

## SHORT COMMIUNICATIONS

## Malaria and haptoglobin content of scrum in a rural population in Upper Volta

Sermm haptoghbin ( Fp ) in healeh and disense has been extemsively inverigated since the disenery of $\mathrm{H}_{\mathrm{p}}$ by Polonowsky and Ja;te in 1938. Studies have been made on the frequeney disuibution of different phenotypers in different populations (Allison et al., 1958 ; Giblet and Steinberg. 1960), and a strikingly low level of Hp reported in some negroid Afrisan populations where malatia is endemic. However, there have been no field studies directly relating $H$ p 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-nephelemetry, using a modification of the method of Engler et al. (1975). Scrum is automatically taken up and diluted $1: 200$ in $9 \% \mathrm{NaCl}$ 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 fluonephelemeter. This mothod can detect Hp levels as low as $7-10 \mathrm{mg} / 100 \mathrm{ml}$. The presence of malaria antiboclies was ascertained by countercurent electrophbresis on a cellulose acetate membrane, using the method of Duilhe of al. (1978). At the same time thick and thin smears were checked for the presence of mataria parasites. The electrophoretic patterns of haemoglobin were also analysed and the (66PD) levels assayed (using a spectrocolorimetric method in accordance with the WFIO requirements, 1967). We arbitrarily defined as 'hypohaptoglohinaemia' (hypoFtp) levels between $7-10$ and $50 \mathrm{mg} / 100 \mathrm{ml}$ and as 'ahaptoglobinatmia' ( aHp ) levels below the sensitivity of the method.

Of our 1510 Hp assats, the mean serum levels were $81.59+5+$ (s.d.) $\mathrm{mg} / 100 \mathrm{ml}$ in men and $81+7-57 \mathrm{mg} 100 \mathrm{ml}$ in women. These values came from 941 subjects, as the remaining 569 samples were ahaptoglobinaemic and so were excluded. Frequency of aHp and hypoHp did not show major variations as a function of age. Neither the geographical region (satannah $i^{\prime}$. sahel), the presence or absence of G6PD deficiency, nor the presence or absence of HbS or nther abnormal haemoglobins, affected the percentage distribution of Hp levels.

In $1+55$ subjects it was pessible to waluate simulaneously the serum $\mathrm{H}_{\mathrm{p}}$ levets, the presence or absence of Plasmodiun and the presence or absence of antimatarial antibodies. The Fp levels were distribued with approximately the same frequency in the groups obtained from the four different combinations of the parasitacmic and immunolegical 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 savammah village (Donsé). A statistically significant difference was found ( $\alpha^{2}$ test $P<0.001$ ). It is apparent from Table 2 that 29.5 and $68.0 \%$ of sera were ahaptoglobinatemic in the absence or presence, respectively, of a heasy parasitacmia; 44 and $57 \%$ of aHp were found in the two groups" with mild and moderate parasitacmia. It is of interest that in the four groups the mean HP levels were superimponable and comparable to those of the overall study pepulation.
 and 2: the lack of dear telatomship betwern abommal Hp level and the presence of matatia, A ate of $37^{\circ \prime}$ of of afp is in agrement with previous data frem Nigeria (Allison


「ГАBIE 1
Distribulion of allp and byonHp in groups in relation (o prescuec) alescner of malaria amtibodies and fresemer/alssenee of parasiles

| Parnvilarmia | Malaria antibadics | $\begin{aligned} & \text { No, } \\ & (14.5 .5) \end{aligned}$ | $\begin{gathered} \operatorname{Sin}_{1} \\ (a H p \div \operatorname{lopollp)} \end{gathered}$ | Percentage |
| :---: | :---: | :---: | :---: | :---: |
|  | . | -- | 13 | 70. |
| - | - | 47 | 33 | 70 |
| - | - | 35 | 24 | 69 |
| - | $\cdots$ | 838 | 605 | 72 |
| 4 | + | 535 | 412 | 77 |

TABLE 2
Ficquency of aHp and mean Hp levels as a function of the purasitic load (thick smonrs: 1 , 1-10 plasmodial50 ficlds: $1-\cdots-11.500$ plasmodial 50 fields; $+1+=-500$ plasmodial 50 fields)

| Smears | 0 | + | $\div+$ | $++\div$ |
| :---: | :---: | :---: | :---: | :---: |
| $-N$ | 173 | - | 122 | 37 |
| allp | 51 | 54 | 21 | 25 |
|  | $: 29.5 \%)$ | $(44.3 \%)$ | $(56.7 \%)$ | 17 |
| Mcan Hp levels* <br> $(\mathrm{mg} / 100 \mathrm{ml})$ | $81 \cdot 3$ | 84.8 | 72.7 | 81.8 |

*aHp subjects excluded from the mean.
below a mean level detected in France ( $120 \mathrm{mg} / 100 \mathrm{ml}$ in Paris) and account for the high prevalence of hypoHp ( $28 \%$ ).

Apart from being a sensitive indicator of intravascular haemolysis ( 5 g of free haemoglobin bind $100 \%$ 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 et al., 1975) are important. The high frequency of aHp in subjects without parasitaemia or anti-Plasmodium antibodies favours a genetic origin for the low levels found in our sample. However this hypothesis camot be certain as longitudinal studies might show that aHp can be a transient finding.

The high frequency of aHp and hypo Hp 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.
acknowledgements. This work was supported by a grant (Nb. 76.7.0846) to Dr. L. Monjour from the Delegation Gencrale à la Recherche Scientifique et Technique, Paris, France.
L. Monjour, J. F. Trape, P. Druilhe, F. Bourdillon, A. Fribourg-Blanc, R. Palminteri, M. Gentilini Parasitologie Médicale, CHU Pitić-Salpétrière, 91, Bd. de l'Hôpital, 75013 Paris, France.
J. M. Kyelem

Ministère de la Santé et des Affaires Sociales, Ouagadougou, République de Haute- Yolta.

MO:NOUR ET AL

## REFERENCCS

Allison, A. C.., Bicmafri, B. S. \& Rees, W. (1958). Nature, 181, 82-t-825.
Drithae, P., Monjolk, LL., Richarl-Levoble, D. \& Gevtilint, M. (1978). Pathologie Biolagipue, 26, 169-172. Engler, R., Wakim, A., Poinms, J., Ronneau, Y'., Jubun, C. \& Jayle, M. F. (197j). Nomells Présse Médicale, 4, 1493-1496.
Giblett, E. R. \& Sthimberg, A. G. (1960;. American Journal of Human Gentics, 12, 100-169.
Polonowshy, M. \& Jayle, M. F. (1938:. Comptes Remdus de la Sociélé de Binlogie (Paris., 129, 457-460.
Ronciemont, A., Qunict, M., Ranqute. Ph. \& Prese, P. (197ti. Bulletin de la Sociélé de Pahologie Exotique, 67, 52-57.
Vu Tien, J., Pisos, G., I.evy, D., Darcos. J. C., Constans. J. \& Matrav-Sexdrati, A. (1975). Comples Rendus de l'Alademí des Sciences de Pari, Sentie D. 280, 2281.
Worid Heqlith Organlatton (1967. Série de Rapporls Techniques, 366, 1-56.

