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International Society of Blood Transfusion Working Party on Red Cell Immunogenetics and Blood Group Terminology: Report of the Dubai, Copenhagen and Toronto meetings.

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Abstract

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Author contribution

As members of the Working Party, all authors have contributed equally to the discussion and conclusions drawn in this paper.

Conflict of Interest declaration

There are no conflicts of interest to declare with regard to the data reported in this paper.

Statement of Disclaimer.

The views expressed do not necessarily represent the view of the National Institutes of Health, the Department of Health and Human Services, or the U.S. Federal Government.

Background and objectives: The International Society of Blood Transfusion (ISBT) Working Party for Red Cell Immunogenetics and Blood Group Terminology meets in association with the ISBT congress and has met three times since the last report: at the international meetings held in Dubai, United Arab Emirates, September 2016 and Toronto, Canada, June 2018; and at a regional congress in Copenhagen, Denmark, June 2017 for an interim session.

Methods: As in previous meetings, matters pertaining to blood group antigen nomenclature and classification were discussed. New blood group antigens were approved and named according to the serologic and molecular evidence presented.

Results and conclusions: Fifteen new blood group antigens were added to eight blood group systems. One antigen was made obsolete based on additional data. Consequently, the current total of blood group antigens recognised by the ISBT is 360, of which 322 are clustered within 36 blood groups systems. The remaining 38 antigens are currently unassigned to a known system. Clinically significant blood group antigens continue to be discovered, through serology/sequencing and/or recombinant or genomic technologies.

Keywords

blood groups; terminology; genetics

A key role for the Working Party on Red Cell Immunogenetics and Terminology is to review reports for blood group systems, antigens and associated blood group alleles. Historically, endorsement has occurred at the biennial ISBT International Congress. Curated blood group antigens and allelic tables are also reviewed by members in the interim who may also meet in the alternate years at regional congress meetings when there is a sufficient member quorum present.

A total of 15 blood group antigens were added to eight of the current blood group systems (Table 1). One antigen was made obsolete following additional investigation of the original antibody. This brings the current total of recognized blood group antigens to 360, of which 322 are clustered within 36 blood groups systems.

Thus, there remain 38 serologically-defined antigens that have not been assigned to a blood group system. Fourteen of those reside in one of five collections (the 200 series), 17 in the low-prevalence series (700) and a further seven are in the 901 series (prevalence >90%).

New Blood Group Antigens

System 2: MNS

One antigen has been added to the MNS system, JENU (MNS49). An alloantibody to a high-prevalence antigen was shown by epitope mapping to be directed at the amino acids ^{38}-SYISSQTNGETG-^{49} of glycophorin B (GPB). The antibody maker was a thalassemia patient of Thai origin who was shown to lack normal GPB, but who was homozygous for the *GYP.Mur* hybrid (*GYP*501*). Thus, JENU is a high-prevalence antigen on GPB that is absent from the GP.Mur hybrid [1]. The epitope defined above is interrupted in the hybrid protein. The name JENU combines JE, from the first two letters of the surname of the

patient who made the JENU antibody, and NU, from the high frequency 'N' and U antigens present on GPB.

System 4: Rh

Identification of a new high-prevalence Rh antigen was made following the investigation of an antibody in the plasma of an elderly South African patient that was nonreactive only with Rh_{null} RBCs [2]. The patient's RBCs typed as RH:−51 however the antibody was not compatible with other examples of RH:−51 RBCs. DNA sequence analysis revealed the nucleotide change *RHCE* c.114A>C that encodes a substitution of p. Leu38Phe in RhCE. The antigen has been assigned the name CEWA (RH62), with CE for RhCE and WA after the proband's name.

Other RH:−51 phenotypes are defined by the amino acid substitutions p.Gln41Arg (C^W+) and p.Ala36Thr (C^X+), and thus the RH51 epitope is likely defined by a peptide that includes amino acids 36–41 of RhCE. The incompatibility of the patient's plasma with D-- RBCs suggests that CEWA is defined by an epitope shared by both RhD and RhCE, analogous to the G antigen.

