Harry's Cosmeticology

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Harry's Cosmeticology

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PART ONE

The Skin and Skin Products

Chapter One

The Skin

Introduction

The skin[1–3] is not simply a protective wrap for the body; it is a busy frontier which mediates between the organism and the environment. It not only controls the loss of valuable fluid, prevents the penetration of noxious foreign materials and radiation and cushions against mechanical shock, but also regulates heat loss and transduces incoming stimuli. Moreover, by its colour, texture and odour it transmits sexual and social signals which may possibly be physiologically enhanceable by cosmetic science but certainly are culturally enhanceable by cosmetic art. For cosmeticians, whether they are concerned with the improvement of the skin by pharmacology or the prevention of damage as a result of artifice, an understanding of skin structure and function is essential.

The total area of the skin ranges from about 2500 cm^2 at birth to 18 000 cm^2 in the adult, when it weighs about 4·8 kg in men and 3·2 kg in women.

There are two main kinds of human skin: hairy and glabrous. Over most of the body the skin possesses hair follicles with their associated sebaceous glands. However, the amount of hair varies greatly; at the extreme, the scalp, with its large hair follicles, may be contrasted with the female face, which has large sebaceous glands associated with very small follicles which produce fine, short vellus hairs. The skin of the palms and soles lacks hair follicles and sebaceous glands, and is grooved on its surface by continuously alternating ridges and sulci which form patterns of whorls, loops or arches, unique to each individual, known as dermatoglyphics (Figure 1.1). Glabrous skin is also characterized by its thick epidermis and by the presence of encapsulated sense organs within the dermis.

The barriers to permeability are situated in the several layers of closely packed cells which form the overlying epidermis; mechanical protection is provided by the thicker underlying dermis which is composed mainly of connective tissue, that is, material secreted by cells and lying outside of them. Isolated epidermis is as impermeable as whole skin, whereas once the epidermis is removed the dermis is completely permeable. If the epidermal layers are progressively stripped by adhesive tape, the permeability of the skin increases, and there is little doubt that the bonded, interlocked, horny cells of the stratum corneum constitute the barrier. It is unlikely that emulsified fat on the skin surface greatly affects permeability, or that the sweat glands and hair follicles are more permeable than the surface epithelium, though material may possibly reach the sebaceous glands by the follicular route.

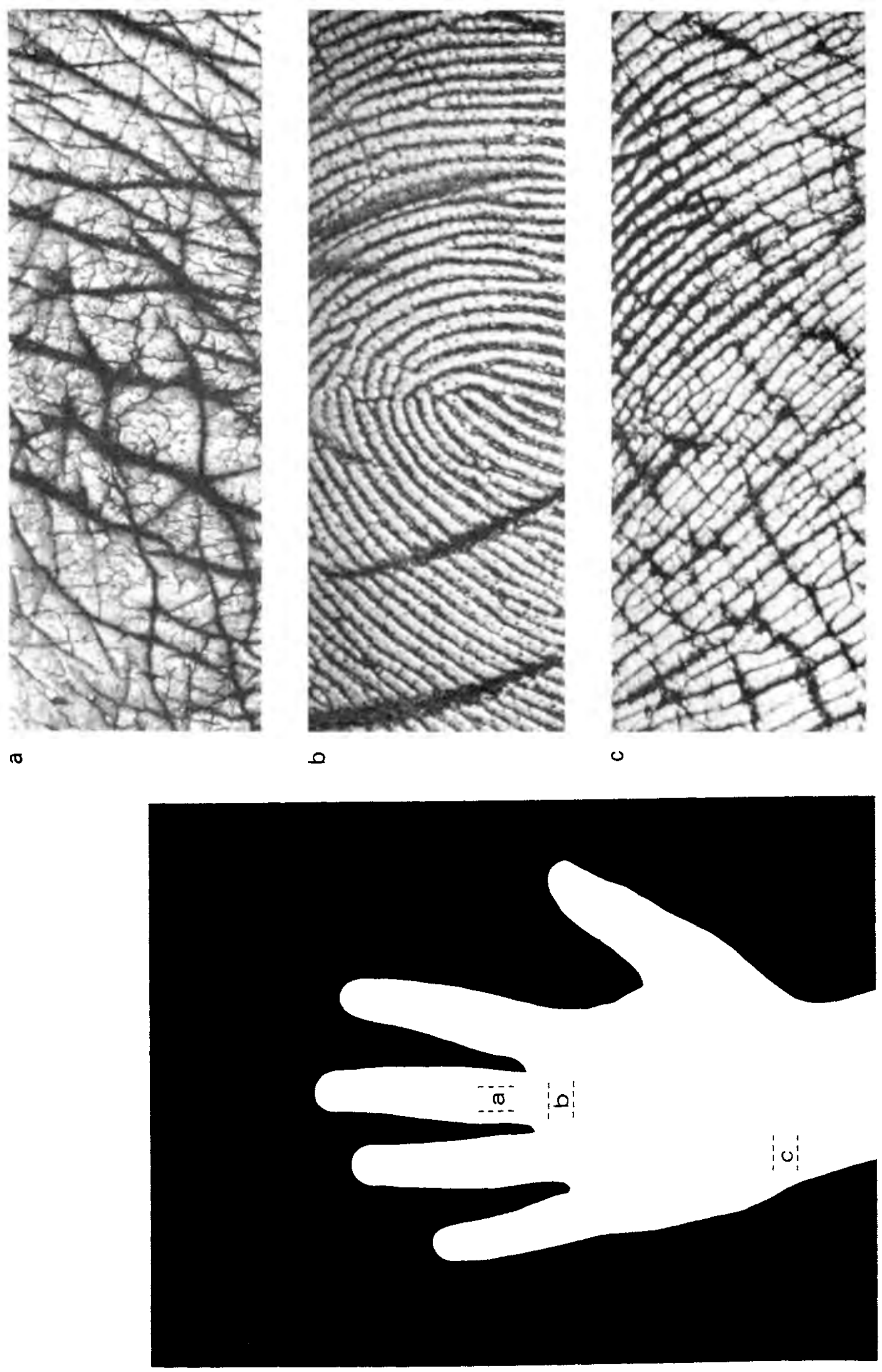

Figure 1.1 Skin patterns of the human hand (magnification × 4.5) *a* Dorsal *b* Palmar *c* Palmar

Epidermis and the Keratinizing System

The epidermis consists of a number of layers. The stratification is the result of changes in the *keratinocytes* as they move outwards from the basal layer, in which they are continuously formed by mitosis, to the skin surface where they are lost.[4–7] Three other cell types are present: *melanocytes* or pigment cells, *Langerhans cells,* which are colourless and dendritic in form, and *Merkel cells,* which are concerned with sensation (see below).

Dermo–Epidermal Junction

The dermo–epidermal junction is undulating in section; so-called rete pegs or epidermal ridges project from the epidermis into the dermis. At the junction is a basement membrane, which under the electron microscope is seen as a convoluted plasma membrane studded with *semi-* or *junctional desmosomes,* separated from the underlying electron-dense *basal lamina* by a clear *lamina lucida.* The basal lamina is anchored in the dermis below by fibrils and bundles of fine filaments.[7,8]

Stratum Basale

The *stratum basale* or *stratum germinativum* is a continuous layer which gives rise to all the keratinocytes. It is usually described as one cell thick,[7] but in thick normal or pathological epidermis it appears that mitosis may not be confined to cells in contact with the basement membrane.[9–11] Do cells destined for differentiation arise as daughters of progenitors permanently committed to cell division? One view is that both daughters equally retain the capacity to divide for a time, but that for each division a basal cell moves into the stratum spinosum, either at random[12,13] or by precedence of age.[14] However, Potten[15] supports the traditional concept of permanent stem-cells, though he concedes that daughters may remain capable of a few 'amplification' divisions before differentiating.

Between one division and the next the cell undergoes a cycle.[16–19] Immediately following mitosis (M) is a growth phase (G_1), which is succeeded by a period of active nuclear DNA synthesis (S) and a short premitotic growth phase (G_2). Each period has a *transit time*; for the complete cycle the term 'cell cycle time' should be used. The expression '*turnover time*', and its synonyms '*regeneration time*', or '*replacement time*', refer to the time for complete replacement of a cell population. Although frequently stated to be equivalent to the cell cycle time, this would only be true if all the cells were continually cycling. In fact, it is likely that there is a substantial compartment (G_0) of non-cycling cells. It is, moreover, important to distinguish the turnover time of the stratum corneum from that of the viable epidermis.

The average duration of the cell cycle has been variously estimated for normal human epidermis as 163 hours,[19] 308 hours,[20] 457 hours[21] and 213 hours, [22] and for psoriatic epidermis as 37 hours.[23] However, these measurements have assumed that in normal epidermis all cells cycle continuously. An alternative explanation is that psoriatic epidermis differs from normal not because of a shorter cell cycle, but because it has a much higher proportion of cycling cells.

