causative agent probably determine the abnormalities found. Histologic and immunologic studies are necessary to explain the clinical differences between primary and secondary syphilis.

REFERENCES

1. Druelle M. Le chancre du cuir chevelu. Thèse, Lyon, 1923.
2. Pareek S S. Syphilitic alopecia in Jarisch-Herxheimer reaction. Br J Vener Dis 1971; 53: 389–390.
3. Peereboom-Wynia J D R. Hair root characteristics of the human scalp hair in health and disease. Thesis, Rotterdam, 1982.
4. Witzel M A, Braun-Falco O. Über den Haarwurzel Status am menschlichen Capillitum unter physiologischen Bedingungen. Arch Klin Exp Dermatol 1963; 216: 221.
5. Zaun H. Diffuses Effluvium kausal Diagnose als Grundlage der Therapie. Akt Dermatol 1975; 1: 119.
6. Braun-Falco O, Fisher C H. Über den Einfluss des Haarwachsens auf das Haarwurzelmuster. Arch Klin Exp Dermatol 1966; 226: 136.
7. Jeerapaet P, Ackerman A B. Histologic patterns of secondary syphilis. Arch Dermatol 1973; 107: 373–377.

Basal Keratinocyte Herniation

LASSE KANERVA

Section of Dermatology, Institute of Occupational Health, Helsinki, Finland

Kanerva L. Basal keratinocyte herniation. Acta Derm Venereol (Stockh) 1987; 67: 254–257.

Basal cell herniations, i.e. basal keratinocyte processes protruding through basal lamina gaps were observed in psoriasis, circinate balanitis, pityriasis rubra pilaris, patch tests, gold dermatitis and conjunctivitis. This indicates that they are not specific to psoriasis and tumours as has been reported. *Key words: Electron microscopy; Allergic and irritant patch tests; Pityriasis rubra pilaris; Psoriasis; Circinate balanitis; Gold dermatitis; Conjunctivitis.* (Received October 20, 1986.)

L. Kanerva, Chief, Section of Dermatology, Institute of Occupational Health, Topeliuksenkatu 41 a A, SF-00250 Helsinki, Finland.

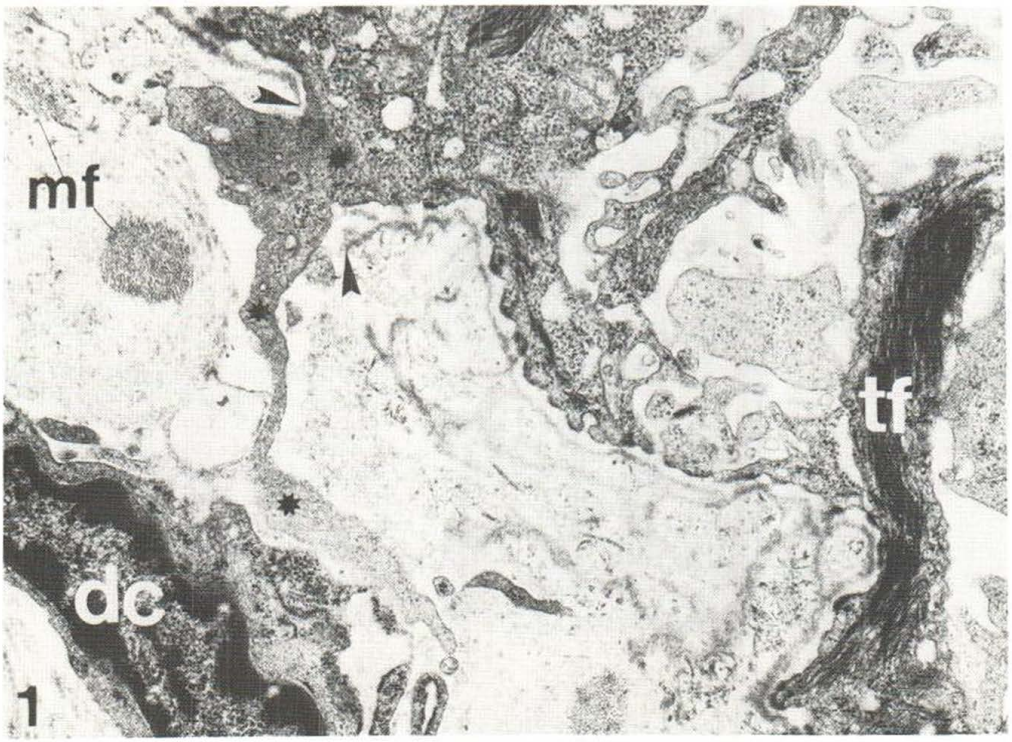
Basal keratinocyte processes protruding through basal lamina gaps have been found in psoriasis (1–3) in Darier's disease (4), and in epithelial tumours (5–9). According to Heng & Kloss (4) these basal cell herniations have not been described in other dermatitides. The purpose of the present report is to give further examples of basal cell herniation.

