A New Rare Rhesus Agglutinogen

ALEXANDER S. WIENER, EVE B. GORDON AND LAURA COHEN

From the Serological Laboratory of the Office of the Chief Medical Examiner of New York City

CORRESPONDING to the three Rh factors, Rh_0, rh′ and rh″, three reciprocally related Hr factors are theoretically possible (Wiener, 1945), of which two, hr′ and hr″, have actually been found (Levine, 1943; Race and Taylor, 1943; Wiener et al., 1945, Mourant, 1945; Wiener and Peters, 1948). Until recently, tests on large random series of blood specimens from Caucasoids as well as family studies appeared to confirm the hypothesis that the blood factor pairs rh′-hr′ and rh″-hr″ are reciprocally related, and behave serologically and genetically like the blood factor pair M-N. Thus, among Caucasoids, blood lacking the rh′ factor invariably contained the hr′ factor, while blood lacking the hr′ factor always contained the rh′ factor (Wiener et al., 1945; Wiener, 1946; Race et al., 1948; Wiener and Gordon, 1951). Moreover, rh′-negative parents had no hr′-negative children, and parents who were hr′ negative had no rh′-negative children (Wiener et al., 1949; Lawler et al., 1951). Similar statements appeared to hold for the factor pair rh″-hr″, but not as many blood specimens have been tested for the hr″ factor (Wiener et al., 1949; Lawler et al., 1951; Race and Sanger, 1950; Wiener, 1950).

Recently, Race et al. (1951) encountered a blood specimen which gave atypical reactions in the Rh-Hr tests. The blood specimen came from a healthy woman, whose serum was found to agglutinate 1400 random blood specimen but did not agglutinate her own cells. She had never received a blood transfusion or blood injection, but her third child had died of erythroblastosis. While the patient's red cells exhibited no unusual reactions outside the Rh-Hr system, the rhesus phenotype was most unusual. The patient's cells proved to contain the Rh_0 factor, but lacked the rh′, rh″, hr′ and hr″ factors. A sample of this blood specimen was kindly sent to us by Drs. Race and Selwyn, and we were able to confirm these findings with the reagents at our disposal.

The discovery of the atypical Rh-Hr agglutinogen described by Race et al. suggests the possible existence of additional related variants of the rhesus agglutinogens. The purpose of the present report is to describe a new rhesus agglutinogen, somewhat similar to the one found by Race et al., which we encountered in the course of routine Rh-Hr typings on blood specimens from Negroids.

MATERIALS AND METHODS

The blood specimens studied came from a series of 743 Caucasoids and 248 Negroid individuals in New York City. The 743 Caucasoids were all professional blood donors. The

248 Negroids were individuals involved in disputed paternity proceedings in the Court of Special Sessions of New York City. (There was also a comparable larger series of 753 Caucasoids who were similarly involved in paternity proceedings, but these were not used for the statistical analysis.) They had denied paternity of the complainant's child, and their blood types were determined in the course of the routine medicolegal examinations. In order that the statistical analysis include only unrelated individuals, the blood specimens from the babies are not included in the present study.

The blood specimens of the professional donors which are used in this study were tested between May, 1950 and October, 1951. Each sample was tested with the following antisera: anti-Rh_0, -rh′, -rh″, -rh^w, -hr′, and -hr″. The blood specimens from the individuals involved in paternity proceedings were tested during the period from September, 1948 to May, 1952. Most of the latter blood specimens were also tested with the six Rh-Hr antisera, except on occasions when there was a shortage of the rarer sera, anti-rh^w and anti-hr″. The tests were carried out by the tube agglutination method. All blood specimens giving negative reactions with anti-Rh_o tube test agglutinating serum, were further tested by the slide method with strong conglutinating anti-Rh_o serum. All blood specimens giving negative reactions with anti-hr′ agglutinating serum were retested with an anti-hr′ conglutinating serum. Those rare blood specimens giving negative reactions with the anti-hr″ serum or positive reactions with anti-rh^w serum were retested several times, by using them as controls in tests carried out on succeeding days. In a number of cases, the absence of the hr″ factor from a blood specimen was verified by the absorption technique.

