

# Organization of the data

Section II is organized into four main parts: (i) the blood group systems; (ii) the blood group collections; (iii) the 700 Series of Low-Incidence Antigens; and (iv) the 901 Series of High-Incidence Antigens. For the systems, facts pertaining to the gene, carrier molecule, and antigens, and the clinical relevance of antibodies are given. For the collections, series of low-incidence antigens, and series of high-incidence antigens, facts pertaining to the antigens and antibodies are given. All are listed in ISBT numerical order. The format for the facts sheets displaying the data about the systems and the antigens is explained below; that for the collections and high-incidence antigens is similar. The 700 Series of Low-Incidence Antigens are given in a table. Section III consists of facts in lists and tables where the information encompasses more than one antigen or blood group system. Other related information is also included. We will use “prevalence” throughout this book, because it is the word used to describe a permanent/inherited characteristic on the phenotypic level.

## ISBT Blood Group Systems

Both the ISBT system symbol and traditional name are used in the section headings. Information is provided under the following headings.

### Number of Antigens

The total number of antigens in the system is indicated. The antigens are listed under the headings: “polymorphic” (in blue font); high prevalence (in red font); and low prevalence (in green font). Table 2.1A lists the blood group systems and antigens currently recognized by the ISBT Working Party for Red Cell Immunogenetics and Blood Group Terminology. If a number assigned to an antigen becomes inappropriate, the number becomes obsolete and is not reused; such antigens are noted with three small dots (...).

### Terminology

Both the traditional name and the ISBT symbol for the blood group system are given as headers. Under this subheading, the ISBT symbol, ISBT number (parenthetically)<sup>1-4</sup>, other, obsolete, names that have been associated with the

**TABLE 2.1A** Blood group antigens assigned to each system

		Antigen number																	
System		001	002	003	004	005	006	007	008	009	010	011	012	013	014	015	016	017	018
001	ABO	A	B	A,B	A1	...													
002	MNS	M	N	S	s	U	He	Mi <sup>a</sup>	M <sup>c</sup>	Vw	Mur	M <sup>g</sup>	Vr	M <sup>e</sup>	Mt <sup>a</sup>	St <sup>a</sup>	Ri <sup>a</sup>	Cl <sup>a</sup>	Ny <sup>a</sup>
003	P1PK	P1	...	p <sup>k</sup>	NOR														
004	RH	D	C	E	c	e	f	Ce	C <sup>W</sup>	C <sup>X</sup>	V	E <sup>W</sup>	G	...	...	...	...	Hr <sub>0</sub>	Hr
005	LU	Lu <sup>a</sup>	Lu <sup>b</sup>	Lu3	Lu4	Lu5	Lu6	Lu7	Lu8	Lu9	...	Lu11	Lu12	Lu13	Lu14	...	Lu16	Lu17	Au <sup>a</sup>
006	KEL	K	k	Kp <sup>a</sup>	Kp <sup>b</sup>	Ku	Js <sup>a</sup>	Js <sup>b</sup>	...	...	Ul <sup>a</sup>	K11	K12	K13	K14	...	K16	K17	K18
007	LE	Le <sup>a</sup>	Le <sup>b</sup>	Le <sup>ab</sup>	Le <sup>bH</sup>	ALe <sup>b</sup>	BLe <sup>b</sup>												
008	FY	Fy <sup>a</sup>	Fy <sup>b</sup>	Fy3	...	Fy5	Fy6												
009	JK	Jk <sup>a</sup>	Jk <sup>b</sup>	Jk3															
010	DI	Di <sup>a</sup>	Di <sup>b</sup>	Wr <sup>a</sup>	Wr <sup>b</sup>	Wd <sup>a</sup>	Rb <sup>a</sup>	WARR	ELO	Wu	Bp <sup>a</sup>	Mo <sup>a</sup>	Hg <sup>a</sup>	Vg <sup>a</sup>	Sw <sup>a</sup>	BOW	NFLD	Jn <sup>a</sup>	KREP
011	YT	Yt <sup>a</sup>	Yt <sup>b</sup>																
012	XG	Xg <sup>a</sup>	CD99																
013	SC	Sc1	Sc2	Sc3	Rd	STAR	SCER	SCAN											
014	DO	Do <sup>a</sup>	Do <sup>b</sup>	Gy <sup>a</sup>	Hy	Jo <sup>a</sup>	DOYA	DOMR	DOLG										
015	CO	Co <sup>a</sup>	Co <sup>b</sup>	Co3	Co4														
016	LW	...	...	...	...	LW <sup>a</sup>	LW <sup>ab</sup>	LW <sup>b</sup>											
017	CH/RG	Ch1	Ch2	Ch3	Ch4	Ch5	Ch6	WH				Rg1	Rg2						

