, glycosaminoglycan water content falls, and the epidermis flattens at its junction with the dermis so the surface folds more easily. Photoageing, chronic ultraviolet exposure, accelerates and dominates the process on exposed sites: ultraviolet generates matrix metalloproteinases that degrade collagen, produces the tangled, dysfunctional elastin material of solar elastosis, and adds pigmentation irregularity. Expression lines form where repeated muscle movement folds the weakening sheet, and gravity and fat redistribution deepen folds with age. The cosmetic response is limited by anatomy: no cream rebuilds the dermis, but photoprotection prevents most photoageing, moisturization plumps the stratum corneum and smooths fine lines optically and physically, and retinoids and related actives used in cosmeceuticals can modestly improve photoaged skin over months. Honest claims say "reduces the appearance of fine lines", which is both the legal and the scientific truth, and examiners reward the distinction.

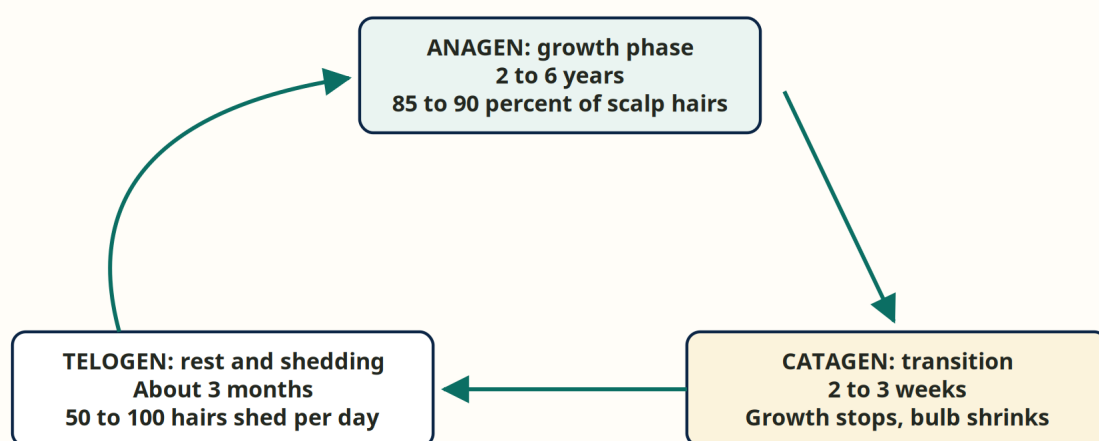
2.7 Body odor: sweat, skin bacteria and the axilla

Fresh sweat is nearly odourless. Body odor is manufactured by skin bacteria, chiefly in the axilla (under arm), where the apocrine glands open into hair follicles and produce a protein and lipid rich secretion on top of the watery eccrine sweat. *Corynebacterium* and *Staphylococcus* species on the warm, moist axillary skin metabolise apocrine secretions and sweat components into volatile odorants, including short chain fatty acids and steroid derivatives; the classic axillary odorant 3-methyl-2-hexenoic acid is released from an odourless precursor secreted by the glands. Foot odor follows the same logic inside occlusive footwear. Two distinct cosmetic strategies follow, and Unit 4 separates them: deodorants reduce odor, by antimicrobial action against the responsible bacteria, by absorbing or masking odorants, and by fragrance; antiperspirants reduce sweat itself, aluminium salts forming a temporary gel plug in the eccrine duct, which also starves the bacteria of moisture. The axilla's skin is thin and often shaved, so irritation and sensitization limits are part of deodorant formulation design.

2.8 Structure of hair and the hair growth cycle

A hair is a keratin filament produced by the hair follicle, an invagination of epidermis reaching into the dermis. The visible shaft has three concentric layers: the outer **cuticle** of overlapping flattened scales pointing towards the tip, the **cortex** of keratin fibres in a protein matrix, which gives the hair its strength, elasticity and its pigment granules, and in coarse hair a central **medulla**. Below the surface, the follicle bulb surrounds the dermal papilla, whose blood supply feeds the rapidly dividing matrix cells that build the shaft; the sebaceous gland opens into the follicle above the bulb, coating the emerging hair with sebum. Hair grows in cycles independent of its neighbours. In **anagen**, the growth phase lasting two to six years on the scalp, the shaft lengthens at roughly a centimetre a month and some 85 to 90 percent of scalp hairs are in this phase. In **catagen**, a brief two to three week transition, growth stops and the bulb shrinks. In **telogen**, a resting phase of about three months, the old hair is held loosely and then shed, normally 50 to 100 hairs a day, as a new anagen hair forms beneath it. The cycle explains both normal shedding and why hair fall products cannot act quickly: a follicle committed to telogen sheds its hair whatever is applied, and any genuine effect on growth appears over a full cycle. Chemical treatments act on the shaft's chemistry: the disulfide bonds of keratin, reduced and reformed in permanent waving and straightening, are what give hair treatments something permanent to change.