In order to economize with the rare anti-rh^w serum,[1] this reagent was salvaged after each test and re-used several times, as described in a previous paper (Wiener and Gordon, 1949). The anti-hr″ serum which we used in the present study came from a woman with hemolytic anemia, and has been described by Wiener and Peters (1948). She was bled at intervals of approximately six months, and since the hr″ antibody titer generally fell with the passage of time, she was restimulated 7 to 10 days before each bleeding by an intravenous injection of a small amount of group O, type rh blood. The most recent specimen obtained from this patient had an anti-hr″ titer of approximately 80 units by the saline tube agglutination method. The serum also contained a potent autoantibody of approximately 100 units titer at body temperature for enzyme-treated human cells (including the patient's own red cells). The autoantibodies did not clump human cells by the conglutination or antiglobulin methods; and the direct antiglobulin test for coating of the patient's cells by autoantibodies was negative. Since the autoantibodies did not clump unmodified red cells, it did not interfere with the tests for the hr″ factor. In order not to waste this rare antiserum, it was diluted 1 to 5 with saline solution and the tests were carried out in narrow tubes (inside diameter 4 mm.). All blood cells giving negative reactions were retested with the full strength serum. Unfortunately, the patient who had supplied the anti-hr″ serum recently died of pneumonia, and at autopsy, unexpectedly, a right mitral stenosis was found.

RESULTS

In table 1 are shown the results of the Rh-Hr typings on the series of 743 professional blood donors. All the blood specimens gave the expected reactions with the six antisera, and the distribution of the types is similar to that observed in our previous studies except for a somewhat higher percentage of type rh individuals. There were 14 blood specimens (1.9 per cent) which were ag-

[1] The anti-rh^w serum used in this study was kindly provided by Dr. J. J. van Loghem, Jr., to whom the authors wish to acknowledge their indebtedness.

glutinated by anti-rh^w serum, and one blood specimen of the rare type Rh_zRh_1. Only 11 blood specimens were not agglutinated with anti-hr″ serum, and all these were of the type Rh_2. There was not a single blood specimen which lacked both blood factors of either of the pairs rh′-hr′ and rh″-hr″. The reactions with anti-hr″ serum were strong except for many blood specimens of type Rh_z (Rh_1Rh_2). The latter could not be ascribed merely to a "gene-dose" effect, because the blood specimens of type Rh_2rh, which likewise have only a single dose of the hr″ factor, almost invariably gave strong reactions with the anti-hr″ serum. Possibly there was some interference from the rh′ factor present

TABLE 1. DISTRIBUTION OF Rh-Hr TYPES IN A SERIES OF 743 CAUCASOIDS*

PHENOTYPES	NUMBER OF INDIVIDUALS	PER CENT	PHENOTYPES FURTHER SUBDIVIDED BY rh^w ANTISERUM		
			Phenotypes	Number	Per cent
Rh_0	18	2.42			
Rh_1 Rh_1rh	241	32.50	Rh_1rh	237	31.96
			Rh_1^wrh	4	0.54
Rh_1Rh_1	143	19.24	Rh_1Rh_1	135	18.17
			$Rh_1^wRh_1$	8	1.07
Rh_2 Rh_2rh	90	12.11			
Rh_2Rh_2	11	1.47			
Rh_z Rh_zRh_0	96	12.92	Rh_zRh_0	94	12.65
			$Rh_z^wRh_0$	2	0.27
Rh_zRh_1	1	0.13			
rh	138	18.55			
rh′ rh′rh	3	0.40			
rh″ rh″rh	2	0.26			
Totals.....	743	100.00	rh^w pos.	14	1.88

* All specimens were tested with sera anti-Rh_0, -rh′, -rh″, -rh^w, -hr′, and -hr″.

in type Rh_1Rh_2 blood, and absent from type Rh_2 blood. This would be comparable to the considerably weaker reaction of group A_2B blood with anti-A serum, as compared with group A_2 blood.

