

International Society of Blood Transfusion Working Party on red cell immunogenetics and blood group terminology: Berlin report

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Introduction

The Working Party met in Berlin, Germany during the 2010 International Society of Blood Transfusion (ISBT) Congress. While no changes were made to the current classification as documented in Blood Group Terminology 2004 [1] and the 2007 update [2], following a meeting of the International Scientific Advisory Committee in Cairo, the Working Party was renamed the Working Party on Red Cell Immunogenetics and Blood Group Terminology

and the following terms of reference were drawn up by Dr Geoff Daniels:

- (1) To advise the ISBT on matters relating to red cell immunogenetics.
- (2) To organize international workshops on blood group molecular genotyping.
- (3) To maintain and monitor a terminology and genetic classification for blood group antigens.
- (4) To develop, maintain and monitor a terminology for blood group genes and their alleles.

A total of 14 new blood group antigens were added to 10 of the 30 current blood group systems (Table 1), and one antigen (FY4) was made obsolete. As described below, a new Collection containing the six MNS-related carbohydrate antigens was created. A new antigen, PX2, was added to Collection 209, from which the P^k antigen was moved to

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	Antigen name	Incidence	Nucleotide change ^a	Amino acid change	Reference
RH58	CELO	High	<i>RHCE</i> 697C>G	Gln233Glu	[7]
RH59	CEAG	High	<i>RHCE</i> 254G>C	Ala85Gly	[8]
LU22	LURC	High	223C>T	Arg75Cys & dependent on Arg77	[9]
KEL35	KELP	High	780G>T	Leu260Phe	[11]
			2024G>A	Arg675Gln	
DI22	DISK	High	1694G>C	Gly565Ala	[12]
D07	DOMR	High	431C>T	Ala144Glu	[13]
			432C>A		
CO4	Co4	High	140A>G	Gln47Arg	[14,15]
GE10	GEPL	High	134C>T	GPC Pro45Leu; GPD Pro24Leu	[16]
GE11	GEAT	High	56A>T	Asp19Val	[16]
GE12	GETI	High	80C>T	GPC Thr27Ile; GPD Thr6Ile	[16]
CROM16	CROZ	High	389G>A	Arg130His	[17]
OK2	OKGV	High	176G>T	Gly59Val	[18]
OK3	OKVM	High	178G>T	Val60Met	[19]
JMH6	JMHQ	High	1040G>T	Arg347Leu	[20]

^aNumbers used for nucleotide and amino acid location are counted from A of the initiating methionine codon and that methionine residue, respectively.

the P blood group system, and was renamed P1PK. This brings the current total of recognized blood group antigens to 328, of which 284 are contained within the 30 blood groups systems.

System 3: P1PK

It has recently been shown that a nucleotide polymorphism upstream of the coding region of the *A4GALT* gene, previously shown to encode the 4- α -galactosyltransferase that synthesizes the P^k antigen [3–5], correlates with P1 antigen expression [6]. A novel short transcript of *A4GALT* revealed a new exon in intron 1, termed exon 2a. Three polymorphisms were identified in the exon, of which one nucleotide change correlates with the P₁/P₂ phenotypes and for the first time, permits genotyping for P1 antigen expression [6]. This finding ties the P1 and P^k antigens, long known to be related serologically and biochemically, to the same gene – *A4GALT*. Based on the molecular evidence (from > 200 donors) linking the two antigens, it was proposed to rename the P system to P1PK. This change was also based on the confusion that would arise if the P1 and P^k antigens but not the P antigen belonged to a system still called P. The P1 antigen (now P1PK1) retains the ISBT number 003001, P^k (P1PK2) becomes 003002, while number 209002 (formerly P^k) is now obsolete.