The replacement time for the whole viable epidermis is probably about 42 days[24] and for the stratum corneum about 14 days,[25,26] and it is generally agreed that the times are considerably less in psoriatic skin.[27–29]

Cells of the stratum basale have large nuclei; under the electron microscope their cytoplasm reveals many ribosomes, mitochondria and, sometimes, smooth membranes. In particular, they contain numerous fine *tonofilaments,* about 5 nm in diameter, which occur mainly in loose bundles, the *tonofibrils.*

Stratum Spinosum

The *stratum spinosum* or prickle cell layer is so called because the cells are given a spiny appearance by the numerous *desmosomes* or attachment plaques at their surfaces. These were once believed to be intercellular bridges through which the tonofibrils maintained the tonus of the epidermis. Ultrastructural studies reveal that they are laminated structures. In the upper region of the stratum spinosum, *membrane-coating granules,*[30,31] also known as lamellated or Odland bodies,[32] make their appearance. These are ovoid bodies about 100–500 nm long. In the stratum intermedium they ultimately migrate towards the periphery of the cell and appear to increase in numbers in the intercellular spaces. Their function is unknown, though they appear to contain mucopolysaccharides and it has been suggested that they may constitute the intercellular cement.[31]

Stratum Granulosum

The stratum spinosum is succeeded by the *stratum intermedium*, or *stratum granulosum,* which contains basophil granules of a material called *keratohyalin.*[33]

Stratum Lucidum

The *stratum lucidum*, unstainable by the usual histological methods, can be recognized only in palmar and plantar skin.

Stratum Corneum

In the *stratum corneum*[6,7,34,35] the keratinocytes have lost their nuclei and virtually all of their cytoplasmic organelles and contents, including the keratohyalin granules. The cells are flattened and completely filled with keratin, in the form of bundles of filaments embedded in an opaque interfilamentous material. At the transition between the stratum intermedium and stratum corneum, transition cells or T-cells[7,36] are recognizable. The cornified cells in their epidermis, though not those of glabrous skin, can be shown to be arranged in regular vertical stacks, which must reflect the underlying dynamic organization.[37–42] Most authors now believe that both the filamentous structures of the lower epidermal layers and the keratohyalin of the stratum intermedium contribute to the formation of keratin.[43,44] Some, however, have held that the fibrils contribute nothing;[45–48] others have questioned the contribution of keratohyalin.[49] The most attractive, if unproven, hypothesis is that the fibrillar material, with helically arranged peptide chains, is transformed in the stratum intermedium by a sulphur-rich matrix which makes possible cystine links.[7] Various attempts to characterize chemically pure 'pre-keratin' have proposed units with molecular weights of 640 000,[50,51] 100 000–200 000[52] or 50 000.[53]

Horny cells are continuously shed from the skin surface. If skin sites are protected by cups for long periods, exfoliated material is trapped, but the thickness of the coherent stratum corneum remains unchanged.[54,55] It seems, therefore, that the horny layer desquamates at a final level which is not much influenced by external forces.

Pigmentary System

Although skin owes some of its colour[56] to red haemoglobin in the blood vessels and yellow carotenoids in the hypodermal fat, the major determinant is a dark pigment, *melanin,* which is the product of special cells known as *melanocytes.* The skin colour of human subjects can be measured by reflectance spectrophotometry.[57]

Melanocytes are derived from the neural crest in the embryo[58,59] and migrate to many tissues of the body, including the basal layers of the epidermis and the hair bulb. They differ from other cells of the stratum basale by the possession of dendritic (that is, finger-like) processes (Figure 1.2), by which they transfer

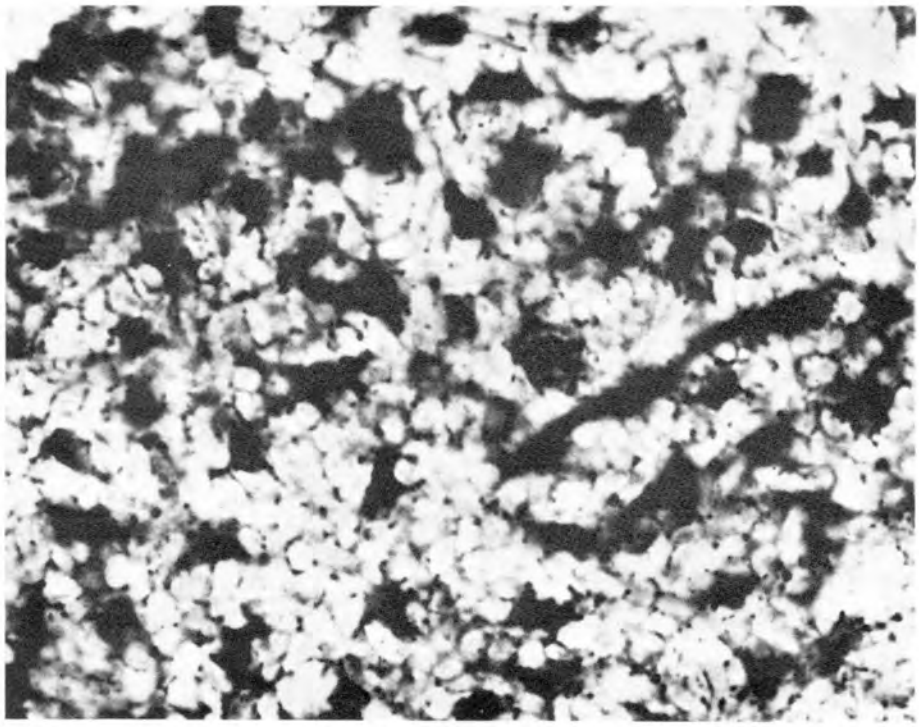

Figure 1.2 Melanocytes on underside of epidermis (magnification × 500): finger-like processes extend from the centres of the melanocytes

pigment to a group of keratinocytes, the whole forming an 'epidermal melanin unit'.[60] They have no desmosomes.

The characteristic feature of melanocytes is a special cytoplasmic organelle known as a *melanosome* (Figure 1.3) on which the melanin is formed by the action of the enzyme tyrosinase. The melanosomes arise as spherical, membrane-bounded vesicles in the zone of the Golgi apparatus. Filaments are at first

Figure 1.3 Melanosomes in various stages of melanization (magnification × 29 000): incompletely melanized granules have a striped appearance

visible in them, but the build-up of melanin ultimately results in a dense structure.[61]

Melanins are quinoid polymers of two kinds. *Phaeomelanins,* which are yellow or red in colour, differ from the brown or black *eumelanins* in being soluble in dilute alkali. Both are formed by the same initial steps which involve oxidation of tyrosine to 3,4-dihydroxyphenylalanine (dopa) and its dehydrogenation to dopa quinone.[62] The formation of eumelanins then involves several further steps to produce indole-5,6-quinone, which polymerizes and becomes linked to protein. It is now believed that eumelanin is not a homopolymer composed solely of indole-5,6-quinone units, but a poikilopolymer which includes several intermediates. Phaeomelanins are formed by a different route. The dopa quinone interacts with cysteine to form 5-S- and 2-S-cysteinyldopa, and these isomers are further oxidized to a series of intermediates which then polymerize.[63,64]

Skin colour has a *constitutive*—that is, genetic—component and a *facultative*—that is, environmental—component. Thus various degrees of pigmentation occur in different ethnic groups; the differences are in the amount of melanin produced, not in the numbers of melanocytes present. Pigmentation can be enhanced by exposure to sun, or by endocrine factors, for example in pregnancy. Melanogenesis is influenced by certain polypeptide hormones of the pituitary[65–67] and to some extent by steroid hormones. From hog pituitary, two melanocyte-stimulating hormones α-MSH and β-MSH, containing respectively 13 and 18 amino acid residues have been isolated.[68,69] The human pituitary lacks α-MSH, but produces a β-MSH with 22 residues. However, it seems likely that the active sequence is actually part of two larger molecules, β-lipoprotein with 91 amino acids and γ-lipotropin with 58.[70]

There are long-standing reports that testosterone increases skin pigmentation in castrated men[71,72] and in women.[73] The same may be true for certain specialized areas of skin in some animals, but experimental studies on the guinea pig failed to reveal any effect of androgens,[74,75] though oestrogens clearly increased skin pigmentation in a number of areas.[76,77]

The major function of melanin is undoubtedly protection against solar radiation.[78–80] In general, pigment is geographically distributed in relation to the solar intensity experienced by the various ethnic groups, being greatest in the tropics, reduced in temperate zones, and partly reappearing in areas of snow-glare.[81] There are exceptions: for example, American Indians do not noticeably differ in colour throughout the continent. The damaging effects of ultraviolet light are well illustrated by the high incidence of epidermal carcinoma in Europeans exposed to the tropical sun. Melanin pigmentation may be useful in two ways. As well as providing direct protection from radiation, it may be activated to a free radical state by incident light and thus could possibly eliminate genetically damaged cells by a phototoxic mechanism.

