REPORT

Blood group terminology 2004: from the International Society of Blood Transfusion committee on terminology for red cell surface antigens

G. L. Daniels,¹ A. Fletcher,² G. Garratty,³ S. Henry,⁴ J. Jørgensen,⁵ W. J. Judd,⁶ C. Levene,⁷ C. Lomas-Francis,⁸ J. J. Moulds,⁹ J. M. Moulds,¹⁰ M. Moulds,¹¹ M. Overbeeke,¹² M. E. Reid,⁸ P. Rouger,¹³ M. Scott,¹⁴ P. Sistonen,¹⁵ E. Smart,¹⁶ Y. Tani,¹⁷ S. Wendel¹⁸ & T. Zelinski¹⁹

¹Bristol Institute for Transfusion Sciences, Bristol, UK

²Growing your Knowledge, Spit Junction, NSW, Australia

³American Red Cross Blood Services, Los Angeles-Orange Counties Region, Los Angeles, CA, USA

⁴Biotechnology Research Centre, Auckland University of Technology, Auckland, New Zealand

⁵Regional Blood Transfusion Center, Department of Clinical Immunology, University Hospital, Århus N, Denmark

⁶Department of Pathology, University Hospitals UH-2G332, Ann Arbor, Michigan, USA

⁷Reference Laboratory for Immunohematology and Blood Groups, National Blood Services Centre, Tel Hashomer, Israel

⁸New York Blood Center, New York, NY, USA

⁹Ortho-Clinical Diagnostics, Raritan, NJ, USA

¹⁰Drexel University College of Medicine, Philadelphia, PA, USA

¹¹Gamma Biologicals Inc (subsidiary of Immunocor Inc), Houston, TX, USA

¹²Central Laboratory of the Netherlands Red Cross Blood Transfusion Service, Amsterdam, the Netherlands

¹³Centre national de Référence pour les Groupes sanguines (CNTS), Paris, France

¹⁴International Blood Group Reference Laboratory, Bristol, UK

¹⁵Finnish Red Cross Blood Transfusion Service, Helsinki, Finland

¹⁶South African National Blood Service, East Coast Region, Pinetown, South Africa

¹⁷Osaka Red Cross Blood Center, Osaka, Japan

¹⁸Blood Bank, Hospital Sirio-Libanes, São Paulo, Brazil

¹⁹Rh Laboratory, University of Manitoba, Winnipeg, Manitoba, Canada

Vox Sanguinis

Received: 17 August 2004, accepted 19 August 2004

Key words: blood groups, terminology, genetics.

Introduction

Human blood groups were discovered over 100 years ago. During the course of the 20th century, a variety of different styles of terminology has been used to denote them. The International Society of Blood Transfusion (ISBT) established a Working Party in 1980 to devise a genetically based numerical terminology for red cell surface antigens. In 1990, the Work-

Correspondence: Geoff Daniels, Bristol Institute for Transfusion Sciences, Southmead Road, Bristol, BS10 5ND, UK E-mail: geoff.daniels@nbs.nhs.uk ing Party published a monograph describing a numerical terminology for 242 red cell antigens [1]. Another monograph listing 254 antigens was published in 1995 [2] and four brief updates have followed [3–6]. In the 9 years since the 1995 report, many amendments to the classification have been necessary: 30 new antigens have been identified and six new systems created. Since 1995, the genes for nine blood group systems have been identified, so that sequenced genes for all 29 systems are now available. All blood group system genes have been located on a specific region of a chromosome (Table 1). The current classification can be found at www.iccbba.com/ppage107.htm.

Table 1	The blood group systems	, the genes that encode them,	their chromosomal I	ocations, and associated CD numbers
---------	-------------------------	-------------------------------	---------------------	-------------------------------------

No.	System name	System symbol	Gene name(s) ^a	Chromosomal location	CD numbers	Refs
001	ABO	ABO	ABO	9q34.2		
002	MNS	MNS	GYPA, GYPB, GYPE	4q31.21	CD235	
003	Р	P1		22q11.2-qter		
004	Rh	RH	RHD, RHCE	1p36.11	CD240	
005	Lutheran	LU	LU	19q13.32	CD239	
006	Kell	KEL	KEL	7q34	CD238	
007	Lewis	LE	FUT3	19p13.3		
800	Duffy	FY	FY	1q23.2	CD234	
009	Kidd	JK	SLC14A1	18q12.3		[7,8]
010	Diego	DI	SLC4A1	17q21.31	CD233	
011	Yt	ΥT	ACHE	7q22.1		
012	Xg	XG	XG, MIC2	Xp22.33, Yp11.3	CD99 ^b	
013	Scianna	SC	ERMAP	1p34.2		[9]
014	Dombrock	DO	DO	12p12.3		[10,11]
015	Colton	CO	AQP1	7p14.3		
016	Landsteiner-Wiener	LW	ICAM4	19p13.2	CD242	
017	Chido/Rodgers	CH/RG	C4A, C4B	6p21.3		
018	Н	Н	FUT1	19q13.33	CD173	
019	Kx	XK	XK	Xp21.1		
020	Gerbich	GE	GYPC	2q14.3	CD236	
021	Cromer	CROM	DAF	1q32.2	CD55	
022	Knops	KN	CR1	1q32.2	CD35	
023	Indian	IN	CD44	11p13	CD44	
024	Ok	OK	BSG	19p13.3	CD147	[12]
025	Raph	RAPH	CD151	11p15.5	CD151	[13–15]
026	John Milton Hagen	JMH	SEMA7A	15q24.1	CD108	[16,17]
027	I	I	GCNT2	6p24.2		[18,19]
028	Globoside	GLOB	B3GALT3	3q26.1		[20,21]
029	Gill	GIL	AQP3	9p13.3		[22,23]

^aAs recognized by the HUGO Gene Nomenclature Committee (www.gene.ucl.ac.uk/nomenclature/); ^bMIC2 product.

The purpose of this monograph is to describe the ISBT terminology for red cell surface antigens and to tabulate the complete current version of the classification. Much of the information provided in the 1995 monograph [1] is reiterated here so that referral back will not generally be required, but only references published after 1995 are provided.

The ISBT numerical terminology

The mandate of the Committee is to provide a numerical terminology for red cell surface antigens. By definition, these antigens must be defined serologically by the use of a specific antibody. Numerical designations and blood group symbols cannot be allocated to nucleotide or amino acid sequences or polymorphisms, even though the presence of specific blood group antigens may be indicated by these techniques. All antigens receiving ISBT numbers must have been shown to be inherited characters.

lood group symbolsEach antigen belonging to a blood group system is identi-
fied by a six-digit number (Tables 1–3). The first three digits
represent the system (e.g. 006 for Kell) and the second three
the specificity (e.g. 006003 for Kp^a). Alternatively, the system
symbol followed by the antigen number may be used

All authenticated antigens fall into one of four classifica-

tions: systems; collections; low-incidence antigens (700 series);

and high-incidence antigens (901 series). A blood group system

consists of one or more antigens controlled at a single gene

locus, or by two or more very closely linked homologous

genes with little or no observable recombination between them. Each system has been shown to be genetically discrete from every other system. Collections consist of serologically,

biochemically, or genetically related antigens, which do not

fit the criteria required for system status. The 700 and 901

series contain low- and high-incidence antigens, respectively,

(e.g. KEL003 or, more usually, KEL3 as sinistral zeros may be

which cannot be included in a system or collection.

