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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 evolutional 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.F.) 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 microlymphocytotoxicity 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 1×10^5 responding and 1×10^5 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 [^{14}C]thymidine (Amersham, England). All experiments were done in triplicate. The MLC was set up in the following module: 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.L.) 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 to few to allow any firm conclusions to be

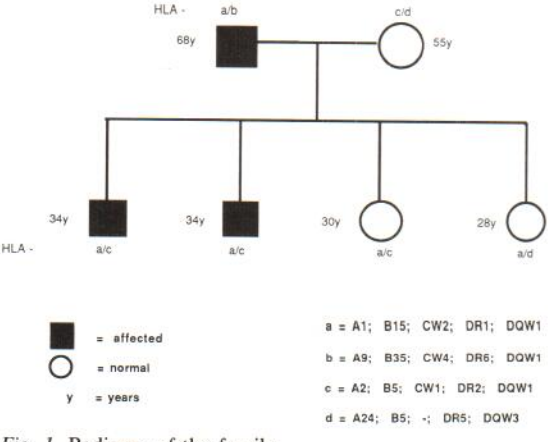

Fig. 1. Pedigree of the family.

drawn about the association of this disease with specific HLA-types.

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