

Scanning Electromicroscopy of Suction Blister-roofs from Psoriatic Lesions and Normal Skin

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Suction blisters were raised on psoriatic lesions and normal appearing skin. The epidermis was separated at the epidermal-dermal junction. Scanning electronmicroscopy of the dermal side of the blister-roofs from normal looking skin and almost healed psoriatic lesions showed stellate cells probably formed by cytoplasmic extensions ending at desmosomes. In non-treated psoriatic plaques the cells were rounded and lacked the stellate extensions. *Key words:* *Cytoplasmic extensions*. (Received January 29, 1985.)

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The suction blister technique has been used to separate epidermis from dermis and for studies of mediators in the blister fluid (1, 2).

When raising blisters on psoriatic plaques the blisters easily rupture if the suction pressure is too high. These increased fragility on psoriatic lesions prompted us to investigate with scanning electron microscopy, the morphology of the dermal side of the separated blister-roof in psoriatic lesions before, during and after treatment.

METHODS

Suction blisters were raised on the abdomen with the technique described by Kiistala and Mustakallio (2) using a subpressure of 200 mmHg. The blisters were raised both on psoriatic lesions and adjacent normal appearing skin. The time for the appearance of the blisters was between 90 and 120 min on both lesional and normal skin. The blister-roofs were removed and fixed in 2.5% glutaraldehyde in PBS at 4°C for 24 h. They were then rinsed in PBS, dehydrated in 30, 50, 70 and 90% graded alcohol and critical point dried before sputtering with gold. Pictures were taken in a Jeol T300 scanning electronmicroscop using 20-30 kV. For light-microscopy a freeze-sectioned part of the blister-roof was used.

PATIENTS

1. Three patients with slightly squamous, reddish, infiltrated psoriatic plaques on the abdomen who had not been given anti-psoriatic treatment for more than one month.
2. Three patients where the lesions were partly healed and almost healed after 2-3 weeks treatment with anthralin in vaseline and/or UV-B irradiation.

RESULTS

The epidermis was separated at the epidermal-dermal junction. In psoriatic lesions the elongated papillae are clearly shown (Fig. 1). Scanning electron microscopy of the basal side of the normal appearing skin showed a typical, stellate form of the cells with cytoplasmic extensions going out from the cell body and forming intercellular bridges (Figs. 5-6). The same picture was seen in partly and almost healed psoriatic plaques (Fig. 4). In non-treated psoriatic plaques the cells were round and cylindrical and no irradiating extensions bridges were seen (Fig. 2). At higher magnification the cells looked like rolls with an uneven surface (Fig. 3) compared to the stellate normal cells.

