**Correspondence**

**HLA B5 and Behçet's disease**

Sir, May we draw your attention again to the inheritance of the HLA B5 antigen and its association with Behçet's disease? The association of the HLA B5 and especially its Bw51 split1-3 with Behçet's disease was established in Japan1-3 and Turkey.4,4 There is also a weak association of B5 and Behçet's disease in British male patients.4,4 There has been a further suggestion by Japanese workers in the field5 that the B5 of Japanese patients is inherited selectively from the mother. We want to present evidence that this is not the case, at least for patients of occidental and Middle Eastern origins.

From a family study of patients with the disease classified according to Japanese criteria6 patients were selected who carried the HLA B5 antigen. We found 8 such Turkish patients and 3 English patients (in all cases except one the antigen carried was Bw51). Tissue typings of these patients and their first-degree relatives were performed in Leiden with a set of 120 typing sera recognising 19 HLA A, 41 HLA B, and 6 HLA C specifications by standard microlymphocytotoxicity tests.

The results are shown in the tables. In Table 1 the HLA typing results on the parents of 8 Turkish probands are given (the mother of case 6 could not be typed, but her HLA genotype was derived from the typing of 6 additional siblings). Of the 3 British probands 2 mothers and 3 fathers could be HLA typed.

The HLA B5 antigen was inherited from the mother alone in 2 Turkish patients, from the father alone in 5, and from both parents in one instance. One British patient inherited the allele from the father, one from the mother, and one from both parents (Table 2).

Ohno et al.7 found that the haplotype carrying the B5 was inherited from the mother alone in 5 patients and from both father and mother in one patient (6 patients and 5 families were studied). We have looked at 11 families of patients carrying the B5 antigen and found no selective inheritance from the mother. In our series 3 subjects inherited the B5 antigen from the mother, 2 inherited it from both parents, and 6 exclusively from the father. We would submit that inheritance is random.

### Table 1 Inheritance of HLA B5 haplotypes in Turkish and British families with Behçet's disease patients

<table>
<thead>
<tr>
<th>Turkish patients</th>
<th>Diag. state</th>
<th>Iritis</th>
<th>HLA typing</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>Sex</td>
<td></td>
<td>Proband</td>
</tr>
<tr>
<td>1</td>
<td>F</td>
<td>I</td>
<td>A26, Bw51, A29, Bw58</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>C</td>
<td>A24, Bw52</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>I</td>
<td>A2, B13, Cw6</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>I</td>
<td>A2, B51, Cw6</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>I</td>
<td>A31, Bw51</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>C</td>
<td>A23, Bw44, Cw5, Bw51, Cw2</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>C</td>
<td>A2, Bw51</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>I</td>
<td>A2, Bw51</td>
</tr>
</tbody>
</table>

**British patients**

<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>Iritis</th>
<th>HLA typing</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>M</td>
<td>S</td>
<td>A28, Bw51</td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>I</td>
<td>A2, B7</td>
</tr>
<tr>
<td>11</td>
<td>F</td>
<td>I</td>
<td>A2, Bw51</td>
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</tbody>
</table>


* Derived haplotypes.
Rheumatoid arthritis and type 1 diabetes mellitus

Sirs, We read with interest the paper by Thomas and colleagues\(^1\) describing an association between rheumatoid arthritis (RA) and type 1 (insulin-dependent) diabetes. In response to their original paper presented to the British Diabetic Association (BDA) in March 1981\(^2\) we had investigated the prevalence of both type 1 and type 2 diabetes in the 1st and 2nd degree relatives of the population of patients with RA attending our clinics in Nottingham. Our findings were presented to the BDA in September 1981\(^3\) and have subsequently been published.\(^4\)

In contrast to Thomas et al. we could find no increased prevalence of type 1 diabetes in the close relatives of 312 patients with RA compared with the relatives of 274 patients with degenerative joint disease. We suspect that the discrepancy between these 2 studies lies in the ascertainment of patients. Our group comprised at least 95% of the Caucasian patients with RA attending our clinics over a 3-month period and were drawn from a defined geographical area. Patients in the study by Thomas et al. were randomly ascertained from 2 separate London clinics by 2 different observers, both hospitals having a particular interest in the genetics of type 1 diabetes. Thus, although there may be sound theoretical reasons for an association between these 2 diseases, based on a common association with HLA-DR4, our data do not support such a hypothesis. It is perhaps unfortunate that the authors did not acknowledge this disagreement or quote the relevant reference in their paper.

As a side issue, this paper illustrates the potential confusion created by the current long publication delay in Annals. Our substantive report was published in Journal of Rheumatology in April 1982, within 4 months of submission. Thomas et al.'s paper, accepted for publication in the same month has unfortunately lain dormant in BMA House for 15 months.

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*Susan Rudge

*The paper by Thomas et al. had not 'lain dormant in BMA House for 15 months' but owing to the great pressure on our space was at the printers awaiting publication.—ED, Annals.

References

HLA B5 and Behçet's disease.

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doi: 10.1136/ard.42.5.602

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