System 5: Lutheran

Three high-prevalence antigens have been added to the Lutheran blood group system. An antibody in the plasma of a male patient of Maori origin was non-reactive with In(Lu) and Lu(a−b−) RBCs. Lutheran specificity was confirmed by the monoclonal antibody immobilisation erythrocyte assay (MAIEA), and subsequent DNA sequencing revealed a novel homozygous mutation c.662C>T in exon 6 of *BCAM*, encoding p.Thr221Ile in the Lu glycoprotein. This antigen has been named LUAC (LU25), LU for Lutheran and AC for Auckland, New Zealand [3].

The second antigen LUBI (LU26) (BI for Birmingham, United Kingdom) was identified following the investigation of multiple antibodies in a Caucasian woman with a history of transfusion. Her plasma contained anti-M, -Fy^a and -Jr^a as well as an unidentified antibody that was defined by MAIEA to be Lutheran-related. Sequence analysis of *BCAM* showed homozygosity for a novel mutation c.1495C>T in exon 12, encoding p.Arg499Trp [3].

Antibodies to an apparent high-prevalence Lutheran system antigen was identified in the plasma of two Turkish sisters during their respective pregnancies. Their RBCs were mutually compatible and subsequent molecular analysis of *BCAM* identified homozygosity for two nucleotide changes. The first is a synonymous change in exon 3, c.324G>A (p.Gly108Gly) known to be present in approximately 3–4% of the general population. The second one was found in exon 9, c.1184G>A, and predicts the amino acid change p.Arg395His on the fourth Ig superfamily domain of the Lutheran glycoprotein. The antigen was named LUYA (LU27) after the proposita [4].

System 6: Kell

A tragic neonatal death due to hemolytic disease led to the identification of an antibody to a novel low-prevalence antigen in the Kell blood group system [5]. The antigen, KEAL

(KEL39; named after the proposita) was shown to be encoded by the substitution c.877C>T (p.Arg293Trp) in exon 8 of *KEL*, and inheritance could be demonstrated in three generations. KEAL is antithetical to the high-prevalence antigen, KHUL (KEL37) [6].

System 11: Yt

The Yt blood group system has more than doubled in size in the past three years. Use of recombinant protein reagents have been very helpful in teasing apart complex mixtures of antibodies, and three new high-prevalence antigens have been added to this system by Thornton and colleagues at the International Blood Group Reference Laboratory. In the first case, soluble recombinant Yt protein (srYt) successfully inhibited an additional unknown antibody to a high-prevalence antigen in plasma from an acute myeloid leukemia patient that contained anti-c, -Fy^b and -Jk^b. Subsequent sequence analysis of *ACHE* demonstrated homozygosity for the mutation c.266G>A in exon 2, encoding an amino acid change p.Gly89Glu in acetylcholine-esterase AChE) [7]. The new antigen is named YTEG (YT03) after the proband's name.

An additional antibody to a high-prevalence antigen was found in the plasma of a pregnant woman with anti-Jk^a. Use of a recombinant soluble antigen panel demonstrated Yt specificity although the patient's RBCs typed Yt(a+b-), YTEG+. *ACHE* sequencing confirmed the proband and her serologically compatible brother to be *YT*A/A* and revealed a novel homozygous mutation c.169G>A in exon 2, encoding p.Gly57Arg change in AChE. The antigen (YT04) has been named YTLI (after the referring centre in Liverpool) [8].

The third addition to the Yt blood group system followed the serological investigation of plasma from a young pregnant woman with sickle cell disease. Her plasma contained multiple antibodies, including anti-Fy^a, -Js^a and an antibody that reacted with all phenotypically similar RBCs. Again, reactivity with the latter was inhibited by srYt although antibodies to Yt^a, YTEG and YTLI were excluded. Sequencing of *ACHE* identified a novel homozygous mutation c.101G>A in exon 2, encoding p.Arg34Gln change in AChE. The antigen (YT05) has been named YTOT, also after the referring laboratory [8].

