## 'Private' Blood Factors, Homozygosis, and the Search for New Blood Groups

Levine[1], in a review of the newer blood factors, classed several of them as 'private'. These, he said, "are characterized by the positive or negative reactions being limited to members of a particular family". He cited as 'private' five antibodies reported by various investigators. J. J. van Loghem and van der Hart[2] have since reported a sixth.

How 'private' a blood factor appears to be may depend upon the population studied with the antibody defining the factor. For example, a year or so ago, we received blood from an American Indian woman who, in her ninth pregnancy, gave birth to a baby who died of erythroblastosis. Her blood contained an antibody which agglutinated the blood of her husband and of some, though not all, of her seven living children, but did not agglutinate the blood of 794 other American Indians. Using the antibody on this population only, the factor it defined fell into the class of a 'private' blood factor; but when the antibody was tested with the blood of whites it was readily shown to be anti-*Kell*. The husband was found to be heterozygous *Kk*. On inquiry it was found that his mother was white. From this observation, we suggest that one should inquire into the racial origin of the stimulator of an antibody defining a 'private' blood factor, and if he be found to be of different race from the person who has developed the antibody, members of his race should be tested.

Sometimes, when questioning does not suggest that a person carrying a particular blood group antigen has a foreign ancestor, a hint may be obtained by detailed blood grouping. When we found anti-*D* in the blood of an *Rh*-negative woman, we genotyped the family. The husband was $R_1R_z$ and the three children $R_zr$. $R_z$ is rare in whites but common in American Indians. When we questioned the woman, she said that her husband's mother was born in Scotland, his father in Canada in a district where many Scots settled 140 years ago. She thought of him as a Scottish Canadian, or, as we would say, Caucasoid. But we know that originally the marriage of a white man to an Indian woman was the rule rather than the exception in the area where the husband's father was born. We have little doubt that his $R_z$ came from an Indian ancestor. The finding of $R_0$ suggests the possibility of a negro ancestor, perhaps via a Mediterranean ancestor[3]. When no racial difference between stimulator and producer of an antibody is proved or suggested, members of as many racial groups as possible should be tested with the antibody, before the factor it defines is set down as 'private'. These 'private' antibodies may be of great ethnological value.

Other observations on American Indians led us to give some thought to homozygosis. In a study of the blood groups of the Blood and Blackfoot tribes of southern Alberta in the summer of 1952, we concluded that pure-bred Indians of these tribes were probably homozygous for the following blood antigens: *k*, $Lu^b$, 'not $Le^a$', *P* and possibly *D*. In such a population, so long as it remains pure-bred, and so long as any member, when transfused, receives blood from his own people only, an immune antibody against a factor for which the population is homozygous cannot be produced. So, for example, the pure-bred Indian cannot produce anti-*k*. On the other hand, the introduction of foreign blood antigens, whether through pregnancy or through transfusion, makes the probability of production of an antibody against one of these antigens greater than in the foreign population. In a random sample of 83 sera of Indian men, women and children, we found a second example of anti-*Kell*, whereas we have identified this antibody only half a dozen times in the examination of sera of some thousands of whites. Further, in such a population, naturally occurring antibodies against a blood factor having a frequency of zero may be present, but cannot be recognized by tests of the population. We have found several examples of anti-$Le^a$ in the sera of Indians, while no Indian blood we have tested has reacted with this antibody. Indian blood, of itself, can give no hint of the *Lewis* system. Many Indian tribes are homozygous for *O*, or, to put it the other way about, have a frequency of zero for *A* and *B*. Their sera, of course, contains anti-*A* and anti-*B*. It comes as something of a shock to think what the history of the *ABO* system would have been had the American Indians been the population of primary investigation: for many tribes there would have been no evidence of its existence; for others the classification could only have been $A+$ and $A-$, for perhaps one tribe in South America $B+$ and $B-$.

Since some Indian populations are homozygous for some blood groups, it seems reasonable to believe that Caucasoid, Negroid, Mongoloid and Australoid populations may be homozygous for other blood groups. This is more likely to be true for populations which have resisted interbreeding or have had, until recently, relatively little opportunity for interbreeding, than it is for those which are highly interbred. In a population exhibiting homozygosis for any blood group, that group must remain unknown so long as tests are carried out only with sera of that population. It is our suggestion that a systematic examination of the sera of persons who have been transfused with the blood of donors of a different race or sub-race, and of the sera of women married to men of a different race or of a mixed race, may bring to light several unrecognized blood-group systems. An experimental programme of inter-racial immunization of volunteers would also be worth while. It need scarcely be said that it will be necessary to test the sera against the blood of persons of the race of the donor, or of the husband. It would be desirable, through exchange of sera between laboratories having access to other racial populations, to test the sera against the blood of several races.

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[1] Levine, P., *Trans. N.Y. Acad. Sci.*, **13**, 205 (1951).
[2] van Loghem, J. J., and van der Hart, M., Bull. Cent. Lab. Bloedtrans. Nederl. Rode. Kr., **2**, 225 (1952).
[3] Mourant, A. E., Cold Spring Harbor Symp. Quant. Biol., **15**, 221 (1950).

## Adsorption of DDT on Suspended Solids in River Water and its Role in Black-fly Control

Recent investigations on the Saskatchewan River[1] indicate that DDT associated with suspended solids gave outstanding results in the control of black flies. Black-fly larvæ have been practically eliminated from other streams and rivers by the application of DDT at rates as low as 0·1 p.p.m.[2]; but the maximum distance of effectiveness was approximately nine

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