LETTERS TO THE EDITOR

Familial Lichen planus

Sir,

Several theories concerning the etiopathogenesis of lichen planus (LP) exist. Hypotheses of an immunologic mechanism are based on recent studies on cell populations in different evolutionary stages of LP lesions, while genetic susceptibility has been suggested by some described familial LP cases and by studies on HLA-antigens. Two recent studies have demonstrated a striking statistically significant increase in the frequency of DR1 antigen in LP patients (1–2).

We observed an Italian family in which the father and two monozygotic twins had the typical skin lesions of LP. The father (F.E.) suffered his first attack of LP in 1975 at the age of 54 years. The cutaneous lesions were morphologically typical and extended over his trunk. There was no involvement of mucous membranes and the diagnosis was confirmed by histological examination. The LP eruption was controlled with topical steroid therapy; after a remission period of one year the lesions recurred in 1976, and the patient was treated with systemic prednisolone. The therapy was stopped in the winter of 1976. During recent years no relapse of the lesions has been observed. The monozygotic twins, born in October 1954, were patients of a practising specialist whose records were obtained. One of them (F.G.F.) developed typical lesions of LP in March 1983. All relevant clinical chemistry analyses were within normal limits. The patient was treated with a topical steroid and with hydroxychloroquine sulfate. Remission of the lesions was observed in October 1983, but relapses occurred in 1984 and 1985.

The second twin (F.E.) developed typical lesions of LP in 1984, and was treated with a systemic steroid. Subsequently no recurrence of the lesions has been observed. All 6 members of the family were HLA-A, B, C, DR, and DQ-typed, and 4 members (the twins and the 2 sisters) were investigated by the mixed lymphocyte culture (MLC) reaction. The HLA typing was done by the microlymphocytoxicity test using test-sera against the majority of known HLA-A, B, C, DR and DQ antigens.

The MLC reaction was performed on sterile NUNC microtitre plates, using $1 \times 10^6$ responding and $1 \times 10^6$ stimulating lymphocytes in 25% human serum RPMI 1640. The time in the tissue culture chamber (5% CO$_2$, 95% air, humidified, 37°C) was 4 days, the last 24 h after addition of 2 nCi [³H]thymidine (Amersham, England). All experiments were done in triplicate. The MLC was set up in the following modules: cells from 4 members of the family were stimulated by each of the others and by two different pools of cells from unrelated individuals in each pool. The numbers of HLA-A, B, C, DR and DQ typings are summarized in Fig. 1. The members of the family suffering of LP shared DR1 antigen and homozygosity for DQ1 antigen. The twins have not reacted to unibi-directional MLC; the first daughter (HLA identical with the monozygotic twins) showed reactivity vis-à-vis the twins with a reduced value (about 50%) compared with values recorded against unrelated persons and the daughter with a different haplotype. Some authors have reported an incidence of familial LP less than 2% (3), whereas others have found a percentage much higher (11%) than that found previously (4). The aetiology and the pathogenetic mechanism of LP are still unknown. However, several recent reports point to immunologic mechanism and genetic predisposition as being of importance. Our results show that the twins and the first daughter (F.E.) are HLA-identical, but differ regarding the minor locus of histocompatibility identifiable by MLC. The numbers of reported cases of familial LP that have been typed for HLA-antigens are as yet too few to allow any firm conclusions to be

---

Fig. 1. Pedigree of the family.

---

Acta Derm Venereol (Stockh) 70
drawn about the association of this disease with specific HLA-types.

REFERENCES

Received November 29, 1989.

R. Valsecchi, M. Bontempielli, A. di Landro, A. Barcella and T. Cainelli, Department of Dermatology, Ospedali Riuniti, I-24100 Bergamo, Italy.

C3d,g In Normal Human Epidermal Basement Membrane

Sir,

We read with interest the recent report of L. Juhlin (Acta Derm Venereol (Stockh) 1989; 69: 492–496) regarding the detection of transferrin and C3d receptors in human skin. In reference to citations concerning our work, we would like to offer the following comments. Our studies have shown that C3d,g was present in normal human epidermal basement membrane, yet absent from dermal microvascular basement membranes, the epidermal basement membrane of a patient with documented congenital C3 deficiency, and papulonodular basal cell carcinoma tumour nest basement membranes (1, 2, 3). In contrast, we have not hitherto found any evidence of C3d receptors in human skin. Specifically, our direct immunofluorescence microscopy studies employing monoclonal antibodies directed against either CR1, CR2, or CR3, have not demonstrated any evidence of these complement receptors in normal skin or in the skin of our patient with congenital C3 deficiency. In addition, we have found that Raji cells (a lymphoblastoid cell line originally derived from a patient with Burkitt’s lymphoma and expressing various C3 receptors including CR2) do not bind normal human epidermal basement membrane or epidermal cells. Nonetheless, it is possible that C3d receptors or C3d receptor analogues are present in human skin and have not been identified by the antibodies or techniques employed in our studies. Further investigation of this issue would clearly be of interest.

In closing, we appreciate Dr Juhlin’s interest in our work and hope these brief comments clarify statements regarding key aspects of our findings.

REFERENCES

Received February 2, 1990.

Nicole Bassett-Seguin1 and Kim B Yancey2, ‘Service de Dermatologie, Hôpital Saint-Charles, Montpellier, France and 2Department of Dermatology, Uniformed Services University of the Health Sciences, Bethesda, Maryland, USA.

I am most grateful for this clarification by Drs Bassett-Seguin and Yancey.

Lenhart Juhlin, Editor.

Acta Derm Venereol (Stockh) 70