System 21: Cromer

A novel Inab-like phenotype has been molecularly characterised after an antibody that was compatible only with IFC-negative RBCs was identified in the plasma of a Druze woman. Her RBCs typed negative with all available antisera to Cromer blood group antigens and were nonreactive with anti-CD55 by flow cytometry. Molecular analysis of *CD55* revealed the mutation c.245T>C, which changes p.Leu82Pro. The WES^b antigen is defined by Leu82, and the antithetical low-prevalence antigen WES^a is defined by c.245T>G (p.Leu82Arg). Thus, this is the third polymorphism at this nucleotide position. Interestingly, Wes(a+b−) RBCs were weakly incompatible with the patient's plasma indicating that the absence of p.Leu82 was insufficient for compatibility, and suggested that p.Leu82 encodes for both WES^b and for another high-prevalence antigen. The antigen (CROM19) has been named CROK after the patient [9].

Exome sequencing was used to resolve an unidentified antibody to a high-prevalence antigen in the plasma of a 103-year old woman of French Corsican descent. Her history showed a

single pregnancy in 1949 and the antibody was first detected in 2002. When new samples were received for investigation in 2016, DNA was isolated and exome sequenced, which revealed homozygosity for c.713G>A in exon 6 of the *CD55* gene, predicted to cause a p.Gly238Glu amino acid change. Her son was found to be heterozygous for the same mutation. Cromer specificity of the antibody was subsequently confirmed when RBCs of the Inab phenotype became available. The antigen (CROM20) was named CORS to reflect the patient's origin [10].

System 23: Indian

INRA (IN5) is a new high-prevalence antigen in the Indian blood group system, and was named after the patient. The defining antibody was present in the plasma of a female patient from Surat in the Indian state of Gujarat and demonstrated the classic absence of reactivity with papain-treated RBCs characteristic of Indian blood group system antibodies. The antibody was compatible only with the RBCs of the patient's brother. Sequence analysis of *CD44* demonstrated homozygosity for a novel homozygous synonymous mutation c.255C>T in exon 3 (p.His85His), and for a novel homozygous missense mutation c.449G>A in exon 5, encoding p.Arg150His [11].

Soluble recombinant CD44 helped to resolve the identity of an antibody to a high-prevalence antigen in the plasma of a Sri Lankan patient with suspected In specificity. Both the patient and his serologically compatible brother typed In(a−b+), INFI+, INRA+. Sequencing of *CD44* revealed homozygosity in both siblings for a mutation c.276C>A, which predicts an amino acid change p.His92Gln. The antigen (IN6) has been named INSL to reflect the origin of the patient [12].

System 36: Augustine

The Augustine system continues to expand since its discovery in 2015 [13]. A case of severe HDFN led to the discovery of a low-prevalence antigen that was shown by targeted exome sequencing to be encoded by *SLC29A1*. DNA from both the infant and father revealed a mutation c.1159A>C, in exon 12 predicting an amino acid change of p.Thr387Pro. In an extended family study, six paternal family members carried the nucleotide substitution; red cells from these six members showed incompatibility with the mother's plasma, demonstrating that serological reactivity segregated with the mutation. The predicted amino acid change is four amino acids from the amino acid that defines the At^a antigen in the fifth extracellular loop. The antigen (AUG3) was named ATML after the initials of the patient [14].

A novel high-prevalence antigen in this system was defined in a follow-up investigation of a long-time unresolved antibody in a female patient. Targeted exome sequencing in this case revealed homozygosity for c.242A>G in the *SLC29A1* gene, that changes p.Asn81Ser. Confirmatory serology showed that the plasma was compatible with AUG:−1 RBCs, which are the null phenotype in this blood group system. The antigen (AUG4) was named ATAM. after the proband's name [15].

Other changes to blood group systems

System 28: GLOB

When PX2 was assigned to this blood group system, it was moved from the GLOB collection (see below) into the GLOB blood group system and assigned the number GLOB2 (028002) [16]. This has caused confusion with old records of rare red blood cells in some laboratories, thus the decision was taken to reassign PX2 as GLOB4, and the numbers GLOB2 and GLOB3 have been made obsolete. A current list of obsolete numbers is shown in Table 2.

