appears to be no *a priori* inconsistency and the predictions which may be made are, in the most part, subject to experimental attack.

FRANCIS BINKLEY,
Lt. (jg), H(S), USNR
U. S. NAVAL MEDICAL RESEARCH UNIT NO. 2,
FPO, SAN FRANCISCO, CALIF.

THEORY AND NOMENCLATURE OF THE Hr BLOOD FACTORS[1]

THE hypothesis proposed by Fisher[2] that there are three different varieties of antisera capable of agglutinating Rh-negative blood, corresponding to the three varieties of anti-Rh sera[3,4] appears to be gaining ground. Therefore, there is need for a clear statement of the status of the Hr factors and an acceptable method of designating the sera and factors postulated under the theory, such as has been devised for the Rh blood factors.[5,6]

Since the three varieties of antisera reacting with Rh-positive bloods are collectively known as Rh antisera, the three varieties postulated by Fisher as acting on Rh-negative bloods may be designated collectively as Hr antisera. The great majority, if not all, Hr antisera found until recently have given reactions resembling those described by Levine and Javert[7] for the original Hr antiserum in that the sera regularly agglutinated bloods not reacting with anti-Rh′ serum. That Levine obtained only 30 per cent. positive reactions with his Hr antiserum, while Race and Taylor[8] obtained 80 per cent. positive reactions with their Hr antiserum (designated by them St after the first two letters of the name of the patient from whom the serum was obtained) has been explained by Wiener[9] as due to the low potency of Levine's serum so that only bloods homozygous for the Hr factor reacted with it. Sera of this specificity will henceforth be designated standard anti-Hr sera, just as Rh antisera giving reactions parallel with the original anti-rhesus serum of Landsteiner and Wiener are known as standard anti-Rh sera.

According to the theory of Race and Taylor, which is now firmly established, standard anti-Hr sera react with the agglutinogens determined by genes Rh_z, Rh'', Rh_0 and rh, but not with the agglutinogens determined by genes, Rh_1, Rh', Rh_y and Rh_z. Therefore, standard anti-Hr serum and anti-Rh′ serum are related serologically and genetically like anti-M and anti-N.[9,10] To indicate this, standard anti-Hr serum may also be designated simply as anti-Hr′. Since the other two Hr antisera postulated by Fisher are related to anti-Rh″ and anti-Rh_0, in the same way that anti-Hr′ (or standard anti-Hr) is related to anti-Rh′, these two antisera may now be designated as anti-Hr″ and anti-Hr_0, respectively.

The reactions of the three sorts of anti-Hr sera with the agglutinogens determined by the six standard Rh allelic genes of Wiener[3,4] and the genes Rh_y and Rh_z of Race and Taylor[11,12] can now be summarized as shown in Table 1.

TABLE 1
THE RH SERIES OF ALLELIC GENES*

Designation of genes†				Reactions with Rh antisera			Reactions with Hr antisera		
1 Preferred	2	3	4	Rh′	Rh″	Rh_0 (standard Rh)	Hr′ (standard Hr)	Hr″	(Hr_0)
rh	*rh*	*rh*	*rh*	−	−	−	+	+	(+)
Rh_0	Rh^0	Rh_0	Rh^0	−	−	+	+	+	(−)
Rh′	*Rh′*	*Rh′*	*Rh′*	+	−	−	−	+	(+)
Rh_1	Rh^1	Rh_0'	$Rh^{0'}$	+	−	+	−	+	(−)
Rh″	*Rh″*	*Rh″*	*Rh″*	−	+	−	+	−	(+)
Rh_2	Rh^2	Rh_0''	$Rh^{0''}$	−	+	+	+	−	(−)
(Rh_y)	(Rh^y)	(Rh''')	(Rh''')	(+)	(+)	(−)	(−)	(−)	(+)
Rh_z	Rh^z	Rh_{12} or Rh_0'''	$Rh^{0'''}$	+	+	+	−	−	(−)

* Does not include the intermediate genes. Reactions, genes and antisera enclosed in parentheses have been predicted but not yet encountered.
† These do not represent different nomenclatures but merely variations of a single method of designating the genes.