Langerhans Cells

Langerhans cells are dendritic cells similar in form to melanocytes but free from pigment and unable to form it when they are incubated with dihydroxyphenylalanine (that is, they are dopa-negative). They were first demonstrated in human

skin by the use of gold chloride[82] and can be stained with ATPase.[83] Under the electron microscope they resemble melanocytes in having a lobulated nucleus, but differ in lacking melanosomes, having instead characteristic granules which are rod- or racquet-shaped.[84–88]

The origin and affinities of Langerhans cells have been much debated, and their function remains undecided. The view that they are effete melanocytes is discarded.[89–91] It is currently believed that Langerhans cells are of mesenchymal origin and equivalent or closely related to dermal histiocytes,[92] in which identical granules have been described.[93–96] Various possible functions have been ascribed to them. For example, opinion is divided about whether they may[97] or may not[98,99] control proliferation of keratinocytes and the pattern of epidermal cell columns. Another suggested role might be the loosening of intercellular connections.[100,101] Langerhans cells are capable of limited phagocytosis, but they should not be regarded as functional macrophages.[102,103] Recently attention has become focussed on the possibility that they are concerned with immune functions.

Dermis

The *dermis*[1,104,105] is a tough and resilient tissue which cushions the body against mechanical injury and provides nutriment to the epidermis and cutaneous appendages. It consists of an association of protein fibres with an amorphous ground substance containing mucopolysaccharide. There are few cells in this matrix; most of them are *fibroblasts* which secrete the dermal constituents; others are *mast cells*, histiocytes or macrophages, lymphocytes and other leucocytes, and melanocytes. The dermis also houses blood, lymphatic and nervous systems, and surrounds the invaginated epidermal appendages, namely the hair follicles, with its associated glands and the eccrine sweat glands.

Collagen

The major fibrous constituent of the dermis, accounting for 75 per cent of the dry weight and 18–30 per cent of the volume, is *collagen*.[105–111] Under the light microscope collagen fibres appear as colourless, branching wavy bands about 15 μm in width. The electron microscope reveals that each fibre is composed of unbranched fibrils about 100 nm (1000 Å) wide and is characteristically cross-striated with a periodicity of 60–70 nm. Collagen fibres can be disintegrated by 0·01 per cent acetic acid, forming molecules with a molecular weight of 300 000–360 000, about 180 nm long. When these acid solutions of *tropocollagen* are neutralized, the 64 nm periodicity reappears, which may be explained on the hypothesis that native collagen is composed of molecules of tropocollagen associated side by side with a regular overlap of a quarter of their length.[112]

Skin collagen is characterized by a high content of glycine, which forms a third of all the residues, and of proline and hydroxyproline, which together make up a further fifth. Tropocollagen molecules[107] consist of three polypeptide chains each containing about 1000 amino acids. The fibroblasts produce a precursor known as *procollagen* which has 300–400 additional amino acids in each of its chains; these extensions are removed after secretion.[113,114]

Elastin and Reticulin

Elastic fibres[115–124] make up 4 per cent of the dry weight and 1 per cent of the volume of the dermis. They are delicate, straight, freely branching fibres which can be stretched by 100 per cent or more but return to their original length when the stress is removed. Elastin differs from collagen in having only about a quarter or a third the amount of basic and acid amino acids, only one tenth the amount of hydroxyproline, a relatively large amount of valine, and an amino acid known as desmosene[125] which appears to be unique to it and to be concerned with cross-linkage.

Not all fibrous constituents can be clearly identified as collagen or elastin on the basis of their tinctorial properties. In addition to true elastin, two other similar fibres have been distinguished and given the names of oxytalan and elaunin.[126] Moreover, about 0·4 per cent of the dry weight of the dermis is made up of fine branching fibres which, unlike collagen, stain black with silver nitrate, and are known as *reticulin*. Their axial periodicity is identical with that of collagen.[116]

Ground Substance

The amorphous ground substance[127–135] in which the fibres and cells lie contains a variety of carbohydrates, proteins and lipids, of which the most important are the acid mucopolysaccharides. These are macromolecules made up of two different saccharide units which alternate regularly. In dermis the major forms are *hyaluronic acid*, in which D-glucosamine, with an acetylated amino group, alternates with D-glucuronic acid, and *dermatan sulphate*, in which L-iduronic acid alternates with D-galactosamine.

Fibroblasts

The term *fibroblast*[105,136,137] should, strictly, designate a cell at any early stage and *fibrocyte* one which is fully differentiated,[138] but most authors use fibroblast to describe an actively secreting cell and fibrocyte for an inactive one.[105] Fibroblasts are derived from the mesenchyme. It is not doubted that fibroblasts secrete collagen.[139] It is probable that they are the source of elastin[140] and, though Asboe-Hansen[141] has implicated the mast cell, also of mucopolysaccharides.[142]

Mast Cells

Mast cells[143–146] also originate from wandering cells of the mesenchyme. They are characterized by a cytoplasm filled with granules which stain metachromatically with basic aniline dyes—purple with methylene blue. They contain, and can release, heparin and histamine. Rupture of the cells, with release of the granules, is observed in many types of skin damage, and histamine is responsible for many of the events associated with inflammation, irritation and other skin disorders. This subject is dealt with at greater length in the next chapter.

Nerves and Sense Organs

The skin is innervated with about one million afferent nerve fibres; most terminate in the face and extremities; relatively few supply the back.

Sensory endings fall into two major groups: corpuscular, which incorporate non-nervous elements, and free, which do not.[147–152] Corpuscular endings, in turn, are subdivided into encapsulated receptors, of which there is a range in the dermis, and non-encapsulated, such as the epidermal Merkel 'touch spot'.[153,154]

The largest encapsulated receptors are the elaborate *Pacinian corpuscles*[155,156] which are ovoid bodies about 1 mm in length and lamellated in cross-section like an onion. Others are the *Golgi-Mazzoni corpuscles* found in the subcutaneous tissue of the finger, the *Krause end-bulbs* in the superficial layers of the dermis, and the *Meissner corpuscles*[147,151,152,157] in the papillary ridges of the glabrous skin. Of somewhat different structure are the branching terminals of *Ruffini.*[151]

Free nerve endings occur both in the dermis and in the epidermis. Hair follicles have nerve terminals of varying degrees of complexity.

The way these miscellaneous receptors function has been much debated. As it is easy to map separate sensory spots for the several kinds of stimuli, the classical view was that receptors were specific for the qualities of touch (Meissner's corpuscles), warmth (Ruffini end-organs), cold (Krause end-bulbs) and pain (free nerve endings). The hypothesis came under attack on the grounds that it did not explain why hairy skin could also distinguish between the stimuli, even though it lacked the encapsulated structures.[158]

The existence of functionally specific afferent units has in recent years been reaffirmed by electrophysiological experiments. Two major categories of units have been established: mechanoreceptors and thermoreceptors,[150,151] and a third category, pain receptors, respond only to high threshold stimulation, mechanical, thermal or chemical. Mechanoreceptors have been further classified into 'slowly adapting', as exemplified by the Ruffini endings and Merkel cells, and 'rapidly adapting', namely the hair follicle receptors, Meissner corpuscles, and the laminated Pacinian and Golgi-Mazzoni corpuscles.[159]

The autonomic nervous system supplies both adrenergic and cholinergic fibres to the arrector pili muscles and the blood vessels. Stimulation of the arrector pili muscle by its associated nerve causes the hair shaft to rise to a more perpendicular position with respect to the skin surface. This slows down the passage of air over the skin and consequently reduces the rate of heat loss. This phenomenon is the cause of 'goose flesh'. Regulation of the amount of blood flowing through the superficial layers of the dermis also influences heat loss (see next section).

Eccrine sweat glands are also richly supplied with nerves.[160] Anticholinergic substances are able to inhibit sweating, and most of the nerves appear to be cholinergic, though a few adrenergic fibres can be demonstrated. It seems likely that the glands of the palms and soles, which secrete sweat to increase the grip of the skin, are influenced by adrenergic fibres, whereas those of the general body surface, which regulate body heat, are under cholinergic control.[161]

Blood Vessels

The arteries entering the skin form a deep plexus, from which a network arises which gives branches to the cutaneous appendages and to a subpapillary plexus, which in turn sends loops into the papillary layer just below the dermo–epidermal frontier. From these capillaries the blood is drained by veins which descend into the intermediate plexuses.[162–164]

All the nutriment for the epidermal cells has to pass through the dermo–epidermal junction; no blood vessels enter the epidermis. The vasculature is much more elaborate than would be necessary solely for nutrition; indeed, the metabolic rate in skin is lower than in many less well-perfused organs. Temperature control thus appears to be a most important function. When the superficial loops are fully dilated, the skin appears flushed and heat loss is at a maximum. However, shunts are provided between arterial and venous systems deeper in the dermis which can carry all or most of the blood when heat loss must be kept to a minimum. In these circumstances the superficial capillary loops are found to be almost completely closed.

