SHORT COMMUNICATIONS

Malaria and haptoglobin content of serum in a rural population in Upper Volta

Serum haptoglobin (Hp) in health and disease has been extensively investigated since the discovery of Hp by Polonowsky and Jayle in 1938. Studies have been made on the frequency distribution of different phenotypes in different populations (Allison et al., 1958; Giblett and Steinberg, 1960), and a strikingly low level of Hp reported in some negroid African populations where malaria is endemic. However, there have been no field studies directly relating Hp levels to recent or past infections. We had the opportunity to study the sera of 1510 inhabitants from savannah and sahel villages in Upper Volta during an epidemiological survey from December 1976 to April 1977 (dry season).

Serum Hp was measured by immuno-nephelometry, using a modification of the method of Engler et al. (1975). Serum is automatically taken up and diluted 1:200 in 9% NaCl solution. Anti-Hp serum, diluted 1:80, is then added. After ten minutes at room temperature the antigen-antibody complexes are measured by a Technicon fluonephelometer. This method can detect Hp levels as low as 7-10 mg/100 ml. The presence of malaria antibodies was ascertained by countercurrent electrophoresis on a cellulose acetate membrane, using the method of Desilets et al. (1978). At the same time thick and thin smears were checked for the presence of malaria parasites. The electrophoretic patterns of haemoglobin were also analysed and the G6PD levels assayed (using a spectrocolorimetric method in accordance with the WHO requirements, 1967). We arbitrarily defined as 'hypohaptoglobinemia' (hypHp) levels between 7-10 and 50 mg/100 ml and as 'ahaptoglobinemia' (aHp) levels below the sensitivity of the method.

Of our 1510 Hp assays, the mean serum levels were 81.59±54 (s.d.) mg/100 ml in men and 81.47±57 mg/100 ml in women. These values came from 941 subjects, as the remaining 569 samples were ahaaptoglobinemic and so were excluded. Frequency of aHp and hypHp did not show major variations as a function of age. Neither the geographical region (savannah vs. sahel), the presence or absence of G6PD deficiency, nor the presence or absence of Hbs or other abnormal haemoglobins, affected the percentage distribution of Hp levels.

In 1455 subjects it was possible to evaluate simultaneously the Hp levels, the presence or absence of Plasmodium and the presence or absence of antimalarial antibodies. The Hp levels were distributed with approximately the same frequency in the groups obtained from the four different combinations of the parasitaemic and immunological factors (Table 1):

The distribution of Hp levels as a function of three levels of parasitic load was studied in 357 subjects of both sexes and different ages from a savannah village (Donsé). A statistically significant difference was found (z² test; P<0.001). It is apparent from Table 2 that 29.5 and 68.4% of sera were ahaaptoglobinemic in the absence or presence, respectively, of a heavy parasitaemia; 44 and 57% of aHp were found in the two groups with mild and moderate parasitaemia. It is of interest that in the four groups the mean Hp levels were superimposable and comparable to those of the overall study population.

Our results indicate: 1) the clear prevalence of aHp and hypHp in Upper Volta and 2) the lack of clear relationship between abnormal Hp levels and the presence of malaria. A rate of 37% of aHp is in agreement with previous data from Nigeria (Allison et al., 1956) and Mali (Rougé et al., 1974). Mean Hp levels of 81 mg/100 ml are
Of below a mean level detected in France (120 mg/l00 ml in Paris) and account for the high apart kom hing a sensitive indicator of intravascular haemolysis (5 g of free haemoglobin bind loo% of Hp) raised .Hp also indicates inflammatory flare-up in rheumatic diseases and cancer. Decreased levels occur in several liver diseases where oestrogens, haemolysis and defective synthesis can all play a role. Finally, genetic factors (Vu Tien cl, 1973) are important. The high frequency of aHp in subjects with out parasitaemia or anti-Plasmodium antibodies favours a genetic origin for the low levels found in our sample. However this hypothesis cannot lx certain as longitudinal studies might show that aHp can be a transient finding.

The high frequency of aHp and hypoHp in subjects with negative blood smears and no antibodies is contrary to the hypothesis that low Hp levels are induced by the presence of autoantibodies. The increased frequency of aHp with increased parasitic load is statistically significant indicating the contribution of Plasmodium to the overall decrease in Hp levels. However, since parasitaemia operates in a population with low baseline Hp values, its contribution is not crucial. Hp levels cannot be used as an indirect diagnostic tool for present or past malarial infection in Upper Volta populations.

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TABLE 1

<table>
<thead>
<tr>
<th>Parasitaemia</th>
<th>Malaria antibodies</th>
<th>No.</th>
<th>No. (aHp - hypoHp)</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>-</td>
<td>-</td>
<td>47</td>
<td>33</td>
<td>70</td>
</tr>
<tr>
<td>+</td>
<td>-</td>
<td>35</td>
<td>24</td>
<td>69</td>
</tr>
<tr>
<td>-</td>
<td>+</td>
<td>838</td>
<td>605</td>
<td>72</td>
</tr>
<tr>
<td>-</td>
<td>+</td>
<td>535</td>
<td>412</td>
<td>77</td>
</tr>
</tbody>
</table>

TABLE 2

Frequency of aHp and mean Hp levels as a function of the parasitic load (thick smears: + - 1-10 plasmodia/50 fields; + - 11 500 plasmodia/50 fields; + - 500 plasmodia/50 fields)

<table>
<thead>
<tr>
<th>Smears</th>
<th>0</th>
<th>+</th>
<th>+</th>
<th>+</th>
<th>+</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>173</td>
<td>122</td>
<td>37</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>aHp</td>
<td>51</td>
<td>54</td>
<td>21</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>Mean Hp levels* (mg/100 ml)</td>
<td>81-3</td>
<td>84-8</td>
<td>72-7</td>
<td>81-8</td>
<td></td>
</tr>
</tbody>
</table>

*aHp subjects excluded from the mean.
REFERENCES