System 30: RHAG

Two independent investigations have shown that the polymorphism RHAG c.808G>A (p.Val270Ile) does not encode a low-prevalence antigen as first described. In a study of *RHD*, *RHCE* and *RHAG* variation examined by whole exome sequencing of samples from children with sickle cell disease, Chou et al. have shown this polymorphism to be common, with 14.6% of samples demonstrating heterozygosity [17]. Supplementary serological investigation of the original sample, reported following a case of severe hemolytic disease of the fetus and newborn (HDFN) [18], revealed the antibody to be anti-s^D (MNS23). Consequently, RHAG4 has been made obsolete.

Changes to the Collections (200), Highs (901) and Lows (700) series

The GLOB collection

The discovery of genetic basis for the GLOB blood group system resulted in the assortment of antigens previously defined as the GLOB collection being placed in the GLOB blood group system. However, since the high-prevalence antigen, LKE remained the only antigen in the GLOB collection, the decision was made to move LKE to the 901 series until the molecular basis is identified. It has been assigned the number 901017. Accordingly, the GLOB collection has been made obsolete.

Gene terminology and conclusion

Clinically significant blood group antigens continue to be discovered, through serology/sequencing and/or recombinant or genomic technologies.

The Working Party continues to update the blood group antigen tables and the allele nomenclature tables. These can be found on the ISBT website.(http://www.isbtweb.org/working-parties/red-cell-immunogenetics-and-blood-group-terminology/) We are working actively with the Locus Genome Reference initiative (https://www.lrg-sequence.org/) to qualify reference sequences for all genes encoding blood group systems. This work in on-going.

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Table 1:

New antigens added to blood group systems

Blood Group System	Antigen number	Alt. name	Prevalence	Molecular basis	Protein change	Reference
MNS	MNS49	JENU	High	*GYPB*; defined as an epitope within ^{38}SYISSQTNGETG^{49}	Interrupted by the amino acids encoded by the *GYPA* exon 3 insertion in the GP.Mur hybrid protein encoded by *GYP*501*.	[1]
RH	RH62	CEWA	High	*RHCE* c.114A>C	RhCE p.Leu38Phe	[2]
LU	LU25	LUAC	High	*BCAM* c.662C>T	BCAM p.Thr221Ile	[3]
	LU26	LUBI	High	*BCAM* c.1495C>T	BCAM p.Arg499Trp	[3]
	LU27	LUYA	High	*BCAM* c.1184G>A	BCAM p.Arg395His	[4]
KEL	KEL39	KEAL	Low	*KEL* c.877C>T	Kell p.Arg293Trp	[5]
YT	YT3	YTEG	High	*ACHE* c.266G>A	AChE p.Gly89Glu	[7]
	YT4	YTLI	High	*ACHE* c.169G>A	AChE p.Gly57Arg	[8]
	YT5	YTOT	High	*ACHE* c.101G>A	AChE p.Arg34Gln	[8]
CROM	CROM19	CROK	High	*CROM* c. 245T>C	CD55 p.Leu82Pro	[9]
	CROM20	CORS	High	CROM c.713G>A	CD55 p.Gly238Glu	[10]
IN	IN5	INRA	High	*CD44* c.449G>A	CD44 p.Arg150His	[11]
	IN6	INSL	High	*CD44* c.276C>A	CD44 p.His92Gln	[12]
AUG	AUG3	ATML	Low	*SLC29A1* c.1159A>C	SLC29A1 p.Thr387Pro	[14]
	AUG4	ATAM	High	*SLC29A1* c.242A>G	SLC29A1 p.Asn81Ser	[15]

Table 2:

Obsolete numbers for blood group antigens organized by blood group system, collections (200 series), low-prevalence antigens (700 series) and high-prevalence antigens (901 series).