 Table 2
 Examples of antigen, phenotype, gene, and genotype designations

 in International Society of Blood Transfusion (ISBT) and traditional
 terminologies

	ISBT	Traditional and 'popular' alternative
Antigen	LU1 or 005001	Lu ^a
Phenotype	LU:-1,2	Lu(a-b+)
Gene	LU*1	Lu ^a
Genotype	LU*2/2	Lu ^b /Lu ^b
	LU* 1,8/2,14	Lu ^a Lu ⁸ /Lu ^b Lu ¹⁴

removed). Phenotypes are represented by the system symbol, followed by a colon, followed by a list of antigens separated by commas. Those antigens shown to be absent are preceded by a minus sign (e.g. KEL:–1,2,–3,4). Genes are designated by the system symbol, followed by an asterisk, followed by the antigen number (e.g. *KEL*3*). Genotypes have the system symbol, followed by an asterisk, followed by alleles or haplotypes separated by a slash (e.g. *KEL*2,3/2,4*). Amorph or null genes are represented by a zero (e.g. *KEL*2,3/0*). Gene and genotype designations are italicized (or underlined). Antigen, phenotype, gene and genotype designations for collections are constructed in the same way. For the 700 and 901 series, 700 or 901 replaces the system symbol.

Symbols for gene loci

The symbol for a gene or cluster of genes controlling a blood group system is the italicized symbol for the system (e.g. CO for the gene controlling the Colton system). The genes controlling all of the blood group systems have been cloned and, in many cases, the gene locus has an alternative symbol (Table 1), usually because the protein product of the gene is of known function or the gene had an existing symbol before the association with the blood group was known. Examples: the genes controlling expression of the Diego and Kidd antigens encode solute carriers with the symbols *SLC4A1* and *SLC14A1*, respectively; the Yt antigens are encoded by the acetylcholinesterase gene ACHE; the Colton and GIL antigens are encoded by AQP1 and AQP3, respectively, the genes producing the water transporters Aquaporin-1 and -3; the presence of H antigen on the red cells is determined by an α -1,2fucosyltransferase gene, FUT1; and the presence of Lewis antigens on red cells is indirectly determined by an α -1, 3/4-fucosyltransferase gene, FUT3. At least two genes, RHD and RHCE, govern the Rh antigens, and three genes, GYPA, GYPB, and GYPE, are involved in the expression of antigens of the MNS system. The symbol for the gene governing a serologically determined blood group antigen comprises the system symbol and antigen number (e.g. RH*1, DI*2, CO^*1). When the gene encoding a blood group antigen is determined by non-serological means, such as nucleotide sequencing, the alternative symbol should be used (e.g. *SLC14A1* instead of *JK*, and *DAF* instead of *CROM*) and allele designations should conform to the established terminologies employed for nucleotide polymorphisms (see refs 24,25 and www.bioc.aecom.yu.edu/bgmut/index.htm).

It is important to remember that when symbols such as RH^*2 or MNS^*10 are used, the symbol does not necessarily represent a single defined gene, but one of a cluster of genes or a hybrid of two genes.

A 'popular', alternative terminology

The numerical terminology was devised primarily for computer storage of information on blood group antigens and to provide a framework for a genetical classification. The numerical terminology is not suitable for everyday communication and many scientists working in the field of human blood groups prefer not to use it in publications. This has led to a variety of alternative names being used for some blood group antigens. In an attempt to introduce some uniformity, a recommended list of alternative names for antigens is provided in Table 3. In most cases, the name or symbol is identical to that originally published, but in a few cases the more commonly used name is provided. In addition, there are recommended formats for describing phenotypes in the alternative terminology. These mostly employ traditional terminology, and examples are shown in Table 4. A more complete list has been published previously [4].

Blood group systems

There are currently 29 systems containing a total of 245 antigens (Tables 1 and 3).

Modifications since 1995: existing systems

002, the MNS system

Five new antigens have been added. Absence of the highincidence antigen MNS39 (ENEP) and presence of the lowincidence antigen MNS41 (HAG) is associated with an Ala65Pro substitution in glycophorin A (GPA) [26]. The absence of MNS42 (ENAV, previously AVIS) and the presence of MNS43 (MARS), its low-incidence antithetical antigen, results from a Gln63Lys substitution in GPA [27]. Both of these substitutions are associated with abnormal expression of DI4 (Wr^b). MNS40 (ENEH) is absent from glycophorin A of the phenotype GP.Vw (Mi.I); the glycophorin A has a Thr28Met substitution and expresses MNS9 (Vw) antigen [28].

003, the P system

Evidence was presented suggesting that the same transferase encoded by *A4GALT*, is responsible for the biosynthesis of

