PATHOLOGY

Susceptibility of Old World Monkeys to Yaba Virus

Bearcroft and Jamieson[1] reported an outbreak of a tumour epidemic in the laboratories of the West African Council for Medical Research in Yaba, Nigeria, among imported rhesus monkeys. Andrewes *et al.*[2,3] showed that this epidemic resulted from a virus of the pox group. Sproul *et al.*[4] classified the tumour as a histiocytoma. Earlier, we reported[5] that Asiatic monkeys (*Macaca mulatta, Macaca irus* and *Macaca speciosa*) are susceptible to the virus, and that a tumour developed, following accidental inoculation, in a laboratory worker as well as in five cancer patients. By contrast, African monkeys (*Cercopithecus aethiops, Cercopithecus aethiops pygerythrus, Cercopithecus mona* and *Cercocebus fuliginosus*) imported from east and west equatorial Africa are resistant. The hypothesis was advanced that this virus may be endemic in certain areas of Africa and that most African monkeys develop immunity at an early age.

As one approach to investigating this hypothesis, a number of young monkeys from African species born in our colony or in zoos in the United States were inoculated subcutaneously with dilution series of Yaba virus suspensions. Animals from the same species imported from Africa were also inoculated. Each virus suspension used in this investigation was also inoculated into at least two *Macaca speciosa* imported from Asia. "Takes" were defined as production of lesions at least 0·5 cm in diameter which persisted for at least 3 weeks, which showed characteristic histological features[4,5] and, when homogenized and injected subcutaneously into *Macaca mulatta*, produced similar lesions.

Table 1

Species	Takes/No. inoculated: African import	U.S. born	Asiatic import
Sooty mangabey (*Cercocebus fuliginosus*)	0/4	2/2	
Vervet monkey (*Cercopithecus aethiops pygerythrus*)	0/4	1/1	
Anubis baboon (*Papio anubis*)	0/3	1/1	
Patas monkey (*Erythrocebus patas*)	0/1	1/1	
Stumptail macaque (*Macaca speciosa*)		1/1	10/10

Table 1 summarizes the results. All African monkeys born in the United States and all Asiatic monkeys developed tumours with 10^{-2}–10^{-4} as the maximal effective dilutions of the virus; none of the imported African monkeys showed any reactions. These results seem to support the hypothesis that African monkeys acquire immunity to Yaba virus in their natural habitat; resistance does not seem to be species specific. It must be kept in mind, however, that in this investigation most of the monkeys born in the United States were young (several months beyond weaning), while most of the imported monkeys appeared somewhat older, although their exact age was unknown. Another weakness of this experiment is the small number of African monkeys born in the United States which were available for investigation.

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Electrophoretic Heterogeneity of Erythrocyte and Leucocyte Glucose-6-phosphate Dehydrogenase in Italians from Various Ethnic Groups

PREVIOUS investigations of drug-induced and fava bean-induced haemolytic anaemia in subjects with a deficiency of glucose-6-phosphate dehydrogenase (G-6-PD) have demonstrated that they are similar both in their clinical characteristics and in the mechanism of genetic transmission of the enzyme defect. Nevertheless, a considerable amount of evidence has accumulated to indicate that this enzyme defect reflects heterogeneous genetic mechanisms in various ethnic groups. It has been demonstrated that the enzyme deficiency may be complete or intermediate, that it may affect erythrocytes alone or involve other tissues[1–3], and that the enzyme protein may differ in its characteristics in cells from subjects of different ethnic groups[3–7]—including its electrophoretic behaviour.

These investigations were almost exclusively undertaken with the erythrocyte enzyme. It is well known that in affected Caucasian male subjects the leucocyte G-6-PD is deficient while it is normal, or almost normal, in Negro subjects of both sexes[3,8]. The enzyme activity is very low (rarely more than 2 per cent of the controls and thus not suitable for assay) in the erythrocytes from sensitive Sardinian subjects which we have extensively studied[6,9–11], and usually in sensitive Caucasian subjects[3,8,12]. Accordingly the leucocyte enzyme is especially suitable for an investigation of the properties of the mutant enzyme in this ethnic group.

In this investigation, a careful study has been made of the properties of leucocyte G-6-PD and a comparison has been made with the erythrocyte enzyme of sensitive subjects from several ethnic groups. The present research is concerned with investigations of a group of twenty-nine subjects from three Sardinian families, from a Sicilian family and from a north Italian family. Our results indicate alterations in electrophoretic patterns of leucocyte G-6-PD from the sensitive subjects, and heterogeneity in the characteristics of the enzyme among the families from Sardinia, Sicily and north Italy.

The families studied were identified when the propositus was admitted to hospital with a haemolytic crisis. The propositi are: case Nos. 5, 11, 12 and 20 for a fava bean-induced haemolytic episode; case No. 24 for a rifocin-induced haemolytic episode; case No. 29 for a pyramidone-induced haemolytic crisis.

Crude samples of the enzymes were prepared as previously described[13]. The modification of the Tsao technique[14] for descending starch-gel electrophoresis which we used has already been described[15], as have the procedures for the G-6-PD and 6-phosphogluconate dehydrogenase (6-PGD) assays used to determine the protein and haemoglobin content[13].

Fig. 1 shows an example of the electrophoretic migration of erythrocyte (case Nos. 9 and 16) and of the leucocyte (case Nos. 3, 12 and 24) G-6-PD from normal and mutant subjects. Two bands were frequently present in the preparations of the leucocyte enzyme, both for male and female normal and mutant subjects. The faster moving band is more intensively stained and results from G-6-PD activity, while the slower one is much less apparent and can be attributed to 6-P-GD. We were able to show that this slower band was in fact 6-P-GD and not a G-6-PD isoenzyme in the following ways: (1) The colour was developed in the usual manner, the slice was then carefully washed and the blue tetrazole stain developed in the presence of 6-phosphogluconate as substrate. The new stain appeared in the same band as the slower component and gave added intensity to the latter. (2) When two symmetrical gel slices were separately stained for G-6-PD and for 6-P-GD, there was a perfect correspondence between the 6-P-GD band and the slower band which appeared in the G-6-PD slice.