COSMETICS AND DERMATITIS

By E. J. MOYNAHAN, M.R.C.P.*

MANKIND HAS used cosmetics since early palaeolithic times, and although we cannot be certain that the connection between health and beauty dates back so far, we know that there has been a close relationship between medicine and the cosmetic art for a long time. It is of some interest that, among the earliest medical writings, we find several cosmetic recipes in the Ebers Papyrus, and many references to such preparations are to be found in the works of the early alchemists and herbalists. Until recently medicine has approached the problem of dermatitis empirically, and it is only within the present century that the scientific foundations for its study have been laid. It had long been known that cosmetics produce reactions in the skin, but there was no scientific proof of this until Kesten and Lazlo showed, in 1931, that nineteen out of twenty-one cases of dermatitis, due to cosmetics, gave positive patch tests to one or more ingredients in the preparation used. We owe a great deal to Sulzberger and his colleagues, who performed no fewer than ten thousand tests on a thousand subjects, and threw considerable light on the commoner causes of trouble with cosmetics.

Very few primary irritants are used in modern cosmetics: these include permanent waving agents, which attack the keratin fibres and break the disulphide linkages and other chemical bonds between adjacent keratin molecules; depilatories, with a closely related chemical action; high concentrations of alkali, which reduce the sulphur in the disulphide links and rupture them in the same way and the oxidation hair dyes, such as paraphenylenediamine. Most cases of dermatitis produced by cosmetics are due to sensitisation to some substance in the preparation. It should be emphasised that such cases are extremely few, considering the large number of people who use cosmetics daily, year in, year out, without any ill effect on their skin. It speaks well for those engaged in the production and manufacture of these preparations that so little trouble follows their use. We doctors have not got such good records in this respect, because a fair number of the preparations we use in the treatment of skin disease produce dermatitis, and some of the newer chemotherapeutic agents, such as sulphonamides and antibiotics (par-

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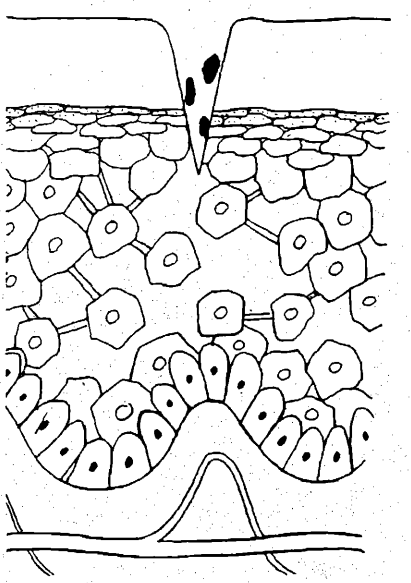
ticularly penicillin), are notorious for the ease with which they produce sensitisation after local application to the skin.

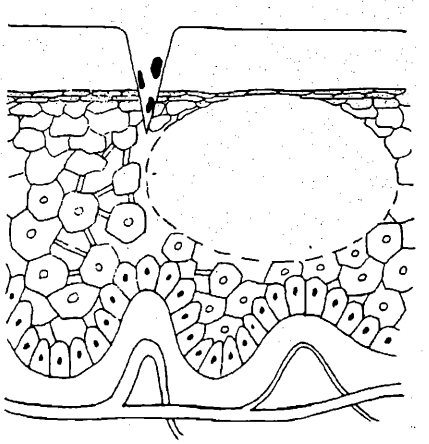
It is important, in view of what follows, to distinguish between a primary irritant and a sensitising agent. Primary irritants are substances which, when applied in sufficient concentration to the skin of a normal individual, will provoke an inflammatory reaction. A sensitising agent, on the other hand, can be applied with impunity to the skin of most individuals without ill effect, but on the skin of a susceptible person, will give rise to a dermatitis. Before going into details it would be useful here to give a brief account of the microscopic structure of the skin.

