eact with all Rh_1Rh_2 bloods. It is a striking coinci-ence that the percentage of positive reactions in the Rh_1 group corresponds exactly with the percentage which we can recognize by means of *St* serum to belong to genotype Rh_1rh, and it seems highly probable that this potent anti-*Hr* serum agglutinates the Rh_1rh fraction of the Rh_1 group. If this is so, it must react with only a single dose of an *St* positive gene, and the failure to agglutinate Rh_1Rh_2 cells cannot be attributable to the presence of only a single *St*-positive component, namely, Rh_2.

It is interesting to compare the observed reactions of this potent anti-*Hr* serum with those predicted for the hypothetical antibody δ which Fisher's formulation anticipated[6].

	Rh-negatives	Rh_1Rh_2	'Rh_1 blood of white individuals' About 60 per cent Rh_1rh and 40 per cent Rh_1Rh_1
Genetic structure in Fisher's scheme	$\frac{cde}{cde}$	$\frac{CDe}{cDE}$	$\frac{CDe}{cde}$ $\frac{CDe}{CDe}$
Observed reactions of anti-*Hr* (Levine)	+	−	+ with "about 60%"
Predicted reactions of δ (Fisher)	+	−	+ with Rh_1rh (60 per cent) −with Rh_1Rh_1 (40 per cent)
Observed reactions of *St* (or γ)	+	+	+with Rh_1rh (60 per cent) −with Rh_1Rh_1 (40 per cent)

The hypothetical δ reacts with *d*. *St* (or γ) reacts with *c*.

It was realized when Fisher proposed his scheme that if the antibody δ were encountered, it would be easily distinguishable from γ because the former would fail to agglutinate Rh_1Rh_2 cells.

The finding by Levine of an antibody possessing the characters of one predicted in Fisher's formulation of the genetics of the *Rh* blood groups greatly increases the probability that this formulation represents the actual state of affairs. That *St* serum and Levine's anti-*Hr* serum contain quite different antibodies is now certain; both react with *Rh*-negative blood, but with different components in its antigenic constitution.

It seems probable that Levine and Wiener are working with different anti-*Hr* sera. Levine's powerful anti-*Hr* appears to be the predicted δ, while Wiener's is probably *St* (γ).

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WITH the complete set of six antibodies now apparently discovered, the recognition of rare genotypes will be immensely easier. All the homozygotes of the seven known allelomorphs can be distinguished unambiguously, and nine of the heterozygotes, namely (omitting *h*'s), R_0r, R_0R_2, R_0R_1, R_2R'', R_2R_z, $R''r$, $R'r$, R_1R_z, R_1R'. Three pairs of heterozygotes are indistinguishable

R_0R''	R_0R'	R_0R_z
R_2r	R_1r	R_1R_2

These leave little practical doubt, since in each case the genotype printed below is about one thousand times more frequent than that above.

Three more heterozygotes are capable of confusion only with a heterozygote involving R_y, which has not been discovered, and is unquestionably very rare:

R_yR_2	R_yR_1	R_yr
R_zR''	R_zR'	$R'R''$

It is among these that R_y may be looked for.

Finally, a group of three known genotypes, and one involving R_y, should react positively with all six reagents. These with their approximate frequencies in the British population are shown below:

R_1R''	R_2R'	R_zr	R_0R_y
0·8%	0·3%	0·4%	very rare indeed.

Thus it is only among these last that genotype recognition will have to rely on pedigree evidence. The interpretation of pedigree evidence also will now be greatly facilitated.

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An Unsuspected Relationship between the Viruses of Vaccinia and Infectious Ectromelia of Mice

IT has been observed that emulsions in saline of the lesions produced on the chorioallantois by the virus of infectious ectromelia of mice have the capacity of agglutinating fowl erythrocytes. As is the case with vaccinia virus preparations[1], only about 50 per cent of individual fowls provide susceptible cells. Cells susceptible to one virus are susceptible to agglutination by the other. Agglutination by infectious ectromelia virus is inhibited by anti-vaccinial immune serum from calves.

The possibility of the infectious ectromelia virus being contaminated with vaccinia virus can be excluded, first by the completely different appearance of the lesions produced by the two viruses on the chorioallantois, and secondly by the fact that liver and spleen emulsions from mice dead of infectious ectromelia give a similar agglutination of susceptible but not of insusceptible fowl cells.

Using a technique similar to that commonly used for hæmagglutination work with influenza virus, the titre of a stock membrane emulsion of ectromelia virus (each chorioallantois ground with 1 ml. of saline) is about 1 : 200. The same emulsion titrated on the chorioallantois gives approximately $1{\cdot}2 \times 10^7$ specific pocks per ml. This corresponds closely to the relationship found between the results with the same two methods of titration of vaccinia virus. Preliminary work suggests that a soluble product rather than the virus itself is responsible for the hæmagglutination.

Although the two viruses differ sharply in host-range and type of lesion produced, their physical qualities agree closely; and there seems to be no adequate reason why our findings should not be taken at their face value as indicating that infectious ectromelia is the murine representative of the mammalian pock diseases.

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