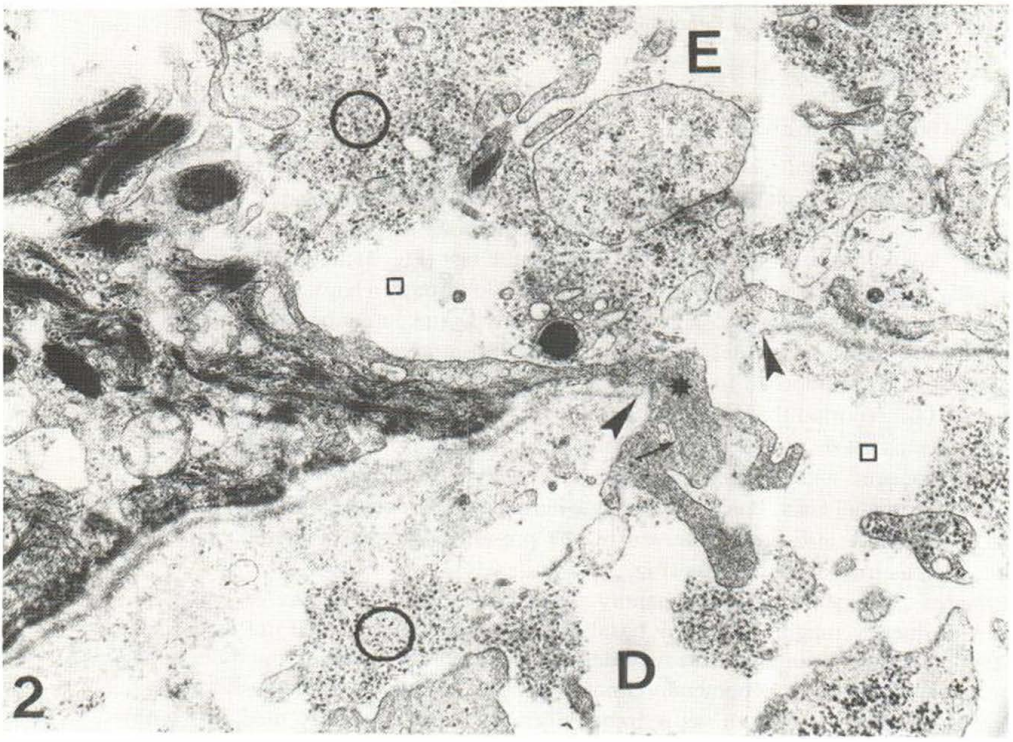
MATERIAL AND METHODS

Electron microscopy of a variety of skin diseases has been performed by the present author since 1979. For ultrahistopathology, 1 mm^3 or smaller blocks have been fixed in cacodylate- or phosphate-

Fig. 1. Pityriasis rubra pilaris. Basal cell process (asterisk) is seen to protrude through the basal lamina (between arrowheads). The distal part of the process is in close contact with a dermal cell (dc). *tf*, tonofilaments; *mf*, microfibrils. ×16600.