The skin is composed of two parts, the epidermis and the dermis. The epidermis arises from the basal layer from which all the overlying layers are derived. This layer rests on the dermis, from which it is separated by a very thin basement membrane. It is anchored to the dermis by certain processes which project from inside the basal cells. These are fibrous in nature but their exact composition remains unknown. Above the basal layer we find several layers of cells known as prickle cells because of the presence of interconnecting fibrils between adjacent cells. These fibrils appear to run through several cells and skirt the nucleus of each. They help to keep the cells together and some authorities believe that they are the precursors of keratin. As we ascend the prickle layer towards the surface of the skin, the cells tend to flatten out and granules accumulate in their cytoplasm. This layer, which may be two or three cells thick, is known as the granular layer, but is not present everywhere in the skin. It is absent, for example, in the eyelids. In certain parts of the skin, such as the palms and soles, there is another layer to be found in the epidermis between the granular layer and the horny layer. The cells composing this layer contain clear granules, and it is known as the stratum lucidum. The horny layer is composed of flat squames with a fibrous protein, keratin, forming a shell or rind round an inner kernel of lipids. The presence of an intact horny layer ensures protection against chemical, mechanical, radiation and other injuries and provides also a defence against bacterial and fungal attack. The sebum, secreted by the sebaceous glands which open into the hair follicles, also helps to protect the underlying living cells against these irritants.

The dermis itself contains the nerves, blood vessels and supporting tissues. The vessels in the upper, or papillary, part of the dermis play a prominent part in the inflammatory response of the skin which we know as dermatitis. Thus, when an irritant penetrates the protective keratin or horny layer, the living cells are irritated and a reaction is seen in the skin which varies from redness (due to dilatation of these vessels), with associated subjective itching, to gross swelling and even the produc-

tion of large blisters in the epidermis. If the irritant is powerful enough, such as a strong acid or alkali, actual death or necrosis of the cells will occur. Because of the predominantly fatty nature of the horny layer and the supernatant sebum, oils and oily emulsions are more likely to penetrate to the deeper layers and thereby give rise to dermatitis. This is why the essential oils of some plants are irritants. On the other hand, aqueous solutions are less likely to give rise to trouble in view of their immiscibility with oil. As a consequence, they are unable to gain access to the living cells in the intact skin. Substances with surface-active properties, such as triethanolamine, sodium lauryl sulphate and the like, by reducing surface tension enable water-soluble agents to penetrate deeper in the skin when they are incorporated in various vehicles. It should be remembered that, if a high concentration of such a wetting agent is incorporated in a preparation, this may lead to an undesirable concentration of the active constituent at or below the prickle layer and thereby give rise to a dermatitis, whereas the same agent, incorporated in a simpler aqueous vehicle, might not penetrate so deep and no trouble would ensue.





The Mechanism of Sensitisation. Figure 1 (above) *represents the entry of chromic acid through an abrasion in the skin. Note oedema and rupture of the intercellular processes in prickle layer. Figure* 2 (below) : *The process further advanced, showing blister formation. See also figures* 5, 6, *and* 7.

CASES OF COSMETIC DERMATITIS

Most of my audience must be familiar with a typical hair-dye dermatitis. The patient, nearly

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Figure 3 (above) *and Figure 4* (opposite page) *illustrate an unusual case of allergic reaction to a face cream, which happened to be a water-in-oil emulsion of the lanolin derivative type. The patient did not himself use the cream : it was his wife's. The illustration above shows the development of dermatitis on the side of the cheek.*