		Antigen number																	
System		001	002	003	004	005	006	007	008	009	010	011	012	013	014	015	016	017	018
018	H	H																	
019	XK	Kx																	
020	GE	...	Ge2	Ge3	Ge4	Wb	Ls <sup>a</sup>	An <sup>a</sup>	Dh <sup>a</sup>	GEIS	GEPL	GEAT	GETI						
021	CROM	Cr <sup>a</sup>	Tc <sup>a</sup>	Tc <sup>b</sup>	Tc <sup>c</sup>	Dr <sup>a</sup>	Es <sup>a</sup>	IFC	WES <sup>a</sup>	WES <sup>b</sup>	UMC	GUTI	SERF	ZENA	CROV	CRAM	CROZ		
022	KN	Kn <sup>a</sup>	Kn <sup>b</sup>	McC <sup>a</sup>	SI1	Yk <sup>a</sup>	McC <sup>b</sup>	SI2	SI3	KCAM									
023	IN	In <sup>a</sup>	In <sup>b</sup>	INF1	INJA														
024	OK	Ok <sup>a</sup>	OKGV	OKVM															
025	RAPH	MER2																	
026	JMH	JMH	JMHK	JMHL	JMHG	JMHM	JMHQ												
027	I	I																	
028	GLOB	P																	
029	GIL	GIL																	
030	RHAG	Duclos	Ol <sup>a</sup>	DSLK	RHAG4														
031	FORS	FORS																	
032	JR	Jr <sup>a</sup>																	
033	LAN	Lan																	

... = Obsolete numbers. For the obsolete antigens see [www.isbt-web.org](http://www.isbt-web.org).

system, the CD number (if any), and a brief description of how the system was named are given.

## Expression

This section relates to the component on which the antigens in a blood group system are carried. Several blood group antigens occur naturally in soluble form in body fluids (e.g., saliva, urine, plasma). If a naturally occurring soluble form of the carbohydrate antigen or carrier protein is available, it will be indicated in this section. Soluble forms of an antigen can be used for inhibition tests to confirm or eliminate antibody activity. If the soluble form of the antigen is to be used for inhibition studies, the substance must be obtained from a person who inherited the antigen of interest and be made isotonic. An ideal negative dilution control for this test would be the particular fluid from a person who did not inherit the antigen of interest<sup>5</sup>. A soluble form of many antigens can be produced through recombinant technology.

Some of the components carrying blood group antigens have been detected on other blood cells and tissues by use of various methods, including testing with polyclonal and monoclonal antibodies or by Northern blot analysis. However, it cannot be assumed that detection of the carrier molecule equates with the expression of the RBC antigen(s).

Under the subheading “Other blood cells,” we refer to cells in the peripheral blood excluding RBCs because it goes without saying that RBCs express all the components carrying blood groups (except in null phenotypes).

## Gene

The chromosomal location for the genes encoding proteins associated with blood group systems is taken from original papers and reviews of the chromosome assignments<sup>6</sup> and how blood groups were cloned<sup>7</sup>. The chromosome number, arm [short p (upper on diagrams); long q (lower on diagrams)], and band number are given. The name of the gene used is that recommended by the ISBT, with alternative names in parenthesis. If the presence of a blood group antigen is detected by serological means, the gene is named by the corresponding ISBT system symbol, an asterisk followed by the antigen number, in italics to indicate the specific allele, e.g., *FY\*01* for the allele encoding Fy<sup>a</sup>. The organization of the gene in terms of number of exons, kilobase pairs of gDNA, and an overview map are provided. The gene product name (and alternatives) is given.

