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## AB0 blood groups in malaria and schistosomiasis haematobium

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### Summary

We sought to determine if there was any relationship between AB0 blood groups and susceptibility to malaria and urinary schistosomiasis. In Epe and outlying villages in south-western Nigeria, we examined 681 people for their blood groups, malaria parasitemia and for the presence of *Schistosoma haematobium* eggs in their urine specimens. Two hundred and sixty-nine individuals were parasitemic for falciparum malaria, 97 subjects had urinary schistosomiasis and 56 people carried concurrent infections of both parasites. Frequencies of the blood groups were 56.68% for group 0, 22.32% for group B, 18.5% for group A and 2.50% for the AB group. The rates of infection with malaria and/or schistosomiasis showed no significant association with the frequencies of the AB0 blood groups.

**Key words:** AB0 blood groups; malaria, schistosomiasis haematobium.

### Introduction

Correlations between the AB0 groups and some infectious diseases have been described, and evidence has been presented to indicate the absence of a relationship in others. No correlation was found between the incidence of loiasis (Ogunba, 1970), Burkitt's lymphoma (Williams, 1966) and the frequencies of AB0 blood groups. However, individuals of blood groups 0 and B were reported to have shown greater susceptibility in three epidemics of influenza A than those of other groups (Frolov et al., 1976). In schistosomiasis mansoni, only a small proportion of infected individuals tend to develop the hepatosplenic form of the disease, and no association of the AB0 blood groups was found with this pathological feature (Katz et al., 1976). But in an endemic area of Brazil, Camus et al. (1977) and Pereira et al. (1979) found higher frequencies of the severe

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form of hepatosplenic schistosomiasis among group A individuals than in any other group. Recent reports that Duffy blood group negative individuals are not susceptible to *Plasmodium vivax* infection (Miller et al., 1976), the conflicting report of Facer and Brown (1979) in *Plasmodium falciparum* infections and the new evidence that human blood group antigens are acquired by *Schistosoma mansoni* in culture media (Goldring et al., 1976), prompted us to examine the relationship of the AB0 blood system to the incidence of malaria and urinary schistosomiasis in an area that is endemic for both diseases.

### Material and Methods

This investigation was carried out as part of a survey for the incidence of parasitic diseases in Epe township and some of its outlying villages. Epe is located in the southwestern part of Nigeria about 100 kilometers northeast of Lagos.

Earlier work had shown the town to be a focus of infection (Okpala, 1961; Gilles et al., 1965). Primary school children between the ages of 7 and 14 years represented the sample population in this survey. With agglutinating A, B, 0 and anti Rh (Anti-A) sera, blood groups of 681 subjects were determined by the direct slide method. All the subjects are of Yoruba (Nigerians) ethnic group and whose blood group frequencies had earlier been reported (Worledge et al., 1974; Araba, 1976). Blood smears from all the children were also examined for microfilaria and malaria parasites. Eosinophil counts were also made from the slide preparations. For the detection of *S. haematobium* infection, duplicate midstream urine specimens were collected in the early afternoon hours. With the volumes noted, and after vigorous shaking, 10 ml aliquots of the urine specimens were carefully examined under a dissecting/binocular microscope for schistosome eggs. The egg counts were recorded as per 10 ml of urine.

### Results

The blood group determinations of the 681 subjects are shown in Table 1. Examination of the blood slides showed that 269 individuals (39.5%) were parasitic for *P. falciparum*. No other species of malaria parasite was encountered in this survey. Microfilaria were not found in any of the blood smears. Table 2 shows separate blood group distributions of individuals with slide demonstrations of falciparum malaria, those with schistosome positive urine

Table 1. AB0 blood group distribution in Epe and environs

Blood groups	No of subjects (Total 681)	Percentage	Gene frequencies
A .....	126	18.5	P = 0.111
B .....	152	22.3	q = 0.133
AB .....	17	2.5	—
0 .....	386	56.7	r = 0.753
Rh- .....	23	3.4	—

Table 2. Frequency of AB0 blood groups in subjects with urinary schistosomiasis and malaria