The regulation of the total blood volume in the skin as opposed to its distribution is mediated by constriction and dilation of the cutaneous circulation, and allows a large reservoir of blood to be made rapidly available for vital central functions in times of stress. The mechanism of constriction of the lumen of a blood vessel in the dermis can be either by a general activation of contractile myoepithelial cells in the wall of the capillary, or by activation of '*glomerae*' which are small contractible cuffs around the vessel, and which effectively strangle the vessel and cut the blood flow. The operations of constriction and dilation are mediated via the local secretion of chemicals (for example acetylcholine) from nerves, hormones (for example adrenalin) and, in cases of skin damage, histamine from the mast cells in the dermis.

As distinct from this widely held view, Ryan[165] has stressed the oxygenating function of the vasculature in a tissue which is exposed to many kinds of injury. Finally, it must be remembered that the blood supply carries all the materials for making the products of the hair follicle and its associated glands, as well as the hormones which influence their manufacture, and the substances which are excreted by the sweat glands.

Eccrine Sweat Glands

Eccrine sweat glands[166] are the most numerous skin appendages and occur over the majority of the body surface. In some areas they number as many as 600 cm^{-2}. They have a cylindrical spiral duct lined with epidermal cells extending from their visible opening in the epidermis down into the deep dermis where the duct becomes coiled and convoluted into a ball (Figure 1.4). Part of the tangled duct is secretory and manufactures the odourless sweat which rises up the duct to be released on the skin surface. It is thought that the duct of the gland has the ability to modify the sweat as it flows upward, by removing salts or water.[167] The analogy with the nephron of the kidney is frequently drawn. Though the lining of the duct is said to be epidermal, it is not highly pigmented even in people with pigmented skin.

The sweat glands of the general body surface are concerned with both control of body temperature and excretion. The evaporation of sweat has a cooling effect. The glands thus respond to environmental temperature, but also to other stimuli, such as ultraviolet light, emotional stress and rises in body temperature due to fevers. On the palms and soles, however, the secretion from the glands serves to increase surface friction. In both areas, sweating is under nervous control, though different types of fibre may be involved (see previous section).

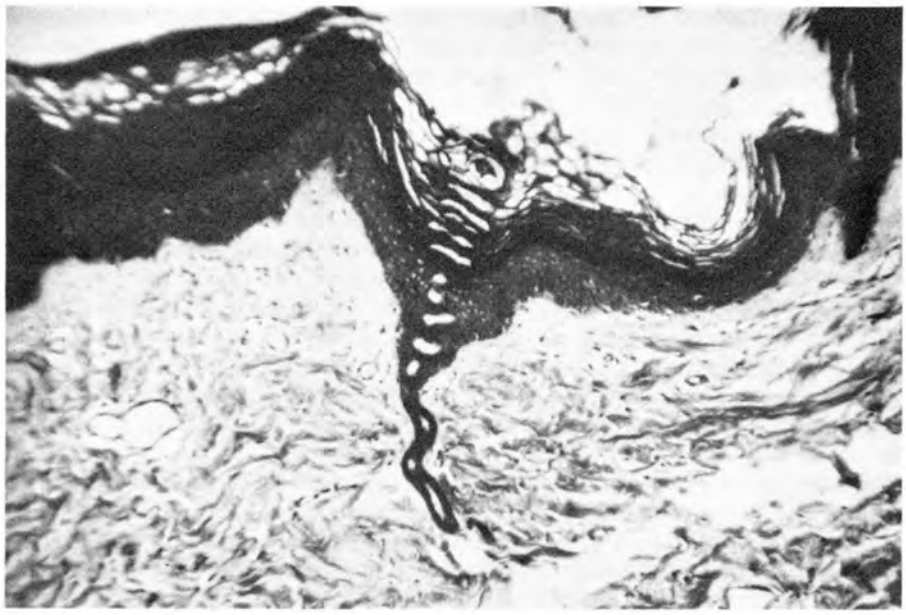

Figure 1.4 Section of human toe skin showing a spiralling sweat duct (magnification × 140): invagination of the epidermal tissue along the length of the duct can be clearly seen

Sweating appears to involve activation of myoepithelial cells which line the ducts of the glands. Although sweating is considered to be a continuous process, it seems that sweat is ejected in small bursts, perhaps 6–7 per minute, suggesting a peristaltic action by the ducts.[168] The composition of eccrine sweat is variable but consists of electrolyte ions, urea, amino acids, small quantities of sugars and possibly some lipid. The normal range of sodium chloride concentration in eccrine sweat is stated to be between 10 and 100 milli-equivalents per litre.[169]

Hair Follicles

Hair follicles are tubular inpushings of the epidermis. The hair is produced by keratinization of cells formed by division in the matrix at the base of the follicle.[170–174] This epidermal matrix surrounds a small dermal papilla which becomes invaginated into its base.

There are about 120 000 follicles on the human scalp. Each one undergoes a cycle of activity[174] in which an active phase (anagen), which lasts for 1 to 3 years or even longer, is followed by a short transition phase (catagen) and a resting phase (telogen) (see Figure 23.2). This process involves a cessation of mitosis in the matrix and the keratinization of the expanded base of the hair to form a 'club', which is retained until the follicle again becomes active, when it is shed (Figure 23.3). Thus about 100 hairs are normally lost from the scalp each day.[175,176]

Such cyclic activity of hair follicle may be considered as a remnant of the moult in other mammals. In contrast to the human scalp, where the activity of each follicle appears to be independent of its neighbours, some animals, such as rats and mice, exhibit wavelike patterns of new hair growth and moulting, which start in the mid-venter and spread over the flanks to the back.[177] These have proved interesting models for experimentation on the factors controlling hair growth, but it should not be supposed that this has any direct relevance to human baldness. It appears that hair follicles have an intrinsic rhythm, of which the mechanism remains undiscovered, but that this can be greatly modified by circulating hormones and thus, in turn, by environmental factors acting through the hypothalamus and the pituitary.[177,178] Thus moulting, like reproductive activity, is seasonally controlled. Perhaps even the human scalp retains a reflection of the seasonal moult, with increased shed of club hairs in the autumn.[179]

In the axillary and pubic regions of both sexes, and on the face of the male, coarse *terminal* hair—as distinct from fine *vellus*—develops at puberty, and continues to increase in amount for several years.[180] The growth of this hair is initiated by and dependent upon androgens (male steroid hormones) which are secreted by the testicles of the male and by the adrenal glands and the ovaries in the female. Male-type body hair is also androgen-dependent, though its amount and distribution vary greatly between individuals. Unacceptable amounts of facial and body hair in women, known as *hirsutism*, may result from abnormal high androgen production, but individual variations in the sensitivity of the target hair follicles is also important. Compounds which block the action of androgen, known as *anti-androgens*, offer possibilities for the alleviation of female hirsutism.[181]

Male pattern alopecia, a condition in which vigorously growing terminal hair is gradually replaced by miserably small and cosmetically useless fibres over areas of the scalp, appears to be hereditary, but requires the presence of male hormone. Hence eunuchs, even if genetically disposed, do not go bald, unless treated with testosterone,[182] and women rarely develop conspicuous bald patches, though they frequently suffer diffuse hair loss which may be the female equivalent. Why male hormones should promote hair growth on the face and body and ruin it on the vertex of the scalp, so far eludes any consistent explanation.

The structure and growth of hair is further considered in Chapter 23.

Sebaceous Glands

Sebaceous glands[183,184] secrete sebum, which forms the majority of the lipid which covers the skin and hair. They occur throughout most of the body and are normally, though not invariably, associated with hair follicles. The greatest concentrations (400–900 cm^{-2}) are found on the scalp, face and upper chest and shoulders, and there are none on the palms and soles.

The glands are *holocrine*, that is to say the cells of the gland pass through a development and maturation stage, during which they accumulate lipid, becoming several times their original size, and subsequently disintegrate completely,

releasing their contents into the lumen of the gland. New cells are formed continually from the lining of the gland by cell division to replace those lost.

Sebaceous gland activity is under hormonal control. It is stimulated by androgens. In human males, the glands are minute during the prepubertal period, but undergo vast enlargement at puberty when the output increases more than fivefold.[185] Eunuchs secrete about half as much sebum as normal males, but substantially more than boys; it seems that the secretion is dependent on adrenal androgens. Adult women secrete only a little less than men; their sebaceous activity appears to be maintained by androgens from the ovary as well as from the adrenal cortex.