## Database accession numbers

Key GenBank accession numbers and the Entrez gene number are given. The Blood Group Antigen Gene Mutation Database (<http://www.ncbi.nlm.nih.gov/gv/rbc/xslcgi.fcgi?cmd=bgmute/home> or enter “dbRBC” in

**TABLE 2.1B** Blood group antigens assigned to each system: extension 1

		Antigen number																			
System		019	020	021	022	023	024	025	026	027	028	029	030	031	032	033	034	035	036	037	038
002	MNS	Hut	Hil	M <sup>v</sup>	Far	s <sup>D</sup>	Mit	Dantu	Hop	Nob	En <sup>a</sup>	ENKT	“N”	Or	DANE	TSEN	MINY	MUT	SAT	ERIK	Os <sup>a</sup>
004	RH	hr <sup>s</sup>	VS	C <sup>G</sup>	CE	D <sup>W</sup>	...	...	c-like	cE	hr <sup>H</sup>	Rh29	Go <sup>a</sup>	hr <sup>B</sup>	Rh32	Rh33	Hr <sup>B</sup>	Rh35	Be <sup>a</sup>	Evans	...
005	LU	Au <sup>b</sup>	Lu20	Lu21	LURC																
006	KEL	K19	Km	Kp <sup>c</sup>	K22	K23	K24	VLAN	TOU	RAZ	VONG	KALT	KTIM	KYO	KUCI	KANT	KASH	KELP	KETI	KHUL	
010	DI	Tr <sup>a</sup>	Fr <sup>a</sup>	SW1	DISK																

**TABLE 2.1C** Blood group antigens assigned to each system: extension 2

		Antigen number																				
System		039	040	041	042	043	044	045	046	047	048	049	050	051	052	053	054	055	056	057	058	059
002	MNS	ENEP	ENEH	HAG	ENAV	MARS	ENDA	ENEV	MNTD													
004	RH	Rh39	Tar	Rh41	Rh42	Crawford	Nou	Riv	Sec	Dav	JAL	STEM	FPTT	MAR	BARC	JAHK	DAK	LOCR	CENR	CEST	CELO	CEAG

**TABLE 2.2** Blood group antigens in the collections

Collection			Antigen number					
Symbol	Name	Number	001	002	003	004	005	006
COST	Cost	205	Cs <sup>a</sup>	Cs <sup>b</sup>				
I	Ii	207	...	i				
ER	Er	208	Er <sup>a</sup>	Er <sup>b</sup>	Er3			
GLOB	Globoside	209	...	...	LKE	PX2		
	Unnamed	210	Le <sup>c</sup>	Le <sup>d</sup>				
VEL	Vel	212	Vel	ABTI				
MN CHO <sup>^</sup>		213	Hu	M <sub>1</sub>	Tm	Can	Sext	Sj

Obsolete Collections: 201 (GE), 202 (CROMER), 203 (IN), 204 (AU), 206 (GY), and 211 (WR). For obsolete antigens see [www.isbt-web.org](http://www.isbt-web.org).

<sup>^</sup> = M and N antigens associated with different sialic acid-carrying oligosaccharides on GPA.

**TABLE 2.3** Series of low-prevalence and high-prevalence antigens

		Antigen number																						
Series		002	003	005	006	008	009	012	014	016	017	018	019	021	028	039	040	044	045	047	049	050	052	054
Low-Incidence	700	By	Chr <sup>a</sup>	Bi	Bx <sup>a</sup>	...	...	...	...	...	To <sup>a</sup>	Pt <sup>a</sup>	Re <sup>a</sup>	Je <sup>a</sup>	Li <sup>a</sup>	Milne	RASM	JFV	Kg	JONES	HJK	HOFM	SARA	REIT
High-Incidence	901	...	At <sup>a</sup>	...	...	Emm	AnWj	Sd <sup>a</sup>	PEL	MAM														

For obsolete antigens see [www.isbt-web.org](http://www.isbt-web.org).

a search engine) is a useful website which gives information about alleles relevant to blood groups and hyperlinks to other websites and original articles.

