and collections of ants and study of the ecology and behavior relations of the stump associations of ants and termites by T. C. Schneirla. During the spring and summer of the current year, Schneirla continued his investigations at the station. The late Henry C. Raven used the laboratory facilities for work toward the completion of his monograph on the anatomy of the gorilla. Raymond B. Cowles, of the University of California, and Charles M. Bogert conducted investigations into the thermal requirements of reptiles, in continuation of research first undertaken in the California deserts in 1939. Edwin H. Colbert made the station his headquarters for study of a subtropical environment in relation to interpretations involving faunal relationships and former ecological conditions of extinct vertebrates, combined with field and laboratory observations on living crocodilians.

The results of much of the foregoing work at the station have been, or will be, published in *Novitates* and the *Bulletin* of the American Museum of Natural History, as part of a subsidized series called "Results of the Archbold Expeditions," of which 51 numbers have appeared to date.

Probably no other locality, and certainly no other established station in southern Florida, offers such a variety of interest for zoological, botanical and ecological research. The station itself presents a rather uniform and long undisturbed sand-scrub habitat of oaks, saw-palmetto, sand-pine and slash-pine, but being centrally situated in a rich and diversified country, it gives easy access to other habitats such as pine flatwoods, bay-gall and cypress swamps, hardwood and cabbage-palm hammocks, dry prairies, wet prairies, freshwater swamps, streams, canals and lakes. A rich representation of wading birds and water birds in the area surrounding the station, and nesting concentrations of herons, egrets and ibis, accessible for observation and experiments, are of special interest to the ornithologist. The station is well equipped not only for the types of research that have been carried out there in the past but for an expanded program. There are provisions for the maintenance of small animals in captivity and large rooms for work indoors. Laboratory equipment includes microscopes, microtome and apparatus for quantitative and qualitative analysis. There are a very complete photographic laboratory and an excellent machine shop. Motor transport is available up to the limit imposed by wartime restrictions.

In matters concerning research at the station, Archbold has the assistance of an advisory board consisting of H. E. Anthony (*chairman*), F. A. Beach, L. J. Brass, Per Höst, Robert Cushman Murphy and H. F. Schwarz, all at the American Museum, and A. L. Rand, at the National Museum, Ottawa. Inquiries from individuals or institutions interested in presenting programs involving research in specific problems which could with advantage be carried out at the station may be sent to Richard Archbold, Box 1309, Lake Placid, Florida, or to any member of the advisory board. Suggestions setting forth ideas for the development of the full potentialities of the station will be welcomed, and given every consideration in plans for the advancement of the aims to which the station is dedicated.

L. J. BRASS

SPECIAL ARTICLES

THE Rh SERIES OF ALLELIC GENES[1]

WITH the aid of the three major varieties of human anti-Rh agglutinins, five variants of the Rh agglutinogen have been identified.[2, 3, 4] According to the writer's genetic theory,[5] which is now established,[6] these blood properties are inherited by a series of six allelic genes, Rh_1, Rh_2, Rh', Rh'', Rh_0 and rh, named after the agglutinogens which they determine. These observations have been substantiated in full by Race *et al.*,[7, 8] working independently, who have in addition defined the reactions given by each Rh variant with so-called anti-Hr[9] or anti-St[10] sera. Moreover, Race *et al.* have partially defined another variant of the Rh agglutinogen, determined presumably by a special allelic gene,[11] tentatively designated Rh_y. As was mentioned in one of the earlier papers by the present writer,[5] occasional bloods have been encountered in our work which give intermediate reactions, suggesting the existence of several additional variants of the Rh agglutinogen. The writer now believes that some of these atypical types are determined by special allelic genes, one of which possibly corresponds to the Rh_y gene of Race *et al.* As will be shown in this re-

[1] From the Serological Laboratory of the Office of the Chief Medical Examiner of New York City. Aided by a grant from the Carnegie Foundation through the Committee on Human Heredity of the National Research Council.

[2] A. S. Wiener and K. Landsteiner, *Proc. Soc. Exp. Biol. and Med.*, 53: 167, 1943.

[3] A. S. Wiener, SCIENCE, 98: 182, 1943.

[4] A. S. Wiener and E. B. Sonn, *Jour. Immunol.*, 47: 461, 1943.

[5] A. S. Wiener, *Proc. Soc. Exp. Biol. and Med.*, 54: 316, 1943.

