

BLOOD GROUPS

“Blood group,” in this dictionary, refers to an entire blood group system consisting of erythrocyte antigens, the specificity of which is controlled by a series of genes, either allelic or else so very closely linked on a single chromosome they cannot be distinguished from alleles using methods now available. “Blood type” and “phenotype” here refer to a specific pattern of reaction to testing antisera within a given system. This usage is not universal. In some current literature, a single system may be referred to in the plural form (i.e., ABO blood groups), or the term “blood group” may be assigned to a single phenotype (i.e., blood group A). Each blood group is thus defined in the terms of reaction to the original antiserum with which the system was discovered.

In discussion of the blood groups themselves, many entries shown here include symbols and shortened forms as they are often found in medical literature, where they often appear with no specification or explanation that a blood group is being discussed. Symbols for genes and genotypes are set in italics. Symbols for gene products or antigens, antisera, and phenotypes are set in ordinary (i.e., roman) type. In the Rh-Hr terminology for the Rh blood group, roman type is also used to designate antigenic substances. **Boldface** type denotes serologic factors and corresponding antibodies. Although most sources follow this way of writing these out, styling is far from uniform, so that all documentation ought to be approached with caution until the orthography being used has been verified by the reader or student.

ABO blood group

This is the most significant blood group system in transfusion practice and the only one for which reciprocal antibodies are consistently present in sera. These antibodies may cause severe intravascular hemolysis if ABO-incompatible blood is transfused into such patients.

In normal human blood, a reciprocal relationship exists between ABO antigens or agglutinogens on the surface of the erythrocyte and the natural antibodies or isoagglutinins found in serum. People with type O have neither A nor B antigens on erythrocytes, although their serum contains both anti-A and anti-B agglutinins. Those with type A have antigen A on erythrocytes and anti-B in serum. People with type B have antigen B in erythrocytes and anti-A in serum. Those with type AB have both A and B antigens on erythrocytes but no isoagglutinins in serum. Types A and AB may be subdivided by anti- A_1 serum into types A_1 as well as A_2 , A_1B , and A_2B . A_2 antigen reacts more weakly than A_1 , but such difference is also qualitative. Production of ABO antigens is controlled by a series of allelic genes A_1 , A_2 , B, and O (sometimes designated I^A , I^{A_1} , I^B , and i ; i^{A_1} , i^{A_2} , i^B , and i ; or A_1 , A_2 , a^B , and a) A_1 is dominant to A_2 , and both are dominant to O; no dominance exists between A_1 and B nor between A_2 and B.

In the usual typing method, a strong anti-A serum that agglutinates cells containing A_1 or A_2 antigen is used; cells agglutinated by this serum but not by anti-B are of type A but may be of genotype A_1A_1 , A_1A_2 , A_1O , A_2A_2 , or A_2O . Cells of people of type A that are agglutinated by anti- A_1 are of type A_1 and may be of genotype A_1A_1 , A_1A_2 , or A_1O ; type A cells not agglutinated by anti- A_1 are of type A_2 and may be of genotype A_2A_2 or A_2O . Cells agglutinated by anti-B but not anti-A are of type B

and may be of genotype BB or BO . Cells agglutinated by both anti-A and anti-B are of type AB and can be divided into types A_1B (genotype A_1B) and A_2B (genotype A_2B) by anti- A_1 . Cells not agglutinated by either anti-A or anti-B are of type O and genotype OO . Cells of type O do not simply lack antigenic substance; most have an antigen called H that is chemically similar to antigens A and B and is probably the precursor antigen that is modified under the influence of genes A_1 , A_2 , and B into their corresponding antigens.

The designation “Bombay” phenotype was assigned to those whose cells lack A, B, and H antigen and whose serum contains anti-A, anti-B, and anti-H; they are also referred to as having the “Oh” phenotype. In addition, weak variants of antigen A have been described with phenotypes designated A_3 , A_4 , A_5 , A_x , and A_z ; more rarely, weak variants of B have been found. The ABO types are of prime importance with respect to blood transfusion, and maternal-fetal incompatibility is a frequent cause of fetal death and erythroblastosis fetalis.