The findings on the smaller series of Negroid blood specimens are shown in table 2. The distribution of types is similar to that observed in our previous studies (Wiener et al., 1943; 1944) and again demonstrate the high percentage of individuals of type Rh_0. Another striking feature is the occurrence of blood specimens giving atypical reactions with anti-Rh_0 serum (Rh_0 variants), again in conformity with our earlier observations (Wiener et al., 1950). The findings on Negroids confirm the reciprocal relationship between factors rh′ and hr′. Only 9 blood specimens failed to react with anti-hr′ serum; 8 of these were of individuals of type Rh_1 and one from an individual of type Rh_1Rh_2. The latter individual, whose phenotype was Rh_zRh_1 and who was therefore presumably a carrier of the R^z gene, was a Puerto-Rican.

On the other hand, the reactions of the blood specimens from Negroids with anti-hr″ serum exhibited certain irregularities in contrast to the blood specimens from Caucasoids. There were as many as 8 blood specimens giving weak or doubtful reactions, and 5 of these were from individuals of type Rh_1rh and

TABLE 2. RESULTS OF TESTS FOR hr″ FACTOR ON A RANDOM SERIES OF BLOOD SPECIMENS FROM NEGROIDS

PHENOTYPE*	NUMBER† OF INDIVIDUALS	PER CENT	REACTIONS WITH ANTI-hr″ SERUM			
			Positive	Negative	Doubtful	Not tested
Rh_0	114	40.14	68	1	4	41
Rh_1rh	75	26.41	41	0	1	33
Rh_1^wrh	1	0.35	1	0	0	0
Rh_1Rh_1	8	2.82	6	0	0	2
Rh_2	47	16.55	36	4	3	4
Rh_z (hr′ pos.)	9	3.17	7	0	0	2
Rh_z (hr′ neg.)	1	0.35	0	0	0	1
rh	17	5.99	11	0	0	6
rh′rh	2	0.70	2	0	0	0
rh″	1	0.35	1	0	0	0
Rh_0 variants	9	3.17	6	0	0	3
Totals	284	100.0	179	5	8	92

* All blood specimens were tested with anti-Rh_0, -rh′, -rh″, -rh^w, and -hr′ serum.

† Includes 23 Puerto-Ricans.

TABLE 3. FAMILY STUDY ON A PUERTO-RICAN WOMAN WITH BLOOD OF TYPE Rh_0 LACKING FACTOR hr″

BLOOD OF	A-B-O GROUP	M-N TYPE	Rh-Hr PHENOTYPE*	REACTION WITH ANTI-hr″ SERUM†
Putative father	O	MN	Rh_1rh	++±
Mother	B	M	Rh_0	−
1st child	B	M	Rh_1rh	+±
2nd child	O	MN	Rh_0	++±

* Based on reactions with anti-Rh_0, -rh′, -rh″, -hr′ sera.

† The intensity of the reactions is indicated by the number of plus signs as follows: +++, one large clump; ++±, several large clumps; ++, many smaller clumps easily visible to the naked eye; +±, numerous smaller clumps just visible to the naked eye; +, clumping distinct but visible only under low power of microscope; ±, doubtful reaction; −, cells separate and distinct microscopically.

type Rh_0, who theoretically should have a double dose of the hr″ factor. In addition, there were 5 other blood specimens giving entirely negative reactions with the anti-hr″ serum. Four of the latter were from individuals of type Rh_2, but one was a blood specimen of type Rh_0 from a Puerto-Rican woman. Since blood of type Rh_0 is supposed to have a double dose of the hr″ factor, this blood specimen was subjected to a thorough study. By absorption tests, it was possible to confirm the absence of the factor hr″ from the blood specimen. More-

over, the individual was recalled and a fresh venous sample was obtained and retested with the full strength anti-hr″ serum. Again it was possible to show that the blood cells contained factors Rh_0 and hr′, but not factors rh′, rh″, or hr″. Thus, for this individual, the reciprocal relationship between factors rh″ and hr″ does not hold, and her blood is similar in that regard to the one described by Race and Selwyn.