[6] A. S. Wiener, E. B. Sonn and R. B. Belkin, *Jour. Exp. Med.*, 79: 235, 1944.

[7] R. R. Race, G. L. Taylor, K. E. Boorman and B. E. Dodd, *Nature*, 152: 563, 1943.

[8] R. R. Race, G. L. Taylor, D. F. Cappell and M. N. McFarlane, *Nature*, 153: 52, 1944.

[9] P. Levine, *Jour. Ped.*, 23: 656, 1943.

[10] R. R. Race and G. L. Taylor, *Nature*, 152: 300, 1943.

[11] R. R. Race and G. L. Taylor, *Nature*, 153, 560, 1944.

port, the newer designations[12] of the Rh antisera and Rh genes have greatly simplified the problem of nomenclature for the "intermediate" genes.

The six standard allelic Rh genes and the reactions that they determine with the three varieties of anti-Rh sera, and with anti-Hr (or anti-St) sera, are shown in Table 1.

TABLE 1
THE Rh GENES

Genes[13]	Reactions with antisera			
	anti-Rh_0 (85 per cent. pos.)	anti-Rh′ (70 per cent. pos.)	anti-Rh″ (30 per cent. pos.)	Anti-Hr (St)[14]
rh	Neg.	Neg.	Neg.	Pos.
Rh$_1$ (*Rh*$_0$′)	Pos.	Pos.	Neg.	Neg.
Rh$_2$ (*Rh*$_0$″)	Pos.	Neg.	Pos.	Pos.
Rh$_0$	Pos.	Neg.	Neg.	Pos.
Rh′	Neg.	Pos.	Neg.	Neg.
Rh″	Neg.	Neg.	Pos.	Pos.

As has already been mentioned, occasional bloods were encountered which gave weak or intermediate reactions with one or more of the Rh antisera. For the sake of simplicity, in our previous reports, these weak reactions were disregarded and all bloods were classified under one of the eight types[15] as determined from the stronger reactions. Now that sufficient evidence has accumulated to establish the theory of the six major allelic genes, more attention has been focussed on the "intermediate" types, and it appears that some of these, at least, are also determined by special allelic genes. A few examples of these rare intermediate types are given in Table 2, and it will be seen that they

TABLE 2
"INTERMEDIATE" Rh GENES

Intermediate types	Reactions with antisera			Formerly classed together with major types:
	anti-Rh_0	anti-Rh′	anti-Rh″	
Rh_1(″)	Pos.	Pos.	Weak	Rh_1
Rh_2(′)	Pos.	Weak	Pos.	Rh_2
Rh_0(″)	Pos.	Neg.	Weak	Rh_0
$Rh_{(0)}$′	Weak	Pos.	Neg.	Rh′

enlarge considerably the series of Rh allelic genes. In selecting designations for the intermediate types and genes, the writer has merely extended the principles employed for naming the major types, with the qualification that all weak or intermediate reactions are indicated by parentheses.[16]

That the intermediate types described are actually determined by special genes can of course be established with certainty only by the investigations of family material. For example, the family of Table 3

TABLE 3
FAMILY ILLUSTRATING TRANSMISSION OF GENE $Rh_{(0)}$′

Blood of	Group and M–N type	Reaction with Rh antisera			Rh Type	Formerly classified as
		anti-Rh_0	anti-Rh′	anti-Rh″		
Mother	OM	±	++	–	$Rh_{(0)}$′	Rh′
Children						
Twin daughter A	OM	±	++	–	$Rh_{(0)}$′	Rh′
Twin daughter B	OM	±	++	–	$Rh_{(0)}$′	Rh′
Daughter	OM	±	++	–	$Rh_{(0)}$′	Rh′
Daughter	OM	++	++	–	Rh_1	Rh_1
Son	OM	++	++	–	Rh_1	Rh_1

(father not living) illustrates the hereditary nature of the intermediate type $Rh_{(0)}$′.