It will be seen that in Table 1, four slightly different methods of designating the genes of the Rh allelic series are given. The reason for this is that certain minor objections have been raised to the first and simplest method of designating the genes—the one preferred by the present writer. According to the convention adopted by American geneticists, genes belonging to the same allelic series are assigned the same basic symbol and are differentiated by superscripts, while subscripts are reserved for genes at different loci having similar effects. Since very few allelic series are known for man in contrast to the situation in Drosophila, there is hardly any danger of confusion from the use of subscripts instead of superscripts in man, but if a change in nomenclature becomes essential in order to satisfy the geneticists, the second set of designations could be adopted. With regard to the third and fourth sets of designations, these are preferred by many workers because the names of the genes more clearly indicate with which Rh antisera the corresponding agglutinogens react. There is no

[1] Aided by a grant from the United Hospital Fund of New York City.
[2] Cited after R. R. Race, *Nature*, 153: 771, 1944.
[3] A. S. Wiener, SCIENCE, 98: 182, 1943.
[4] A. S. Wiener, *Proc. Soc. Exp. Biol. and Med.*, 54: 316, 1943.
[5] A. S. Wiener, SCIENCE, 99: 532, 1944.
[6] A. S. Wiener, *Jour. Amer. Med. Asn.*, 127: 294, 1945.
[7] Cited by P. Levine, *Jour. Ped.*, 23: 656, 1943.
[8] R. R. Race and G. L. Taylor, *Nature*, 152: 300, 1943.
[9] A. S. Wiener, I. Davidsohn and E. L. Potter, *Jour. Exp. Med.*, 81: 63, 1945.
[10] P. Levine, SCIENCE, 102: 1, 1945.
[11] R. R. Race and G. L. Taylor, *Nature*, 153: 560, 1944.
[12] J. Murray, R. R. Race and G. L. Taylor, *Nature*, 155: 112, 1945.

doubt that these alternative methods of designating the genes are helpful to individuals learning the Rh genetic and serologic theory for the first time, but for general use the first and second sets of symbols are preferred because they are simpler. It is obviously a disadvantage to use a cumbersome symbol such as Rh_0' or $Rh^{0\prime}$ as the name for the most common Rh type and gene. To individuals accustomed to learning abbreviations like t. i. d., t. p. r., etc., an abbreviation like Rh_1 should not be too difficult to master. The advantages of a streamlined nomenclature as far as facility in referring to the Rh types verbally or in writing should certainly be obvious, and the extra effort entailed in learning the designations under column 1 or 2 in the table will soon be repaid.

The recent work on the various Hr antisera does not in any way affect the nomenclature of the Rh genes. The reason for this is that there is a reciprocal relationship betwen the Rh and Hr antisera, so that if the reactions of an agglutinogen with the Rh antisera are known, the reactions with the three Hr antisera are obvious on inspection. Since the purpose of a name is to identify and not to give a complete description, it is neither necessary nor desirable to change the designations of the Rh genes in order to include the reactions with the Hr antisera.

