

Regional variations in contact sensitization

The main prerequisites for allergic contact sensitization are (a) a genetically susceptible individual,¹ (b) a hapten absorbed through the skin in an appropriate concentration, and (c) normally functioning Langerhans cells in the epidermis.

See also the paper by Jaworsky et al (pp 443–444)

The concentration-dependent sensitization risk for strong allergens is well-established.^{2,3} Any physiological factor that tends to increase the absorption of chemicals through the skin increases the risk of allergic contact sensitization. There can be great variation in absorption depending on the anatomical site. The penetration rate for hydrocortisone on the scrotum, for example, is 100 times greater than the penetration rate on the soles of the feet.⁴ This point is also well illustrated in leg ulcer patients where the skin is exposed to topical medicaments under occlusion for prolonged periods. Contact sensitization, even to weak allergens such as lanolin, parabens, and neomycin, is frequent in this particular group of patients compared with patients who have dermatitis elsewhere.⁵ Leg ulcer patients sensitized to parabens often tolerate cosmetic products preserved with parabens when these are applied to normal skin.⁶

Sensitization to occupational contact allergens such as epoxy resin and chromate typically occurs on the hands and may spread to the face and other areas. In epidemiological studies, it is important to recognize that contact allergy to specific haptens is related to the area of skin involved and is either due to the exposure pattern or to

physiological variations in the skin in different regions of the body.

In order to make it possible to compare patch test studies, Wilkinson et al⁵ introduced the MOHL index where M = percentage of males, O = percentage of occupational cases, H = percentage with hand eczema, and L = percentage of patients with leg ulcer or stasis eczema. Patch test materials from different clinics should be classified according to this index before any comparisons are made among them. Modern computer technology allows more variables to be included in such analysis.⁷

Reactivity to diagnostic patch testing differs greatly according to the anatomical site of the test. Since skin response on the back is more pronounced than reactions seen on the arms and thighs, the upper back is recommended for routine diagnostic patch testing.⁸

It is essential both for primary sensitization and for the elicitation of contact allergy that there are normally functioning Langerhans cells in the epidermis.⁹ Berman et al¹⁰ investigated the densities of T6 antigen-bearing Langerhans cells in human skin. The number of Langerhans cells was low in the soles compared to other areas such as the face, chest, back, and extremities. Ashworth et al¹¹ confirmed this finding and further established that the density of Langerhans cells was equally low in the palms and soles, compared to other areas.

Glutaraldehyde in a 25% concentration is a popular remedy for treating hyperhidrosis, plantar warts, and other skin diseases on the soles of the feet. Contact dermatitis from the use of this high concentration of glutaraldehyde on the soles is rare. Maibach and Prystowsky¹² performed usage tests with glutaraldehyde on soles and antecubital fossa of six previously documented glu-

taraldehyde-sensitive subjects. All had negative usage tests to 25% glutaraldehyde on the soles, but reacted to a concentration of 2.5% glutaraldehyde applied to the antecubital fossa. The strong binding of glutaraldehyde to keratin probably minimizes absorption in the soles. But the small number of Langerhans cells in this area suggests another explanation for the clinical observations made.

In this issue, Jaworsky et al¹³ describe the first case of glutaraldehyde sensitization due to a cosmetic product. Allergic contact dermatitis from hair cosmetics is due to dyes, hair bleaches, permanent waving chemicals, preservatives, antidandruff agents, perfumes, and, rarely, detergents. Such cases are easily overlooked because of low clinical suspicion as dermatitis from hair cosmetics frequently spares the scalp, but involves the face and neck.¹⁴⁻¹⁶ The fact that up to 1% concentrations of glutaraldehyde are used in 74 different cosmetic products is entirely new information for most dermatologists. In Europe, where preservatives are not listed on labels, it would have been difficult to establish the correct diagnosis, as there would have been no suspicion of such sensitization.

Glutaraldehyde must now be regarded as one of the ubiquitous allergens. Only by employing routine patch testing on a large number of patients with contact dermatitis can the numerical significance of the problem be established.

TORKIL MENNÉ, M.D., PH.D.
Consultant Dermatologist
Department of Dermatology
Gentofte Hospital
DK-2900 Hellerup
DENMARK

References

1. Menné T, Holm NV. Genetic susceptibility in human allergic contact sensitization. *Semin Dermatol* 1986; **5**:301-306.
2. Marzulli FN, Maibach HI. The use of graded concentrations in studying skin sensitizers. Experimental contact sensitization in man. *Food Cosmet Toxicol* 1974; **12**:219-227.
3. Cardin CW, Weaver JE, Bailey PT. Dose-response assessments of Kathon biocide. II. Threshold prophetic patch testing. *Contact Dermatitis* 1986; **15**:10-16.
4. Feldmann RJ, Maibach HI. Regional variations in percutaneous penetration of ¹⁴C cortisol in man. *J Invest Dermatol* 1967; **48**:181-183.
5. Wilkinson JD, Hambly EM, Wilkinson DS. Comparison of patch test results in two adjacent areas of England. II. Medicaments. *Acta Derm Venereol (Stockh)* 1980; **60**:245-249.
6. Fisher AA. The paraben paradoxes. *Cutis* 1973; **12**:830-832.
7. Edman B. Sites of contact dermatitis in relation to particular allergens. *Contact Dermatitis* 1985; **13**:129-135.
8. Magnusson B, Hersle K. Patch test methods. II. Regional variations of patch test responses. *Acta Derm Venereol (Stockh)* 1965; **45**:257-261.
9. Choi KL, Sauder DN. The role of Langerhans cells and keratinocytes in epidermal immunity. *J Leukocyte Biol* 1986; **39**:343-358.
10. Berman B, Chen VL, France DS, Dotz WI, Petroni G. Anatomical mapping of epidermal Langerhans cell densities in adults. *Br J Dermatol* 1983; **109**:553-558.
11. Ashworth J, Turbitt ML, Mackie R. The distribution and quantification of the Langerhans cell in normal human epidermis. *Clin Exp Dermatol* 1986; **11**:153-158.
12. Maibach HI, Prystowsky SD. Glutaraldehyde (pentanedial) allergic contact dermatitis. Usage test on sole and antecubital fossa: regional variations in response. *Arch Dermatol* 1977; **113**:170-171.
13. Jaworsky C, Taylor JS, Evey P, Handel D. Allergic contact dermatitis to glutaraldehyde in a hair conditioner. *Cleve Clin J Med* 1987; **54**:000-000.
14. Storrs FJ. Permanent wave contact dermatitis: contact allergy to glyceryl monothioglycolate. *J Am Acad Dermatol* 1984; **11**:74-85.
15. Andersen KE, Roed-Petersen J, Kamp P. Contact allergy related to TEA-PEG-3 cocamide sulfate and cocamidopropyl betaine in a shampoo. *Contact Dermatitis* 1984; **11**:192-193.
16. Brandrup F, Menné T. Zinc pyrithione (zinc omadine) allergy. *Contact Dermatitis* 1985; **12**:50.