## Molecular bases of antigens and phenotypes

Tables listing alleles encoding blood group variants, together with relevant information, are given. The alleles and their names are those assigned by the ISBT Working Party as of December 2011. For updates, go to the ISBT website ([www.isbt-web.org](http://www.isbt-web.org)). The accession number for the reference allele is indicated, and key nucleotide differences are noted for each allele. Cases where the nucleotide substitution introduces or ablates a restriction enzyme site (indicated, respectively, by “+” or “-”) are documented. A blank indicates that, despite our best efforts, the information could not be ascertained and not, necessarily, that there is no change. The approximate prevalence of the allele in different ethnicities are noted using the following general terms: many = 13 or more examples; several = 6 to 12 examples; few = 2 to 5; and rare = 1. Amino acid changes (using the three-letter code; see Table 2.2) are also given for each variant. In line with the ISBT allele nomenclature, silent nucleotide changes are not given unless there is an effect on antigen expression. For each protein-based blood group system, the reference allele encodes all high prevalence antigens in that system. The protein encoded by a variant allele will differ by the unique expression, or absence, of an antigen. The variant protein will express all high-prevalence antigens of that blood group system, except for the specific variant antigen, e.g., the protein encoded by *KEL\*02.06* lacks Js<sup>b</sup>, expresses the antithetical antigen Js<sup>a</sup>, and also all other Kell high-prevalence antigens, k, Kp<sup>b</sup>, Ku, K11, etc.

The molecular bases for each antigen are also given on the individual antigen pages. Other tables list alleles encoding so-called mod (greatly reduced antigen expression that may require adsorption/elution for detection) or null phenotypes. If the allelic backbone of the altered allele is known, this is noted, e.g., *CO\*01N.03* and *CO\*01N.04* means that the silencing nucleotide change(s) is on a Co(a+) background. If the background was not reported, the allele is written, e.g., *CO\*N.01* and *CO\*N.02*. “Compound heterozygosity” means two different alleles encoding a weak phenotype, one allele encoding a weak phenotype and one allele encoding a null phenotype or two different null alleles.

For all alleles, the numbering for nucleotides and amino acids follows the ISBT system, i.e., nucleotides are counted as #1 being the “A” of the initiation “AUG” and amino acids are counted as #1 from the initiation methionine. This ISBT consistency policy means that the numbers may differ from those published; for example, nucleotide numbers in Kell decrease by 120 and in Knops decrease by 27, and amino acids numbers in MNS increase by 19, in LW they increase by 30 (see Section III for the complete list).

For references to original reports of the alleles, go to dbRBC and use the hyperlinks.

### Amino acid sequence

The amino acid sequences in this section are shown in the single-letter code (Table 2.4). The predicted transmembrane sequence for a single-pass

**TABLE 2.4** Amino acids and their three-letter and single-letter codes<sup>8</sup>

Amino acid	Three-letter code	Single-letter code	Properties	Molecular weight (Daltons)
Alanine	Ala	A	Nonpolar	89
Arginine	Arg	R	Polar, positively charged	174
Asparagine	Asn	N	Polar, uncharged	132
Aspartic acid	Asp	D	Polar, negatively charged	133
Cysteine	Cys	C	Polar, uncharged	121
Glutamine	Gln	Q	Polar, uncharged	146
Glutamic acid	Glu	E	Polar, negatively charged	147
Glycine	Gly	G	Polar, uncharged	75
Histidine	His	H	Polar, positively charged	155
Isoleucine	Ile	I	Nonpolar	131
Leucine	Leu	L	Nonpolar	131
Lysine	Lys	K	Polar, positively charged	146
Methionine	Met	M	Nonpolar	149
Phenylalanine	Phe	F	Nonpolar	165
Proline	Pro	P	Nonpolar	115
Serine	Ser	S	Polar, uncharged	105
Threonine	Thr	T	Polar, uncharged	119
Tryptophan	Trp	W	Nonpolar	204
Tyrosine	Tyr	Y	Polar, uncharged	181
Valine	Val	V	Nonpolar	117



membrane protein is underlined. Amino acids are counted as number 1 being the initiation methionine. The number of amino acids that are believed to be cleaved, and thus, not present in the mature membrane-bound protein is stated.