Auberger (Au) blood group

This erythrocyte antigen was first found in the serum of a Frenchwoman, one Madame Auberger, who had had many transfusions. This blood group is associated with the Lutheran system; Au^a and Au^b are considered an allelic pair within the Lutheran focus (now designated as Lu18 and Lu19). Au^a antigen is inherited as a dominant trait and occurs in about 80% of whites and blacks.

Bg blood group

This group was discovered and classified in 1967. Three specificities were determined: Bg^a , Bg^b , and Bg^c . Its antibodies direct themselves to the human leukocyte antigen.

Cartwright blood group

Discovered in 1956, it involves the Yt system, which is composed of two antigens, Yt^a (98% incidence in the general population) and Yt^b (8% incidence in the same population). Anti- Yt^a and Yt^b are IgG antibodies that are clinically important because they are predominantly stimulated by erythrocytes.

CDE blood group

This group consists of Rh system antigens. D is the most important erythrocyte antigen after A and B. Anti-D is formed from exposure, through transfusion, and in pregnancy to erythrocytes containing the D antigen. SEE ALSO Rh blood group.

Chido-Rodgers blood group

This classification includes nine antigens as follows: six Chido, two Rodgers, and WH. High incidence antigens are CH1, CH2, CH3, RG1, and RG2 (CH1 is found in nearly 100% of random populations, and RG1 in nearly 98%).

All are poorly expressed on cord cells, adsorbed onto the red blood cell membrane in people with antigen-positive plasma, and sensitive to treatment with ficin, papain, and pronase. The presence of anti-Ch/Rg antibodies often complicates testing undertaken before transfusions and also in antibody identifica-

tion because they tend to obscure detection of more important underlying antibodies such as anti-K, Jk, Fy, Rh, or Vel.

Colton blood group

This system comprises three antigens: Co^a, Co^b, and Co³ (the last was formerly designated as Co^{ab}.) Co^a and Co^b are antithetical partners inherited as codominant antigens on chromosome 7. Co^a is a high-frequency antigen occurring in 99.9% of most random populations. Co^b occurs in about 10% of most random populations. Anti-Co^a and anti-Co^b are encountered only rarely and have been associated with transfusions reactions.

Cromer blood group

Delineated first in 1965, this system has seven high-incidence antigens (Cr^a, Tc^a, Dr^a, Es^a, IFC, UMC, and Wes^b). Three low-incidence antigens have also been isolated: Tc^b, Tc^c, and WES^a. These antigens are to be found in serum, plasma, leukocytes, and in placental tissue, although they may be depressed during pregnancy. Another antigen, GUTI, was more recently isolated using reverse transcriptase polymerase chain reaction.

Diego blood group

The erythrocyte antigen defined by anti-D^a antibody was first isolated in Venezuela, where it caused erythroblastosis fetalis. An antibody with antithetical reactions, anti-D^b, was discovered in 1967. The antigen system is controlled by two alleles, Di^a and Di^b. The blood group antigens of this system are caused by a single amino acid variation in the *SLCA1* gene, which encodes the band-3 protein of the erythrocyte membrane. The Di^a antigen is common in Native Americans and Asians but is apparently absent in whites. Two independent pairs of antigens, Dia/Dib and Wra/Wrb, comprise the Diego system. Antibodies to Dia can cause significant hemolysis, including hemolytic disease of the newborn.

Dombrock blood group

This group includes antigens Doa and Dob and is found slightly more often in white patients. The chromosomal location has not, according to some authorities, been assigned. Antibodies to DO are seldom encountered but are more amenable to identification using polyethylene glycol or with enzyme enhancement. Antibodies to Do antigens are implicated in a very mild form of hemolytic disease of the newborn, and the development of anti-Do antibodies may cause severe hemolytic transfusion reactions. They are more often encountered in combination with other antibodies than alone.

