haemozoin of normal *P. berghei* is a heterogeneous mixture of porphyrin–peptide compounds derived from the degradation of the host erythrocyte haemoglobin at various points in its chain by proteolytic enzymes of the parasite. Whether a part of the haemoglobin is completely degraded in the process to yield a soluble porphyrin is not known but would not be beyond the realms of possibility. The work of Ball *et al.*[11] with *P. knowlesi* would, however, suggest that, at least in this species, "the parasite retains within its cell all of the hematin that it splits off from hemoglobin". (These authors were, of course, of the then current opinion that the porphyrin and globin portions were completely split by the malaria parasite.) It is difficult to visualize, therefore, how such a mechanism as complexing of antimalarial drugs by ferrihaemic acid can account for the development of drug resistance by *P. berghei* although it may play a part once resistance has developed. If, then, free porphyrin is released by the parasite following the more complete digestion of haemoglobin in the food vacuoles that apparently occurs in our *RC* and *M* and Jacobs's[4] quinine-resistant strains, it might conceivably inactivate some of the drug with which it is in contact in the artificially high concentration that is used in such experimental conditions in mice. Some other, so far undefined mechanisms must underlie the ability of the parasites to survive such a normally lethal environment. This order of drug concentration is certainly never likely to be encountered in the human subject with *P. falciparum* malaria.

Of immediate practical importance is the possibility that primaquine may prove to be relatively ineffective for the treatment of infection by other species of malaria parasites such as *P. falciparum* that are resistant to pyrimethamine, proguanil or its dihydrotriazine metabolite.

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HAEMATOLOGY

Haptoglobin Johnson in Australian Aborigines

The rare haptoglobin phenotype, Johnson, discovered by Giblett[1] in a Negress and her daughter, has since been described in Chinese, Jewish and Caucasian populations[2]. During the course of a population study of the blood groups and serum proteins of Australian Aboriginals of Central Australia[3], two sera taken from members of the Pintubi tribe were found to have this phenotype. Afterwards, samples from the family of one of these were obtained. While there is some White admixture among Central Australian Aboriginals, the propositus himself is, so far as can be ascertained from his blood groups and general appearance, wholly Aboriginal.

The haptoglobin phenotypes of the members of the pedigree are indicated in Fig. 1. The starch-gel pattern designated H (for hypohaptoglobinaemia) was very similar to that of the serum of a member of the pedigree reported by Ramot *et al.*[4] in which the parents were also Hp 2–1

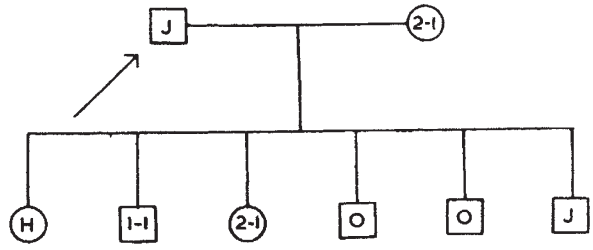

Fig. 1. Pedigree of an Australian Aboriginal family involving the Johnson haptoglobin type

and Johnson. Their pedigree and that reported here are in agreement with the previously proposed hypothesis[5] that Johnson is genotypically $Hp^2{}^JHp^1$. Further, both pedigrees agree with the suggestion[4] that the genotype $Hp^2{}^JHp^2$ is either hypohaptoglobinaemic or ahaptoglobinaemic.

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Low Concentrations of Serum Haptoglobin in Mothers of Children with Haemolytic Disease of the Newborn

The ante-partum prognosis of foetal haemolytic disease is very difficult. The presence and titre of antibodies discovered in pregnancy are not always comparable with the seriousness of the clinical manifestations in the newborn; in the case of an incompatibility in the ABO system not even this guide is available, so that for this purpose new methods are persistently being sought[1].

In foetal erythroblastosis the foetus is attacked by antibodies. The damage found after delivery varies in degree from anaemia[2] to hydropic foetus and, in the extreme, to stillbirth. The disintegration products of foetal erythrocytes are supposed to be transported out of the foetus by the placenta. The transmission of unconjugated bilirubin through the placenta was directly shown by Schenker *et al.*[3] in the guinea-pig foetus. This is compatible with placental transmission of free haemoglobin molecules, which obviously represent the main fraction of the foetal extracorpuscular haemoglobin in the absence of haptoglobin—traces of which are only seldom found in the cord blood by special methods[4]. The transmission of free foetal haemoglobin into the maternal