System 4: Rh

Two high-incidence antigens have been added to the Rh system. RH58 (CELO) is antithetical to the low-incidence

Table 1 New blood group antigens

antigen, RH43 (Crawford), and is not encoded by *RHCE*^{*}-*ceCF* (48C, 697G, 733G) and very weakly expressed on the Dc(e) phenotype encoded by *RHCE*^{*}*ceBP* [7]. Two patients, one who was homozygous for *RHC E*^{*}*ceCF* the other who had *RHCE*^{*}*ceCF* in trans to a silenced *RHCE*^{*}*ceE*, had each made an anti-Rh17-like antibody that was shown to be anti-CELO. Furthermore, the amino acid changes encoded by *RHCE*^{*}*ceCF* (Trp16Cys, Gln233Glu, Leu245Val) give rise to partial c and e antigens, which have permitted the production of alloanti-c and alloanti-e, respectively. CELO is also absent from Rh_{null}, D–, and DC^W– phenotype RBCs.

RH59 (CEAG) is a high-incidence antigen associated with an allele encoding a partial e antigen [8]. It was identified following the investigation of three multiply transfused patients whose plasma contained alloanti-e but whose RBCs typed e+. The absence of CEAG is defined by the single nucleotide change *RHCE*^{*}*ce* 254G>C that encodes an amino acid exchange of Ala85Gly.

System 5: Lutheran

A new high-incidence antigen, LU22 (LURC), has been added to the Lutheran system. The molecular change resulting in the LU:–22 phenotype was shown to be a nucleotide change in exon 3, 223C>T that changed Arg75Cys [9]. The RBCs of the proband phenotyped as Lu(a+b^{weak}), and sequencing revealed heterozygosity for this mutation carried on a *LU*^{*}*B* allele and for an *LU*^{*}*A* allele bearing a previously described Lu^a-related polymorphism, 586G>A (Val196Ile) [10]. The Lu^a/Lu^b amino acid polymorphism occurs at residue 77, where Arg77 defines Lu^b and His77

defines Lu^a. The proximity of Arg75Cys presumably interrupts and weakens the expression of the Lu^b antigen encoded by this allele. Furthermore, the occurrence of the LURC-negative phenotype on a heterozygous *LU^a/LU^b* background demonstrates the reliance of LURC on the Lu^b antigen residue, Arg77 for expression.

System 6: Kell

Another high-incidence antigen was added to the Kell system: KEL35 (KELP), defined by an antibody that was nonreactive only with K₀ RBCs and those of the antibody maker [11]. The patient's RBCs typed KEL:–1,2–3,4,6,11,–12,22, although sequence analysis did not confirm the K:–12 phenotype. However, the proband was homozygous for two novel mutations: 780G>T in exon 8, which encodes Leu260Phe, and 2024G>A in exon 18, encoding Arg 675Gln. Leu260Phe is located on the Kell glycoprotein at a position distal from the membrane and modelling predicts a change in the molecular surface of Kell. However, amino acid position 675 lies close to or in the membrane. Both mutations are seemingly unconnected with Arg548 that defines K12 and the effect of these two novel mutations on K12 expression remains unresolved.

System 8: Duffy

Following the criteria that an antibody to a blood group antigen must be extant for that antigen to be recognized, and based on only scant original evidence for its existence, FY4 has been made obsolete.

System 10: Diego

A high-incidence antigen antithetical to DI9 (Wu) has been identified. The new antigen, DI22 (DISK), was characterized by an apparently naturally occurring, strongly agglutinating antibody reactive both at 18 and 37°C and by the indirect antiglobulin test [12]. DISK was shown to be sensitive to α-chymotrypsin treatment, but resistant to other commonly used proteases. Targeted sequence analysis of *SLC4A1* exon 14 revealed homozygosity in the proband and heterozygosity in a sample from her brother, for the mutation 1694G>C that encodes Gly565Ala. The RBCs of her brother reacted more weakly with her antibody, suggesting that the anti-DISK exhibits dosage in DI:9,22 individuals.