## Carrier molecule

Molecules carrying blood group antigens are glycoconjugates (the carbohydrate portions of glycolipids or glycoproteins), single-pass membrane proteins, multi-pass membrane proteins or glycosylphosphatidylinositol (GPI)-linked proteins. Single-pass proteins can be oriented with the N-terminus outside (type I) or inside (type II) the membrane (Figure 1.1).

Carbohydrate antigens are depicted by the critical immunodominant sugars and linkages. Proteins carrying blood group antigens are depicted by a stick diagram within a gray band that represents the RBC membrane lipid bilayer. The inside (cytoplasmic surface) of the membrane is always to the bottom of the page and the outside (exofacial surface) is to the top of the page. The predicted topology of the protein in the membrane is shown in the models. The predicted orientation of the N-terminus and the C-terminus is indicated, as are the total number of amino acids. O-glycans are depicted by an open circle (○) and N-glycans by a closed circle on a line (lollipop). Glycosylphosphatidylinositol linkage will be depicted by the zigzag symbol. On the RBC membrane, the presence of a third fatty acid chain on GPI-linked proteins makes the protein harder to cleave by phospholipases. For background reading about membrane proteins the interested reader is referred to Alberts, et al.<sup>8</sup>

The locations of single amino acid substitutions for antigens within the blood group system are shown on a diagram. While one protein molecule can carry numerous high-prevalence antigens, with few exceptions, it is unlikely to carry more than one antigen of low prevalence. Certain characteristics will be given:

*M<sub>r</sub> (SDS-PAGE):* The relative molecular mass (*M<sub>r</sub>*) of a protein as determined by SDS-PAGE. The *M<sub>r</sub>* of a protein (and in particular a glycoprotein) usually differs from the actual molecular weight and the molecular weight calculated from the amino acid sequence deduced from the nucleotide sequence.

*Glycosylation:* Potential N-linked glycosylation sites (Asn-X-Ser/Thr where X is any amino acid except Pro) are indicated. O-linked glycosylation occurs at Ser and Thr residues. Not all Ser and Thr residues are glycosylated.

*Cysteine residues:* The total number present is indicated.

*Copies per RBC:* The number of copies in the RBC membrane of the protein carrying a blood group antigen is indicated. Human polyclonal antibodies to a specific antigen and monoclonal antibodies to the protein have been used as intact immunoglobulin molecules and as Fab fragments to ascertain copy number. Depending on the technology used, these numbers

can vary dramatically in different publications. This is particularly true of carbohydrate antigens. The figures given are only a guide, and the interested investigator is encouraged to perform a thorough literature search.

## Function

Function of the carrier protein, or the predicted function, based on homology with other proteins of known function, is given.

## Disease association

This entry includes diseases caused by an absence of the protein or carbohydrate carrying the blood group antigens and disease susceptibilities associated with an absence, an altered form or a reduced number of copies/RBC of the protein. We also include diseases associated with altered carbohydrate structures.

## Phenotypes

The prevalence of phenotypes associated with the blood group system, the null phenotype, and any unusual phenotypes (unusual in expression, not in prevalence) are given. In general, the figures given are for Caucasian populations (northern European), because that is the best studied group by hemagglutination but where possible and where particularly relevant due to differences between populations, other ethnic groups have also been included. Information was obtained from original publications, *Blood Groups in Man*<sup>9</sup>, and the *AABB Technical Manual*<sup>5</sup>. The numbers are an average estimate.

In addition, tables with useful facts pertaining to antigens or phenotypes in one system are given here.

## Comments

Any fact or interesting information relevant to the blood group system and/or the carrier protein or carbohydrate that does not fit elsewhere is placed here.

## References

It is incompatible with the format of this book to provide a comprehensive list of references. However, when appropriate, key or recent references and reviews have been included. For references for antigens and/or alleles, go to GenBank, dbRBC, Rhesus Base (*RHD*), or nybloodcenter.org (*RHCE*) for hyperlinks to the original publications. Certain reference books have been used throughout and rather than list them all on each set of sheets they are listed below. Some references for specific antigens are not given on the system pages, but are given in the antigen pages. These texts are also a good source of references<sup>5,9-17</sup>.

