

the mesoblastic, the hepatic and the medullary period. Perhaps these three periods relieve one another—in sequence as mentioned—corresponding to the appearance of the three types of haemoglobin. Provided that there are really correlations, one must distribute one of the haemoglobin types to each of the three periods of foetal haemopoiesis.

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<sup>1</sup> Halbrecht, J., and Klibanski, C., *Nature*, **178**, 794 (1956).

<sup>2</sup> Drescher, H., and Künzer, W., *Klin. Wschr.*, **32**, 92 (1954).

<sup>3</sup> Allison, A., *Science*, **122**, 640 (1955).

### Blood Group Antigens $Mi^a$ and $Vw$ and their Relation to the $MNSs$ System

THE blood group antigen  $Mi^a$  was discovered by Levine, Stock, Kuhmichel and Bronikovsky<sup>1</sup> in 1951. Anti- $Mi^a$ , the antibody necessary for the identification of the antigen, was made by a mother in response to immunization by her  $Mi(a+)$  foetus which, as a result, had severe haemolytic disease. The antigen was shown to be inherited as a dominant character. The antigen was evidently rare, for no example was found in testing 425 random people.

The blood group antigen  $Vw$  was discovered by van der Hart, Bosman and van Loghem<sup>2</sup> in 1954. The antibody, anti- $Vw$ , was made by a mother in response to immunization by her foetus. The father had the antigen and so had several members of his family. The antigen was not present in 740 blood samples from random Dutch people. The antigen was shown to be inherited as a dominant character. Sanger, studying the pedigree, noticed that the gene  $Vw$  was linked to the  $MNSs$  locus and was travelling with  $Ns$ : there were 12 non-cross-overs and no cross-overs.

It was then found<sup>3</sup> that those members of the Dutch family who were  $Vw+$  were also  $Mi(a+)$ , so it was naturally assumed that  $Vw$  was identical with the antigen  $Mi^a$ , discovered several years before.

We find that the antigens  $Mi^a$  and  $Vw$ , though related, are not the same: about half  $Mi(a+)$  people are  $Vw+$  and about half are  $Vw-$ . No  $Mi(a-)$   $Vw+$  person has been found in several thousand tests on white people.

Though the antigens are rare (the combined incidence of the two phenotypes is one in about a thousand white people) the antibody anti- $Mi^a$  is relatively common and, rather surprisingly, can be present in the serum of people who have not been exposed to either antigen by pregnancy or by transfusion.

From tests on families it has become clear that the gene or genes responsible for both phenotypes,  $Mi(a+) Vw+$  and  $Mi(a+) Vw-$ , are linked to the  $MNSs$  locus. The linkage is of the extremely close type found between the  $CDE$  genes and between the  $MNSs$  genes: this was suggested by the absence of cross-overs in the families and confirmed by the observation that in all chromosomes so far analysed the gene or genes responsible for the  $Mi(a+) Vw+$  reaction are accompanied by  $N$  and  $s$ , while those responsible for the  $Mi(a+) Vw-$  reaction are accompanied by  $M$  and  $S$ .

Because of the rarity of the two phenotypes it may be some time before their precise genetic back-

ground is established. We can think of three possibilities. The genes responsible could be alleles: they would fit the  $CC^w$  behaviour in the Rh system. (The antigen  $Mi^a$  could be like  $C$  and the antigen  $Vw$  like  $C^w$ ; anti- $Mi^a$  could be like anti- $C$ , which reacts with  $Cc$  and with  $C^wc$ , and anti- $Vw$  could be like anti- $C^w$ , which reacts only with  $C^w$ .) On the other hand, the genes may be representatives of adjacent loci like  $D$  and  $C$  in the Rh system. (The antigen  $Mi^a$  could be like  $D$  and the antigen  $Vw$  like  $C$ ; anti- $Mi^a$  could be like anti- $D+C$  and anti- $Vw$  like anti- $C$ .) The available genetical and serological details would fit either arrangement; they would not fit so well the third possibility that one gene alone is responsible and that the difference between  $Mi(a+) Vw+$  and  $Mi(a+) Vw-$  reflects a position effect exerted by the  $Ns$  and by the  $MS$  genes respectively.

The antigens  $Mi^a$  and  $Vw$ , together with the antigens  $Hu$  and  $He$ , are demonstrating the complexity of the region of chromosome responsible for the  $MNSs$  blood groups and it now appears that at least four loci must be involved.

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<sup>1</sup> Levine, P., Stock, A. H., Kuhmichel, A. B., and Bronikovsky, N., *Proc. Soc. Exp. Biol., N.Y.*, **77**, 402 (1951).

<sup>2</sup> v. d. Hart, M. A., Bosman, Hélène, and van Loghem, J. J., *Vox Sanguinis*, **4**, 108 (1954).

<sup>3</sup> Levine, P., Robinson, E. A., Layrisse, M., Arends, T., and Sisco, E. D., *Nature*, **177**, 40 (1956).

### The Diego Blood Factor in Negroid Populations

A GREAT number of the Negroes brought to South American shores during Colonial times came from West Africa (Gold Coast, Guinea, Dahomey, Nigeria, etc.)<sup>1</sup>. These Negroes mixed in large proportion with Indians and Caucasoids, mainly Spaniards and Portuguese. The racial intermixture was so intense that to-day it is almost impossible to find a pure Negro population in any South American country, including Venezuela. Indian admixture was assumed by Layrisse and Arends<sup>2</sup> as an explanation of the eleven Diego-positive cases found in 150 random