Duffy blood group

These antigens were first isolated in a patient named Duffy who had hemophilia. Antigens Fy^a and Fy^b on the Duffy glycoprotein can result in four possible phenotypes, as follows: Fy(a+ b-), Fy(a+ b+), Fy(a- b+), and Fy(a- b-). The Duffy glycoprotein acts as the receptor for *Plasmodium vivax*, a malarial parasite; the resistance of people in West Africa to *P. vivax* malaria appears to be due to natural selection of the Fy(a- b-) phenotype.

Blood from whites is agglutinated by one or both antigens; most blacks, however, and some Yemeni Jews have negative test reactions to both antigens. It is therefore assumed that the pro-

duction of Duffy antigens is controlled by a series of at least allelic genes: Fy^a, Fy^b, and Fy, with antibodies specific for the first two of the series now known. People whose blood reacts positively to anti-Fy^a and negatively to anti-Fy^a are of phenotype Fy(a+ b-), and their genotype may be either Fy^aFy^a or Fy^aFy. Those of phenotype Fy(a+ b+) are of genotype Fy^aFy^b. Those of phenotype Fy(a- b+) are of genotype Fy^bFy^b or Fy^bFy. Those of phenotype Fy(a- b-) are of genotype FyFy. Duffy antibodies occasionally cause transfusion reactions or erythroblastosis fetalis.

Gerbich blood group

This complex system comprises three high-incidence antigens (Ge2, Ge3, and Ge4) and four of low-incidence (Wb, Ls^a, An^a, and Dh^a). Incidence of the three high-incidence antigens is found in all populations, except in Melanesians. Antibodies to them are predominantly IgG, erythrocyte stimulated and variably bind complement. Anti-Ge2 and anti-Ge3 are noted to elicit acute reactions on transfusion but have not been identified in hemolytic disease of newborns.

High-frequency blood groups

This group of antigens is found in most people but is absent in members of a very few families. Because of such high frequency, these antigens are often called "public antigens." The antibodies usually have been found in the serum of a patient lacking the antigen who has become immunized by transfusion or pregnancy. Names or symbols applied to public antigens include Vel, Yt^a, Ge, Lan, and Sm. SEE ALSO low-frequency blood groups.

I blood group

These erythrocyte antigens are defined by reactions to antibodies designated anti-I and anti-i. Antigen I differs from other blood group antigens in the slowness of its development and in its wide range of strength in different people; the range approximates a normal distribution curve. I antigens are present in subterminal regions of oligosaccharides, which are converted to H, A, and B antigens. Anti-I occurs in a wide range of strength and in two forms: autoanti-I is the antibody usually found in the serum of patients with acquired hemolytic anemia of the "cold agglutinin" type and in sera described as containing "nonspecific complete cold autoagglutinin"; natural anti-I or isoanti-I occurs regularly in the serum of people with phenotype i. Phenotype I may be divided into types i₁ and i₂, both rare in adults.

Indian blood group

This is the most recently discovered blood group. It is composed of two antithetical antigens, In^a, which is of somewhat low incidence, and In^b, which is of high incidence (96% of whites, and 96% of Indians). The antigens are inherited by codominant alleles on chromosome 11 and are denatured by most enzymes. Anti-In^b has been linked with hemolytic transfusion reactions. Anti-In^a has not been so linked, however. Neither has been linked to hemolytic disease of the newborn.

Kell blood group

These erythrocyte antigens are defined by an immune antibody, anti-K, first found in the serum of a Mrs. Kellacher, which reacted with the erythrocytes of her newborn infant, her older daughter, her husband, and some 96% of the random population. Its antithetical partner, k, is also called Cellano. Later, two additional antithetical antigens, Kp^a and Kp^b , were described. The null phenotype, designated K_0 , was discovered at around the same time. The Kell system also included J_s^a and J_s^b antigens.

The K antigen is present in 90% of whites and 2% of blacks. It is strongly immunogenic and is frequently found in sera of transfused patients. As a cause of transfusion reactions and erythroblastosis fetalis, the Kell blood group is next in clinical importance after the ABO and Rh blood groups.

The antigens K and k, which may play an important role in the structure and function of cell membranes, are controlled by a pair of allelic genes without dominance, hence three genotypes (KK , Kk , kk) may be recognized by agglutination of erythrocytes by anti-K alone, both anti-K and anti-k, or anti-k alone.