Circumstantial evidence from man, and experimental evidence from animals, indicates that pituitary hormones may also influence sebaceous secretion. Sebum secretion is abnormally high in acromegalics.[186] The response of the rat sebaceous glands to testosterone is greatly diminished when the pituitary is removed. Bovine growth hormone[187] and synthetic α-MSH[188,189] have each been shown to have some direct effect on sebaceous secretion, and to facilitate the response of the glands to testosterone.

Oestrogens, or anti-androgens such as cyproterone acetate, will inhibit sebaceous secretion in man[181] as well as in rats.[190]

Human sebum[191] is composed of glycerides and free fatty acids (57·5 per cent), wax esters (26·0 per cent), squalene (12·0 per cent), cholesterol esters (3·0 per cent) and cholesterol (1·5 per cent). Lipid produced from the superficial epidermis differs in lacking wax esters and squalene, and having much higher proportions of cholesterol esters and cholesterol. Skin lipids appear to differ greatly between species.

Apocrine Glands

The so-called *apocrine glands*[192] are tubular glands attached to the hair follicle and, like the sebaceous glands, developed in association with it. Though rudiments are formed throughout the body in the foetus, the glands become canalized and functional almost exclusively in the axillary, anal and genital regions and in the areola of the nipple; few are found elsewhere. The axillary glands only become functional at puberty and it seems probable that, like similar derivatives in other animals, for example the rabbit,[193] they are androgen-sensitive.

The secretion of human apocrine glands is milky, viscous and at first without noticeable odour, which is said to develop through bacterial action. Secretory activity is controlled by adrenergic nerves.

The function of the glands in the human species has been much debated. In many other mammals they constitute or contribute to scent glands. Odour is undoubtedly important in human communication,[194,195] though little information has been recorded since Havelock Ellis wrote down his entertaining if anecdotal evidence.[196]

Common Disorders of the Skin

The cosmetic chemist is concerned not with serious clinical disorders of the skin but with lesser, if often chronic, conditions that affect large numbers of the

population and which are only presented to the clinician when extremely severe. Discussion in this chapter is confined to a few which appear to come within the purview of the cosmetic scientist. For detailed accounts of these and of other disorders the reader is referred to textbooks of dermatology.[197,198]

Pigmentary Disorders

Ephelides, Lentigens and Moles. It is not easy to discover a consistent classification for the small hyperpigmented areas which occur on the skin of most Caucasians. It is generally agreed that freckles (ephelides) are pale, variably coloured, not usually raised, and harmless. Their pigmentation is due to an increased local synthesis of melanin in the epidermis. The predispositions for these are apparently genetically determined. They are found predominantly on the exposed areas of fair or red-haired people and are stimulated by exposure to UV or X-irradiation. Children do not usually have freckles until after their sixth year of life.

It is usually considerations of degree which differentiate between freckles and the more pronounced lentigens which are generally associated with age, and moles (junctional naevi). These latter are usually more heavily pigmented, fewer in number and are associated with a thickening of the epidermis. They are rarely present at birth, and in women become considerably darker during pregnancy, as do other areas.[200] It should be mentioned that in the most severe cases these naevi can become malignant but this will not be considered further in this book.

Vitiligo. Apart from hyperpigmented disorders there are considerable cosmetic problems associated with hypopigmentation diseases, the most common of which is vitiligo, a patchy depigmentation of the skin afflicting a considerable number of non-Caucasians. Although it does occur in Caucasians, it is not usually cosmetically troublesome. This condition has been referred to by a former Prime Minister as 'India's national disease'. It is the more distressing on account of its resemblance to the early stages of leprosy, when depigmentation also occurs, and therefore it can carry a social stigma without any foundation.

Vitiligo is usually associated with an absence not only of melanin but of melanocytes in the affected areas. The aetiology is unknown. It frequently exhibits a degree of bilateral symmetry and is also seen to follow superficial nerve trunks, but there is little support for the hypothesis that it is linked with nerve function.[201] An autoimmune hypothesis is based on its clinical association with a number of other supposedly autoimmune disorders.[202]

Vitiligo has been treated by systemic psoralens (photosensitizing compounds obtained from certain umbelliferous plants) followed by exposure to the sun or UV radiation,[203] or by topical corticosteroid preparations.[204] Treatments are usually not very satisfactory, and cosmetic camouflage is often the best recourse.

Disorders of the Sebaceous and Sweat Glands

Acne vulgaris[205–207] is a chronic disorder of the pilosebaceous follicles which is so frequent among Caucasoids as to be regarded as physiological in adolescents.

Appendix

Proprietary Materials Cited in this Book

Entries in italics are the CTFA adopted names as listed in the *CTFA Cosmetic Ingredient Dictionary*; they are reprinted with the permission of the Cosmetic, Toiletry and Fragrance Association, Inc., Washington DC, USA.

It should be noted that CTFA does not 'approve', 'certify' or 'endorse' particular ingredients for use in cosmetic products. CTFA has a committee that assigns 'adopted names' to certain cosmetic ingredients for inclusion in the *CTFA Cosmetic Ingredient Dictionary*, which is recognized by regulations of the United States Food and Drug Administration (Title 21, Code of Federal Regulations, Section 701.3) as an authoritative reference of proper nomenclature of an ingredient for purposes of cosmetic ingredient labelling. Assignment of an 'adopted name' and inclusion in the *Dictionary* establishes only proper *nomenclature* for an ingredient for purposes of ingredient labelling on cosmetic products sold in the United States; it does *not* signify CTFA (or FDA) 'approval' of the ingredient.

In the interests of achieving uniform nomenclature for ingredients available for use in the cosmetic industry, the CTFA adopted names are becoming increasingly used in countries other than the United States of America.

Proprietary name	**Chemical description**	**Supplier**
Acetol	*Acetylated lanolin alcohol*	Emery
Actamer	2,2-Thiobis-(4,6-dichlorophenol)	Monsanto
Aduvex 2211	2-Hydroxy-4-methoxy-4′-methyl-benzophenone	Ward Blenkinsop
Aerosil	Finely divided silica	Degussa
Aerosol OT	Dioctyl sodium sulphosuccinate	American Cyanamid
Aethoxal	Ethoxylated propoxylated fatty alcohol *PPG-10-Ceteareth-20*	Henkel
Alcloxa	Aluminium chlorhydroxy allantoinate *Alcloxa*	Hoechst
Alrosal	Fatty acid amide concentrate	Ciba-Geigy
Amerchol CAB	Cholesterol absorption base *Petrolatum* and *Lanolin Alcohol*	Amerchol
Amerchol L-101	Mineral oil and lanolin alcohols	Amerchol
Aminoxid WS35	Cocamidopropylamine oxide	Goldschmidt

Proprietary name	Chemical description	Supplier
Ammonyx 4002	Benzyldimethylstearylammonium chloride *Stearalkonium Chloride*	Onyx
Amphomer	*Octylacrylamide/Acrylates/Butylaminoethyl Methacrylate Polymer*	National Starch
ANM starch powders	Starch ethers	Neckar-Chemie
Anobial	3′,4′,5-Trichlorosalicylanilide	Firmenich
Anobial TFC	3-Trifluoromethyl-4, 4′-dichlorocarbanilide	Firmenich
Antara opacifiers		GAF
Antifoam AF	Mixture of dimethylpolysiloxane (*dimethicone*) and silica gel *Simethicone*	Dow Corning
Antiviray	Homomenthyl salicylate (*Homosalate*), benzyl salicylate and methyl eugenol	Bush Boake Allen
Arlacel A	Mannide mono-oleate	ICI
Arlacel C	*Sorbitan Sesquioleate*	ICI
Arlacel 83	*Sorbitan Sesquioleate*	ICI
Arlacel 165	Glycerylmonostearate and polyoxyethylene stearate *Glyceryl Stearate* and *PEG-100 Stearate*	ICI
Arlamol E	Fatty acid propoxylate *PPG-15 Stearyl Ether*	ICI
Arlatone T	Sorbitan polyoxyethylene fatty acid ester *PEG-40 Sorbitan Peroleate*	ICI
Arlex	*Sorbitol*	ICI
Arochlor 5460		Monsanto
Arquad 2HT	Dimethyldi (hydrogenated tallow) ammonium chloride *Quaternium-18*	Armak
Arquad 16	Alkyltrimethylammonium chloride *Cetrimonium Chloride*	Armak
Arquad S50	Trimethylsoyammonium chloride *Soyatrimonium Chloride*	Armak
Atlas G-271	N-soya-N-ethylmorpholinium ethosulphate *Quaternium-2*	ICI
Atlas G-1086	Polyethylene glycol sorbitol hexaoleate	ICI
Atlas G-1425	Polyoxyethylene sorbitol lanolin derivative	ICI