		Antige	n numbe	er																					
System		001	002	003	004	005	006	007	800	009	010	011	012	013	014	015	016	017	018	019	020	021	022	023	024
001	ABO	А	В	A,B	A1																				
002	MNS	Μ	Ν	S	S	U	He	Mi ^a	Mc	Vw	Mur	M ^g	Vr	Me	Mt ^a	St ^a	Ri ^a	Cla	Ny ^a	Hut	Hil	Mv	Far	sD	Mit
003	Р	P1																							
004	RH	D	С	Е	с	e	f	Ce	Cw	C×	V	Ew	G					Hr _o	Hr	hr ^s	VS	C^G	CE	D^w	
005	LU	Lu ^a	Lu ^b	Lu3	Lu4	Lu5	Lu6	Lu7	Lu8	Lu9		Lu11	Lu12	Lu13	Lu14		Lu16	Lu17	Au ^a	Au ^b	Lu20	Lu21			
006	KEL	К	k	Kp ^a	Кр ^ь	Ku	Js ^a	Js ^b			Ula	K11	K12	K13	K14		K16	K17	K18	K19	Km	Кр ^с	K22	K23	K24
007	LE	Le ^a	Le ^b	Leab	Le ^{bH}	ALe ^b	BLe ^b																		
800	FY	Fy ^a	Fy ^b	Fy3	Fy4	Fy5	Fy6																		
009	JK	Jk ^a	Jkb	Jk3																					
010	DI	Dia	Dib	Wr ^a	Wr ^b	Wd ^a	Rb ^a	WARR	ELO	Wu	Bp ^a	Mo ^a	Hg^{a}	Vg ^a	Sw ^a	BOW	NFLD	Jn ^a	KREP	Tr ^{a*}	Fr ^a	SW1			
011	ΥT	Yt ^a	Yt ^b																						
012	XG	Xg ^a	CD99																						
013	SC	Sc1	Sc2	Sc3	Rd	STAR																			
014	DO	Do ^a	Do^b	Gy ^a	Hy	Jo ^a																			
015	CO	Co ^a	Co ^b	Co3																					
016	LW					LW ^a	LW ^{ab}	LW ^b																	
017	CH/RG	Ch1	Ch2	Ch3	Ch4	Ch5	Ch6	WH				Rg1	Rg2												
018	н	Н										5	5												
019	XK	Kx																							
020	GE		Ge2	Ge3	Ge4	Wb	Ls ^a	An ^a	Dh ^a	GEIS															
021	CROM	Cr ^a	Tc ^a	Tc ^b	Tc ^c	Dr ^a	Es ^a	IFC	WES ^a	WES ^b	UMC	GUTI	SERF	ZENA											
022	KN	Kn ^a	Кп ^ь	McC ^a	SI1	Yk ^a	McC ^b	SI2	SI3*																
023	IN	In ^a	In ^b																						
024	ОК	Ok ^a																							
025	RAPH	MER2																							
026	JMH	JMH																							
027	1	1																							
028	GLOB	P																							
029	GIL	GIL																							
		025	026	027	028	029	030	031	032	033	034	035	036	037	038	039	040	041	042	043	044	045	046		
002	MNS	Dantu	Нор	Nob	Ena	En ^a KT	'N'	Or	DANE	TSEN	MINY	MUT	SAT	ERIK	Osa	ENEP	ENEH	HAG	ENAV	MARS	0 17	0 10	0.10		
002	RH		c-like	cE	hr ^H	Rh29	Go ^a	hr ^B	Rh32	Rh33	Hr ^B	Rh35		Evans		Rh39	Tar	Rh41	Rh42	Crawford	Nou	Riv	Sec		
004	KEL	VLAN	TOU	RAZ	VONG	11123	00		mijz	11155		11155	DC	Lvans		11155	101	111-71	111-7Z	crawfolu	nou	1117	JCC		
		047	048	049	050	051	052	053	054	055	056														
004	RH	Dav	JAL	STEM	FPTT	MAR	BARC	JAHK	DAK	LOCR	CENR														
004	ΝП	DdV	JAL	SIEIVI	FFII	WAR	DAIL	ЛЛИГ	DAK	LUCK	CEINK														

... = obsolete (see Table 9); *provisional.

Numerical terminology	Alternative terminology
ABO:-1,-2,-3	0
ABO:1,-2,3,4	A ₁
ABO:1,-2,3,-4	A ₂
MNS:1,2,-3,4,5,-6,7	M+ N+ S- s+ U+ He- Mi(a+) (in ISBT order)
	Symbols such as Mi.III or GP.Mur, En(a–), M ^k are also acceptable
P:1	$P1+ \text{ or } P_1$
P:-1	P1– or P ₂ (if shown to be GLOB:1)
RH:1,2,-3,4,5,-8,32,33,-36	D+ C+ E- c+ e+ C ^w - Rh:-32,33 Be(a-) (in ISBT order)
	The order D C c E e would be an acceptable alternative. Probable genotypes as
	phenotypes (e.g. R_1R_2 or DCe/DcE; $R_1r C^w$ + or DCe/dce C^w +) are acceptable, providing
	it is made clear that they are only probable genotypes based on haplotype frequencies
	Null and mod phenotypes: Rh _{null} ; Rh _{mod} .
LU:1,2,3,4	Lu(a-b+) Lu:3,4.
	Null phenotype: Lu _{null} or Lu(a–b–)
KEL:-1,2,-3,4,5,-6,7,11,12,13,-17,-21	K– k+ Kp(a–b+c–) Ku+ Js(a–b+) K:11,12,13,–17,–21
	Null and mod phenotypes: K _o or Kell _{null} ; K _{mod} .
FY:1,2,3	Fy(a+b+) Fy:3
FY:-1,-2,-3	Fy(a–b–) Fy:–3
	Fy ^x may be used as a phenotype
DI:1,2,-3,4,-5,-6,-7	Di(a+b+) $Wr(a-b+)$ $Wd(a-)$ $Rb(a-)$ WARR-
D0:1,2,3,4,5	Do(a+b+) Gy(a+) Hy+ Jo(a+)
LW:5,6,-7	LW(a+b-) LW(ab+)
CH/RG:1,2,-7,11,12	Ch:1,2 WH- Rg:1,2
H:1	H – The symbol O _h may be used for the true Bombay phenotype (red cells totally
	H-deficient, ABH non-secretors). Otherwise the terms 'Red cell H-deficient
	secretor' and 'Red cell H-deficient non-secretor' are recommended.
XK:-1	Kx– or McLeod
GE:2,3,4,-5,-6,-7,-8,-9	Ge:2,3,4 Wb- Ls(a-) An(a-) Dh(a-) GEIS-
GE:2,3,4	Ge:-2,-3,4 or Gerbich phenotype
GE:2,3,4	Ge:-2,3,4 or Yus phenotype
GE:-2,-3,-4	Ge:-2,-3,-4 or Leach phenotype
RAPH:1	MER2+
l:1	l adult
l:-1	i adult or cord

both P1 and P^k (209002 of the GLOB Collection) [29]. However, because of reports of conflicting evidence [30] (L. Tilley, G. Daniels, unpublished) P^k has not joined the P system.

004, the Rh system

Five antigens of low incidence have been added: RH52 (BARC) is produced by most $D^{VI}Ce$ haplotypes, but is not produced by $D^{VI}cE$ haplotypes [31]; RH53 (JAHK) is associated with the rare Rh phenotype r^{G} [32]; RH54 (DAK) with DIIIa, DOL, and some other rare Rh phenotypes [33]; RH55 (LOCR, previously 700053) with weak expression of RH4 (c) and RH5 (e) [34]; and RH56 (CENR) with an *RHCE-D* hybrid gene associated with RH8 (C^W) and altered expression of RH2 (C) and RH5 (e) [35].

A new terminology has been devised for epitopes of RH1 (D) [36]. Basically, each epitope has two numbers, one representing the conventional epD1 to epD9 system proposed by

Lomas *et al.* [37], the other representing subdivisions of those numbers. The complete numerical terminology for epD1 is 004001.001.001 (abbreviated to epD1.1) and the epD1 subsplit caused by DFR is 004001.001.002 (epD1.2). Partial D antigens will be designated by up to four upper case letters or Roman numerals on the line; for example DIIIa, DIV, DHAR, DFR.

005, the Lutheran system

Absence of a new Lutheran antigen of high incidence, LU21, is associated with homozygosity for an *LU* mutation encoding Asp94Glu [38].

006, the Kell system

KEL25 (VLAN) and KEL28 (VONG) are low-incidence antigens associated with Arg248Gln and Arg248Trp, respectively, in the Kell glycoprotein [39,40]. KEL26 (TOU) and KEL27 (RAZ) are antigens of high incidence, absent from the Kell glycoprotein with Arg406Gln [41] and Glu299Lys [39] substitutions, respectively.