Kidd blood group

These erythrocyte antigens, also referred to as the JK blood group, are defined by reactions to an antibody designated anti- Jk^a , discovered in the serum of a Mrs. Kidd who had an infant with erythroblastosis and by reactions to its antithetical serum, anti- Jk^b . The antigens are controlled by a pair of codominant genes, Jk^a and Jk^b , that are genetically independent of other blood group genes.

People with erythrocytes that are agglutinated by anti- Jk^a but not by anti- Jk^b are of phenotype $Jk(a+ b-)$ and genotype $Jk^a Jk^b$. This carries a double dose of the antigen, which can give inconclusive panel results if only single dose panel cells are used in the procedure.

Knops blood group (KN)

This group comprises five antigens: Kn^a (Knops), Kn^b , McC^a (McCoy), Si^a , and Yk^a (York). All except Kn^b have been described as high-incidence antigens. KN antibodies are characterized as IgG, erythrocyte stimulated. They react weakly and highly variably.

Xk blood group

The Xk blood group system contains the Kx antigen, the expression of which increases when Kell antigens are denatured.

Landsteiner-Wiener blood group

This is the Rh blood group system. The antibody Rh was reported by Landsteiner and Wiener in 1940 after it had been produced by guinea pigs and rabbits that had been transfused with rhesus monkey erythrocytes.

Lewis blood group, Le blood group

These antigens of erythrocytes, saliva, and certain other body fluids are defined by reactions to anti- Le^a antibody, first found in the serum of a Mrs. Lewis by reactions to related sera, particularly anti- Le^b , and by interactions with the secretor factor. Lewis antigens are formed in tissue under control of genes designated Le and le (Le dominant to le) and released into body

fluids, where they may be absorbed onto the surface of erythrocytes and determine the reactions of erythrocytes to antisera. Lewis erythrocyte types in children may not develop fully until about 6 six years of age. Lewis genes are genetically independent of those controlling the secretor factor (Se and se), but their products interact in certain phenotypic effects. Several theories have been proposed to account for the complex immunologic and genetic observations. The theory of Grubb and Ceppellini was summarized by Race and Sanger, as shown in Table 1.

Variant antibodies of this system include the following: anti- Le^x (originally called anti-X), which seems to be anti- Le^a plus anti- Le^b ; anti- Le^c , an immune rabbit serum that agglutinates $Le(a-b-)$ cells; and the Magard antiserum, obtained from a patient with carcinoma of the stomach, that agglutinates strongly the cells of $A_1 Le(a-b-)$ secretors and less strongly those of $A_2 Le(a-b-)$ secretors. Lewis antibodies have occasionally been implicated in transfusion reactions.

Low-frequency blood groups

In this group of erythrocyte antigens, each is defined by a specific antiserum, and each is found only in members of a very few families. Because of their rarity, these antigens are often referred to as "private antigens." The antibodies usually have been found in the sera of patients who have received transfusions or in mothers of infants with erythroblastosis. These antigens are often named for the family in which they were first discovered. The names or symbols assigned to some private antigens are Levay, Jobbins, Becker, Ven, Chr^a , Wright or Wr^a , Be^a , By, Swann or Sw^a , Good, Biles or Bi , Tr^a , Stobo, Ot, Ho, and Webb. SEE ALSO High-frequency blood groups.

Lutheran blood group

Lutheran blood group, or Lu blood group, includes Lu^a and Lu^b antigens. Anti- Lu^a and anti- Lu^b are uncommon. They are produced after pregnancy or transfusion but have also occurred with apparent erythrocyte stimulation. Only mild hemolysis has been reported with these antibodies.

Table 1: Secretor-Lewis Interactions*

Genotypes	Antigens				Of Erythrocytes
	Of Saliva				
	ABH	Le^a	Le^{bL}	Le^{bH}	
$SeSe LeLe$ $SeSe Lele$ $Sese LeLe$ $Sese Lele$	+	+	+	+	$Le(a-b+)$
$sese LeLe$ $sese Lele$	-	+	-	-	$Le(a+b-)$
$SeSe lele$ $Sese lele$	+	-	-	+	$Le(a-b-)$
$sese lele$	-	-	-	-	$Le(a-b-)$

* From Race RR, Sanger R. *Blood Groups in Man*, 4th ed. Oxford, England: Blackwell Scientific Publications; 1962.