Table 4 illustrates the hereditary nature of type

TABLE 4
FAMILY ILLUSTRATING TRANSMISSION OF GENE Rh_1(″)

Blood of	Group and M–N type	Reactions with antisera				Rh type	Most probable genotype
		anti-Rh_0	anti-Rh′	anti-Rh″	anti-Hr		
Mother ...	A_2M	++±	++±	+	+±	Rh_1(″)	*Rh*$_1$(″)*rh*
Children:							
Daughter*	$A_{1,2}M$	++±	++	+	–	Rh_1(″)	*Rh*$_1$(″)*Rh*$_1$
Son	$A_{1,2}M$	++±	++	++±	++	Rh_1Rh_2	*Rh*$_1$(″)*Rh*$_2$
Daughter	$A_{1,2}M$	++±	++±	+	–	Rh_1(″)	*Rh*$_1$(″)*Rh*$_1$
Son	A_2M	++±	++	+	–	Rh_1(″)	*Rh*$_1$(″)*Rh*$_1$

* Mother of an erythroblastotic baby,[17] and having anti-Hr agglutinins in her serum.

Rh_1(″), determined by the corresponding gene *Rh*$_1$(″), which may be the same as the gene *Rh*$_y$ of Race *et al.*

Unfortunately, in this family also the father was not alive, but his probable Rh-type (Rh_1Rh_2) can be surmised from the types of the mother and children.

It should be mentioned that the anti-Rh′ and anti-Rh″ sera used in these studies were really anti-Rh_0′ and anti-Rh_0″ sera with weak anti-Rh_0 agglutinins whose action was eliminated by simple dilution. Recent studies reveal that some bloods containing Rh_0 factor react distinctly with traces of anti-Rh_0 agglutinin insufficient to clump other bloods containing the

[12] A. S. Wiener, SCIENCE, 99: 532, 1944.

[13] The alternative designations, *Rh*$_0$′ and *Rh*$_0$″ are used whenever necessary for the sake of clarity. As a rule, the simpler designations, *Rh*$_1$ and *Rh*$_2$, will be found preferable and unambiguous.

[14] Recently, through the courtesy of Dr. I. Davidsohn, an anti-Hr (St) serum became available to the writer and it was possible to corroborate Race and Taylor's report (*Nature*, 152: 300, 1943) concerning the properties of such antisera.

[15] Of the eight Rh types, the theoretically rarest type, Rh′Rh″, until recently had not been encountered. F. Stratton (*Nature*, 153: 773, 1944) now reports finding four such individuals, two of them siblings.

[16] An alternative method of indicating the intermediate reactions is with the aid of dots. The four types listed in Table 2 would then be designated as Rh_1··, Rh_2·, Rh_0·· and Rh.′ respectively.

[17] I. Davidsohn, E. Potter and A. S. Wiener, unpublished case.

Rh_0 factor. In this way reactions could occur due to residual anti-Rh_0 agglutinins in the serum, which might erroneously be attributed to the anti-Rh′ or anti-Rh″ agglutinin. This pitfall can now be eliminated with the aid of potent anti-Rh_0 blocking serum,[18] because if such a serum is mixed with a diluted anti-Rh_0′ or anti-Rh_0″ serum, the action of the anti-Rh_0 agglutinin will be completely eliminated. An alternative technique is to retest the blood sample with the anti-Rh′ and anti-Rh″ sera after the blood has been treated with anti-Rh_0 blocking serum in order to block the Rh_0 antigen.

The author feels that these observations concerning the intermediate genes probably complete the picture of the Rh blood types. Should, however, other Rh antisera be encountered with specificities different from those of the known antisera, it should not be too difficult to adjust the nomenclature in order to include them in the scheme. For example, if a new agglutinin anti-Rh‴ is discovered, analogous to anti-Rh′ and anti-Rh″, it would only be necessary to postulate the existence of two additional genes Rh_3 (or Rh_0‴) and Rh‴, and the scheme of 8 Rh types would be enlarged to a scheme of 14 types as shown in Table 5.

TABLE 5

SCHEME OF THE RH TYPES EXTENDED TO INCLUDE A HYPOTHETICAL AGGLUTININ ANTI-RH‴

Blood lacking Rh_0					Blood containing Rh_0				
	Reactions with antisera					Reactions with antisera			
Types	Anti-Rh′	Anti-Rh″	Anti-Rh‴	Anti-Rh_0	Types	Anti-Rh′	Anti-Rh″	Anti-Rh‴	Anti-Rh_0
Rh-neg.	−	−	−	−	Rh_0	−	−	−	+
Rh′	+	−	−	−	Rh_1(Rh_0′)	+	−	−	+
Rh″	−	+	−	−	Rh_2(Rh_0″)	−	+	−	+
Rh‴	−	−	+	−	Rh_3(Rh_0‴)	−	−	+	+
Rh′Rh″	+	+	−	−	Rh_1Rh_2	+	+	−	+
Rh′Rh‴	+	−	+	−	Rh_1Rh_3	+	−	+	+
Rh″Rh‴	−	+	+	−	Rh_2Rh_3	−	+	+	+