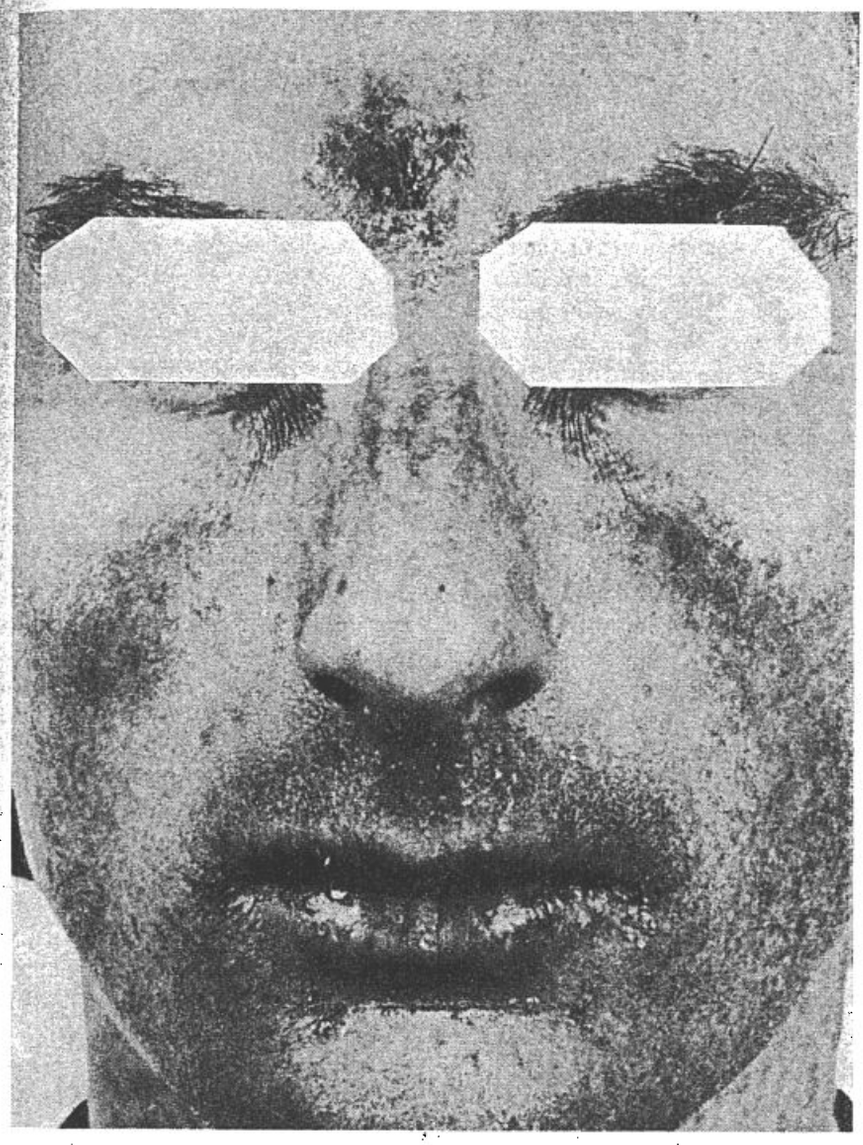

Figure 4. Simple contact dermatitis around the lips. See also opposite page.

always a middle-aged, grey-haired woman (but men get it too !) decides to undo the ravages of time and, ignoring the instructions on the bottle of hair-dye, or reading them in a cursory manner, dabs a little

of the dye behind her ear and asks her husband to inspect the result a few minutes later. As no reaction will be visible after this short time, she assumes that all is well and proceeds to dye her hair. She retires to bed, satisfied with her operation, but, in the early hours of the morning, she wakes feeling as if her scalp was on fire. She is further alarmed by the fact that she can scarcely open her eyes, due to the considerable swelling of her eyelids. After an anxious night she then presents herself to the nearest skin clinic, where the dermatologist can usually make the diagnosis as he passes her by.

Similar reactions follow exposure to other agents and the site of the eruption may vary with the part of the skin which comes in contact with the offending substance. It should be remembered that thick-skinned areas are less likely to show inflammatory changes than are thin-skinned areas, such as the eyelids. It is quite a frequent experience, for example, to find a patient, sensitive to the dye in nail lacquer preparations, who never shows skin changes in the hands at all. The eruption is usually seen on the thinner skin of the eyelids, which may become quite swollen as a result of contact with the dye, brought about, for example, by the nervous habit of tapping or rubbing the lids with the fingers.