MNSs blood group

This system of erythrocyte antigens was originally defined by reactions to immune rabbit sera designated anti-M and anti-N and has since been extended by reactions to sera anti-S, anti-s, and certain others. These low-incidence antigens are present on glycoproteins A and B. When tested with the readily available anti-M and anti-N sera only, the erythrocytes of all people may be assigned to one of three classes, M, N, or MN, depending on whether they are agglutinated by one or both antisera.

Production of M and N antigens is controlled by two allelic genes, M and N; people of genotype *MM* are type M, those of genotype *NN* are type N, and those of the heterozygous genotype *MN* are type MN. Because anti-M and anti-N sera are commercially available and the reactions are simple and reliable, they are widely used for medicolegal blood testing in disputed paternity actions and for genetic linkage and population studies. The *MN* locus is not genetically linked to loci of other major blood group systems. Anti-M antibody rarely causes hemolysis, whereas anti-S antibodies can cause significant hemolysis.

With the discovery of anti-S serum and its reciprocal anti-s, it was shown that the MNS group is complex and that nine phenotypes representing ten genotypes may be defined by the antisera. In addition, nearly 1% of blood samples in blacks will lack both S and s. An antibody designated anti-U has been found that reacts with both S and s antigens. Weak variants of the M and N antigens that react with some anti-M or anti-N sera but not with others have been designated *M*₂ or *N*₂. A qualitative variant of M has been designated *M*₁. An antigen that gives intermediate reactions has been designated *M*^c. An extremely rare variant of M detected with a special serum has been designated *M*^g.

Antigens designated *Hu* and *He* are detected in sera obtained from rabbits immunized with the blood of certain blacks and are found almost entirely in people of African ancestry. Anti-*Hu* gives distinct reactions only with cells that contain N, and most people who are positive for anti-*He* have both N and S. Other rare antigens that are associated with or are variants of the MNS group have been designated *Mi*², *Vw* (identical to *Gr*), and *Mu*.

P blood group

This group includes PI antigen only (P, Pk, and LKE antigens, formerly part of the P system, have been assigned to the globoside category of antigens.) People who are PI negative commonly have the anti-PI antibody. Anti-PI is predominantly immunoglobulin M, so it does not cross the placenta, does not cause hemolytic diseases of the newborn, and only rarely causes hemolysis.

Rh blood group

This complex system of erythrocyte antigens was originally defined by reactions to serum from rabbits or guinea pigs immunized with blood of the rhesus monkey, but it now defined by reactions to a series of human antisera usually obtained from people immunized by transfusion or pregnancy. (SEE ALSO discussion at Landsteiner-Weiner blood group.)

The nomenclature of genes, antigens, and antisera of this system has undergone an evolution reflecting the acquisition of knowledge since 1940, and conflicting systems of nomenclature have been used. The most widely used systems of nomenclature are those proposed by Wiener (Rh-Hr system) and by Fisher and Race (CDE system), both of which have been frequently modified and extended, and that proposed by Rosenfield and col-

Table 2: Gene Designations in Three Nomenclature Systems*

Rh-Hr (Wiener)	CDE (Fisher-Race)	Numeric (Rosenfield <i>et al.</i>)
<i>r</i>	<i>cde</i>	<i>R</i> ^{-1, -2, -3, 4, 5}
<i>R</i> ¹	<i>CDe</i>	<i>R</i> ^{1, 2, -3, -4, 5}
<i>R</i> ²	<i>cDE</i>	<i>R</i> ^{1, -2, 3, 4, -5}
<i>R</i> ⁰	<i>cDe</i>	<i>R</i> ^{1, -2, -3, 4, 5}
<i>r</i> '	<i>Cde</i>	<i>R</i> ^{-1, 2, -3, -4, 5}
<i>r</i> ''	<i>cdE</i>	<i>R</i> ^{-1, -2, 3, 4, -5}
<i>R</i> ^z	<i>CDE</i>	<i>R</i> ^{1, 2, 3, -4, -5}
<i>r</i> ^y	<i>CdE</i>	<i>r</i> ^{-1, 2, 3, -4, -5}