## ISBT Blood Group Antigens

### Terminology

The traditional name for the blood group antigen is given at the top of the page. Also given are the ISBT symbol, ISBT number (parenthetically), other, obsolete, names that have been associated with the antigen, and a brief history about the antigen, including how it was named.

As use of the ISBT number without sinistral zeros was originally stated to be an acceptable terminology, we have given this as well as the more commonly used six-digit number<sup>18</sup>.

### Occurrence

Antigen and phenotype prevalence are often obtained by averaging several series of tests, and are given as a guide. The prevalence of an antigen is given for Caucasians; notable ethnic differences also are given. Where no ethnicity is given, the figures refer to all populations tested. In general, the information was obtained from *Blood Groups in Man*<sup>9</sup>, *Distribution of the Human Blood Groups and Other Polymorphisms*<sup>19</sup>, the *AABB Technical Manual*<sup>5</sup>, and original articles.

### Antithetical antigen

If an antigen is polymorphic, the antithetical partner is indicated.

### Expression

Entries here relate to serologically detectable antigens; however, in most instances the information also will apply to the carrier molecule.

### Molecular basis associated with antigen

The name and position of specific amino acid(s) (using the three-letter code) associated with the antigen and the position of the base pair (bp) change(s) are both indicated. For the amino acid associated with the allele encoding a blood group antigen, it will be necessary to refer to the pages for the antithetical antigen. For those antigens that do not have defined antithetical partners, the amino acid associated with the wild-type protein or with the absence of a high-prevalence antigen, are given. Where the nucleotide or amino acid number has changed due to counting #1 from the initiation codon, we give this information.

For antigens on hybrid molecules or that are more complex than a single amino acid change, a stick diagram is given.

## Effect of enzymes and chemicals on intact RBCs

The options for entries are: resistant; sensitive; weakened; variable. In those instances when reactions between antibody and antigen are markedly enhanced, this information is noted; enhancement may not have been studied for all antigens in a particular system. If no information is available, we have taken the liberty of using “presumed,” and extrapolated our interpretation based on the behavior of other antigens in the same system. The information given is to be used only as a guide, because with all chemical treatment of RBCs, the effect varies depending on the exact conditions of treatment, purity of reagents, and the age (condition) of the RBCs. It should be noted that the effect of enzymes on an isolated protein may not be the same when the protein is within the milieu of the RBC membrane.

We provide information regarding the effect of pronase treatment of RBCs only when it is known to differ from the effect of papain or ficin treatment. Namely, this applies to antigens in Lutheran, Diego (3rd extracellular loop), Dombrock, Landsteiner-Wiener, and Cromer blood group systems and Ge3. Similarly, we only give the effect of sialidase treatment of RBCs when the antigen is sensitive to such treatment.

It is important to remember that antibodies to enzyme-treated RBCs exist naturally in some plasma, which can cause false-positive results. This is particularly true for pronase.

If an antigen is sensitive to treatment of RBCs with 200mM dithiothreitol (DTT), an additional entry will be made for the effect of 50mM DTT. Other thiol-containing reagents, which include 2-mercaptoethanol (2-ME) and 2-aminoethylisothiuronium bromide (AET), would be expected to give similar results to those indicated for DTT treatment. The commonly used reagents, WARM™ and ZZAP<sup>20</sup>, are a combination of DTT and papain.

The effect of acid treatment on antigens is included for those who wish to type RBCs after *in vivo* bound immunoglobulin has been removed by EDTA/glycine/acid treatment<sup>21</sup>. RBCs treated in this way do not express antigens in the Kell blood group system, the Er collection or Bg antigens.

Most information for this section was obtained from original papers and from references<sup>11,20,22,23</sup>.

## *In vitro* characteristics of alloantibody

An alloantibody can be made by a person who lacks the corresponding antigen. The immunoglobulin class of a blood group antibody is usually IgG and/or IgM. Blood bank techniques do not routinely include methods to detect IgA antibodies. IgD and IgE have not been described as blood group specific antibodies. In general, naturally-occurring antibodies are IgM and react best by direct agglutination tests, while immune antibodies are IgG and react best by the indirect antiglobulin test (IAT). Readers who are

interested in information about the IgG subclass of blood group antibodies are referred to Petz and Garratty<sup>16</sup>.