Some primary irritants may in their turn become sensitising agents. A common example of this is seen in industry with chromium. Chrome ulcers or "chrome holes" are usually due to small amounts of chromic acid or other chromium salts which reach the living cells of the prickle layer through a small abrasion or incised wound in a worker's hand. The presence of these salts produces a local painless ulceration of the skin which slowly enlarges unless the lesion is treated. Some individuals, however, subsequently develop a rash composed of vesicles which are intensely irritating, in the neighbourhood of and remote from the original chrome ulcer. They have become sensitised to chromium and then, wherever chromium comes into contact with their skin, they develop an irritating dermatitis.

The mechanism involved in sensitisation is not fully understood, but it would seem that many substances have the property of forming loose complexes with the proteins in the skin of susceptible subjects. This protein complex acts as an antigen. Antigens are substances which in some way disturb protein synthesis in the cell, so that antibodies are produced instead of normal proteins. Haurowitz mentions that the surface of antibody molecules is adapted complementarily to the determinant molecular groups on the surface of the antigen molecule. These appear to be strongly polar groups consisting mainly of negatively charged acid groups. Such groups must possess a definite shape in order to cause sensitisation, thus Landsteiner has shown that the flexible paraffin chain of a fatty acid cannot act as an antigen. Furthermore, there is always a latent period between the

first contact with the antigen and the development of sensitisation, during which further application of the antigen to the skin will produce no obvious reaction. This period is round about ten days and may be shorter or longer.

We are unable, unfortunately, to determine in advance which substances are likely to produce sensitisation, and except for the azo dyes and the amine intermediate dyes, we are not able to correlate sensitising properties with chemical structures. It is therefore necessary to try out a new preparation by patch testing several hundred normal people before one can determine its sensitising powers. In this respect it is worth remembering that, if a substance produces a skin reaction in at least one in two hundred normal subjects, its commercial development should not be proceeded with until a further test on two hundred more subjects has shown that the risk of sensitisation is less than 0·5 per cent. Should this percentage be exceeded, a careful review of the composition of the cosmetic should be undertaken, to see which ingredient is responsible for sensitisation, and this should be excluded. Prophetic patch tests are undesirable in industry, however, even though they have been popular in the United States. Patch tests intended to be carried out by the user at home are unlikely to be done properly, and in any case patch tests require skill and experience in their interpretation.

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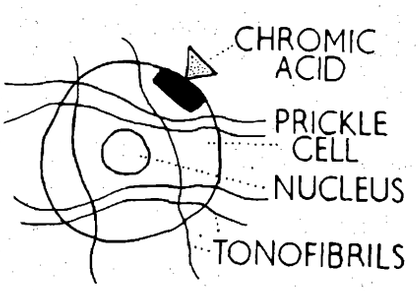

Figure 5. Theory of sensitisation mechanism. Chromic acid in contact with skin protein on surface of a prickle cell.

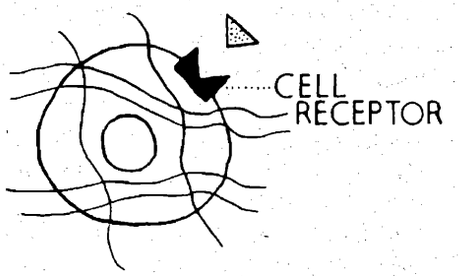

Figure 6 (above) : An antibody has been produced at cell surface. Note diagrammatic representation of "complementary fit." Figure 7 (below) : Antibody has united with antigen (chromic acid) leading to release of histamine and other substances.

Reviewing Cosmetics as a Class

Cosmetics as a class are relatively harmless. Most of them are quite innocuous when applied to the skin, as mentioned above, and in practice are responsible for very few cases of dermatitis.

Occupational dermatitis due to cosmetics is very rare; the chief reason for this freedom from occupational risk is probably to be found in the preponderance of automatic methods used in mixing, filling and packing the various preparations.