Proprietary name	Chemical description	Supplier
Atlas G-1441	Polyoxyethylene sorbital lanolin derivative *PEG-40 Sorbitan Lanolate*	ICI
Atlas G-1690	Polyoxyethylene ether of alkyl phenol	ICI
Atlas G-1790	Polyoxyethylene lanolin condensate *PEG-20 Lanolin*	ICI
Atlas G-2132	Polyoxyethylene lauryl ether	ICI
Atlas G-2240	Polyoxyethylene sorbitol	ICI
Atlas G-2320	Polyoxyethylene sorbitol	ICI
Atlas G-2859	Polyoxyethylene sorbitol 4, 5-oleate	ICI
Atlas G-3721	Polyoxyethylene-2-butyl octanol	ICI
Atlas G-7596J	Polyoxyethylene sorbitan monolaurate *PEG-10 Sorbitan Laurate*	ICI
Avicel	Microcrystalline cellulose	FMC
Avitex ML	Cationic emulsifying agent	DuPont
Bentones	Quaternary hectorites	NL Industries
Bentonite	Clay mineral, montmorillonite *Bentonite*	Berk
Betadine	Iodophor (i.e. mixture of iodine with surface-active agent)	Berk
BHT	Butylated hydroxytoluene (i.e. 2,6-di-*tert*-butyl-4-methylphenol) *BHT*	Kodak Chemical
Bithionol	2,2′-thio-*bis*-(4,6-dichlorophenol)	Hilton Davis Chemicals
Bradosol	β-Phenoxyethyldimethyldodecyl-ammonium bromide	CIBA
Brij 30	Polyoxyethylene lauryl ether *Laureth-4*	ICI
Brij 35	Polyoxyethylene lauryl ether *Laureth-23*	ICI
Brij 93	Polyoxyethylene (2) oleyl ether *Oleth-2*	ICI
Bronopol	*2-Bromo-2-Nitropropane-1, 3-Diol*	Boots
BTC	Lauryldimethylbenzylammonium chloride	Onyx
BTC 2125M	Tetradecyldimethylbenzylammonium and dodecyldimethyl-*p*-ethylbenzylammonium chlorides *Myristalkonium Chloride* and *Quaternium-14*	Onyx
Cab-o-sil	Fumed silica	Cabot

Proprietary name	Chemical description	Supplier
Calflo E	Calcium silicate	Johns-Manville
Carbopol 934	Polymer of acrylic acid cross-linked with a polyfunctional agent *Carbomer-934*	Goodrich
Carbopol 940	Polymer of acrylic acid cross-linked with a polyfunctional agent *Carbomer-940*	Goodrich
Carbopol 941	Polymer of acrylic acid cross-linked with a polyfunctional agent *Carbomer-941*	Goodrich
Carbopol 960	Ammonium salt of Carbopol 934 *Carbopol-960*	Goodrich
Carbowax 400	Ethylene oxide polymer *PEG-8*	Union Carbide
Carbowax 1000	Ethylene oxide polymer *PEG-20*	Union Carbide
Carbowax 1500	Ethylene oxide polymer *PEG-6-32*	Union Carbide
Carbowax 1540	Ethylene oxide polymer *PEG-32*	Union Carbide
Carbowax 4000	Ethylene oxide polymer *PEG-75*	Union Carbide
Carbowax 6000	Ethylene oxide polymer *PEG-150*	Union Carbide
Catrex	Interpolymer of aminoethylacrylate phosphate and acrylic acid *Aminoethylacrylate Phosphate/Acrylate*	National Starch
Ceepryn	Cetylpyridinium bromide or chloride	Merrell
Celacol	Sodium carboxymethylcellulose	British Celanese
Cellofas	Sodium carboxymethylcellulose	ICI
Cellosize	*Hydroxyethylcellulose*	Union Carbide
Ceraphyl 60	γ-Gluconamidopropyldimethyl-2-hydroxyethylammonium chloride *Quaternium-22*	Van Dyk
Ceraphyl 65	Mink aminopropyldimethyl-2-hydroxyethylammonium chloride *Quaternium-26*	Van Dyk
Cetavlon	Cetyltrimethylammonium bromide	ICI
Cetiol HE	Polyol fatty acid ester *PEG-7 Glyceryl Cocoate*	Henkel
Chlorhexidine	1,6-di-(N-*p*-chlorophenylguanidino)hexane *Chlorhexidine*	ICI

Proprietary name	Chemical description	Supplier
Chlorhydrol	Aluminium chlorhydrate, 50% solution *Aluminium Chlorohydrate*	Reheis
Comperlan HS	Stearic monoethanolamide	Henkel
Comperlan KD	Cocamide diethanolamide *Cocamide DEA*	Henkel
Courlose	Sodium carboxymethylcellulose	British Celanese
Crillet 3	Mixture of stearate esters of sorbitol and sorbitol anhydrides condensed with approx. 20 moles of ethylene oxide *Polysorbate 60*	Croda
Crodafos N3 acid	Phosphated oleyl ether (3EO) *Oleth-3 Phosphate*	Croda
Crodafos N3 neutral	Diethanolamine salt of a complex mixture of esters of phosphoric acid and *Oleth-3* (which is the polyethylene glycol ether of oleyl alcohol) *DEA-Oleth-3 Phosphate*	
Crodalan IPL	*Isopropyl lanolate*	Croda
Crodamol CSP	Cetostearyl palmitate	Croda
Crodamol IPP	*Isopropyl Palmitate*	Croda
Crodamol ML	*Myristyl Lactate*	Croda
Crodamol OP	*Octyl Palmitate*	Croda
Crodaterge LS (now named Crodasinic LS)	*Lauroyl Sarcosine*	Croda
Crodaterge OS (now named Crodasinic OS)	*Oleoyl Sarcosine*	Croda
Crodesta F70, F160 and F110	Sucrose esters of palmitic acid and stearic acids	Croda
Cromeen	Substituted alkyl amine derivative of various lanolin acids	Croda
Crotein Q	Quaternized hydrolysed animal protein	Croda
DC 200	Dimethylpolysiloxane	Dow Corning
Dehyquart SP	Ethoxylated quaternary ammonium phosphate *Quaternium-52*	Henkel
Deriphat 170C	Lauryl aminopropionic acid *Lauraminopropionic Acid*	General Mills
Detergent 1011	Secondary amide of lauric acid	
Dichlorophene	2,2-methylene-*bis*-(4-chlorophenol)	Givaudan
Dicrylan 325	Acrylate/acrylamide polymer	Ciba-Geigy

Proprietary name	Chemical description	Supplier
Diometam	Di-(n-octyl)-dimethylammonium bromide	British Hydrological
Dioxin	6-Acetoxy-2, 4-dimethyl-*m*-dioxane	Givaudan
Dowfax 2A		Dow
Dowicil 200	1-(3-chloroallyl)-3,5,7-triaza-l-azoniaadamantane chloride *Quaternium-15*	Dow
Duponol C	Sodium lauryl sulphate	DuPont
Duponol WA	Sodium lauryl sulphate	DuPont
Duponol WAT	Triethanolamine salt of lauryl sulphate *TEA-Lauryl Sulfate and TEA-Oleyl Sulfate*	DuPont
Edifas	Sodium carboxymethylcellulose	ICI
Emcol CD-18	Propoxylated polyol	Witco
Emcol E-607	*N*-(Acylcolaminoformylmethyl) pyridinium chloride	Witco
Emcol E-6075	*N*-Stearoylcolaminoformyl-methylpyridinium chloride *Quaternium-7*	Witco
Emerest 2400	*Glyceryl Stearate*	Malmstrom
Emersol 132	Stearic acid	Malmstrom
Empicol LZ	Sodium lauryl sulphate	Albright & Wilson
Empigen BB	Alkyl dimethyl betaine	Albright & Wilson
Empigen BT	Alkyl amido betaine	Albright & Wilson
Empigen CDR10	Coconut imidazoline betaine	Albright & Wilson
Empigen CDR30	Modified coconut imidazoline betaine	Albright & Wilson
Emsorb 6915	*Polysorbate 20*	Malmstrom
Escalol 506	Amyldimethyl-*p*-aminobenzoic acid *Amyl Dimethyl PABA*	Van Dyk
Estol 1461	Glycerol monostearate nse	Unichema
Ethomeen C/25	*PEG-15 Cocamine*	Armak
Eutanol G	2-Octyldodecanol *Octyl Dodecanol*	Henkel
Eutanol LST		Henkel
Evanol	Proprietary cream base	Evans Chemetics
Extrapones	Herbal extracts	Dragoco
Fentichlor	*Bis*-(2-hydroxy-5-chlorophenyl) sulphide	Cocker Chemical
F.H.P.	Sodium carboxymethylcellulose	Hercules