The properties in the red cells reacting with the three varieties of Rh antisera, anti-Rh_0, anti-Rh′ and anti-Rh″, are known as the Rh factors, Rh_0, Rh′ and Rh″, respectively.[5, 6] Similarly, one may postulate the existence of three Hr factors corresponding to the three varieties of Hr antisera, namely Hr_0 and Hr′ and Hr″. Each of these six blood factors, considered individually, is inherited as a simple Mendelian dominant. For example, factor Rh′ may be considered to be inherited by a pair of allelic genes, $\overline{Rh'}$ and $\overline{rh'}$. (The bar is placed over designations of the collective genes $\overline{Rh'}$ and $\overline{rh'}$ in order to distinguish them from the gene Rh' of the series of allelic genes listed in Table 1. The collective gene $\overline{Rh'}$ includes the genes Rh', Rh_1, Rh_y and Rh_z of Table 1, while collective gene $\overline{rh'}$ includes genes rh, Rh_0, Rh_2 and Rh''.) Similarly, factor $\overline{\text{Hr}'}$ may be considered to be inherited by means of a pair of allelic genes, $\overline{Hr'}$ and $\overline{hr'}$. It is obvious on inspection that gene $\overline{Rh'}$ is identical with gene $\overline{hr'}$, and gene $\overline{rh'}$ is identical with gene $\overline{Hr'}$. In fact, if tests are made only with sera anti-Rh′ and anti-Hr′, three types are distinguished, $\overline{\text{Rh}'}$, $\overline{\text{Rh}'\text{Hr}'}$, and $\overline{\text{Hr}'}$, inherited by a pair of allelic genes $\overline{Rh'}$ and $\overline{Hr'}$, so that, as has already been mentioned, the situation is identical with that which exists for the three M-N types.

From the foregoing, it is evident that the percentage of positive reactions that a particular Hr antiserum would be expected to give in a population can readily be calculated if the percentage of positive reactions that the corresponding Rh antiserum gives is known. Thus,

$$\text{Hr}+ = 1 - [1 - \sqrt{(\text{Rh}-)}]^2.$$

For example, it is known that Rh′ antisera give 70 per cent. positive reactions on blood samples from white individuals. Therefore, the expected frequency of positive reactions of standard Hr antiserum (anti-Hr′) on white individuals equals $1-[1-\sqrt{(0.30)}]^2 = 1-(1-0.55)^2 = 1-0.20 = 80$ per cent., approximately. Similarly, since anti-Rh″ sera give 30 per cent. positive reactions on white individuals, it would be expected that anti-Hr″ sera should give approximately 97 per cent. positive reactions. Applying these computations also to anti-Rh_0 and anti-Hr_0 sera, and to the various populations for which data concerning the distribution of the Rh blood types have been compiled, Table 2 was constructed. In Table 3 are summarized the results of the present writer's investigations on the distribution of the Hr factor, using two different Hr antisera with parallel specificities. It will be seen that the findings confirm the conclusion that the antisera used in these studies were anti-Hr′ sera, or correspond to what we have defined as standard Hr antiserum.

TABLE 2

RELATIONSHIP BETWEEN RH AND HR ANTISERA

Racial group	Observed* percentages of positive reactions with Rh antisera			Expected percentages of positive reactions with Hr antisera		
	Rh′	Rh″	Rh_0 (standard)	Hr′ (standard)	Hr″	Hr_0
Whites	70	30	85	80	97	63
Negroes	28	27	90	97.7	97.5	54
American Indians; Chinese; Japanese	85 to 95	40 to 60	99 to 100	43 to 63	86 to 95	0 to 20
Asiatic Indians ...	85	18	93	63	99.1	45

* A. S. Wiener, E. B. Sonn and R. B. Belkin, *Proc. Soc. Exp. Biol. and Med.*, 54: 238, 1943; A. S. Wiener, J. P. Zepeda, E. B. Sonn, and H. R. Polivka, *Jour. Exp. Med.*, 81: 559, 1945.

TABLE 3

DISTRIBUTION OF THE HR FACTOR IN VARIOUS RACES

Population	Investigators	Number of persons tested	Percentage of positive reactions	
			Observed	Expected
Whites	Wiener, Davidsohn and Potter*	239	72.0	80.0
	Wiener†	350	80.0	80.0
Negroes	Wiener, Davidsohn and Potter*	49	98.0	97.7
Mexican Indians	Wiener, Zepeda, Sonn and Polivka‡	98	55.8	53.8

† Unpublished data.
‡ *Jour. Exp. Med.*, 81: 559, 1945.
* *Jour. Exp. Med.*, 81: 63, 1945.