As has been pointed out elsewhere,[19] the anti-Hr sera have a place in the scheme of the Rh blood types similar to that of the anti-O sera in the blood group scheme. The newer knowledge of the Hr factor[20] has therefore helped to clarify the problem of the nature of the anti-O sera. If, as seems probable, anti-O sera react with the properties determined by genes O and A_2, but not with those determined by genes A_1 and B, then it is clear that only bloods of genotypes A_1B, A_1A_1 and BB will fail to react with potent anti-O sera (*cf.* Table 6). The confusion that existed up to now

[18] A. S. Wiener, *Proc. Soc. Exp. Biol. and Med.*, 56: 173, 1944.

[19] A. S. Wiener and H. Karowe, *Jour. Immunol.*, 49: 51, 1944.

[20] A. S. Wiener, I. Davidsohn and E. L. Potter, *Jour. Exp. Med.*, in press.

TABLE 6

REACTIONS OF ANTI-O SERA WITH BLOODS OF THE VARIOUS GROUPS

Blood of group	Reactions with antisera				Reaction with anti-O serum
	anti-A	anti-A_1	anti-B	Genotype	
O	Neg.	Neg.	Neg.	*OO*	Strong
A_1	Pos.	Pos.	Neg.	*A_1A_1*	Neg.
				A_1A_2	Weak
				A_1O	
A_2	Pos.	Neg.	Neg.	*A_2A_2*	Strong
				A_2O	
B	Neg.	Neg.	Pos.	*BB*	Neg.
				BO	Weak
A_1B	Pos.	Pos.	Pos.	*A_1B*	Neg.
A_2B	Pos.	Neg.	Pos.	*A_2B*	Weak

was caused by the fact that anti-O sera, like anti-Hr sera, are usually of low potency, so that consistent positive reactions were obtained only with bloods of groups O and A_2. Wtih more potent anti-O sera, the reactions obtained agree closely with the predictions under the theory proposed above.

BROOKLYN, N. Y. ALEXANDER S. WIENER

ANTIBACTERIAL SUBSTANCES IN ORGANS OF HIGHER PLANTS

THE presence in higher plants of substances with antibacterial properties has been reported in previous publications.[1, 2, 3, 4, 5, 6, 7] The work of Fleming, Florey, Waksman and many others has shown that such substances present in lower plants have important biological significance. Hence, a more intensive effort in investigating the vast field offered by higher plants seemed to be warranted. The authors have for the past six months been engaged in a systematic review of members of families of higher plants with the aim of discovering and isolating substances with antibacterial properties. Osborn's[8] work in the same field was not known to the authors when this search was begun, hence some duplications occurred.

The hypothetical foundation of this search was twofold. (1) It was known that in the rizosphere of higher plants the growing roots regularly survive the actions of innumerable micro-organisms capable of destroying them. It was concluded that a mechanism must be present in the plants enabling them to counteract potentially destructive microorganisms. One such mechanism is undoubtedly manifested in the secretion of acids within the rizosphere. Other protective mechanisms in plants might be based on antibiotic substances of a specific nature. (2) For centuries plant drugs have been used in all parts of the

[1] E. Glaser and F. Prinz, *Fermentforsch.*, 9: 64, 1926.
[2] O. Stickl, *Zeits. Hyg. Infektkr.*, 108: 566, 1928.
[3] F. Boas, *Ber. D. Bot. Ges.*, 52: 126, 1934.
[4] F. Boas and R. Steude, *Biochem. Zeits.*, 279: 417, 1935.
[5] F. Boas, *Ber. D. Bot. Ges.*, 57: (100), 1939.
[6] O. E. Böcker, *Zeits. Hyg. Infektkr.*, 121: 166, 1939.
[7] Other references are given in the paper of Huddleson *et al.*, published in *Jour. Vet. Asn.*, 105: 394, 1944.
[8] E. M. Osborn, *Brit. Jour. Exp. Path.*, 24: 227, 1943.