Proprietary name	Chemical description	Supplier
Filtrosol A-1000 and B	UV screens—proprietary mixtures	Norda
Fixanol C	Cetylpyridinium bromide or chloride	ICI
Fixanol VR	Tetradecylpyridinium bromide	ICI
Fluilanol	Lanolin oil and *Oleth-3* (which is the polyethylene glycol ether of oleyl alcohol)	Croda
Fluorophene	3,5-dibromo-3′-trifluoromethylsalicylanilide *Fluorosalan*	Pfister
F.M.P.	Sodium carboxymethylcellulose	Hercules
Foromycen F10	Antifungal agent	Petrosin Laboratorium
Fungicide DA	Undecylenamide diethanolamide *Undecylenamide DEA*	Dragoco
Fungicide UMA	Undecylenamide monoethanolamide *Undecylenamide MEA*	Dragoco
Gafquat 734 and 755	Quaternary ammonium polymers formed by the reaction of dimethyl sulphate and a copolymer of vinyl pyrrolidone and dimethylaminoethylmethacrylate *Quaternium-23*	GAF
Gantrez ES-225	Monoethyl ester of methylvinylether/maleic acid copolymer (50% in ethanol) *Ethyl ester of PVM/MA copolymer*	GAF
Gantrez ES-425	Monobutyl ester of methylvinylether/maleic acid copolymer (50% in ethanol) *Butyl ester of PVM/MA copolymer*	GAF
Genapol S200		Hoechst
Germall 115	Imidazonidyl urea	Sutton Laboratories
Giv-tan F	2-Ethoxy-*p*-methoxycinnamate *Cinoxate*	Givaudan
Glucam-P20	*PPG-20 methylglucose ether*	Amerchol
HD Eutanol	Oleyl alcohol	Henkel
Hexachlorophene	2,2′-methylene-*bis*-(3,4,6-trichlorophenol)	Givaudan
Hibitane	1,6-di-(N-*p*-chlorophenylguanidino) hexane	ICI
Hostaphat KL340N	Triester of the polyethylene glycol ether of lauryl alcohol and phosphoric acid *Trilaureth-4 Phosphate*	Hoechst

Proprietary name	Chemical description	Supplier
Hostapur SAS	Secondary alkane sulphonate	Hoechst
Hyamine 10X	Methylbenzethonium chloride	Rohm & Haas
Hyamine 1622	*p*-Diisobutylphenoxyethoxyethyl-dimethylbenzylammonium chloride *Benzethonium Chloride*	Rohm & Haas
Hyamine 2389	Alkyltolylmethyltrimethylammonium chloride *Quaternium-28* and *Quaternium-29*	Rohm & Haas
Ionol	Butylated hydroxytoluene, i.e. 2,6-di-*tert*-butyl-4-methylphenol *BHT*	Shell
Ionol CP	Purified grade of Ionol *BHT*	Shell
Irgasan BS200	3,3′,4,5′-Tetrachlorosalicylanilide	Ciba-Geigy
Irgasan CF-3	3-Trifluoromethyl-4,4′-dichlorocarbanilide *Cloflucarban*	Ciba-Geigy
Irgasan DP-300	2,4,4′-Trichloro-2″-hydroxydiphenylether *Triclosan*	Ciba-Geigy
Isopar E	Isoparaffinic solvent	Esso
Isopropylan 33	*Isopropyl Lanolate* and *Lanolin oil*	Robinson-Wagner
Isothan Q	Alkylisoquinolinium bromide	Onyx
Isothan Q-15	*Lauryl Isoquinolinium Bromide*	Onyx
Kelzan	*Xanthan gum*	Kelco
Klucel	*Hydroxypropylcellulose*	Hercules
Klucel HA	Hydroxyalkylcellulose	Hercules
L-43 Silicone		Union Carbide
Laneto 50, 100	Polyethylene glycol-50 lanolin *PEG-50 Lanolin*	R.I.T.A.
Lanoquats	Quaternized fatty acid amides derived from lanolin acid	Malmstrom
Lanoquat DES	Lanolin quaternary	Malmstrom
Lantrol	Liquid lanolin *Lanolin oil*	Malmstrom
Laponite	Hectorite-type synthetic clay	Laporte
Lathanol LAL	*Sodium Lauryl Sulphoacetate*	Stepan
Lexate TA	Glyceryl stearate, isopropyl myristate and stearyl stearate	Inolex
Lexein X-250	Hydrolysed animal protein	Inolex

Proprietary name	Chemical description	Supplier
Lexemul AR	Glycerol sterate	Inolex
Lexemul AS	Glyceryl stearate and sodium lauryl sulphate	Inolex
Liquid Base CB. 3929	Mineral oil and lanolin alcohols	Croda
Loramine DU 185	Undecylenamide diethanolamide	Rewo
Loramine OM 101	Monoalkylolamide of mixed fatty acids	Rewo
Loramine SBU 185	Disodium monoundecylenamido monoethanolamide sulphosuccinate	Rewo
Loramine U 185	Undecylenamide monoethanolamide	Rewo
Luviset CE 5055	Vinyl acetate/crotonic acid copolymer	BASF
Marinol	Alkyldimethylbenzylammonium chloride	Berk
Maypon 4C	Potassium cocylhydrolysed animal protein	Stepan
Merquat-550	Polymeric quaternary ammonium salt consisting of acrylamide and dimethyldiallyl ammonium chloride monomers *Quaternium-41*	Merck
Merquat resins	Dialkyldimethyl ammonium chloride cyclopolymers	Merck
Methocel	*Hydroxypropylmethylcellulose*	Dow
Methofas	Methylcellulose	ICI
Microdry	Aluminium chlorhydrate fine powder *Aluminum Chlorohydrate*	Reheis
Miranol C2M, C2M-SF	A long-chain imidazoline type of zwitterion *Amphoteric-2*	Miranol
Miranol SM	Capryl imidazoline derivative	Miranol
Mirapol A15	Polyquaternary ammonium compound	Miranol
Modulan	*Acetylated Lanolin*	Amerchol
Morpan CHSA	Cetyltrimethylammonium bromide	Glovers
Myacide SP	2,4-Dichlorobenzyl alcohol	Boots
Myrj 45	Polyoxyethylene monostearate *PEG-8 Stearate*	ICI
Myrj 52	Polyoxyethylene monostearate *PEG-40 Stearate*	ICI
Myverol 18-17	Molecularly distilled monoglyceride	Eastman

Proprietary name	Chemical description	Supplier
Nacconal NRSF	Alkylaryl sulphonate	Allied Chemical
Natrosol	*Hydroxyethylcellulose*	Hercules
Neo-fat 18-55	*Stearic Acid*	Armak
Neo-PCL, water-soluble	Ethoxylated alkyl phenol and polyethylene glycol ester of 2-ethylhexanoic acid *Nonoxynol-14* and *PEG-4 Octanoate*	Dragoco
Neosyl	Finely divided silica	Crosfield
Neutronyx 600	Ethoxylated alkyl phenol *Nonoxynol-9*	Onyx
Nimlesterol D	*Mineral Oil* and *Lanolin Alcohol*	Malmstrom
Ninol 2012	Fatty acid alkanolamine concentrate	Stepan
Nipagin M	Methyl ester of *p*-hydroxybenzoic acid *Methylparaben*	Nipa
Nipagin P		Nipa
Nipasol M	Propyl-*p*-hydroxybenzoate *Propylparaben*	Nipa
Nonic 218	Polyethylene glycol *tert*-dodecyl thioether	Sharples Chem.
Novol	*Oleyl Alcohol*	Croda
Oat-Pro	*OatFlour*	Quaker Oats
Octaphen	*p-tert*-Octylphenoxyethoxyethyl dimethylbenzylammonium chloride	Ward Blenkinsop
Omadine	1-Hydroxypyridine-2-thione	Olin Mathieson
Omamids	Polyamide resins	Olin Mathieson
Onamer	Poly(dimethylbutenylammonium chloride)-α, ω-*bis*(triethanolamine chloride)	Onyx
Oracid	Urea-formaldehyde foam	Chemische Fabrik Frankenthal
Orvus WA	*Sodium Lauryl Sulfate*	Procter & Gamble
Ottasept extra	*Chloroxylenol*	Ottawa Chemical
PCL Liquid	*Cetearyl Octanoate*	Dragoco
Phemerol	*p-tert*-Octylphenoxyethoxyethyl dimethylbenzylammonium chloride	Parke-Davis
Phenonip	Combination of parabens and phenoxyethanol	Nipa
Pluronic F-127	A polyoxyethylene–polyoxypropylene block polymer *Poloxamer-407*	BASF-Wyandotte

