## Erosive pustular dermatosis of the leg—a definition

SIR, We enjoyed the letter on 'Erosive pustular dermatosis of the scalp—a definition',[1] but we are sorry that Burton and his colleagues think we have scalped their clinical entity. This was never our intention.

One of the reasons for presenting our patients was to try and cast a little light on the pathogenesis of erosive pustular dermatosis whatever its site. It is our experience that erosive pustular dermatosis of the scalp in the U.K. almost always occurs on previously damaged and clinically atrophic skin. Common forms of preceding damage to the scalp include excessive ultraviolet light, shingles, or even physical trauma sufficient to produce scarring. Exactly the same situation holds good for the legs, so it is not surprising that dermatoliposclerosis was quite often present on our patients. Another feature in common between scalp and shin is that the skin in both these areas is fairly tightly applied to underlying bone.

We quite agree that our patients with erosive pustular dermatosis of the leg are not cases of erosive pustular dermatosis of the scalp, but nevertheless they do show the changes of erosive pustular dermatosis clinically. However, we concede that a scalp, even to a Northerner, looks quite different to a leg. In the meantime, we suggest that erosive pustular dermatosis of the leg should be defined as follows: chronic erosive pustulation confined to the legs of patients with a long-standing history of gravitational ulceration, dermatoliposclerosis and associated skin atrophy. No currently recognized cause of pustulation is present and the histology is non-specific.

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## Expression of the myelomonocytic antigens L1 and CD36 in human epidermis

SIR, We read with interest the paper by Kirkham *et al.*[1] detailing the epidermal distribution of the myelomonocyte L1 antigen in inflamed skin as assessed by immunohistochemical staining with the monoclonal antibody Mac 387. Their results were essentially identical to those previously published.[2,3] Although L1 antigen is cytoplasmic, we were struck by the close similarity between its pattern of expression in keratinocytes and the distribution of CD36 (OKM5), a surface antigen, in inflammatory skin diseases such as psoriasis, allergic contact dermatitis and lichen planus as we have reported.[4] In both cases, keratinocyte expression was mainly suprabasal and confined to those areas immediately overlying the dermal inflammatory infiltrate. Keratinocyte CD36 expression has been reported to be induced *in vitro* by interferon-gamma (IFN-$\gamma$),[5] a product of activated T lymphocytes, and we have suggested that this finding provided further evidence that cytokine-activated keratinocytes participated in skin immune responses. Kirkham *et al.*[1] arrived at similar conclusions based on their results for L1 antigen expression.

More recently, however, we have explored the distribution of keratinocyte CD36 expression immunohistochemically in tumours of epidermal origin (unpublished data) and found striking but consistent variations in the patterns of expression. In well-differentiated squamous cell carcinoma (SCC) the neoplastic cells, together with adjacent normal keratinocytes expressed CD36, while in poorly differentiated SCC, CD36 expression was absent from the tumour cells. In basal cell carcinoma (BCC), CD36 expression by the tumour cells was absent while strong expression by normal keratinocytes at the shoulders of the lesions was consistently observed in a suprabasal pattern, even in the absence of an inflammatory infiltrate. Subsequently, we examined epidermal tumours for expression of L1 antigen,

using Mac 387 antibody and found an identical pattern of expression as for CD36, albeit cytoplasmic, including uninvolved keratinocytes adjacent to SCC and BCC in the absence of an inflammatory infiltrate. In this regard, our results are consistent with those of Gabrielsen *et al.*[6]

We have also attempted to induce keratinocyte CD36 and L1 antigen expression *in vitro* by incubating cultured, normal human keratinocytes with a range of cytokines including IFN-$\gamma$, tumour necrosis factor-$\alpha$ and interleukin 1. In all experiments, expression of these antigens could not be induced, despite strong expression of ICAM-1 and HLA-DR.[7] *In vivo*, intradermal administration of IFN-$\gamma$[8] failed to induce keratinocyte expression of CD36.

Thus, it appears that rather than being specific for inflammatory cutaneous disease, keratinocyte expression of these two myelomonocytic antigens is widespread in diseases involving the epidermis even in the absence of a conspicuous mononuclear cell infiltrate. Furthermore, since expression is confined to suprabasal keratinocytes in areas of epidermal damage and is unrelated to the underlying inflammatory infiltrate, we suggest that L1 and CD36 expression, rather than being directly associated with cytokines produced by infiltrating mononuclear cells in the papillary dermis, may represent a direct response of keratinocytes, depending on their state of differentiation, to an assortment of environmental cues (injurious stimuli). In support of this hypothesis, keratinocytes respond *in vitro* to a variety of low molecular weight substances including phorbol esters, retinoids and urushiol (the catechol responsible for poison ivy dermatitis) to produce a range of pro-inflammatory cytokines including tumour necrosis factor-$\alpha$ and interleukin 8. This hypothesis would explain the absence of L1 expression in normal skin, but positive L1 staining of normal-appearing oral mucosa where repeated trauma probably exists.[9]

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## Topical capsaicin for psoriasis

Sir, The nervous system appears to affect psoriasis through peptides such as substance P (SP) which is released from cutaneous sensory nerve endings.[1] SP-immunoreactive nerve fibres are reported to be increased in the lesional skin in psoriasis,[1,2] and SP binds to receptors on mast cells, inducing degranulation.[1] In a developing lesion, mast cell degranulation is one of the early histological changes,[3] and the release of inflammatory mediators may enhance epidermal and endothelial cell proliferation.[3] SP induces local axon reflex vasodilatation though release of histamine from mast cells and released histamine