007, the Lewis system

Three antigens, previously known for many years, were added to this system. LE4 (Le^{bH}) is defined by antibodies that react with LE:2 [Le(b+)] cells, but only when H1 (H) is strongly expressed (group 0 and A_2 phenotypes). LE5 (ALe^b) and LE6 (BLe^b) are expressed when A type 1 and B type 1 are modified by the product of the Lewis gene, *FUT3* [42].

010, the Diego system

Seventeen new antigens have been added, all of low incidence and all associated with mutations in *SLC4A1* encoding amino acid substitutions in the erythrocyte anion exchanger (Table 5). The DI19 designation for Tr^a is still provisional because only one case was analysed.

012, the Xg system

CD99, a glycoprotein encoded by a gene on both X and Y chromosomes, is recognized by monoclonal antibodies and human alloantibodies [54,55]. *MIC2*, the structural gene for CD99, is closely linked to *XG* and shares substantial sequence homology with *XG*. Consequently, CD99 became XG2.

013, the Scianna system

Following the discovery that the Scianna antigens are expressed by the red cell adhesion protein, human ERMAP, expression of the low-incidence antigen, 700015 (Rd), was found to be associated with a mutation in *ERMAP* encoding

Table 5 Antigens of the Diego System assigned since 1995
--

Antigen		Previous number	Amino acid substitution	References
DI5	Wd ^a	700030	Val557Met	[43,44]
DI6	Rb ^a	700027	Pro548Leu	[44]
DI7	WARR	700055	Thr552lle	[45]
DI8	ELO	700051	Arg432Trp	[46,47]
DI9	Wu	700013	Gly565Ala	[48]
DI10	Bp ^a	700010	Asn569Lys	[47]
DI11	Mo ^a	700022	Arg656His	[47]
DI12	Hg ^a	700034	Arg656Cys	[47]
DI13	Vg ^a	700029	Tyr555His	[47]
DI14	Sw ^a	700004	Arg646GIn or Trp	[49]
DI15	BOW	700046	Pro561Ser	[47,50,51]
DI16	NFLD	700037	Glu429Asp, Pro561Ala	[51]
DI17	Jn ^a	700014	Pro566Ser	[52]
DI18	KREP		Pro566Ala	[50]
DI19	Tr ^a *	700008	Lys551Asn	[44]
DI20	Fr ^a	700026	Glu480Lys	[53]
DI21	SW1	700041	Arg646Trp	[49]

Pro60Ala [9]. 700015 has become SC4. A new antigen of high incidence, STAR, is associated with Glu47Lys in ERMAP [56] and numbered SC5.

018, the Hh system

The system name has been changed to H.

020, the Gerbich system

A new antigen of low incidence, GEIS, associated with Thr32Asn in GPC and Thr11Asn in GPD [57], has been numbered GE9.

021, the Cromer system

Three new antigens of high incidence have been added: CROM11 (GUTI) [58], CROM12 (SERF) [59], and CROM13 (ZENA) [60] are associated with Arg206His, Pro182Leu and His208Gln in CD55, respectively.

022, the Knops system

Three new antigens have been added: KN6 to KN8. KN6 (McC^b) represents Lys1590Glu in CD35, which is also associated with the absence of KN3 (McC^a) [61]. KN4 (previously Sl^a, now Sl1) requires Arg1601 on CD35, KN7 (previously Vil, now Sl2) requires Gly1601, and KN8 (Sl3) requires both Arg1601 and Ser1610 [61,62]. The number KN8 is provisional because further complications are predicted.

Modifications since 1995: new systems

024, the Ok system

The high-incidence antigen OK1 (Ok^a , previously 901006) became the sole antigen of the Ok system following identification of the gene (*BSG*) encoding the protein and of the amino acid substitution (Glu92Lys) responsible for the OK:-1 phenotype [12].

025, the Raph system

This new system contains one antigen: RAPH1 (MER2, previously 901011). Distinction of RAPH from the other blood group systems was demonstrated by family studies and by the positioning of *CD151*, the controlling gene, at 11p15.5 [13–15].

026, the John Milton Hagen system

JMH1 (JMH, previously 901007) is carried on the semaphorin CD108 [16]. The gene encoding CD108, *SEMA7A*, is located on the long arm of chromosome 15 [17], which distinguishes JMH1 from all existing blood group systems. JMH1 represents the antigen detected by antibodies produced by JMH:-1 individuals.

027, the I system

GCNT2, the gene encoding the *N*-acetylglucosaminyltransferase responsible for converting i-active straight chains to I-active branched chains, has been cloned and some mutations responsible for the i adult phenotype identified [18,19]. I1 antigen (I, previously 207001) is the only antigen of the I system. The i antigen, the precursor of I1, provisionally remains as 207002.

028, the Globoside system

The gene (*B3GALT3*) encoding the *N*-acetylgalactosaminyltransferase (globoside synthase) responsible for converting P^k (209002) to P (previously 209001, globoside) has been cloned and inactivating mutations responsible for the P^k (Pnegative) phenotype have been identified [20,21]. P has become GLOB1, the only antigen of the Globoside system.

029, the GIL system

GIL 1, the only antigen of the GIL system, is an antigen of high incidence located on the glycerol transporter aquaporin 3, and the GIL:–1 phenotype results from an inactivating mutation within the *AQP3* gene [22,23]. *AQP3* is on chromosome 9p13, so GIL is genetically discrete from all other blood group systems.

Guidelines for the establishment of new blood group systems

For an antigen to form a new blood group system it must be defined by a human alloantibody, be an inherited character, the gene encoding it must have been identified and sequenced, and its chromosomal location must be known. In addition, the gene must be different from, and not a closely linked homologue of, all other genes encoding antigens of existing blood group systems.

All established blood group systems meet these criteria.

Guidelines for the inclusion of a new specificity in an established system

All antigens awarded an ISBT number must have been shown to be inherited, and at least one of the following four criteria must be met.

(1) An antithetical relationship between a new antigen and one already assigned to the system.

(2) Demonstration that expression of the antigen is associated with a variation in the nucleotide sequence of the gene controlling the system.

(3) Evidence, from a linkage analysis of family data, that the controlling allele is probably a newly recognized form of the pertinent gene, and supporting serological or biochemical information.

(4) Demonstration that an antigen is located on a protein or glycoprotein that carries other antigens belonging to the system. It must be remembered, however, that this could result from post-translational modification of a gene product, such as glycosylation, which would not support inclusion within the system.

Blood group collections

There are six blood group collections (Table 6).

A new Collection, 211 or Vel, was created to include two serologically related antigens of high incidence, Vel (VEL1, previously 901001) and ABTI (VEL2, previously 901015) [63,64].

An antibody produced by a patient with ER:-1,-2 red cells and with the characteristics of antibodies of the ER Collection reacted with ER:1,-2, ER:-1,2, and the only other example of ER:-1,-2 red cells [65]. The antigen recognized by this antibody has become ER3 (208003) of the ER Collection.