Fig. 2. Eczematous gold dermatitis reaction. Basal keratinocyte herniation (asterisk) through basal lamina (between arrowheads). Oedema □ and granular substance (inside ring beyond and below dermo-epidermal junction) is often observed in eczematous reactions. *D*, dermis; *E*, epidermis; →, microtubule. ×20000.


mf
tf
dc
1


E
D
2

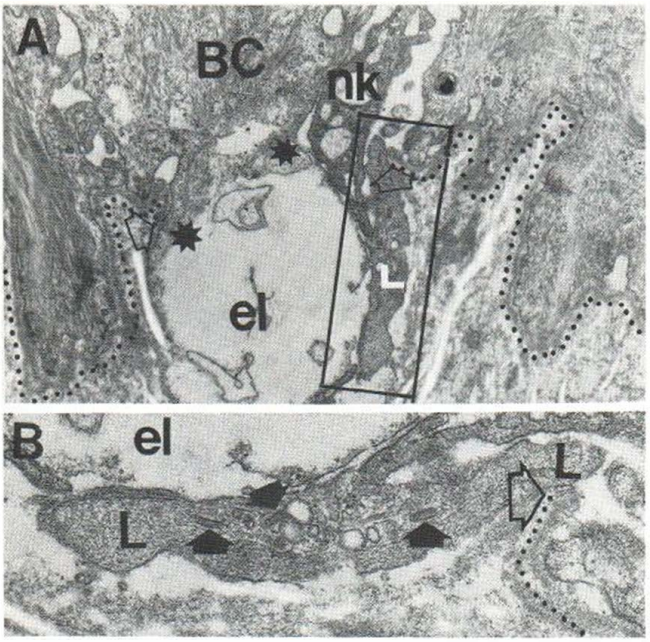


Fig. 3. Irritant patch test provoked by dithranol. Cytoplasmic process (*) from basal keratinocyte (*BC*) protruding into the dermis through gap in basal lamina (between arrows). A Langerhans cell (*L*) is crossing the basal lamina and is associated with electron lucent area (*el*). nk=non-keratinocyte associated with Langerhans cell in the gap of basal lamina. Inset in Fig. 3 A is seen at higher magnification in Fig. 3 B. Fig. 3 A, ×5900 Fig. 3 B, ×24000.

buffered (0.1 M, pH 7.2–7.3) 2.5% glutaraldehyde (osmolality about 530 mOsm/kg H^2O) at 4°C and postfixed with 1% osmium tetroxide and processed in routine manners as previously described (10). Irritant patch tests were provoked by dithranol (0.2% in pet.) as previously described (11).

RESULTS AND DISCUSSION

To date, the present author has observed basal cell herniation in psoriasis, circinate balanitis, pityriasis rubra pilaris (see ref. no. 10), allergic and irritant patch tests, gold dermatitis and conjunctivitis. Basal keratinocyte herniations as seen in pityriasis rubra pilaris (Fig. 1), gold dermatitis (Fig. 2) and irritant patch test (Fig. 3) are illustrated. Short to moderately long processes (Fig. 1) are seen to protrude through a basal lamina gap. The cytoplasmic processes possess the same electron density as the parent basal keratinocyte. Microtubuli were observed in the processes (Fig. 2). Basal keratinocyte herniation processes were seen to be apposed to dermal cells (Fig. 1).

Heng et al. (12) reported that basal cell herniations were often associated with electron lucent areas in psoriasis, suggesting protoeolytic autodigestion. These electron lucent areas were associated with Langerhans' cells, lymphocytes, neutrophils, dermal macrophages, and endothelial cells. Basal keratinocyte herniations associated with cells crossing the basal lamina have also been observed by the present author in other inflammatory disorders, such as irritant patch tests (Fig. 3).

No attempts have been made to quantify these herniations, but Heng & Kloss (4) reported a frequency ranging from 3–9 basal lamina herniations per 500 μm epidermal length. It is probable that in our cases the number was much lower, but on the other hand this report shows that this phenomenon is not unusual. The present author believes that when the basal lamina is broken, as is frequently seen in inflammatory disorders with invasion of leukocytes into the epidermis, keratinocyte herniation can occur. It has been suggested that these cytoplasmic processes may be a stimulus for cell proliferation (3), but

it might merely represent a mechanical sequence of basal lamina rupture without specific significance. In tumours it might represent an invasion mechanism in the preliminary stages of tumour growth (5–9).