Until recently no antisera had been encountered giving reactions corresponding to anti-Hr″ or anti-Hr_0. Just a few months ago, Mourant[13] reported that he had obtained an Hr antiserum from an Rh-positive mother of an erythroblastotic infant that gives only 3 per cent. negative reactions. Moreover, all the negatively reacting bloods proved to be homozygous for the gene Rh_2, so there seems hardly any doubt concerning the correctness of Mourant's conclusion that the serum in question corresponds to anti-Hr″. On the other hand, the present writer is convinced that Race *et al.'s*[14] contention that Levine's Hr antiserum corresponds to anti-Hr_0 is incorrect. The contention of Race *et al.* is based on the statement[15] that a "potent" anti-Hr serum of Levine's gave negative reactions consistently with bloods of type Rh_1Rh_2 and positive reactions with 60 per cent. of type Rh_1 bloods. Wiener's earlier interpretation that Levine's bizarre results (such as his incorrect statement[16] that in cases of hemolytic disease due to the Hr factor, the mother is Rh positive and the infant Rh negative) were due to the use of weak anti-Hr sera is supported by the statement that "almost all colored individuals tested possessed the Hr factor," which corresponds to the expectations for anti-Hr′ serum and not for anti-Hr_0 serum (*cf.* Table 2). The more consistent results obtained by Levine on Negro bloods are readily understandable because most are homozygous for the Hr′ factor and so their bloods would be expected to react strongly even with weaker Hr′ antisera. On the other hand, the high percentage of persons heterozygous for the Hr′ factor among whites explains Levine's erratic findings in tests on such individuals.

In conclusion, a brief discussion of the medicolegal application of the Rh and Hr blood tests in cases of disputed paternity may be of interest. For the past six months, the present writer has been applying the Hr test (Hr′) in such cases, alongside of the tests for the blood groups, subgroups of A, M-N types and Rh types, and the results in the first 23 cases are given in Table 4. It will be seen that in one case the man was shown not to be the father by the Hr test, and in this case he would not have been excluded had the examination been limited to the grouping, M-N and Rh tests. A man falsely accused of paternity now has better than a 50 per cent. chance of proving his innocence, if tests for the A-B-O blood groups, subgroups of A, M-N types, Rh types and standard Hr factor are performed. When and if the factors Hr″ and Hr_0 become available, the chances of exclusion will be further increased. Under the genetic theory of the Rh and Hr factors, exclusions of paternity would be based on the following two laws: (1) Factors Rh′, Rh″, Rh_0, Hr′, Hr″ and Hr_0 can not appear in the blood of a child unless present in the blood of one or both parents; (2) parents with blood lacking a particular