(Paired combinations of the above give rise to 36 genotypes)

Table 3: Rh Antiserum Designations in Three Nomenclature Systems

Rh-Hr (Wiener)	CDE (Fisher-Race)	Numeric (Rosenfield <i>et al.</i>)
Standard Antisera		
Rh ₀	D	Rh1
rh'	C	Rh2
rh''	E	Rh3
hr'	c	Rh4
hr''	e	Rh5
Other Antisera		
hr	f, ce	Rh6
rh	Ce	Rh7
rh ^{w1}	C ^w	Rh8
rh ^x	C ^x	Rh9
hr ^v	V, ce ^s	Rh10
rh ^{w2}	E ^w	Rh11
rh ^G	G	Rh12
Rh ^A	No term	Rh13
Rh ^B	No term	Rh14
Rh ^C	No term	Rh15
Rh ^D	No term	Rh16
Hr ₀	No term	Rh17
Hr	No term	Rh18
hr ^S	No term	Rh19
Hr ^H plus hr ^v	VS, e ^s	Rh20
No term	C ^G	Rh21

* Tables 2 and 3 are adapted from Rosenfeld RE, Allen FH Jr., Swisher SN, Kochwa S. A review of Rh serology and presentation of a new terminology. *Transfusion*. 1962;2:287.

leagues (numeric system). Certain differences of theoretic interpretations of both immunology and genetics are associated with the different systems of nomenclature. It is agreed that the production of Rh antigens is controlled by a complex series of genes located on a single chromosome, that eight major genes or gene complexes control production of qualitatively different antigens, that the paired arrangements of these eight genes give rise to 18 qualitative phenotypes or erythrocyte patterns recognizable by reactions with five standard commercially available antisera, and that many more antigens or antigen combinations exist, with both qualitative and quantitative differences recog-

Table 4: Rh Phenotypes Defined by Five Standard Antisera

Antiserum Reactions					Phenotypes		
Rh. D	rh' C	rh'' E	hr' c	hr'' e	Rh-Hr	CDE	Numeric
Rh1	Rh2	Rh3	Rh4	Rh5			
-	-	-	+	+	rh	cde/cde	Rh: -1, -2, -3, 4, 5
+	+	-	+	+	Rh ₁ rh	CDe/cde*	Rh: 1, 2, -3, 4, 5
+	+	-	-	+	Rh ₁ Rh ₁	CDe/CDe*	Rh: 1, 2, -3, -4, 5
+	+	+	+	+	Rh ₂ Rh ₀	CDe/CDe*	Rh: 1, 2, 3, 4, 5
+	-	+	+	+	Rh ₂ rh	cDe/cde*	Rh: 1, -2, 3, 4, 5
+	-	-	+	+	Rh ₀	cDe/cde*	Rh: 1, -2, -3, 4, 5
+	-	+	+	-	Rh ₂ Rh ₂	CDe/CDe*	Rh: 1, -2, 3, 4, -5
-	+	-	+	+	rh'rh	Cde/cde	Rh: -1, 2, -3, 4, 5
-	-	+	+	+	rh''rh	cDe/cde	Rh: -1, -2, 3, 4, 5
+	+	+	-	+	Rh ₂ Rh ₁	CDE/CDe*	Rh: 1, 2, 3, -4, 5
+	+	+	+	-	Rh ₂ Rh ₂	CDE/CDe*	Rh: 1, 2, 3, 4, -5
-	+	+	+	+	rh'rh	Cde/cde*	Rh: -1, 2, 3, 4, 5
-	+	-	-	+	rh'rh'	Cde/Cde	Rh: -1, 2, -3, -4, 5
-	-	+	+	-	rh''rh''	cde/cde	Rh: -1, -2, 3, 4, -5
-	+	+	-	+	rh ₁ rh'	CDe/Cde	Rh: -1, 2, 3, -4, 5
-	+	+	+	-	rh ₂ rh''	CDe/cde	Rh: 1, 2, 3, 4, -5
+	+	+	-	-	Rh ₂ Rh ₂	CDE/CDE*	Rh: 1, 2, 3, -4, -5
-	+	+	-	-	rh ₁ rh ₂	CDe/CDe	Rh: -1, 2, 3, -4, -5

* Expressed as most common of two or more genotypes of this phenotype

nizable by a series of other antisera not generally available with reaction specificities that reflect wide immunologic and genetic variation.