In table 3 is presented an analysis of the Rh-Hr reactions of the two children of the woman with atypical Rh_0 blood lacking hr″ factor, in order to show the reactions with anti-hr″ serum of the blood cells of these two children. One child belongs to type Rh_0 like the mother, but his blood reacts typically with anti-hr″ serum; the blood of the second child who belongs to type Rh_1rh is also agglutinated by the anti-hr″ serum, but the reactions are only of moderate intensity. The putative father could not be excluded by the blood tests (cf. table 3), but of course this does not necessarily mean that he is actually the father of the two children. To explain the failure of the mother's blood to agglutinate in anti-hr″ serum, one must postulate that her blood contains a special kind of Rh agglutinogen, which may be designated Rh_0^u in order to indicate the presence of the hr′ factor but the absence of the hr″ factor.[2] This individual is presumably homozygous for the corresponding gene R^{ou}, while each of the two children therefore have at least a single dose of the gene.

The existence of individuals with red cells lacking both factors rh″ and hr″ has an important implication for the medicolegal application of these tests in cases of disputed parentage. If atypical agglutinogens like Rh_0^u and Rh_0^x are not taken into account, this could lead to erroneous exclusions of parentage. In fact, two instances were encountered by us in the series of Negroids involved in disputed paternity proceedings, which might have been misinterpreted in this manner. As shown in table 4, in both these cases the mother's blood reacted as type Rh_0, while the child's blood gave reactions corresponding to type Rh_2Rh_2; that is, in these two families, the mother's blood was rh″ negative while her child's blood was hr″ negative, contradicting the postulated reciprocal relationship between factors rh″ and hr″. The findings are readily explained if one assumes that these two mothers are carriers of the rare gene R^{ou}, and that the two children are both of the genotype R^2R^{ou}. It is significant that the blood of one of the mothers gave only weak reactions with anti-hr″ serum suggesting the presence of hr″ factor in single dose.

Thus, among the fewer than 300 Negroid individuals tested with anti-hr″

[2] In the very first study on the heredity of the Rh blood types (Wiener et al., 1944) the letters "U" and "V" were used to represent the factors rh′ and rh″, respectively. Accordingly, the small letters "u" and "v" could be used for factors hr′ and hr″. Therefore, when type Rh_0 blood is found to contain hr′ factor but to lack hr″ factor, as in the case described here, we indicate this by the superscript "u", namely Rh_0^u. The superscript "v" will be used, on the other hand, for hypothetical blood of type Rh_0 possessing factor hr″ but lacking factor hr′. When type Rh_0 blood lacks both Hr factors the superscript "x" will be used, e.g., the blood described by Race et al. is designated Rh_0^x.

serum, individuals presumably carrying the gene R^{ou} were detected three times, and one of these three individuals was homozygous for the gene. No doubt the number of individuals in this series carrying the R^{ou} gene was actually considerably larger, but the types of mother and child were not always favorable for demonstrating the presence of the gene. At any rate, the gene appears to be not uncommon among Negroids. On the other hand, as shown in table 1, the gene is rare among Caucasoids, if it occurs at all. In this connection, the second series of 753 Caucasoids (involved in disputed paternity proceedings) has been analyzed; of these, 563 individuals were tested with anti-hr″ serum. Thirteen persons were encountered with blood lacking the hr″ factor; all of them belonged to type Rh_2. As has already been mentioned, weak reactions with anti-hr″ serum are not uncommon among individuals of type Rh_1Rh_2.