System 14: Dombrock

D07 (DOMR) is defined by an antibody to a high-incidence antigen produced in a patient, homozygous for two adjacent nucleotide changes 431C>A, 432C>A that encode a change of Ala144Glu [13]. These changes are present with a

*DO*B-WL* allele (*DO*793G*, *DO*323G*, *DO*350C*, *DO*547T*, *DO*898G*). The *DO*B-WL* allele is predicted to encode the phenotype Do(a–b+), Hy+, Jo(a+), DOYA+, however, the patient's RBCs typed Do(a–b+), Hy+^w, Jo(a+^w), DOYA+^w. Her RBCs also typed Gy(a–) with a polyclonal antibody but were reactive with several monoclonal antibodies to the Dombrock glycoprotein. Further complexity was demonstrated by the plasma antibody that was nonreactive or only very weakly reactive with six Hy– samples, nonreactive with Gy(a–) RBCs but reactive with Jo(a–) and DOYA– RBCs.

System 15: CO

An antibody compatible with Co(a–b–) RBCs but weakly incompatible with Co(a–b+) RBCs was identified in the plasma of a patient whose RBCs typed Co(a–b–) [14]. Molecular analysis of *AQP1* revealed homozygosity for 140A>G, encoding the amino acid change Gln47Arg, in close proximity to the Co^a/Co^b polymorphism at residue 45. Samples from a second Co(a–b–) proband homozygous for the same 140A>G mutation were investigated following detection of an antibody to a high-incidence antigen initially identified as anti-Co3 [15]. Unexpectedly, her RBCs were shown to have functional aquaporin 1. Expression studies with *AQP1* encoding Arg47 demonstrated a loss of Co^a antigen expression despite the presence of the Co^a-specific Ala45, consistent with the observed phenotype. Arnaud *et al.* [15] also re-evaluated three antisera classified as anti-Co3 in their collection and re-identified one of the three sera as anti-Co4. PCR-RFLP analysis revealed that this sample also was homozygous for *AQP1* 140G. Thus, this polymorphism defines a new high-incidence antigen, CO4, in the Colton blood group system.

System 20: Gerbich

Three novel high-incidence antigens among four individuals have been added to the Gerbich blood group system: GE10 (GEPL), GE11 (GEAT), GE12 (GETI) [16]. GE10 was identified following the investigation of an individual whose RBCs typed GE:2,3,4 but whose plasma contained an apparent alloanti-Ge3. Immunoblotting analysis showed the presence of both normal GPC/GPD and two unexpected bands that were approximately 2 kDa smaller than normal. DNA sequence analysis revealed a single point mutation in *GYPC*, 134C>T, which encodes an amino acid change Pro to Leu at positions 45 and 24 of GPC and GPD, respectively.

Anti-Ge11 was nonreactive with GE:–2,–3 and GE:–2,–3,–4 RBCs, but demonstrated variable reactivity with GE:–2,3 RBCs. Bands corresponding to GPC and GPD of apparently normal mass were shown by immunoblotting and DNA sequence analysis revealed homozygosity for a

point mutation 56A>T that encodes an amino acid change of Asp19Val in GPC.

Anti-Ge12 was identified in the plasma of two siblings and an unrelated individual whose RBCs typed GE:–2,3,4 (although typing for Ge3 and Ge4 on the siblings' RBCs was weaker than controls). Immunoblotting analysis of RBC membranes from the siblings revealed normal GPC and GPD as well as a broad band similar to that seen in GE:–2,3,4 RBCs, although the RBCs of the unrelated patient were normal. DNA sequence analysis revealed that the siblings showed compound heterozygosity for two mutations: 80C>T in exon 2 that encodes Thr27Ile in GPC and Thr6Ile in GPD and a transition in the invariant exon-adjacent nucleotide of intron 1, IVS2 –1g>a. This change most likely causes exon skipping and results in an aberrant protein, similar to GE:–2,3,4 RBCs in which exon 2 is deleted and explains the unusual blotting results. The unrelated patient was homozygous for 80C>T. Thus, Thr27Ile and Thr6Ile define the GE12 (GETI) antigen.

System 21: Cromer

An antibody directed at an apparently Cromer-related high-incidence antigen was shown to detect a new antigen in that system, CROM16 (CROZ) [17]. DNA sequence analysis of *DAF* revealed a missense transition, 389G>A in exon 3 that encodes an amino acid change of Arg130His.