The Rh-Hr nomenclature implies that all Rh genes constitute a single set of multiple alleles located at a single chromosome locus and that a single gene may control production of an erythrocyte antigen or agglutinin possessing many factors, each of different antibody specificity. The CDE nomenclature implies three contiguous chromosome loci or areas of genetic information, each with a series of two major and probably several minor alleles, each controlling production of a specific erythrocyte antigen. The numeric system assigns an arbitrary number to each known antiserum, uses numerals 1 through 5 for the five standard antisera and higher numbers for others, and classifies people by positive or negative reactions to the antisera used. The correspondences of the three systems of nomenclature with respect to designations for genes, phenotypes, and antisera are given in Tables 2, 3, and 4.

The symbol Du is used in the CDE nomenclature to designate antigens that are agglutinated by some anti-D sera and not by others, and these apparently represent weak or incomplete forms of the D antigen. Many varieties of Du exist. People with Du may be mistyped as D negative (d), but Du blood may stimulate the formation of anti-D if it is transfused to a D-negative recipient. Some people with Du have made anti-D when they were transfused with D-positive blood. In the Rh-Hr nomenclature, weak variants are indicated by using Germanic (i.e. black letter or fraktur) type (e.g., **Rh₀**).

Scianna blood group

This is composed of Sc1, Sc2, and Sc3 antigens. Sc1 and Sc2 are antithetic antigens inherited as codominant alleles on chromosome 1. Sc1 is a high-incidence antigen that occurs in virtually 100% of random populations. Anti-Sc1 and anti-Sc2 are rarely seen.

Sutter blood group

This erythrocyte antigen, part of the Kell blood group, is defined by reaction to an antibody designated anti-Js^a, first found in the serum of a Mr. Sutter, who had been previously transfused. The Js^a antigen is inherited as a dominant trait. It occurs in about 20% of American blacks but is rare in other ethnic groups. Sutter antibodies have been implicated in transfusion reactions and in hemolytic disease of the newborn. Js^a is a high-frequency antigen that is antithetical to Js^a.

Vel blood group

This high-frequency antigen occurs in more than 99.9% of random populations. Its high-frequency antigens are only rarely associated with antibodies directed against them because only rare patients lack these antigens. Other high-frequency antigens include k and Lu^b.

Wright blood group

This classification includes two allelic antigens: Wr^a (low incidence) and Wr^b (high incidence). Anti- Wr^a has frequently been reported as naturally occurring IgM and immune-stimulated IgG. IgG anti- Wr^a can cause hemolytic disease of the newborn, as well as transfusion reactions.

XG blood group

This erythrocyte antigen is defined by reaction to an antibody designated anti-Xg^a, which was found in the serum of a patient who had received many transfusions. In contrast to all other known blood group antigens, Xg^a proved to be controlled by a gene located on the X chromosome and is thus the only known sex-linked blood group. Anti-Xg^a has been used for genetic linkage studies of diseases caused by sex-linked genes. Anti-Xg^a antibody is uncommon. It usually reacts only to antiglobulin testing, and has not been reported to cause significant hemolysis.

YT blood group

Also called Cartwright blood group system. Composed of two antigens: Yta (high incidence) and Ytb (low incidence). There are three phenotypes: (Yt(a+b-), Yt(a+b+), and Yt(a-b-). No null phenotype [Yt(a-b-)] has been observed. The Yta antigen is not well developed at birth. Anti-Yta and anti-Ytb are both IgG antibodies considered clinically important because they are predominantly stimulated by erythrocytes.