As far as the user is concerned, face creams very rarely give trouble except when irritant perfume materials have been incorporated in them; the same is true of face powders. Permanent waving agents give most trouble. This is due to the fact that they are potent keratolytic agents and therefore tend to damage the horny layer. The oxidation hair dyes are also liable to give trouble, because of their sensitising properties. Allergic dermatitis produced by hair dyes is dramatic in its onset and character, as the case outlined above shows, but it is usually, and fortunately, easy to treat. Lipstick dermatitis is often provoked by exposure to strong sunlight, either by the seaside or in the high mountains, and is due to the eosin or similar agent incorporated to give the colour to the lipstick. These substances have photochemical properties and sensitise the individual to the dye in the presence of light. They may also, of course, produce dermatitis without the aid of light. Another photodynamic substance used in cosmetics is bergamot oil, and this gives rise to a fairly characteristic brown pigmentation in areas exposed to sunlight. Nail lacquer dermatitis is usually met with on the thinly keratinised skin around the eyes and on the chin, but rarely on the fingers.

Several constituents of plastics are known to be sensitising agents and some of the nail cosmetics produce brittleness in the nails with splitting of their free margins. It should, however, be borne in mind that the commoner cause of nail brittleness is the injudicious use of certain of the newer detergents by the housewife rather than, as is popularly supposed, the nail varnish solvents; although the latter do sometimes give rise to trouble. Rouge is quite innocuous in character and very rarely provokes signs of dermatitis. Depilatories, on the other hand, are likely, by their very nature, to give rise to trouble if they are not used carefully. The keratin in hair is tougher than that of the horny layer of the skin, and as the depilatory has to destroy this keratin, it is likely to damage the horny layer if it is allowed to remain too long in contact with the skin. (In clinical practice I prefer to use electrolysis for depilation, but this is impractical for axillary hair in women.) An unusual type of dermatitis may follow the use of brilliantine. This consists of acne-like lesions, commonly seen on the forehead of the user and is due to the hydrocarbons incorporated in the brilliantine. It

seems to be a commoner condition in France than in this country and it may well be due to some difference in the composition of the brilliantine in the two countries.

DETERGENTS AND DISINFECTANTS

I have already referred to the injudicious use of some of the newer detergents. It should here be mentioned that most of the anionic detergents used in cosmetics are satisfactory in the proportions usually employed, but much trouble has been caused by other detergents and wetting agents, used in the washing and treating of fabrics, and this is probably due to the fact that they all tend to remove the protective sebum from the surface of the skin, and some have a high *p*H which renders the keratin fibre more vulnerable to chemical attack.

Some mention of barrier creams would not be out of place here. On the whole they are disappointing, because they cannot replace entirely the keratin and lipid of the skin. They are, however, better than nothing and they help to protect the skin from potential irritants, which are then removed by ordinary washing when the wearer finishes work for the day. In this way they limit the duration of contact of the irritant with the worker's skin and reduce the risk of dermatitis. Unfortunately there is no really effective barrier cream available for wet work, and the production of one would be a great boon to housewives, who suffer from alkali dermatitis, as well as to the industrial worker. Housewife's dermatitis is probably the commonest occupational dermatosis.

I would like to make some remarks about the incorporation of disinfectants in cosmetic preparations. Unless there is a very good reason, disinfectants should not be used in cosmetics, and in any case their concentration should be kept as low as possible. We are meeting more and more cases of dermatitis due to disinfectants of the chloroxylenol group, as their use becomes more widespread. They commonly give rise to itching when used indiscriminately in baths, and may produce extensive dermatitis, e.g., in nurses who "scrub up" with these disinfectants many times each day. On the whole disinfectants should be kept off the skin, and reliance should be placed on the detergent properties of soaps and the like to remove the bacteria. With regard to soaps, a known factor in the production of dermatitis is the rosin which is incorporated in some of the household soaps. This substance is widely used and one may find that the patient who is sensitive to rosin is also sensitive to such things as adhesive plasters, which may also contain a fair amount of rosin. It must be admitted, however, that soaps give less trouble as sensitising agents than as occasional primary irritants.

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