Proprietary name	Chemical description	Supplier
Pluronic L64D	A polyoxyethylene–polyoxypropylene block polymer *Poloxamer-184*	BASF-Wyandotte
Polawax	Nonionic emulsifying wax	Croda
Polawax A.31	Blend of cetyl stearyl alcohol and EO condensate products	Croda
Polychol 5	Polyethylene glycol condensate of wool wax alcohols *Laneth-5*	Croda
Polychol 15	Polyethylene glycol ester of lanolin alcohol with an average ethoxylation value of 15 *Laneth-15*	Croda
Polyglycol 400	Polyethylene glycol 400	Hoechst
Polymer JR	Cationic cellulose ether derivative *Quaternium-19*	Union Carbide
Povidone-Iodine	*PVP-iodine*	Berk
Procetyl AWS	Polyoxypropylene, polyoxyethylene ether of cetyl alcohol *PPG-5-Ceteth-20*	Croda
Promulgen D	Cetylstearyl alcohol and its polyethoxylated (20) derivative *Cetearyl Alcohol* and *Ceteareth-20*	Robinson-Wagner
Prosolal S8	Octyl cinnamate sunscreen	Dragoco
Prosolal 58	Mixture of phenylacrylic esters and oxybenzoic acid esters	Dragoco
Protolate WS	PEG-75-lanolin and hydrolysed animal protein *PEG-75 Lanolin oil*	Malmstrom
PVP-VA E-735	*PVP/VA Copolymer*	GAF
Quadramer		American Cyanamid
Renex	Polyoxyethylene esters of mixed resin and fatty acids	ICI
Resyn 28-1310	*Vinyl Acetate/Crotonic Acid Copolymer*	National Starch
Resyn 28-2930	*Vinyl Acetate/Crotonic Acid/Vinyl Neodecanoate Polymer*	National Starch
Rewopol SBFA 30	Disodium lauryl alcohol polyglycol ether sulphosuccinate	Rewo
Roccal	Alkyldimethylbenzylammonium chloride *Benzalkonium Chloride*	Bayer

Proprietary name	Chemical description	Supplier
Sandopan DTC acid	α-(Carboxymethyl)-ω-(tridecyloxy) poly (oxy-1,2-ethanediyl) *Trideceth-7-Carboxylic Acid*	Sandoz
Sandopan TFL	Sulphoamidobetaine *Amphoteric-7*	Sandoz
Santicizer 8	Sulphonamide plasticizer	Monsanto
Santicizer 160	*Butyl Benzyl Phthalate*	Monsanto
Santocel 54	Hydrated silica	Monsanto
Santolite MHP	Toluenesulphonamide/formaldehyde resin	Monsanto
Santolite MS 80%	Toluenesulphonamide/formaldehyde resin	Monsanto
Schercoquat	Quaternized fatty acid amides derived from isostearic acid	Scher Chemicals
Silicone fluid DC-556	Polyphenylmethyl siloxane *Phenyl Dimethicone*	Dow Corning
Silicone fluid L-45	Dimethyl silicone *Dimethicone*	Union Carbide
Sodium silicate 'O'	*Sodium Silicate*	Philadelphia Quartz
Softigen 767	Ethoxylated partial glyceride fatty acid ester *PEG-6 Caprylic/Capric Glycerides*	Dynamit-Nobel
Solprotex	Digalloyl trioleate	Firmenich
Solulan 98	Acetylated polyoxyethylene (10) lanolin alcohol *Laneth-10 Acetate*	Amerchol
Sorbo	*Sorbitol*	ICI
Span 20	Sorbitan monolaurate *Sorbitan Laurate*	ICI
Span 60	Sorbitan monostearate *Sorbitan Stearate*	ICI
Span 80	Sorbitan monooleate *Sorbitan Oleate*	ICI
Span 85	*Sorbitan Trioleate*	ICI
Standapol OLP	Oleyl betaine	Henkel
Steinapon AM-B13	Alkylamido betaine	Goldschmidt
Stepanhold R-1	*PVP/Ethyl Methacrylate/Methacrylic Acid Polymer*	Stepan Chemical
Sunscreen 3573	Oil-soluble suncreen	Merck
Surfynol 82	*Dimethyl Octynediol*	Air Products
Syloid 72	*Hydrated Silica*	Grace
Syncrowax ERLC	*C18-C36 Acid Glycol Esters*	Croda
Syncrowax HGLC	*C18-C36 Acid Triglyceride*	Croda

Proprietary name	Chemical description	Supplier
Syncrowax HRC	*Glyceryl Tribehenate*	Croda
Syncrowax PRLC	Propylene glycol ester of mixed fatty/wax acids	Croda
TBS	3,4′,5-Tribromosalicylanilide	Theodore St. Just
TCC	3,4,4′-Trichlorocarbanilide *Triclocarban*	Monsanto
TCS	2,3,3′, 5-Tetrachlorosalicylanilide	Ciba-Geigy
Tegin 515	Glyceryl monostearate *Glyceryl Stearate*	Goldschmidt
Tego 103S		Goldschmidt
Tegobetaine L7	Cocamidopropyl betaine	Goldschmidt
Temasept IV	3,4′, 5-Tribromosalicylanilide	Fine Organics
Tergitol NPX	Alkylaryl polyethylene glycol ether *Nonoxynol-10*	Union Carbide
Texapon Extract N25	Sodium lauryl ether sulphate *Sodium Laureth Sulfate*	Henkel
Timica	Mica coated with titanium dioxide	Mearl
Tinuvin P	UV-absorber	Ciba-Geigy
Tiona G	Titanium dioxide, oil-dispersible	Laporte
Topanol O	Butylated hydroxytoluene, i.e. 2,6-di-*tert*-butyl-4-methylphenol *BHT*	ICI
Topanol OC	Purified grade of Topanol O	ICI
Triton X-100	Alkylated aryl polyether alcohol *Octoxynol-9*	Rohm and Haas
Triton X-200	Sodium salt of alkylated aryl polyether sulphate *Sodium Octoxynol-3 Sulfonate*	Rohm and Haas
Triton X-400	Stearyldimethylbenzylammonium chloride *Stearalkonium Chloride*	Rohm and Haas
Tuasal 100	3,4′, 5-Tribromosalicylanilide	Dow
Tween 20	Polyoxyethylene sorbitan monolaurate *Polysorbate-20*	ICI
Tween 40	Polyoxyethylene sorbitan monopalmitate *Polysorbate-40*	ICI
Tween 60	Polyoxyethylene sorbitan monostearate *Polysorbate-60*	ICI
Tween 65	Polyoxyethylene sorbitan tristearate *Polysorbate-65*	ICI

Proprietary name	Chemical description	Supplier
Tween 80	Polyoxyethylene sorbitan monooleate *Polysorbate-80*	ICI
Tylose	Sodium carboxymethylcellulose	Hoechst
Ucon LB-1715	Polypropylene glycol butyl ether *PPG-40 Butyl Ether*	Union Carbide
Ucon 50-HB-660	Polyoxypropylene, polyoxyethylene monobutyl ether *PPG-12-Buteth-16*	Union Carbide
Ultrawet 60L	Triethanolamine salt of alkylaryl sulphonate *TEA-Dodecylbenzene Sulfonate*	ARCO
Uvinul D-50	2,2',4,4'-Tetrahydroxybenzophenone *Benzophenone-2*	GAF
Uvistat	2-Hydroxy-4-methoxy-4'-methyl benzophenone	Ward Blenkinsop
Vancide 89RE	N-Trichloromethylthio-4-cyclohexene-1,2-dicarboximide *Captan*	Vanderbilt
Vancide BL	2,2'-Thiobis-(4,6-dichlorophenol)	Vanderbilt
Vantoc AL	Alkyltrimethylammonium bromide	ICI
Vantoc B	Tetradecylpyridinium bromide	ICI
Vantoc CL	Alkyldimethylbenzylammonium chloride	ICI
Veegum	*Magnesium Aluminum Silicate*	Vanderbilt
Veegum HV	*Magnesium Aluminum Silicate*	Vanderbilt
Veegum K	*Magnesium Aluminum Silicate*	Vanderbilt
VEM resin	*PVP/Ethyl Methacrylate/Methacrylic Acid Polymer*	Barr-Stalfort
Versamids	Polyamide resins	General Mills
Versene	Ethylenediamine tetraacetic acid *EDTA*	Dow
Virac	Iodophor (i.e. mixture of iodine with surface-active agent)	Ruson Laboratories
Volatile Silicone 7158, 7207	Cyclic dimethyl polysiloxane compound *Cyclomethicone*	Union Carbide
Volpo N3	Oleyl alcohol/ethylene oxide condensate	Croda
Volpo N5	Polyoxyethylene oleyl ether	Croda

Proprietary name	**Chemical description**	**Supplier**
Volpo S10	Polyoxyethylene (10) stearyl ether *Steareth-10*	Croda
Volpo S20	Polyoxyethylene (20) stearyl ether *Steareth-20*	Croda
Wescodyne	Iodophor (i.e. mixture of iodine with surface-active agent)	Bebgue
Zephiran	Alkyldimethylbenzylammonium chlorice	Bayer
Zephirol	Alkyldimethylbenzylammonium chloride	Bayer
Zetesol 856T	Alkyl ether sulphate	Zschimmer & Schwarz
78-4329 (now named Celquat)	Cationic polymer grafted on a cellulosic chain	National Starch

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