Since 1995, 207001 (I1, I) has formed the I system and retains the symbol I1, and 209001 (GLOB1, P) has formed the Globoside system and retains the symbol GLOB1. The creation of the I and Globoside systems means that these two systems have identical symbols, I and GLOB, to those of Collections 207 and 209. This should not cause confusion, however, as I1 and GLOB1 cannot refer to 207001 and 209001, as those numbers are now obsolete. Collection 207 now contains only one antigen. These anomalies will be resolved when a separate carbohydrate collection is created.

700 series, low-incidence antigens

There are currently 19 antigens in the 700 series (Table 7). Since 1995, no new numbers have been added and 18 numbers have been made obsolete: 16 antigens have joined the Diego

Table 6 Collections of antigens

Collection			Antigen					
No.	Name	Symbol	No.	Symbol	Incidence percentage			
205	Cost	COST	205001	Cs ^a	95			
			205002	Cs ^b	34			
207	li	I	207002	i	×			
208	Er	ER	208001	Er ^a	> 99			
			208002	Er ^b	< 1			
			208002	Er3	> 99			
209		GLOB	209002	P ^k	> 99*			
			209003	LKE	98			
210			210001	Le ^c	1			
			210002	Le ^d	6			
211	Vel	VEL	211001	Vel	> 99			
			211002	ABTI	> 99			

*By standard serological tests, may appear to be low incidence. Obsolete collections: 201 Gerbich; 202 Cromer; 203 Indian; 204 Auberger; 206 Gregory; 211 Wright.

© 2004 Blackwell Publishing Ltd. Vox Sanguinis (2004) 87, 304-316

No.	Name	Symbol	No.	Name	Symbol
700002	Batty	Ву	700040	Rasmussen	RASM
700003	Christiansen	Chr ^a	700043	Oldeide	Ola
700005	Biles	Bi	700044		JFV
700006	Box	Bx ^a	700045	Katagiri	kg
700017	Torkildsen	To ^a	700047	Jones	JONES
700018	Peters	Pt ^a	700049		HJK
700019	Reid	Re ^a	700050		HOFM
700021	Jensen	Je ^a	700052		SARA
700028	Livesay	Li ^a	700054		REIT
700039	Milne				

Table 8 The 901 series: high-incidence antigens

No.	Name	Symbol	No.	Name	Symbol
901002	Langereis	Lan	901012	Sid	Sd ^a
901003	August	At ^a	901013	Duclos	
901005		Jr ^a	901014		PEL
901008		Emm	901016		MAM
901009	Anton	AnWj			

system (Table 5); 700015 (Rd) has become SC4; and 700053 (LOCR) has become RH55.

Criteria for inclusion in the 700 series:

(1) An incidence of < 1% in most populations tested.

(2) Distinction from all other numbered low-incidence antigens of the 700 series as well as those of the blood group systems and collections.

(3) Demonstration of inheritance through at least two generations.

901 series, high-incidence antigens

There are currently nine antigens in the 901 series (Table 8). Since 1995, two new antigens, both of high incidence, have been added: 901015 (ABTI) [63] and 901016 (MAM) [66], although 901015 (ABTI) has now become VEL2 of the 211 collection. Three antigens became the sole antigens of new blood group systems and their 901 numbers are obsolete: 901006 (Ok^a) has become OK1; 901007 (JMH) has become JMH1; and 901011 (MER2) has become RAPH1 (Table 1).

The gene encoding the β 1,4-*N*-acetylgalactosaminyltransferase responsible for 901012 (Sd^a) antigen has been cloned [67,68], but 901012 has not formed a new blood group system because the molecular basis for the Sd^a polymorphism has not been determined.

Criteria for inclusion in the 901 series:

(1) An incidence of > 90% in most populations tested.

(2) Distinction from all other numbered high-incidence specificities.

(3) Demonstration that the antigen is lacking from the red cells of at least 2 sibs, i.e. that the negative phenotype is genetically determined.

Obsolete numbers

There is no recycling of blood group numbers: once a number has been allocated to a specificity, that number cannot be subsequently used for any other specificity. Consequently, if the number of a specificity becomes inappropriate, then that number becomes obsolete. Obsolete numbers are listed in Table 9.

Procurement of ISBT numerical designations

Choice of symbol

Symbols for designations of new specificities will consist of three to six on-line capital letters and must not duplicate, alphabetically or phonetically, any current or obsolete symbols shown in the tables. Also, symbols used in related fields, such as those used for platelet and leucocyte antigens, must be avoided. Symbols for specificities that may herald new blood group systems, and thus new genes, have the further constraint that they must differ from any symbols given to genes by the HUGO Nomenclature Committee (http://www. gene.ucl.ac.uk/nomenclature/).

Procedures for acquisition of an ISBT number

The initial stipulation is that materials for defining the new specificity should be available for either circulation or inhouse testing; the futility of defining a new specificity from which future new specificities cannot be distinguished is obvious. Proposals should be submitted with supporting data to the members of the Committee listed below, who are authorized to allocate numbers provisionally in consultation with the Chair. All decisions must be ratified by the Committee before being finalized.

- For a new blood group system or collection: Dr Geoff Daniels.
- For a specificity number within an established system: Dr Marion Reid for the MNS system; Dr Sylvano Wendel or Mrs Marilyn Moulds for the Rh system; Dr Jan Jørgensen for other systems.
- For a specificity number in a current collection: Dr Geoff Daniels.
- For a 700 number: Dr Teresa Zelinski.
- For a 901 number: Dr Geoff Daniels.

Table 9 Obsolete numbers

Obsolete no.	Symbol or previous symbol	Current no.	Obsolete no.	Symbol or previous symbol	Current no
001005	Н	018001	700010	Bp ^a	010010
003002	Р	209001	700011	Or	002031
003003	P ^k	209002	700012	Gf	Obsolete
004013	Rh ^A	Obsolete	700013	Wu	010009
004014	Rh ^B	Obsolete	700014	Jn ^a	010017
004015	Rh ^C	Obsolete	700015	Rd	013004
004016	Rh ^D	Obsolete	700016	Heibel	Obsolete
004024	ET	Obsolete	700020	An ^a	020007
004025	LW	Obsolete, see system 16	700022	Mo ^a	010011
004038	Duclos	901013	700023	Hey	Obsolete
005010	Singleton	Obsolete	700024	RI^{a} (= Ls^{a})	020006
005015	Anton	901009	700025	In ^a	023001
006008	Kw	Obsolete	700026	Fr ^a	010020
006009	KL	Obsolete	700027	Rb ^a	010006
006015	Кх	019001	700029	Vq ^a	010013
016001	LW, phenotype	Obsolete	700030	Wda	010005
016002	LW ₂ phenotype	Obsolete	700031	Dh ^a	020008
016003	LW ₃ phenotype	Obsolete	700032	POLL	Obsolete
016004	LW ₄ phenotype	Obsolete	700033	Os ^a	002038
020001	Ge1	Obsolete	700034	Hq ^a	010012
Collection 201	GE	System 020	700035	Tc ^b	021003
Collection 202	CROMER	System 021	700036	Tc ^c	021004
Collection 203	IN	System 023	700037	NFLD	010016
Collection 204	AU	005018 & 005019	700038	Hov (= Wu)	700013
205003	Yk ^a	022005	700041	SW1	010021
205004	Kn ^a	022001	700042	WES (WES ^a)	021008
205005	Кп ^ь	022002	700046	BOW	010015
205006	McC ^a	022003	700048	FPTT	004050
205007	Sl ^a	022004	700051	ELO	010008
Collection 206	GY	014003 & 014004	700053	LOCR	004055
207001	I	027001	700055	WARR	010007
Collection 211	WR	010003 & 010004	901004	Jo ^a	014005
209001	Р	028001	901006	Ok ^a	024001
700001	Wr ^a	010003	901007	JMH	026001
700004	Sw ^a	010013	901010	Wr ^b	010004
700007	Ls ^a	020006	901011	MER2	025001
700008	Tr ^a	010019	901001	Vel	211001
700009	Wb	020005	901015	ABTI	211002

