8. Kripke ML. Immunologic effects of UV-radiation and their role in photocarcinogenesis. In: Smith KL, ed. Photochemical and photobiological reviews. Plenum Publ. Corp. 1980; 5: 257.
9. Cormane RH, Siddiqui AH, Westerhof W, Schutgens RBH. Phenylalanine and UVA for te treatment of vitiligo. Arch Dermatol Res 1984; 277: 126.
10. Cormane RH, Siddiqui AH, Nengerman IM. Photochemotherapy of vitiligo with oral phenylalanine. J Invest Dermatol 1983; 80: 367.
11. Zelickson AS, Mottaz JH. Epidermal dendritic cells: a quantitative study. Arch Dermatol 1968; 98: 652.
12. Mishima V, Kawasaki H, Pinkus H. Dendritic cell dynamics in progressive depigmentation. Arch Derm Forsch 1972; 243: 67.
13. Harrist TJ, Muhlbauer JE, Murphy GF, Mihm MC, Bahn AH. T6 is superior to Ia (HLA-DR) as a marker for Langerhans cells and indeterminate cells in normal epidermis: a monoclonal antibody study. J Invest Dermatol 1983; 80: 100.

Association between HLA-B16 and Psoriatic Spondylitis

E. CRIVELLATO and T. ZACCHI[1]

Department of Dermatology, University of Treste, and [1]Tissue-Typing Laboratory, General Hospital, Trieste, Italy

Crivellato E, Zacchi T. Association between HLA-B16 and psoriatic spondylitis. Acta Derm Venereol 1986: 66: 262–264.

The frequency distribution of HLA antigens was studied in a group of 40 patients with psoriatic arthritis from a region in North-eastern Italy. Our results indicate: 1) a strong association, in this population, between HLA-B16 and inflammatory spinal involvement; 2) a failure to confirm previous reports on HLA-B27 increased frequency in psoriatic spondylitis and/or sacroilitis. *Key words: Psoriatic arthritis; HLA-B16; HLA-B27.* (Received September 7, 1985.)

E. Crivellato, Department of Dermatology, University of Trieste, Trieste, Italy.

In Caucasians, the HLA-B27 antigen has been closely linked to susceptibility to seronegative spondyloarthropathies, particularly to ankylosing spondylitis and Reiter's syndrome (1). A statistically significant association has also been reported between HLA-B27 and the axial type of arthritis often seen in psoriatic patients (2, 3, 4).

In this study we were unable to find any significant increase of HLA-B27 in a group of Italian patients with psoriatic arthritis. Conversely results indicate a significantly increased frequency of HLA-B16, even more marked in the group with spondylitic involvement.

MATERIAL AND METHODS

A total of 40 unrelated psoriatic arthritis patients (24 males, 16 females) from the Trieste area (North-eastern part of Italy) were studied. The Moll & Wright (5) criteria for the diagnosis of psoriatic arthritis and the New York criteria (6) for spondylitis were used. Twenty-four (60%) patients had peripheral arthropathy alone (PA); ten (25%) had radiographically proved spondylitis and sacroiliitis (SS) while the remaining six (15%) had sacroiliitis without spondylitis (SA).

HLA typing was performed by microlymphocytotoxicity test following N.I.H. technique (7). 32 antigens were studied for A, B and C loci. Antisera were provided by the Italian Cooperative Group for Tissue Typing, the Behring-Werke and Biotest. Antigen frequencies were compared with 341 controls.

The statistical analysis was made by using the Fisher's exact test. The corrected p-values were obtained by multiplying the p-values by the number of HLA antigenic specificities tested for.

Table I. *Result presentation and statistical evaluation (in each group comparison is made with controls)*

	No. (%)	rr	p	Controls No. (%)
Total (40)				
A1	18 (45)	2.8	$<2.8\times10^{-3}$ [b]	77 (23)
B16	8 (20)	16.8	$<6.6\times10^{-6}$ [a]	5 (1.5)
B27	6 (15)	1.97	NS	28 (8)
B37	3 (7)	27.6	$<4.1\times10^{-3}$ [b]	1 (0.3)
PA (24)				
A1	10 (42)	2.4	NS	
B16	3 (12)	9.6	NS	
B27	2 (8)	1	NS	
B37	2 (8)	30.9	NS	
SS (10)				
A1	4 (40)	2.3	NS	
B16	5 (50)	67.2	$<1.4\times10^{-6}$ [c]	
B27	3 (30)	4.8	NS	
B37	0	0	–	
SA (6)				
A1	4 (67)	6.9	NS	
B16	0	0	–	
B27	1 (17)	2.2	NS	
B37	1 (17)	68	NS	

[a] Corrected $p<2.1\times10^{-4}$.
[b] Corrected p=not significant.
[c] Corrected $p<4.2\times10^{-5}$.

