

LETTERS TO THE EDITORS

The Editors do not hold themselves responsible for opinions expressed by their correspondents. No notice is taken of anonymous communications

A New Blood-Group Antibody, Anti- Jk^b

THE human blood-group system called *Kidd* was discovered in 1951 by Allen, Diamond and Niedziela¹, who found an antibody, anti- Jk^a , which distinguished two phenotypes, $Jk(a+)$ and $Jk(a-)$. Family investigations² showed that the antigen Jk^a is inherited and that the gene Jk^a is capable of expressing itself in single and in double dose; the existence of an allelomorphic gene Jk^b was assumed. In tests on 390 unrelated persons³, the phenotype frequencies were $Jk(a+)$ 76.92 per cent and $Jk(a-)$ 23.08 per cent, from which the following gene and genotype frequencies were calculated:

Genes		Genotypes	
Jk^a	0.5196	Jk^aJk^a	0.2700
Jk^b	0.4804	Jk^aJk^b	0.4992
		Jk^bJk^b	0.2308

The finding of the antibody corresponding to the antigen, and gene, Jk^b is here reported. It has been found in the serum of Mrs. W. Mrs. W. gave birth to full-term healthy infants in 1937 and 1940 and had miscarriages in 1944 and 1951. There is no history of hæmolytic disease of the newborn. She was transfused with blood after the second and again after the fourth delivery. The latter transfusion had to be stopped owing to a rigor, and because of this her serum was examined.

The blood groups of Mrs. W. are *O*, *MsMs*, *P+*, *cDE/cde*, *Lu(a-)*, *K-*, *Le(a-b+)*, *Fy(a-)*, $Jk(a+)$. Her serum contains anti- Fy^a as well as anti- Jk^b . The more powerful anti- Fy^a was recognized first; the presence of anti- Jk^b was suspected from the results of tests on $Fy(a-)$ red cells. The anti- Fy^a was removed by absorption, leaving in the serum anti- Jk^b alone. The cells used for this absorption were from a person known to be homozygous Fy^aFy^a and thought, from previous dosage work with anti- Jk^a sera, to be homozygous Jk^aJk^a .

That the antibody left in the absorbed serum is indeed anti- Jk^b is shown as follows.

(1) Ninety-eight members of the staff of the Lister Institute have been tested, with the following results:

Mrs. W.'s serum absorbed {	+	$Jk(a+)$	$Jk(a-)$
	-	$\frac{44}{35}$	$\frac{19}{0}$

If the new antibody were not related to the *Kidd* groups the 'exact' probability of getting such a result would be only 1 in 14,129. There is no association with any of the other blood-group antigens, with sex or with the ability to taste phenyl thio-carbamide.

(2) All 19 $Jk(a-)$, that is, Jk^bJk^b , persons so far tested are positive, and so are all of 22 persons known from family studies to be heterozygous Jk^aJk^b . (Seven of these heterozygotes are in the Lister 98; a further 15 were selected and do not appear in the 2×2 table.) If the new antibody were not associated with Jk^b the chance that all 41 persons should be positive is but one in many thousands.

(3) The positive reactions of the new antibody are, on the average, a little stronger with $Jk(a-)$ than with $Jk(a+)$ red cells.

The new antibody in the serum of Mrs. W. is therefore anti- Jk^b . Of the 79 $Jk(a+)$ persons in the

2×2 table it can be calculated that about 51 should be Jk^aJk^b , and therefore positive with anti- Jk^b , while 28 should be Jk^aJk^a and therefore negative; in fact, 44 were positive and 35 negative, which is not a significant departure from expectation ($\chi^2 = 2.9$ for 1 degree of freedom).

The presence of anti- Jk^b in this serum was first demonstrated by the indirect anti-globulin method, but was not detectable by the trypsin or other tests. The reactions were weak. It was then found, however, that an indirect anti-globulin test on trypsinized cells, a combination of tests recommended by Unger³, gave very clear results.

The existence of the antibody anti- Jk^b promotes the *Kidd* system to fourth place in the order of 'usefulness' of the nine blood-group systems. 'Usefulness' is a measure, suggested by Fisher, of the potential value of the systems in human genetics and in problems of identity, parentage and paternity.

We are grateful to Mr. Norman White and Dr. Martin Hynes, of the Royal Northern Hospital, Holloway, for their co-operation; we also acknowledge, once again, the kindness of our long-suffering colleagues at the Lister Institute in giving innumerable samples of blood.

GERTRUDE PLAUT

North London Blood Transfusion Centre,
Deansbrook Road,
Edgware, Middlesex.

ELIZABETH W. IKIN
A. E. MOURANT

Medical Research Council
Blood Group Reference Laboratory,

RUTH SANGER
R. R. RACE

Medical Research Council
Blood Group Research Unit,
Lister Institute,
Chelsea Bridge Road,
London, S.W.1.

Dec. 13.

¹ Allen, F. H., Diamond, L. K., and Niedziela, Bevely, *Nature*, **167**, 482 (1951).

² Race, R. R., Sanger, Ruth, Allen, F. H., Diamond, L. K., and Niedziela, Bevely, *Nature*, **168**, 207 (1951).

³ Unger, L. J., *J. Lab. Clin. Med.*, **37**, 825 (1951).

⁴ Race, R. R., and Sanger, Ruth, "Blood Groups in Man" (Oxford, 1950).

Effect of Hyaluronidase on the Mortality from Experimental Burns

IN treating secondary shock, the intravenous route of fluid administration may not be possible under certain conditions, as, for example, in small infants or in the event of a catastrophe when sufficient trained personnel are not available. In lieu of the intravenous route, the interstitial administration of fluids containing hyaluronidase to assist assimilation has been used clinically in recent years¹. With the addition of this enzyme to break down hyaluronic acid, the fluid spreads more rapidly throughout the connective tissue, thereby entering the capillaries more rapidly, with a consequent increase in the effectiveness of the shock therapy.

If hyaluronidase is to be used in hypodermoclysis, however, it is important to establish that it exhibits no adverse effects otherwise on the shocked individual as, for example, in burns. For this reason, the effect of hyaluronidase on burned, shock-treated animals was studied experimentally.