Conclusions

Although the primary function of the Committee is to monitor and update the terminology, there are two main issues that need to be tackled in the future: updating and redefining the terminology for blood group genes and alleles; and devising a collection for those carbohydrate antigens that do not belong to a system.

The Committee strongly urges that all authors in the field of human red cell blood groups should use the terminology provided in Tables 1–8 in either the alphabetical/numerical format (e.g. FY2) or the 'popular' alternative format (e.g. Fy^b). For clarity, it is often advisable when using the more traditional terminology to include the ISBT terminology in parentheses, for example An^a (GE7).

All members of the Committee, listed in Appendix I, are prepared to handle requests for advice or assistance in any aspect of an investigation or to direct them to the most appropriate authority. The Chair of the Committee would be happy to receive any comments or criticisms of the terminology and would raise them at a Committee meeting. Membership of the Committee is open to colleagues who are considered international experts in the field of blood groups or related fields and who are prepared to participate in the activities of the Committee.

Acknowledgements

We are grateful to the following colleagues who have retired from the Committee since 1995: Prof. David Anstee, Prof. Jean-Pierre Cartron, Prof. Dr Wolfgang Dahr, Dr Peter Issitt, Dr Lief Kornstad, Dr Marie Lin, Dr Anatole Lubenko, Ms Delores Mallory, Dr Yasuto Okubo, Dr Siegfried Seidl, and Dr Graeme Woodfield.

References

- 1 Lewis M, Anstee DJ, Bird GWG, Brodheim E, Cartron J-P, Contreras M, Crookston MC, Dahr W, Daniels GL, Engelfriet CP, Giles CM, Issitt PD, Jørgensen J, Kornstad L, Lubenko A, Marsh WL, McCreary J, Moore BPL, Morel P, Moulds JJ, Nevanlinna H, Nordhagen R, Okubo Y, Rosenfield RE, Rouger Ph, Rubinstein P, Salmon Ch, Seidl S, Sistonen P, Tippett P, Walker RH, Woodfield G, Young S: Blood group terminology 1990. From the ISBT Working Party on Terminology for Red Cell Surface Antigens. *Vor Sang* 1990; 58:152–169
- 2 Daniels GL, Anstee DJ, Cartron JP, Dahr W, Issitt PD, Jørgensen J, Kornstad L, Levene C, Lomas-Francis C, Lubenko A, Mallory D, Moulds JJ, Okubo Y, Overbeeke M, Reid ME, Rouger P, Seidl S, Sistonen P, Wendel S, Woodfield G, Zelinski T: Blood Group Terminology 1995: From the ISBT Working Party on Terminology for Red Cell Surface Antigens. *Vox Sang* 1995; 69:265–279
- 3 Daniels GL, Anstee DJ, Cartron JP, Dahr W, Henry S, Issitt PD, Jørgensen J, Judd WJ, Kornstad L, Levene C, Lomas-Francis C, Lubenko A, Mallory D, Moulds JM, Moulds JJ, Okubo Y, Overbeeke M, Reid ME, Rouger P, Seidl S, Sistonen P, Wendel S, Zelinski T: Terminology for red cell surface antigens. Makuhari report. *Vox Sang* 1996; 71:246–248
- 4 Daniels GL, Anstee DJ, Cartron JP, Dahr W, Garratty G, Henry S, Jørgensen J, Judd WJ, Kornstad L, Levene C, Lomas-Francis C, Lubenko A, Moulds JJ, Moulds JM, Moulds M, Overbeeke M, Reid ME, Rouger P, Scott M, Seidl S, Sistonen P, Tani Y, Wendel S, Zelinski T: Terminology for red cell surface antigens. ISBT Working Party Oslo report. *Vox Sang* 1999; 77:52–57
- 5 Daniels GL, Anstee DJ, Cartron JP, Dahr W, Fletcher A, Garratty G, Henry S, Jørgensen J, Judd WJ, Kornstad L, Levene C, Lin M, Lomas-Francis C, Lubenko A, Moulds JJ, Moulds JM, Moulds M, Overbeeke M, Reid ME, Rouger P, Scott M, Sistonen P, Smart E, Tani Y, Wendel S, Zelinski T: Reports and guidelines. International Society of Blood Transfusion Working Party on terminology for red cell surface antigens. *Vox Sang* 2001; 80:193 – 196
- 6 Daniels GL, Cartron JP, Fletcher A, Garratty G, Henry S, Jørgensen J, Judd WJ, Levene C, Lin M, Lomas-Francis C, Moulds JJ, Moulds JM, Moulds M, Overbeeke M, Reid ME, Rouger P, Scott M, Sistonen P, Smart E, Wendel S, Zelinski T: International Society of Blood Transfusion Commitee on terminology for red cell surface antigens. *Vax Sang* 2003; 84:244–247
- 7 Olivès B, Mattei M-G, Huet M, Neau P, Martial S, Cartron JP, Bailly P: Kidd blood group and urea transport function of

human erythrocytes are carried by the same protein. *J Biol Chem* 1995; **270**:15607 – 15610