Blood groups	<i>Schistosoma haematobium</i> infection		Malaria parasitemia		Concurrent malaria and schistosomiasis		% expected for either infection*
	No. observed	% observed	No. observed	% observed	No. observed	% observed	
A	19	19.6	47	17.5	11	19.6	18.5
B	16	16.4	56	20.8	9	16.1	22.3
AB	2	2.1	6	2.2	0	0.0	2.5
O	60	61.9	160	59.5	36	64.3	56.7
Total	97	$\chi^2 = 1.71$	269	$\chi^2 = 0.33$	56	$\chi^2 = 5.34$	

\* These figures are based on the frequency distribution of the blood groups in the total survey population of 681.

specimens and those in whom the two infections were demonstrated. Chi-square analysis showed no statistical association between the incidence of falciparum malaria, schistosomiasis haematobium and the population frequencies of the AB0 blood groups. There were also no significant differences in the blood group frequencies and the rates of infection with respect to sex of the sample population.

Of the control individuals without demonstrable infections, 19.2% were group A, 23.3% were group B, 54.9% were group 0 with 2.7% belonging to the AB group. There were no statistical differences between the infected and the control groups. Eosinophilia from the sample population ranged from 2 to 36%, with a mean of 13.1%. Differences in the mean slide eosinophil counts for the various blood groups were not significant.

## Discussion

We have demonstrated in this study that the AB0 blood group system has no relationship to the prevalence of both falciparum malaria and schistosomiasis haematobium. The frequency distributions of the AB0 blood group antigens in individuals with one or both infections were not significantly different from those of the controls, and are in agreement with those of Worlledge et al. (1974) and Araba (1976) for the same Yoruba ethnic group. Recent studies have shown that the frequencies of the AB0 and other blood group antigens, in children with severe falciparum malaria, were also not different from the controls (Martin et al., 1979; Facer and Brown, 1979). The Duffy blood group antigens have, however, been implicated in the susceptibility to vivax malaria. Miller et al. (1976) reported that African and American blacks who were Duffy positive were resistant to *P. vivax* infection. Martin et al. (1979) have found that Duffy negative individuals are, however, susceptible to falciparum malaria.

Only the AB0 group system has so far been investigated in the susceptibility to schistosomiasis mansoni. Although Katz et al. (1967) did not find any association between the AB0 blood groups and hepatosplenic schistosomiasis, Camus et al. (1977) and Pereira et al. (1979) reported that group A individuals in their study population were more prone to a severe form of the disease than any other group. We did not find that individuals with high *S. haematobium* egg counts were confined to a particular blood group. Goldring et al. (1976) have shown that *S. mansoni* schistosomula would acquire human A, B, and H erythrocyte antigens when cultured in blood of appropriate specificities. Such an acquisition of host blood antigens could shield the parasites from host immune responses and ensure their survival in individuals having any of the four AB0 blood groups. With the same chances of infection in an endemic environment, the blood group distributions of infected individuals would therefore not vary significantly from those of the general population. The group distributions

found in this study are similar to those previously reported for the same Nigerian Yoruba population (Worlledge et al., 1974; Gilles, 1965; Araba, 1976). These results suggest that the AB0 blood antigens are not linked with human *P. falciparum* infections and contrary to Camus et al. (1977) and Pereira et al. (1979) there is no relationship between genetic factors and the evolution of *S. haematobium* infections in humans. We thus confirm the results of Katz et al. (1967) in schistosomiasis and Martin et al. (1979) and Facer and Brown (1979) in falciparum malaria.

Available evidence suggests that *Anopheles gambiae* recognises AB0 blood group variation, with a preference for blood Group 0 (Wood and Harrison, 1972). In the present study, there is no evidence to suggest the importance of this selectivity in our results because the percentage malaria positivity observed is similar to the value expected (Table 2).

If anything, the preponderance of blood group 0 in the study population (Worlledge et al., 1974) may explain the high percentage of those with malaria parasitaemia belonging to blood group 0, or it may be that the sample size could not detect this preference and/or the relationship between the infections and AB0 blood antigens.

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