TABLE 4

RESULTS OF TESTS IN CASES OF DISPUTED PATERNITY

Case	Putative Father	Mother	Children	Interpretation
1.	(a)† $A_1MRh_1Rh_2Hr^+$ (b) $OMNRh_1Rh_2Hr^+$	$OMNRh_1Hr^+$	(a) $OMRh_1Rh_2Hr^+$ ♀ (b) $OMRh_1Hr^-$ ♀ (c) $A_1MRh_1Hr^-$ ♀	Putative father (b) excluded by A–B–O tests for child (c). No conclusion possible for the other two children
2.	$A_2MNRh_2Hr^+$	$A_2NRh_1Hr^+$	$BMNRh_1Rh_2Hr^+$ ♂	Exclusion by A–B–O tests.
3.	$A_2BMRh_1Hr^+$	$OMNRh_1Rh_2Hr^+$	$A_2MNRh_1Hr^-$ ♂	No exclusion
4.‡	$A_1MNRh_1Hr^-$	$BMNRh_1Rh_2Hr^-$	$A_1BMNRh_1Rh_2Hr^-$ ♀	" "
5.	$OMNRh_1Hr^-$	$OMNRh_1Hr^-$	$OMRh_1Hr^-$ ♀	" "
6.	$A_1BMRh_1Hr^-$	$ONRh_1Hr^-$	$BMNRh_1Hr^-$ ♂	" "
7.	$OMNRh^-Hr^+$	$OMRh_1Hr^-$	a) $OMRh_1Hr^+$ ♂ b) $OMNRh_1Hr^+$ ♀	" "
8.*	$A_1MNRh_2Hr^+$	$OMNRh_1Rh_2Hr^+$	$A_1MNRh_2Hr^+$ ♀	" "
9.	$OMRh_1Hr^+$	$OMNRh_1Rh_2Hr^+$	$OMRh_1Rh_2Hr^+$ ♂	" "
10.*	$BMNRh_0Hr^+$	$BMRh_0Hr^+$	$BMRh_0Hr^+$ ♀	" "
11.	$A_1MRh_1Rh_2Hr^+$	$BMNRh^-Hr^+$	$A_1MRh_1Hr^+$ ♂	" "
12.	$BMRh^-Hr^+$	$A_1BNRh_1Hr^-$	$BMNRh_1Hr^+$ ♀	" "
13.	$A_2MRh^-Hr^+$	$A_1NRh_1Hr^-$	$A_1MNRh_1Rh_2Hr^+$ ♀	Exclusion by Rh tests
14.	$OMRh_1Hr^-$	$A_1BNRh_0Hr^+$	$BMNRh_1Hr^+$ ♀	No exclusion
15.	$A_1MRh_2Hr^+$	$A_1NRh_1Hr^+$	a) $A_1MNRh_2Hr^+$ ♀ b) $A_1MNRh_2Hr^+$ ♀	" "
16.	$OMNRh_1Hr^+$	$OMNRh_1Hr^+$	$OMNRh_1Hr^+$ ♀	" "
17.	$BMNRh_1Hr^-$	$A_1NRh_1Hr^+$	$BNRh_1Hr^-$ ♂	" "
18.*	$OMRh_0Hr^+$	$BNRh_2Hr^+$	$A_2MNRh_1Hr^+$ ♂	Double exclusion by A–B–O and Rh tests
19.	$A_1MNRh_1Hr^-$	$BMNRh_1Hr^-$	$OMRh_1Hr^-$ ♂	No exclusion
20.*	$OMRh_0Hr^+$	$ONRh_1Rh_2Hr^+$	$OMNRh_2Hr^+$ ♂	" "
21.	$A_1MNRh_1Hr^+$	$A_1MNRh_1Rh_2Hr^+$	$A_1MNRh_1Rh_2Hr^+$ ♀	" "
22.	$A_1MNRh_1Hr^+$	$OMRh_1Rh_2Hr^+$	$OMRh_2Hr^+$ ♀	" "
23.	$A_1MRh_1Hr^-$	$OMNRh_1Hr^+$	$OMNRh_0Hr^+$ ♂	Exclusion by Hr-Rh tests.

* Negroes.
† Husband.
‡ Mother and child in case 4 both carry the rare gene Rh_2.

[13] E. Mourant, *Nature*, 155: 544, 1945.
[14] R. R. Race, M. N. McFarlane, D. F. Cappell and R A. Fisher, *Nature*, 155: 543, 1945.
[15] R. K. Waller and P. Levine, SCIENCE, 100: 453, 1944.
[16] P. Leyine, *Human Fertility*, 9: 65, 1944.

Rh factor can not have children with blood lacking the corresponding Hr factor; and parents lacking any of the Hr factors can not have children lacking the corresponding Rh factors.

ALEXANDER S. WIENER
SEROLOGICAL LABORATORY,
OFFICE OF CHIEF MEDICAL EXAMINER,
NEW YORK, N. Y.