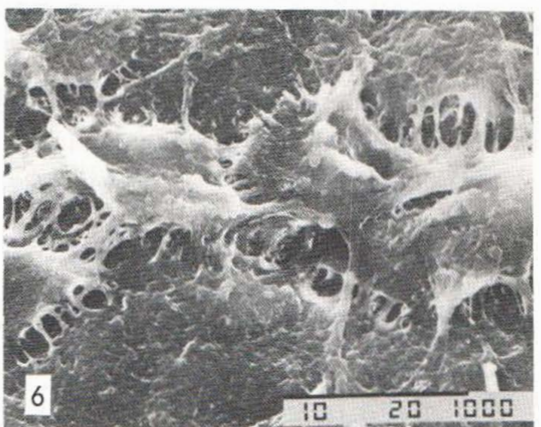
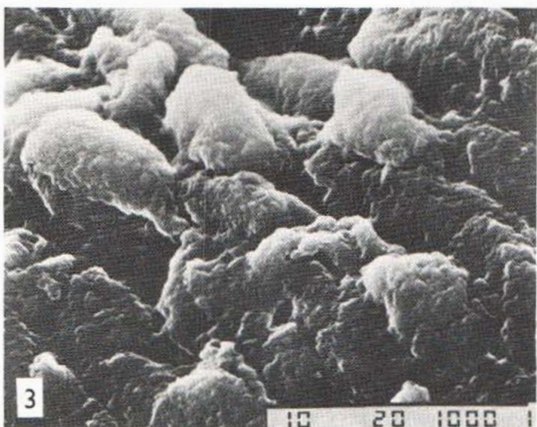
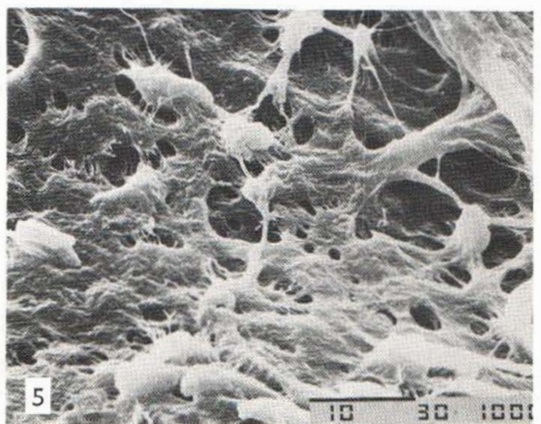
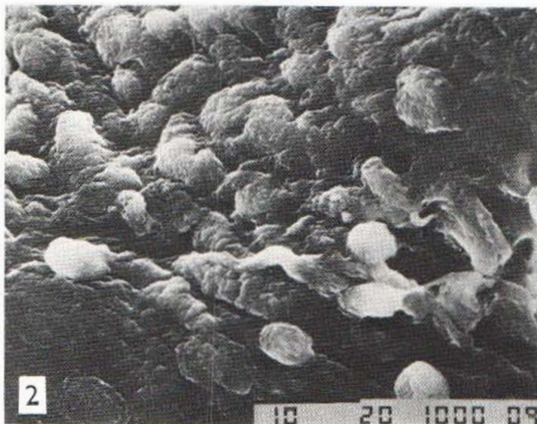
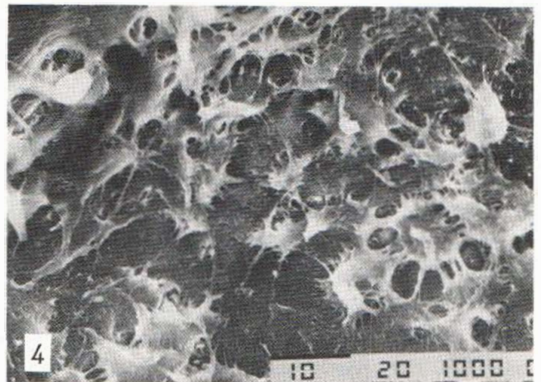
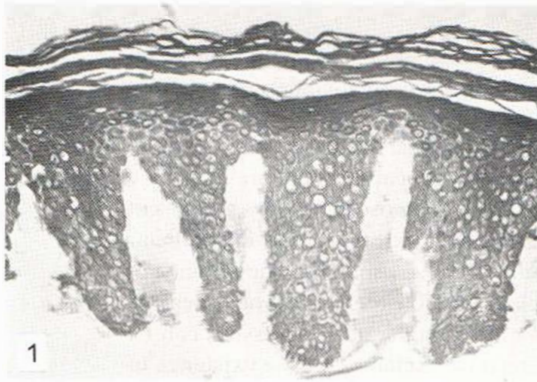


Fig. 1. Section of blister-roof from psoriatic lesion.

Fig. 2. SEM of blister roof from psoriatic plaque with round cylindrical cells on dermal side of suction blister-roof. Bar below indicates length of 10 μ m, 20 kV.

Fig. 3. As 2 but higher magnification showing rolls of cells with an uneven surface.

Fig. 4. Blister roof from almost healed psoriatic plaque with stellate cytoplasmic extensions.

Fig. 5. Normal appearing skin showing irradiating cytoplasmic extension.

Fig. 6. Enlargement of normal skin as in Fig. 5.

COMMENTS

Kiistala & Mustakallio (2) showed that the blisters in normal skin splits below the basal membrane. In psoriatic lesions we also found the rupture at the dermal-epidermal junction. The surface of the basal cells in non-treated psoriatic lesions showed a striking difference from non-lesional skin or treated lesions. The reason is not clear.

The intercellular bridges seen in treated and normal appearing skin are probably cytoplasmic extensions containing tonofilaments ending at desmosomes. They are absent in non-treated psoriatic lesions. This might be associated with the presence of intracellular oedema and/or the rapid proliferation in the basal cells in active psoriasis. The absence of elongated processes and desmosomes can also explain why blisters on psoriatic lesions easily rupture as soon as they are formed and that only a moderate subpressure can be used (1). That partly healed lesions rarely rupture could be explained by the existence of normal structures. One could have expected that the blisters form more rapidly in psoriatic lesions. The reason that no marked difference in time for appearance of blisters was seen between normal and lesional skin could possibly be due to that the increased thickness of the plaques gives a decreased suction force in the deeper layers of the epidermis.

REFERENCES

1. Hensby C, Juhlin L, Civier A, Berne B, Michaëlsson G, Vahlqvist C. Arachidonic acid and prostanoids in suction blister fluid from psoriatic lesions and unaffected skin. *Arch Dermatol Res* 1983; 275: 350.
2. Kiistala U, Mustakallio KK. Dermo-epidermal separation with suction. *J Invest Dermatol* 1967; 48: 466.