may relate to the type of anti-Ia antibody used. We hypothesize that during the inflammatory reaction occurring after hapten application, the density of Ia molecules progressively increases with time and that early after elicitation keratinocytes express a low density of Ia antigens which could be under the sensitivity threshold of some but not all anti-Ia antibodies. The precise role of Ia+ keratinocytes in the initiation of the ACD reaction remains to be clarified.

REFERENCES

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Immunohistochemical Screening of Neuropeptides in Cutaneous Macular Lesions of Leprosy

Sir,

Leprosy peripheral neuropathy is caused by an inflammatory involvement of peripheral nerves; however, the interaction of mononuclear leukocytes and the neural fibres as well as the functional molecular disturbances that take place in nerves affected by the disease remain to be determined. The first study of neuropeptide expression in the biopolar spectrum of leprosy was carried out by Karanth et al. (1), who could detect significant alterations of calcitonin gene-related peptide (CGRP), neuropeptide tyrosine (NPY), vasoactive intestinal polypeptide (VIP), substance P (SP) and protein gene product (PGP) 9.5 immunoreactivity in lepromatous, tuberculoid and indeterminate leprosy patients.

We also think that immunohistochemical screening of early leprous cutaneous macular lesions for changes of neuropeptide expression may contribute to the understanding of initial leprosy nerve alterations.

Therefore, we studied the cutaneous macular lesions of 5 patients using the indirect immunofluorescence technique for detecting abnormalities in the neural fibre immunoreactivity for somatostatin, methionine-enkephalin, α-, β- and γ-melanocyte stimulating hormone (MSH), peptide histidine isoleucine amide (PHI), growth-associated protein (GAP) 43, and galanin. Skin punch biopsies (5 mm) were obtained under local anaesthesia (Xylocain/adenalin, 20 mg/ml + 12.5 μg/ml). Age-, sex-, and body region-matched pieces of skin from normal individuals were taken as controls.

Our study did not show any difference between pathological and normal skin regarding the expression of these neuropeptides, which is in contrast to the ones investigated by Karanth et al. (1).

Alteration of MSH immunoreactivity associated with leprosy pigmentation disorders, as well as disturbances in the production of additional neuropeptides, would be likely to occur in early leprosy; however, immunohistochemical methods may not be sensitive enough to detect early modifications of these neuropeptides. Thus, our results must be interpreted with some caution.

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REFERENCES

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4 For offprint request

Acta Derm Venereol (Stockh) 74