The Hair Growth Cycle



Each follicle cycles independently; a telogen hair is shed whatever is applied

© 2026 PharmacoLearn, pharmacolearn.fyi

Figure: The hair growth cycle. A follicle in telogen sheds its hair on schedule, which is why hair products cannot show quick effects.

2.9 Common problems of the oral cavity

The mouth is a cosmetic territory with medical borders. **Dental caries** begins when plaque bacteria, principally *Streptococcus mutans*, ferment dietary sugars to acid, dropping plaque pH below the critical value (about 5.5 for enamel) at which enamel mineral dissolves; repeated acid attacks produce the cavity. **Gingivitis**, inflammation of the gums with bleeding on brushing, is the response to plaque accumulated at the gum margin, and untreated it can progress to periodontitis, destruction of the tooth's support, which is dental disease beyond cosmetics. **Halitosis** (mouth odor) comes mainly from anaerobic bacteria on the tongue coating and in periodontal pockets degrading proteins to volatile sulfur compounds such as hydrogen sulfide and methyl mercaptan. **Dentin hypersensitivity** (sensitive teeth) arises when gum recession or enamel wear exposes dentinal tubules; fluid movement in the tubules in response to cold, heat or touch triggers sharp pain. Add to these extrinsic tooth staining from tea, coffee and tobacco, and calculus, mineralised plaque that only professional scaling removes, and the oral care formulator's targets are complete. Unit 3 builds the toothpaste that addresses them and Unit 4 turns each problem into a cosmeceutical design.

2.10 Cleansing and care needs across body sites

One skin biology, many sites: the care programme differs because thickness, sebum output, exposure and function differ.

Site	Distinguishing features	Cleansing and care needs
Face	Thin skin, high sebum in the T zone, fully exposed to sun and pollution	Gentle cleansing that does not strip, moisturization matched to skin type, daily photoprotection, eye area products kept mild
Eye lids	Thinnest skin of the body, no subcutaneous fat, products migrate towards the eye	Very mild, tear compatible cleansers and removers, no harsh surfactants or strong fragrance near the eye
Lips	No stratum corneum of normal thickness, no sebaceous or sweat glands, lose water rapidly	Occlusive and emollient lip salves, no detergent cleansing, sun protection for prolonged exposure
Hands	Palms thick and gland rich, backs thin and exposed; frequent washing at work	Syndet or superfatted cleansing, rich replenishing creams after washing, barrier creams for wet work

Site	Distinguishing features	Cleansing and care needs
Feet	Thick plantar stratum corneum, occluded by footwear, high sweat output	Antifungal hygiene where needed, odor control, urea based softening of thick callus, drying between toes
Nails	Hard keratin plate over a sensitive bed, cuticle seals the base	Gentle removers, cuticle care without cutting injury, moisturization of the nail plate to prevent brittleness
Scalp	Dense follicles and sebaceous glands, dandruff prone, hair covered	Sebum and scale cleansing by shampoo, conditioning of the shaft, anti dandruff and soothing products where required
Neck	Thin, sun exposed, often neglected in product application	Same moisturization and photoprotection as the face
Body	Large area, variable thickness, friction zones	Soap or syndet cleansing matched to frequency, moisturization after bathing, talc or powders in folds in hot climates
Under arm	Thin, occluded, apocrine glands, frequently shaved	Mild cleansing, deodorant or antiperspirant care, irritation aware formulation

The table is the practical summary of this unit: every product category of Units 3 and 4 exists because one of these sites has a need its biology creates.

*More free M.Pharm notes and unit wise PDFs at pharmacolearn.fyi
Free video lectures on the PharmacoLearn YouTube channel*