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LETTERS TO THE EDITOR

QUANTITATION OF EPIDERMAL DENDRITIC CELLS

To the Editor:

We have read with great interest and benefit, the recent paper by R. M. MacKie and M. L. Turbitt concerning the quantitation of epidermal dendritic cells by anti-Ia-like and HTA-1 antigen monoclonal antibody staining [1].

The method of peroxidase-positive epidermal dendritic cell numeration proposed by the authors consisted of evaluation of such a cell count per 200 basal layer keratinocytes. The number of the counted cells was equally expressed per "linear mm of epidermis" which, as we understand from the data included in Table I (200 basal keratinocytes ≃ 1.8 mm), also corresponded to the epidermal section length measured along a basement membrane.

Since some lesions show an important degree of clubbing phenomenon, the demonstrated differences to "normal" would be even more significant if the peroxidase-positive cells were expressed by the epidermal section length unit measured along the surface of viable epidermis. Additionally, such a count would reflect a skin-surface distribution and density of positively stained cells (as if viewed from above the skin surface) [2].

Extremely interesting findings concerning double staining (with anti-Ia-like and HTA-1 antigen monoclonal antibodies) in poikilodermatous mycosis fungoides support a thesis of keratinocyte–dendritic cell–lymphocyte interaction disturbances in this disease [3]. It seems, however, that the resident and passenger epidermal cell interactions may require also another, additional approach. The quantitative relations between the above-mentioned cell populations (and thus proportions of various cell types) may differ according to the changes of epidermal thickness (acanthosis) without influence on the stained-population cell count expressed per epidermal section length unit, and vice versa.

The problem has arisen when we have tried to quantify the LCs in psoriatic epidermis [4], where possible LC-mediated control of the keratinization process was discussed, and for this reason it appeared necessary to express the data per "epidermal section surface unit" (to demonstrate a relative stained-cells/other epidermal cells ratio) [5].

REFERENCES

1. MacKie RM, Turbitt ML: Quantitation of dendritic cells in normal and abnormal human epidermis using monoclonal antibodies directed against Ia and HTA antigens. J Invest Dermatol 81:216–220, 1983
2. Haftek M, Schmitt D, Brochier J, Faure M, Thivolet J: A specific method of Langerhans cells quantification in human epidermis. Differences in expression of HLA-DR & T6 antigens (abstr). J Invest Dermatol 78:332, 1982
3. Schmitt D, Thivolet J: Lymphocyte-epidermis interactions in malignant epidermotropic lymphomas: I. Ultrastructural aspects. Acta Derm Venereol (Stockh) 60:1–11, 1980
4. Haftek M, Faure M, Schmitt D, Thivolet J: Effects of aromatic retinoid treatment on epidermal Langerhans cells in psoriasis. A quantitative study (abstr). J Invest Dermatol 78:327, 1982
5. Haftek M, Faure M, Schmitt D, Thivolet J: Langerhans cells in skin from patients with psoriasis: quantitative and qualitative study of T6 and HLA-DR antigen-expressing cells and changes with aromatic retinoid administration. J Invest Dermatol 81:10–14, 1983

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REPLY

In reply to the comments by Dr. Haftek and Professor Thivolet on our recent article (J Invest Dermatol 81:216–220, 1983), we would like to make the following points.

There are many methods by which proportions of different cells present in an area of tissue may be quantified, and we are grateful to the correspondents for their contributions in this field. Our conclusions, however, were for the most part based on comparing the numbers of positive cells stained with two different antibodies (anti-HTA and anti-Ia) on the same sections or between similar lesions. The correction factor for thickness of the section in these results would therefore be negligible.

The degree of acanthosis normally seen in cutaneous T cell lymphoma is not so marked as in psoriasis, where expression of results per epidermal section surface unit is undoubtedly of value.

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DETERMINATION OF SKIN THICKNESS

To the Editor:

The paper published in *The Journal of Investigative Dermatology* by Alexander and Miller (Determining skin thickness with pulsed ultra sound. 72:17–19, 1979) was the first publication to describe the use of ultrasound in the measurement of skin thickness.

Alexander and Miller measured thickness with ultrasound and x-rays in 10 volunteers and supplied the data in Table I of their paper. A correlation coefficient (r) of 0.99 was quoted. We have used the same data to correlate the correlation coefficient, using a least-squares linear regression, but computed r to be 0.70. We have repeated this work using 16 volunteers and obtained a value for r of 0.74 between x-ray and ultrasound data. We therefore agree with Alexander and Miller's findings but feel that as their paper is frequently referred to in the current literature [1,2] their inaccurate statistical analysis is rather misleading.

REFERENCES

1. Tan CY, Marks R, Payne P: Comparison of xeroradiographic and ultrasound detection of corticosteroid induced dermal thinning. J Invest Dermatol 76:126–128 1981
2. Kirsch JM, Hanson ME, Gibson JR: The use of conventional diagnostic ultrasound equipment to determine skin thickness. Br J Dermatol 109 (suppl 24):42, 1983

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HLA-DR AND HERPES GESTATIONIS

To the Editor:

In a recent article Shornick, Stastny, and Gilliam [1] reported an increased frequency of HLA-DR2 in the husbands of patients with herpes gestationis (HG) and they suggested that the husband's HLA type may be associated with the development of HG. They also noted a high frequency of anti-HLA antibodies in patients with HG.

We have conducted similar studies and we agree that an immunological reaction to paternally derived fetal antigens may well be important in the etiology of HG. This possibility was highlighted for us in five of our cases, who had experienced normal pregnancies but developed HG when they changed their sexual partner [2]. However, in contrast to Shornick, Stastny, and Gilliam, we do not feel that paternal expression of HLA-DR2 plays a role in the etiology of HG. In the husbands of 16 of our cases the frequency of HLA-DR2 was normal (20%; $p = 0.8$), as was the frequency of the other HLA antigens [3]. In addition to our normal findings we would like to stress that the results of HLA studies in husbands may not necessarily reflect the antigens expressed by the fetus. To assume this presupposes that the husband is the sexual consort, which may not be the case. In Shornick, Stastny, and Gilliam's study we were able to check this possible source of error by comparing the HLA antibodies in the patients with the HLA antigens in the husbands. As the patients had not received transfusions,