TABLE 4. TWO EXAMPLES OF ATYPICAL Rh-Hr REACTIONS IN MOTHER AND CHILD, EXPLAINED BY POSTULATING THE PRESENCE OF GENE R^{ou}

BLOOD OF	A-B-O GROUP	M-N TYPE	Rh-Hr TYPE	REACTION WITH ANTI-hr″ SERUM	PROBABLE GENOTYPE
Putative father.....	A_1	M	Rh_zRh_0	++	R^1R^2
Mother.....	A_1	MN	Rh_0	++	$R^{ou}r$ or $R^{ou}R^o$
Child.....	A_1	M	Rh_2Rh_2	−	R^2R^{ou}
Putative father.....	A_1	MN	Rh_2rh	++	R^2r or R^2R^o
Mother.....	B	MN	Rh_0	±	$R^{ou}r$ or $R^{ou}R^o$
Child.....	O	M	Rh_2Rh_2	−	R^2R^{ou}

In addition, five individuals of type Rh_1Rh_1 and one of type Rh_1rh unexpectedly gave weak reactions with the anti-hr″ serum. Since these were not further tested, they may represent accidental technical deviations, but even if one assumes the presence of an R^{ou} gene, it is evident that this gene is but rarely present among Caucasoids in comparison to Negroids. Since at least among Negroids the postulated reciprocal relationship between the rh″ and hr″ factors does not always hold, the anti-hr″ serum may be used in medicolegal cases of disputed parentage only with certain reservations. Thus, only when the accused man belongs to type rh or rh′ and the child to type Rh_2Rh_2, or when the accused man belongs to type Rh_2Rh_2 and the child to type rh or rh′, would it be safe to conclude that the paternity is excluded, because then the question of the rare R^{ou} gene would not arise.

The situation with regard to the factor pair rh′-hr′ is more satisfactory, as far as the medicolegal application is concerned. Not a single exception to the theory has been encountered in our extensive series of families, nor has any been found by Race and his coworkers. However, the discovery by Race and Selwyn of the rare individual of type Rh_0^x indicates that such exceptions may actually exist. This theoretical, rare possibility should not prevent the use of

the Rh-Hr tests in cases of disputed parentage, but it does perhaps call for some mild qualification when the rh′-hr′ tests exclude an accused man. On the other hand, no qualification is necessary for the dominance rule of inheritance of the various Rh-Hr factors.

COMMENT

While studying the serological behavior of the blood from the individual of type Rh_0^x (—D—), Race et al. observed an unexpected phenomenon. In tests with blocking anti-Rh_0 sera, it was found that clumping occurred also in saline media with most such antisera. Race et al. conclude "The curious ability of these cells to be agglutinated in saline by incomplete anti-D suggests that the incomplete antibody is not monovalent as it is so frequently represented. It suggests rather that some hindrance to agglutination in saline by incomplete anti-D is removed when all C and E antigens are absent from the cells."

We have been able to confirm the report regarding this anomalous behavior of the Rh_0^x blood in tests with blocking anti-Rh_0 serum, but we cannot agree with Race's interpretation (cf. Sturgeon, 1952). A long time ago, we noticed that there is a reciprocal relationship between the agglutinability of Rh-positive red cells by anti-Rh sera and the ease with which they are blocked by Rh_0 univalent antisera. For example, old cell suspensions give lower titers in conglutination and agglutination tests, but higher titers in titrations carried out by the blocking technique. On the other hand, fresh cells homozygous for the Rh_0 factor generally give higher titers by the conglutination and agglutination methods and lower titers by the blocking technique. Almost all Rh_0 antisera contain a mixture of the univalent and bivalent forms of the antibody. In fact, many so-called "pure" blocking or "incomplete" Rh_0 antisera actually contain a certain amount of anti-Rh_0 agglutinin, which is masked by the more potent blocking antibody. With highly agglutinable cells such as the homozygous Rh_0^x (—D—/—D—) cells of Race et al., the hidden Rh_0 agglutinin will cause clumping of cells in tests carried out in saline media despite the presence of Rh_0 blocking antibody. This, in fact, is an important source of error in Rh-Hr blood typing, since most anti-rh′ and anti-rh″ reagents contain anti-Rh_0 blocking antibody. In tests against homozygous bloods of type Rh_0, which are not uncommon among Negroids, such reagents may give weak clumping in tests carried out in saline media. If the true nature of these reactions is not recognized, such blood specimens may be incorrectly classified as type Rh_1, Rh_2, or Rh_1Rh_2 instead of type Rh_0.