Obsolete number	Symbol or previous symbol	Subsequent number (Current number in bold)	Year reported obsolete	Obsolete number	Symbol or previous symbol	Subsequent number (Current number in bold)	Year reported obsolete
Antigens in blood group systems				Antigens in collections			
001005	H	**018001**	1991	Collection 201	GE	System 020	1991
003002	P	209001	1991	Collection 202	CROMER	System 021	1991
003003	P^k	209002	1991	Collection 203	IN	System 023	1995
004013	Rh^A		1995	Collection 204	AU		1991
004014	Rh^B		1995	204001	Au^a	**005018**	1991
004015	Rh^C		1995	204002	Au^b	**005019**	1991
004016	Rh^D		1995	205003	Yk^a	**022005**	1993
004024	E^T		1995	205004	Kn^a	**022001**	1993
004025	LW		1985	205005	Kn^b	**022002**	1993
004038	Duclos	901013	1995	205006	McC^a	**022003**	1993
005010	Singleton		1991	205007	Sl^a	**022004**	1993
005015	AnWj (Anton)	**901009**	1991	Collection 206	Gregory		1993
006008	Kw		1991	206001	$GY1/Gy^a$	**014003**	1993
006009	KL		1985	206002	GY2/Hy	**014004**	1993
006015	Kx	**019001**	1991	207001	I	**027001**	2003
008004	FY4		2010	Collection 209	GLOB		2018
016001	LW_1 phenotype		1985	209001	P	**028001**	2003
016002	LW_2 phenotype		1985	209002	p^k	**003003**	2010
016003	LW_3 phenotype		1985	209003	LKE	**901017**	2018
016004	LW_4 phenotype		1985	209004	PX2	**028002**	2014
020001	Ge1		1991	Collection 211	WR		1995
030004	RHAG4		2018	211001	$WR1/Wr^a$	**010003**	1995
				211002	$WR2/Wr^b$	**010004**	1995

Obsolete number	Symbol or previous symbol	Subsequent number (Current number in bold)	Year reported obsolete	Obsolete number	Symbol or previous symbol	Subsequent number (Current number in bold)	Year reported obsolete
				Collection 212	Vel/ABTI		2014
				212001	Vel	**034001**	2014
				212002	ABT1	**901015 (reverted)**	2014
Antigens in the low-prevalence (700) series				Antigens in the low-prevalence (700) series			
700001	Wr[a]	**010003**	1991	700035	Tc[b]	**021003**	1991
700004	Sw[a]	**010013**	1999	700036	Tc[c]	**021004**	1991
700007	Ls[a]	**020006**	1991	700037	NFLD	**010016**	1999
700008	Tr[a]	**010019**	2004	700038	Hov (= Wu)	**010019**	1999
700009	Wb	**020005**	1991	700041	SW1	**010021**	2001
700010	Bp[a]	**010010**	1999	700042	WES (WES[a])	**021008**	1991
700011	Or	**002031**	2008	700046	BOW	**010015**	1999
700012	Gf		1991	700048	FPTT	**004050**	1995
700013	Wu	**010009**	1999	700051	ELO	**010008**	1999
700014	Jn[a]	**010017**	1999	700053	LOCR	**004055**	2003
700015	Rd	**013004**	2003	700055	WARR	**010007**	1996
700016	Heibel		1990	700052	SARA	**004047**	2014
700020	An[a]	**020007**	1991				
700022	Mo[a]	**010011**	1999	Antigens in the high-prevalence (901) series			
700023	Hey		1995	901001	Vel	212001	2004
700024	R[a] (= Ls[a])	**020006**	1991	901002	Lan	**033001**	2012
700025	In[a]	**023001**	1995	901003	At[a]	**036002**	2015
700026	Fr[a]	**010020**	2001	901004	Jo[a]	**014005**	1993
700027	Rb[a]	**010006**	1996	901005	Jr[a]	**032001**	2012
700029	Vg[a]	**010013**	1999	901006	Ok[a]	**024001**	1999
700030	Wd[a]	**010005**	1996	901007	JMH	**026001**	2001
700031	Dh[a]	**020008**	1991	901010	Wr[b]	**010004**	1991
700032	POLL		1991	901011	MER2	**025001**	1999
700033	Os[a]	**002038**	1995	901013	Duclos	**030001**	2008

Obsolete number	Symbol or previous symbol	Subsequent number (Current number in bold)	Year reported obsolete
700034	Hg[a]	**010012**	1999

Obsolete number	Symbol or previous symbol	Subsequent number (Current number in bold)	Year reported obsolete
901015	ABTI	212002	2004