EFFECT OF DDT, SULPHUR AND LETHANE DUSTS ON GERMINATION OF SUGAR-BEET AND ONION POLLENS

GERMINATION tests were made with sugar-beet pollen collected from portions of sugar-beet seed fields that had been given a single application (20 pounds per acre) of a dust containing 5 per cent. DDT and 95 per cent. pyrophyllite in comparison with similar tests with pollen from undusted portions of the field. Sugar-beet pollen throughout the entire blooming period had shown very poor germinations, making it difficult to obtain exact quantitative data. However, the results of numerous tests were appraised as showing approximately the same germination ratings for pollen from dusted plots as for pollen from undusted plots. The germination tests with sugar-beet pollen were made on an agar medium containing 40 per cent. sucrose, found to be the optimum sucrose concentration to use. Companion tests with onion pollen showed excellent germinations regardless of whether the flowers had been exposed to DDT dust or not. An agar medium containing sucrose at concentrations ranging from 15 to 32 per cent. was used. The indications are that germination of these two kinds of pollen was not adversely affected by the DDT dust. No observations were made on the effects of DDT upon the insects that frequent the sugar-beet and onion flowers. Obviously, DDT should not be used in onion-seed fields after the flowers begin to open because of its known lethal effect upon the insects that pollinate onion flowers.

Sulphur dust as a single application of superfine sulphur at the rate of 20 to 30 pounds per acre, shortly after the blooming period of sugar beets commenced, appeared in the preliminary tests to slow up or inhibit germination of sugar-beet pollen. However, if care was taken not to get sulphur particles on the agar medium along with the pollen, the rate of germination and energy of germination were not greatly affected as a result of the field having been dusted with sulphur. When sugar-beet fields are dusted heavily with sulphur, some of the dust falls upon the anthers and stigmas, so that direct inhibitory effects of sulphur comparable to those observed on artificial media may be a factor in nature.

Lethane B 71 (an organic thiocyanate dust containing beta, beta-dithiocyanodiethyl ether) used at the rate of 30 to 40 pounds per acre on onion fields during the blooming period did not adversely affect germination of onion pollen. During the first few tests agar plates receiving a considerable amount of Lethane dust together with the onion pollen showed no germinating pollen grains. To avoid these direct effects of the dust on the agar medium, both broken and intact anthers from onion flowers were removed individually and placed on the agar medium. Pollen from broken anthers germinated abundantly whether removed 2, 3 or 5 hours after exposure to the fumes of the Lethane dust. The germination energy of pollen from plants dusted with Lethane appeared to be slightly higher than that of pollen from control plants. The material for the pollen studies with onion was supplied by the Division of Horticulture of the New Mexico Agricultural Experiment Station.

ERNST ARTSCHWAGER
DIVISION OF SUGAR PLANT INVESTIGATIONS,
BUREAU OF PLANT INDUSTRY, SOILS AND
AGRICULTURAL ENGINEERING,
AGRICULTURAL RESEARCH ADMINISTRATION,
U. S. DEPARTMENT OF AGRICULTURE

THE DEVELOPMENT OF LITOMOSOIDES CARINII FILARIID PARASITE OF THE COTTON RAT IN THE TROPICAL RAT MITE[1, 2]

VARIOUS blood-sucking arthropods have been explored as possible vectors of the cotton rat (*Sigmodon hispidus litoralis*) filariid, *Litomosoides carinii*. To date, only in the mite, *Liponyssus bacoti*, has development of the filariid been demonstrated.

In mites fed on infected rats, all stages of development have been recovered. The microfilariae grow in length from 69 μ (not including the sheath) to 105–109 μ while there is a rather gradual increase in width from 5.5 μ to 7 μ. At this point in the development there is a sudden expansion in width to 13.2 μ and a typical sausage form with a short sickle-shaped tail is formed. The width increases and individual variations ranging from 15.6 μ to 20.8 μ were found among those larvae between 125 μ and 510 μ in length. From this point on in the development of the worm the width appears to become fixed at 15.6 μ. Those forms which were presumed to be the infective stage reached a length of 800 μ. Further studies are being made on the development of the worm within the mite and its transmission to the cotton rat.

Other ectoparasites of rats that were examined, including fleas, lice and ticks, did not harbor developing filariae. In five species of mosquitoes (*Aedes aegypti, A. taeniorhynchus, A. sollicitans, A. albo-*

[1] This study was made possible through the financial support of the John and Mary R. Markle Foundation.
[2] The authors wish to express their gratitude to the Hegener Research Supply Company of Sarasota, Florida, for the helpful assistance rendered.