- 8 Sidoux-Walter F, Lucien N, Olivès B, Gobin R, Rousselet G, Kamsteeg EJ, Ripoche P, Deen PM, Cartron JP, Bailly P: At physiological expression levels the Kidd blood group/urea transporter protein is not a water channel. *J Biol Chem* 1999; 274:30228 – 30235
- 9 Wagner FF, Poole J, Flegel WA: The Scianna antigens including Rd are expressed by ERMAP. *Blood* 2002; 101:752–757
- 10 Eiberg H, Mohr J: Dombrock blood group (*D0*): assignment to chromosome 12p. *Hum Genet* 1996; **98**:518–521
- 11 Gubin AN, Njoroge JM, Wojda U, Pack SD, Rios M, Reid ME, Miller JL: Identification of the Dombrock blood group glycoprotein as a polymorphic member of the ADP-ribosyltransferase gene family. *Blood* 2000; 96:2621–2627
- 12 Spring FA, Homes CH, Simpson KL, Mawby WJ, Mattes MJ, Okubo Y, Parsons SF: The Ok^a blood group antigen is a marker for the M6 leukocyte activation antigen, the human homolog of OX-47 antigen, basigin and neurothelin, an immunoglobulin superfamily molecule that is widely expressed in human cells and tissues. *Eur J Immunol* 1997; 27:891–897
- 13 Daniels GL, Tippett P, Palmer DK, Miller YE, Geyer D, Jones C: MER2: a red cell polymorphism defined by monoclonal antibodies. *Vox Sang* 1987; 52:107 – 110
- 14 Daniels GL, Levene C, Berrebi A, Schechter Y, Moulds M, Sela R, Poole J, Lacey P, Atkins CJ: Human alloantibodies detecting a red cell antigen apparently identical to MER2. *Vox Sang* 1988; 55:161–164
- 15 Karamatic Crew V, Burton N, Kagan A, Green CA, Levene C, Flinter F, Brady LR, Daniels G, Anstee DJ: CD151, the first member of the tetraspanin (TM4) superfamily detected on erythrocytes, is essential for the correct assembly of human basement membranes in kidney and skin. *Blood* 2004; 104: 2217–2223.
- 16 Mudad R, Rao N, Angelisova P, Horejsi V, Telen MJ: Evidence that CDw108 membrane protein bears the JMH blood group antigen. *Transfusion* 1995; 35:566–570
- 17 Yamada A, Kubo K, Takeshita T, Harashima N, Kawano K, Mine T, Sagawa K, Sugamura K, Itoh K: Molecular cloning of a glycosylphosphatidylinositol-anchored molecule CDw108. J Immunol 1999; 162:4094–4100
- 18 Yu L-C, Twu Y-C, Chang C-Y, Lin M: Molecular basis of the adult i phenotype and the gene responsible for the expression of the human blood group I antigen. *Blood* 2001; 98:3840–3845
- 19 Yu L-C, Twu Y-C, Chou M-L, Reid ME, Gray AR, Moulds JM, Chang C-Y, Lin M: The molecular genetics of the human *I* locus and molecular background explaining the partial association of the adult I phenotype with congenital cataracts. *Blood* 2003; 101:2081–2088
- 20 Okajima T, Nakamura Y, Uchikawa M, Haslam DB, Numata Furukawa K, Urano T, Furukawa K: Expression cloning of human globoside synthase cDNAs. Identification of β3Gal-T3 as UDP-*N*-acetylgalactosamine: globotriosylceramide β1,3-*N*acetylgalactosaminyltransferase. *J Biol Chem* 2000; 275:40498– 40503
- 21 Hellberg Å, Poole J, Olsson ML: Molecular basis of the globoside-deficient P^k blood group phenotype. Identification of four inactivating mutations in the UDP-N-acetylgalactosamine:

globotriaosylceramide 3- β -N-acetylgalactosaminyltransferase gene. J Biol Chem 2002; 277:29455–29459

- 22 Daniels GL, DeLong EN, Hare V, Johnson ST, LePennec PY, Mallory D, Marshall MJ, Oliver C, Spruell P: GIL: a red cell antigen of very high frequency. *Immunohematology* 1998; 14:49–52
- 23 Roudier N, Ripoche P, Gane P, Le Pennec PY, Daniels G, Cartron JP, Bailly P: AQP3-deficiency in humans and molecular basis of a novel blood group system, GIL. J Biol Chem 2002; 277:45854– 45859
- 24 den Dunnen JT, Antonarakis E: Nomenclature for the description of human sequence variations. *Hum Genet* 2001; 109:121–124
- 25 Garratty G, Dzik W, Issitt PD, Lublin DM, Reid ME, Zelinski T: Terminology for blood group antigens and genes-historical origins and guidelines in the new millennium. *Transfusion* 2000; 40:477–489
- 26 Poole J, Banks JA, Bruce LJ, Ring SM, Tanner MJA, Levene C: A novel glycophorin A polymorphism affecting Wr^b expression. *Transfusion* 1995; 35:40S (Abstract)
- 27 Jarolim P, Moulds JM, Moulds JJ, Rubin HL, Dahr W: MARS and AVIS blood group antigens: polymorphism of glycophorin A affects band 3 glycophorin A interaction. *Blood* 1996; 88:182a (Abstract)
- 28 Spruell P, Moulds JJ, Martin M, Gilcher RO, Howard PB, Blumenfeld OO: An anti-En^aTS detected in the serum of an *Mi^I* homozygote. *Transfusion* 1993; 33:848–851
- 29 Iwamura K, Furukawa K, Uchikawa M, Sojka BN, Kojima Y, Wiels J, Shiku H, Urano T, Furukawa K: The blood group P1 synthase gene is identical to the Gb3/CD77 synthase gene: a clue to the solution of the P1/P2/, p. puzzle. *J Biol Chem* 2003; 45:44429–44438
- 30 Hellberg Å: Sixteen polymorphic sites in the 5'/3'-UTR of the P^k gene do not correlate with P1/P2 phenotypes. *Vox Sang* 2004; 87 (suppl 3):81 (Abstract)
- 31 Tippett P, Lomas-Francis C, Wallace M: The Rh antigen D: partial D antigens and associated low incidence antigens. *Vox Sang* 1996; 70:123 – 131
- 32 Green C, Coghlan G, Bizot M, Kasulke D, Bombail-Girard M, Wallace M, Lomas-Francis C, Daniels G: JAHK: a low frequency antigen associated with the r^G complex of the Rh blood group system. *Transfus Med* 2002; 12:55–61
- 33 Reid ME, Storry JR, Sausais L, Tossas E, Rios M, Hue-Roye K, Gloster ES, Miller ST, Wolf C, Lomas-Francis C: DAK, a new lowincidence antigen in the Rh blood group system. *Transfusion* 2003; 43:1394–1397
- 34 Coghlan G, Zelinski T: DNA microsatellite and linkage analysis supports the inclusion of LOCR in the Rh blood group system. *Transfusion* 2003; **43**:440–444
- 35 Westhoff CM, Storry JR, Walker P, Lomas-Francis C, Reid ME: A new hybrid *RHCE* gene (CeNR) is responsible for expression of a novel antigen. *Transfusion* 2004; 44:1047 – 1051
- 36 Scott ML, Anstee DJ, Cartron J-P, Dahr W, Daniels G, Fletcher A, Garratty G, Henry S, Jorgensen J, Judd WJ, Levene C, Lin M, Lomas-Francis C, Moulds JJ, Moulds JM, Moulds M, Overbeeke M, Reid ME, Rouger P, Sistonen P, Smart E, Tani Y, Wendel S, Zelinski T: International Society of Blood Transfusion Working Party on Terminology for Red Cell Surface Antigens – terminology for epitopes and variant antigens of Rh D (004001; RH1). Vor Sang 2004; in press

