Distribution of ABO Blood Groups, G6PD Deficiency, and Abnormal Haemoglobins in Leprosy

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The extensive literature on the association of ABO blood groups and leprosy has been reviewed by Salzano (1967). He listed 27 series and reported an apparent excess of blood group O and a lack of B among leprosy patients compared to normal controls. In his list the highest level of significant association was observed in Japanese patients. Among the Japanese workers, Omichi (1928) did not find any association whereas Hasegawa (1937) observed a higher frequency of B and Sato (1949) a lower frequency of B among leprosy patients compared to O groups. In the remaining 24 in the series listed by Salzano there was hardly any O:B association. Recently Vogel (1970) calculated the association levels of ABO blood groups in leprosy from 33 published series and reported an O:A association (p ≤ 0.0027).

In an earlier analysis of genetic association in pulmonary tuberculosis (Saha and Banerjee, 1968) the authors could find a significant association only in the case of Chinese patients, and it has been suggested that the question of genetic association may further be complicated by an ethnic factor.

In view of the limited and controversial literature of the association of ABO blood groups in Japanese leprosy patients, we thought it worthwhile to examine the question of ABO association in another Mongolian race, namely the Chinese.

It has been reported that there is a higher incidence of G6PD deficiency among lepers (Kher and Grover, 1969). We thought it worthwhile to verify this in a Chinese population and also to study the incidence of abnormal haemoglobins in lepers compared to healthy subjects.

**Materials and Methods**

All the 459 Chinese ward patients of both sexes at the Trafalgar Home, Singapore which is the only leprosarium in the Republic, were investigated for the distribution of ABO blood groups, G6PD deficiency and abnormal haemoglobins. The details of the method have been published elsewhere (Saha and Banerjee, 1968; Saha 1969 and 1970). The Chinese control series of pulmonary tuberculosis was used as the control series for the present study.

**Results and Discussion**

The results are presented in the Table, which shows the distribution of ABO blood groups in leprosy patients and in the control group.

**TABLE**

<table>
<thead>
<tr>
<th>Blood Groups</th>
<th>Controls*</th>
<th>Leprosy Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>O</td>
<td>6644</td>
<td>43-5</td>
</tr>
<tr>
<td>A</td>
<td>3967</td>
<td>26-0</td>
</tr>
<tr>
<td>B</td>
<td>3814</td>
<td>25-0</td>
</tr>
<tr>
<td>AB</td>
<td>837</td>
<td>5-5</td>
</tr>
<tr>
<td>Total</td>
<td>15,261</td>
<td></td>
</tr>
</tbody>
</table>

* Series of Chan (1962).  
χ² (1df) lepromatous and non-lepromatous O:B = 1-5.  
χ² (1df) control and leprosy O:B = 2-4.

From the Table it can be seen that there is no significant difference in the frequency distribution of ABO blood groups between leprosy patients and normal controls. The distribution was 40-5 and 43-5% with blood group O; 26-1 and 26-0% with group A; 27-9 and 25-0% with group B; 5-4 and 5-5% with group AB. There was also no significant difference in the frequency distribution of ABO blood groups between 338 patients with lepromatous leprosy and 121 patients with non-lepromatous leprosy. From this investigation it is suggested that there is no association of ABO blood groups and leprosy.

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There was no incidence of abnormal haemoglobins among 459 Chinese subjects investigated. Among healthy Chinese in Singapore there is also a very low frequency of abnormal haemoglobins (Vella, 1962; Saha, 1970).

Of 300 male patients tested for G6PD deficiency only 5 (1-5%) had G6PD deficiency as against 3-64% with G6PD deficiency in healthy males (Saha, 1969).

Summary

Four hundred and fifty-nine Chinese patients of both sexes suffering from leprosy were investigated for distribution of ABO blood groups, G6PD deficiency, and abnormal haemoglobins. There was no significant association between these genetic markers and leprosy, nor any difference of frequency distribution between lepromatous and non-lepromatous patients.

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