The Fisher-Race school have committed themselves to the theory that the Rh-Hr factors are inherited in threes by means of corresponding triply linked genes. The present observations provide further evidence refuting this concept. Unfortunately, instead of modifying their theory to conform with the observations, British workers have chosen to attempt to force the facts to fit their

preconceived ideas. They apparently are not disturbed by their inability to procure anti-Hr_0 (d) serum, or by the existence of agglutinogens such as Rh_1^w with four Rh-Hr blood factors instead of three (cf. table 5). To account for the agglutinogen Rh_0^x with but a single Rh-Hr factor, they have been com-

TABLE 5. PRESENT STATUS OF THE Rh SERIES OF ALLELIC GENES

GENE	CORRESPONDING AGGLUTINOGEN	BLOOD FACTORS PRESENT	REMARKS	INCIDENCE
1. *r*	rh	hr′ and hr″	Only 2 factors demonstrable	One of most common Rh genes in Caucasoids. Less common in Negroids. Rare or absent in Mongoloids
2. *r′*	rh′	rh′ and hr″	Only 2 factors demonstrable	Rare gene
3. *r″*	rh″	rh″ and hr′	Only 2 factors demonstrable	Rare gene
4. r^y	rh_y	rh′ and rh″	Only 2 factors demonstrable	Extremely rare gene
5. R^0	Rh_0	Rh_0, hr′, and hr″		Most common gene among Negroids; relatively uncommon in Caucasoids and Mongoloids
6. R^1	Rh_1	Rh_0, rh′, and hr″		Most common gene in Mongoloids; common also in Caucasoids and Negroids
7. R^2	Rh_2	Rh_0, rh″ and hr′		Relatively common gene in all races
8. R^z	Rh_z	Rh_0, rh′ and rh″		Very rare gene in Caucasoids and Negroids; less rare in Mongoloids
9. R^{1w}	Rh_1^w	Rh_0, rh′, rh^w and hr″	4 blood factors demonstrable	Relatively uncommon gene
10. $\mathfrak{R}^0$	$\mathfrak{R}h_0$	$\mathfrak{R}h_0$, hr′ and hr″		Rare in Caucasoids; more common in Negroids
11. $\mathfrak{R}^1$	$\mathfrak{R}h_1$	$\mathfrak{R}h_0$, rh′, and hr″		Rare in Caucasoids; more common in Negroids
12. $\mathfrak{R}^2$	$\mathfrak{R}h_2$	$\mathfrak{R}h_0$, rh″ and hr′		Rare in Caucasoids; more common in Negroids
13. R^{ou}	Rh	Rh_0 and hr′	Only 2 factors demonstrable	Rare gene present in Negroids; not yet found in Caucasoids
14. R^{oz}	Rh_0^x	Rh_0	Only a single blood factor demonstrable	Extremely rare agglutinogen in Caucasoids

pelled to postulate a deletion involving so-called gene pairs *C-c* and *E-e*. Similarly, no doubt, to account for the observations reported here, they will postulate a deletion involving the so-called gene pair *E-e*. On the other hand, if one takes into account the distinction between blood factor and agglutinogen and adopts the concept of inheritance by multiple allelic genes, one avoids the

necessity of resorting to such far-fetched supplementary assumptions. (cf. Wiener and Wexler, 1952).

SUMMARY

A new rhesus agglutinogen is described which is characterized by the presence of blood factors Rh_0 and hr′ and the absence of blood factors rh′, rh″, and hr″. This agglutinogen is not uncommon among Negroids but has so far not been encountered among Caucasoids. A simple method of designating the new agglutinogen as well as the related agglutinogen reported by Race et al. is proposed. The implications of the observations for the serology and genetics of the Rh-Hr types are discussed, and especially the effect on the medicolegal application of the tests in cases of disputed parentage.