REFERENCES

1. Brody I. The ultrastructure of the epidermis in psoriasis vulgaris as revealed by electron microscopy. I. The dermoepidermal junction and the stratum basale in parakeratosis without keratohyalin. J Ultrastruct Res 1962; 6: 304–323.
2. Cox AJ. The dermal-epidermal junction in psoriasis. J Invest Dermatol 1969; 53: 428–435.
3. Heng MCY, Kloss SG, Kuehn CS, Chase DG. Sequence of events in psoriatic plaque formation after tape-stripping: A light and electron microscopic study. Br J Dermatol 1985; 112: 517–532.
4. Heng MCY, Kloss SG. Basal keratinocyte herniations in Darier's disease. J Am Acad Dermatol 1985; 13: 307–310.
5. Luibel FJ, Sanders E, Ashworth CT. An electron microscopic study of carcinoma in situ and invasive carcinoma of the cervix uteri. Cancer Res 1960; 20: 356–361.
6. Tarin, D. Sequential electron microscopical study of experimental mouse skin carcinogenesis. Int J Cancer 1967; 2: 195–211.
7. Sugar, J. An electronmicroscopic study of early invasive growth in human skin tumors and laryngeal carcinoma. Eur J Cancer 1968; 4: 33–38.
8. Woods DA, Smith CJ. Ultrastructure of the dermoepidermal junction in experimentally induced tumors and human oral lesions. J Invest Dermatol 1969; 82: 259–263.
9. Schenk P, Konrad K. Zur Ultrastruktur der Tumor-Stromagrenze des invasiven Larynxkarzinoms. Laryng Rhinol 1979; 58: 575–582.
10. Kanerva L, Lauharanta J, Niemi K-M, Lassus A. Ultrastructure of pityriasis rubra pilaris with observations during retinoid (etretinate) treatment. Br J Dermatol 1983; 108: 653–663.
11. Kanerva L, Ranki A, Lauharanta J. Lymphocytes and Langerhans cells in patch tests. An immunohistochemical and electron microscopic study. Contact Dermatitis 1984; 11: 150–155.
12. Heng MCY, Kloss SG, Kuehn CS, Chase DG. Significance and pathogenesis of basal keratinocyte herniations in psoriasis. J Invest Dermatol 1986; 87: 362–366.

Immunological Studies in Chronic Mucocutaneous Candidiasis before and after Ketoconazole Treatment

HÅKAN MOBACKEN,[1] LEIF LINDHOLM[2] and SVEN MOBERG[1]

[1]*Department of Dermatology, Sahlgren's Hospital, Gothenburg, and* [2]*Department of Clinical Immunology and Institute of Medical Microbiology, University of Gothenburg, Sweden*

Mobacken H, Lindholm L, Moberg S. Immunological studies in chronic mucocutaneous candidiasis before and after ketoconazole treatment. Acta Derm Venereol (Stockh) 1987; 67: 257–260.

Immune functions were studied in eight patients with chronic mucocutaneous candidiasis representing a broad clinical spectrum of this disease. Clinical improvement after ketoconazole for 6 months was not associated with amelioration of cutaneous delayed hypersensitivity to Candida antigen or the in vitro lymphocyte responses to Candida antigen of T-cell mitogens. *Key words: Polyendocrine deficiency syndrome; Immune function.*

(Received June 12, 1986.)

H. Mobacken, Department of Dermatology, Sahlgren's Hospital, S-413 45 Göteborg, Sweden.

Chronic mucocutaneous candidiasis (CMC) is a rare condition with persistent *C. albicans* infections of the skin, mucous membranes and nails. It is often associated with autoimmune or endocrine disorder. It has been surmised that a defective host defence, particular-