Significance was assigned to frequency differences when p-values were less than 0.01. Relative risk (rr) was calculated using the Woolf method.

RESULTS

HLA-B16, A1 and B37 were recognized as the most frequent HLA antigens. Detailed results are presented in Table I.

DISCUSSION

The results of this study show a significant increase in the frequency of HLA-B16 in psoriatic patients with inflammatory involvement of the axial skeleton. As we found 3 B16 positive patients in the PA group, we cannot conclude that this antigen is a "marker" of psoriatic spondylitis. However, in a pilot study we were unable to observe any significant increase of B16 in a psoriatic group without arthritis. Therefore our findings suggest that psoriatic patients carrying the B16 antigen have an increased risk of developing arthritic lesions particularly of the spondylitic type.

The second significant information which results from this study is the failure to find any significant increase of B27 frequency in the SS and SA groups. We believe that these data reflect some genetic differences between our psoriatic population and other (Scandinavian, English and North-American) psoriatic populations (2, 3, 4, 8, 9). It is otherwise well

known that B27 frequency is lower in idiopathic ankylosing spondylitis patients from the Mediterranean area in comparison to patients from the North-European regions (10).

Therefore, the ethnic background seems to influence the linkage between psoriatic arthritis and distinct alleles of the major histocompatibility system.

REFERENCES

1. Brewerton DA. HLA-B27 and the inheritance of susceptibility to rheumatic disease. Arthritis Rheum 1976; 19: 656–668.
2. Brewerton DA, Caffrey M, Nicholls A, Walters D, James DCO. HL-A27 and arthropathies associated with ulcerative colitis and psoriasis. Lancet 1974; i: 956–957.
3. Marcusson J. Psoriasis and arthritic lesions in relation to the inheritance of HLA genotypes. Acta Derm Venereol (Stockh) 1979; 59: Supp 82: 25–27.
4. Lambert JR, Wright V, Rajah SM, Moll JMH. Histocompatibility antigens in psoriatic arthritis. Ann Rheum Dis 1976; 35: 526–530.
5. Moll JMH, Wright V. Psoriatic arthritis. Sem in Arthritis Rheum 1973; 3: 55–78.
6. Bennet PH, Wood PHN (eds.). Population studies of the rheumatic diseases. (Proceedings of the 3rd International Symposium. New York, 1966.) International Congress Series, Excerpta Medica Foundation, Amsterdam, 1968: 453–463.
7. Terasaki PI, Park MS. NIAID manual of tissue typing techniques. Ray JG, Hare DB, Pedersen PD, Mullally DI, eds. Government Printing Office, DHEW Publication No (NIH) 76–545, 1976: 69–78.
8. Karvonen J, Lassus A, Sievers U, Tiilikainen A. HL-A antigens in psoriatic arthritis. Ann Clin Res 1974; 6: 304–307.
9. Eastmond CJ, Woodrow JC. The HLA system and the arthropathies associated with psoriasis. Ann Rheum Dis 1977; 36: 112–120.
10. Ferri S, Conighi C, Buzzanca G, Albertazzi R, Mattiuz PL. Contribution a l'étude de la fréquence de l'antigène HLA B27 dans la spondylite ankylosante en Italie. Rev Rhum 1982; 49: 355–358.

HLA-B 16 in Hailey-Hailey's Disease

REINHARD MALCHUS,[1] WOLFGANG CH. MARSCH[2] and GÜNTER EHLERS[3]

[1]*Bloodbank and* [2]*Department of Dermatology, Klinikum Charlottenburg, Freie Universität Berlin, and* [3]*Department of Dermatology, Krankenhaus Neukölln, Bereich Britz, Berlin, FRG*

Malchus R, Marsch W C, Ehlers G. HLA-B 16 in Hailey-Hailey's disease. Acta Derm Venereol (Stockh) 1986; 66: 264–266.

Hailey-Heiley's disease is an autosomal hereditary disease of the skin for which only few data exist in regard to genetic markers concerning the HLA system. We report on HLA-A, B and C typing results finding an increased frequency of 55.5% HLA-B 16 positive patients compared to 8.2% in healthy controls. *Key words: MHC; HLA-B locus.* (Received December 23, 1985.)

R. Malchus, Medizinische Klinik und Poliklinik, Klinikum Charlottenburg, Freie Universität Berlin, Spandauer Damm 130, D-1000 Berlin 19, FRG.

Hailey-Hailey's disease (chronic benign familial pemphigus) is an autosomal dominant hereditary cutaneous disease with irregular penetrance (1, 3). The disorder is based on a defect of intercellular coherence of the epidermal keratinocytes. The histological hallmark is a suprabasal blister formation with acantholytic keratinocytes. Clinically the disease is manifested by physical and chemical insults and preferentially affects the intertriginous areas. However, the disease can be expressed in a generalized fashion (5).