REFERENCES

LAWLER, S. D., BERTINSHAW, D., SANGER, R., & RACE, R. R. 1951. Inheritance of the *Rh* blood groups; 150 families tested with anti-C-c-C^w-D-E and anti-e. *Ann. Eugen.* LOND. 15: 258–270.

LEVINE, P. 1943. The pathogenesis of erythroblastosis fetalis. *J. Pediat.* 23: 656–675.

MOURANT, A. E. 1945. A new rhesus antibody. *Nature* LOND., 155: 542.

RACE, R. R., MOURANT, E. E., LAWLER, S. D., & SANGER, R. 1948. The Rh chromosome frequencies in England. *Blood,* 3: 689–695.

RACE, R. R., & SANGER, R. 1950 *Blood Groups in Man.* Blackwell Scientific Publ., Oxford.

RACE, R. R., SANGER, R., & SELWYN, J. A. 1951. A possible deletion in a human Rh chromosome; a serological and genetical study. *Brit. J. Exp. Path.,* 32: 124–135.

RACE, R. R., & TAYLOR, G. L. 1943. A serum that discloses the genotype of some Rh-positive people. *Nature* (Lond.), 152: 300.

STURGEON, P. 1952. Studies on a "complete" and an "incomplete" Rh antiserum with cDe/cde and -D-/-D- cells. I. The native sera. *J. Immunol.,* 68: 277–285.

WIENER, A. S. 1945. Theory and nomenclature of the Hr blood factors. *Science,* 102: 479–482.

WIENER, A. S. 1946. Recent developments in the knowledge of the Rh-Hr blood types; tests for Rh sensitization. *Am. J. Clin. Path.,* 16: 477–497.

WIENER, A. S. 1950. Blood grouping in disputed parentage with special reference to the Rh-Hr blood types. *Proceedings of the International Society of Hematology,* 207–220, Grune and Stratton, N. Y. C.

WIENER, A. S., BELKIN, R. B., & SONN, E. B. 1944. Distribution of the A_1-A_2-B-O, M-N, and Rh blood types among negroes in New York City. *Am. J. Phys. Anthrop.,* 2 (N.S.): 187–194.

WIENER, A. S., DAVIDSOHN, I., & POTTER, E. L. 1945. Heredity of the Rh blood types. II. Observations on the relation of factor Hr to the Rh blood types. *J. Exp. Med.,* 81: 63–72.

WIENER, A. S., & GORDON, E. B. 1949. Studies on the blood factor rh^w. *Amer. J. Clin. Path.,* 19: 621–629.

WIENER, A. S., & GORDON, E. B. 1951. Repartition des facteurs de groupes sanguine dans une population de New York City. *Rev. d'Hêmatol.,* 6: 46–50.

WIENER, A. S., GORDON, E. B., & HANDMAN, L. 1949. Heredity of the Rh blood types.

VIII. Additional family studies with special reference to factor rh^{w}. *Am. J. Human Genet.*, 1: 127–140.

WIENER, A. S., & PETERS, H. R. 1948. Intragroup incompatibility due to the hr″ factor. *Am. J. Clin. Path.*, 18: 533–536.

WIENER, A. S., SONN, E. B., & BELKIN, R. B. 1943. Heredity and distribution of the Rh blood types. *Proc. Soc. Exp. Biol.* 54: 238–240.

WIENER, A. S., SONN, E. B., & BELKIN, R. B. 1944. Heredity of the Rh blood types. *J. Exp. Med.* 79: 235–253.

WIENER, A. S., UNGER, L. J., & SONN, E. B. 1945. New data on the distribution of the Rh blood types. *Proc. Soc. Exp. Biol.* 58: 89–92.

WIENER, A. S., & WEXLER, I. B. 1952. The mosaic structure of red blood cell agglutinogens. *Bact. Rev.* 16: 69–87.