The optimal technique for detection of an antibody to a given antigen is listed as room temperature (RT) or IAT. “RT” means incubation at ambient temperature followed by centrifugation and examination for hemagglutination. “IAT” represents the indirect antiglobulin test, regardless of which enhancement medium (e.g., LISS, albumin, PEG) was used. “Enzymes” means that the antibody agglutinates protease-treated (usually ficin or papain) RBCs, usually after incubation at 37°C. Enzyme-treated RBCs also may be used by the IAT. If column agglutination technology is being used, then “RT” indicates use of the neutral (buffer) cassette, and “IAT” indicates use of the antiglobulin cassette.

Complement binding is used to convey whether the alloantibody is known to bind complement during the *in vitro* interaction with its antigen. It is not intended to indicate the potential of an alloantibody to cause *in vivo* hemolysis of transfused antigen-positive blood.

## Clinical significance of alloantibody

This section summarizes the type and degree of transfusion reaction(s), and the degree of clinically significant hemolytic disease of the fetus and newborn (HDFN) that have been associated with the alloantibody in question. Many factors influence the clinical significance of a blood group antibody, and the interested reader is referred to the following references<sup>14,24,25</sup>.

Under “Transfusion reaction” the entries are: No/+DAT/mild/moderate/severe; immediate/delayed/hemolytic, and no data. “Severe” usually means an immediate transfusion reaction as indicated by symptoms that may include but are not limited to lower back pain, change in blood pressure, shortness of breath, feeling of “impending doom,” nausea, and/or vomiting, restlessness, flush, passing of red or dark urine. Hemoglobinemia, hemoglobinuria, rapid drop of haptoglobin, reduced RBC count or hematocrit can often be registered. This type of reaction may be fatal. “Delayed” transfusion reaction means a reduced RBC survival associated with a positive DAT, absence of the expected rise in hemoglobin levels, and/or shortened transfusion interval (for chronically transfused patients). This type of reaction is typically not dramatic and often asymptomatic, although jaundice and fatigue may develop. Hemoglobinemia and hemoglobinuria may be recorded but is not typical, whilst a reduced RBC count or hematocrit is expected. The options for HDFN are No/+DAT but no clinical HDFN/mild/moderate/severe and rare.

## Autoantibody

If autoantibodies directed to the antigen in question have been described, they are indicated here<sup>11,16,26</sup>.

## Comments

Any fact or interesting information relevant to the antigen and that does not fit elsewhere is placed here.

## References

It is incompatible with the format of this book to provide a comprehensive list of references. However, appropriate key references for reviews or recent papers have been selected as a source of further relevant references. References given on the system page will not necessarily be repeated on each antigen page. Where no reference is given, refer to the system page. For references for alleles, go to GenBank or dbRBC for hyperlinks to the original publications. Certain textbooks have been used throughout, and rather than list them on each antigen page they are listed below. These textbooks are a good source of references<sup>5,9-17</sup>.

## ISBT Blood Group Collections

Antigens within each collection have a serological, biochemical or genetic relationship, but do not fulfill the criteria for system status. Information relating to each collection of blood group antigens [COST, I, ER, GLOB, Unnamed, VEL, and MN CHO (MNS carbohydrate antigens)] are given on separate pages.

## ISBT 700 Series of Low-Incidence Antigens

Antigens in this section occur in less than 1% of most populations studied, and are not known to belong to a blood group system.

## ISBT 901 Series of High-Incidence Antigens

Antigens in this section occur in more than 90% of the population, and are not known to belong to a blood group system. Originally, this Series had number 900 but was renumbered 901 in 1988 after many of the antigens were relocated to Systems or Collections.

## References

- <sup>1</sup> Daniels, G., et al., 2007. International society of blood transfusion committee on terminology for red cell surface antigens: Cape Town report. *Vox Sang* 92, 250–253.
- <sup>2</sup> Daniels, G.L., et al., 2004. Blood group terminology 2004. *Vox Sang* 87, 316.
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