System 24: OK

Two new high-incidence antigens have been identified in the OK blood group system; OK2 (OKGV) and OK3 (OKVM). The OK:–2 phenotype described in 2003 was shown to arise from a nucleotide substitution in exon 2 of the basigin gene, *BSG* 176G>T, which encodes the amino acid change Gly59Val [18]. This mutation was found only in the proposita, together with two additional silent single nucleotide exchanges; 195C>T (Asp65) and 234G>C (Ser78). Interestingly, an additional silent transition, 327T>C (Ala108), was identified in one of the control samples sequenced in this study.

Karamatic Crew *et al.* [19] also determined the molecular basis of the OK:–3 phenotype: the proband's plasma was nonreactive with three examples of Ok(a–) RBCs tested, although her RBCs typed Ok(a+). Sequence analysis of *BSG* revealed homozygosity for another mutation in exon 2: 178G>A, causing a Val60Met change in the OK glycoprotein (basigin, CD147).

System 26: JMH

A sixth antigen has been added to the increasingly polymorphic JMH blood group system; JMH6 (JMHQ). An antibody with JMH-related specificity in the plasma of four

unrelated native Americans led to the investigation of *SEMA7A* [20]. The molecular basis was identified as a transversion, 1040G>T, which encodes a change of Arg347Leu. Recombinant forms of *SEMA7A* were constructed and used in an ELISA, which confirmed reactivity of the antibodies with wild-type *SEMA7A*, while the JMHQ– isoform was nonreactive with the plasma samples.

Collection 209: GLOB

A new antigen, PX2 has been added to the GLOB Collection (209). This antigen is a high-incidence antigen defined by a terminal β 3-*N*-acetylgalactosamine (β 3GalNAc) on paragalloboside and is known to be abundant on PP1P^k– (p phenotype) RBCs [21, 22]. Recent data have revealed that rare individuals lacking the P antigen make antibodies not only against globoside but also against this 'new' antigen, which makes their plasma incompatible with p phenotype RBCs (J. R. Storry, T. Peyrard, A. H. Hult, Å. Hellberg, M. L. Olsson, manuscript in preparation).

Collection 213

A new Collection, number 213 (Table 2), has been created to recognize the carbohydrate antigens associated with the M and N antigens in MNS (ISBT system 002). These antigens have previously been defined biochemically as different substitutions of the sialic acid-carrying oligosaccharides on glycoprotein A [23].

Allele terminology

To address the fourth term of reference listed at the beginning of this article, a subcommittee has drafted guidelines for blood group allele terminology that can be found on the Blood Group Terminology website at <http://ibgri.blood.co.uk/ISBTPages/ISBTHome.htm>. Proposed allele terminology for 26 of the 30 blood group systems can also be found here and comments/feedback are encouraged. The proposed guidelines are intentionally noninclusive of silent single nucleotide polymorphisms (SNPs) and other

Table 2 Collection 213: Antigens that are associated with M and N antigens and that are the result of different substitutions of the sialic acid-carrying oligosaccharides on glycoprotein A [23]

Number	Antigen name
213001	Hu
213002	M ₁
213003	Tm
213004	Can
213005	Sext
213006	Sj

polymorphisms that do not affect the protein product. The primary goal is to establish an allele name for use by transfusion medicine practitioners that is readily recognizable. Significantly, the recommendations state that an allele encoding an antigen can be named even when only a small region of the gene had been sequenced or analysed by a SNP-specific method. This criterion was based on current laboratory tests, both in-house and commercial assays, which generally focus only, or mainly, on the molecular polymorphism(s) that gives rise to the antigen. The intent is to make the terminology acceptable for use in laboratory reports of analysis, other correspondence and in publications relating to transfusion medicine. All comments should

be sent to the Working Party chair at the address listed in Appendix 1. Tables giving the allele names will eventually reside on the ISBT website and not at the IBGRL website. At the time of submission, the address was not available, but it will be posted at the web address given above. Comments on the proposed nomenclature are welcomed before the next meeting of the Working Party in Mexico City, 2012.

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