- 37 Lomas C, McColl K, Tippett P: Further complexities of the Rh antigen D disclosed by testing category DII cells with monoclonal anti-D. *Transfus Med* 1993; 3:67–69
- 38 Karamatic Crew V, Poole J, Banks J, Reed M, Daniels G: LU21: a new antigen in the Lutheran blood group system. *Vox Sang* 2004; 87:109–113.
- 39 Lee S, Reid ME, Redman CM: Point mutations in KEL exon 8 determine a high-incidence (RAZ) and a low-incidence (KEL25, VLAN) antigen of the Kell blood group system. *Vox Sang* 2001; 81:259–263
- 40 Grey D, Poole J, Martin P, Condon J, Allwright J, O'Day S, Daniels G: Haemolytic disease of the newborn caused by a new Kell antigen. *Transfus Med* 2003; 13 (suppl 1):30 (Abstract)
- 41 Lee S: Molecular basis of Kell blood group phenotypes. *Vox Sang* 1997; **73**: 1 11 and *Vox Sang* 1998; **74**: 58
- 42 Henry S, Oriol R, Samuelsson B: Lewis histo-blood group system and associated secretory phenotypes. Vox Sang 1995; 69:166–182
- 43 Bruce LJ, Zelinski T, Ridgwell K, Tanner MJA: The low-incidence blood group antigen, Wd^a, is associated with the substitution Val₅₅₇ → Met in human erythrocyte band 3 (*AE1*). *Vox Sang* 1996; **71**:118-120
- 44 Jarolim P, Murray JL, Rubin HL, Smart E, Moulds JM: Blood group antigens Rb^a, Tr^a, and Wd^a are located in the third ectoplasmic loop of erythroid band 3. *Transfusion* 1997; 37:607–615
- 45 Jarolim P, Murray JL, Rubin HL, Coghlan G, Zelinski T: A Thr₅₅₂ → Ile substitution in erythroid band 3 gives rise to the Warrior blood group antigen. *Transfusion* 1997; 37:398–405
- 46 Zelinksi T, Punter F, McManus K, Coghlan G: The ELO blood group polymorphism is located in the putative first extracellular loop of human erythrocyte band 3. *Vox Sang* 1998; **75**:63–65
- 47 Jarolim P, Rubin HL, Zakova D, Storry J, Reid ME: Characterization of seven low incidence blood group antigens carried by erythrocyte band 3 protein. *Blood* 1998; **92**:4836–4843
- 48 Zelinski T, McManus K, Punter F, Moulds M, Coghlan G: A Gly₅₆₅ → Ala substitution in human erythrocyte band 3 accounts for the Wu blood group polymorphism. *Transfusion* 1998; 38:745-748
- 49 Zelinski T, Rusnak A, McManus K, Coghlan G: Distinctive Swann blood group genotypes: molecular investigations. *Vox* Sang 2000; 79:215–218
- 50 Poole J, Bruce LJ, Hallewell H, Kusnierz-Alejska G, Zupanska B, Daniels GL, Tanner MJA: Erythrocyte band 3 mutation Pro561 → Ser givers rise BOW antigen Pro566 → Ala to a novel antigen KREP. *Transfus Med* 1998; 8 (suppl 1):17 (Abstract)
- 51 McManus K, Pongoski J, Coghlan G, Zelinski T: Amino acid substitutions in human erythroid protein, band 3 account for the low-incidence antigens NFLD and BOW. *Transfusion* 2000; 40:325–329
- 52 Poole J, Hallewell H, Bruce L, Tanner MJA, Zupanska B, Kusnierz-Alejska G: Identification of two new Jn(a+) individuals and assignment of Jn^a to erythrocyte band 3. *Transfusion* 1997; 37:90S (Abstract)
- 53 McManus K, Lupe K, Coghlan G, Zelinski T: An amino acid substitution in the putative second extracellular loop of RBC band 3 accounts for the Froese blood group polymorphism. *Transfusion* 2000; 40:1246–1249
- 54 Tippett P, Ellis N: The Xg blood group system: a review. *Transfus* Med Rev 1998; 12:233–257

- 55 Uchikawa M, Tsuneyama H, Tadokoro K, Juji T, Yamada M, Maeda Y: An alloantibody to 12E7 antigen detected in 2 healthy donors. *Transfusion* 1995; 35:23S (Abstract)
- 56 Hue-Roye K, Chaudhuri A, Velliquette RW, Thomas R, Balk M, Reid ME: A novel high prevalence antigen in the Scianna blood group system. *Vox Sang* 2004; 87 (suppl 3):40 (Abstract)
- 57 Yabe R, Uchikawa M, Tuneyama H, Ogasawara K, Toyoda T, Suzuki Y, Shimizu H, Uchida S, Nakajima K: IS: a new Gerbich blood group antigen located on the GPC and GPD. *Vox Sang* 2004; 87 (suppl 3):79 (Abstract)
- 58 Storry JR, Sausais L, Hue-Roye K, Mudiwa F, Ferrer Z, Blajchman MA, Lublin DM, Ma B-W, Miquel JF, Nervi F, Pereira J, Reid ME: GUTI: a new antigen in the Cromer blood group system. *Transfusion* 2003; 43:340–344
- 59 Banks J, Poole J, Ahrens N, Seltsam A, Salama A, Hue-Roye K, Storry JR, Palacajornsuk P, Ma B-W, Lublin DM, Reid ME: SERF: a new antigen in the Cromer blood group system. *Transfus Med* 2004; 14:313–318
- 60 Hue-Roye K, Powell V, Barnes J, Chung A, Fung Kee Fung K, Kinney J, Lublin D, Belaygorod. L, Reid M: ZENA: A new high prevalence Cromer blood group antigen. *Transfusion* 2004; 44: 26A (Abstract)
- 61 Moulds JM, Zimmerman PA, Doumbo OK, Kassambara L, Sagara I, Diallo DA, Atkinson JP, Krych-Goldberg M, Hauhart RE, Hourcade DE, McNamara DT, Birmingham DJ, Rowe JA, Moulds JJ, Miller LH: Molecular identification of Knops blood group polymorphisms found in long homologous region D of complement receptor 1. *Blood* 2001; 97:2879–2885
- 62 Moulds JM, Zimmerman PA, Doumbo OK, Diallo DA, Atkinson JP,

Krych-Goldberg M, Hourcade DE, Moulds JJ: Expansion of the Knops blood group system and subdivision of Sl^a. *Transfusion* 2002; **42**:251–256

- 63 Schechter Y, Chezar J, Levene C, Poole J, Moulds M, Daniels G: ABTI (901015), a new red cell antigen of high frequency. *Transfusion* 1996; 36:25S (Abstract)
- 64 Banks J, Poole J, Das Gupta C, Lonicer C, Salama A: Two new cases of nti–ABTI showing an association between ABTI and Vel. *Vox Sang* 2004; **87** (suppl 3):38 (Abstract)
- 65 Arriaga F, Mueller A, Rodberg K, Ciesielski D, Poole J, Banks J, de La Rubia J, Carpio N, Marty ML, Garratty G: A new antigen of the Er collection. *Vox Sang* 2003; 84:137–139
- 66 Montgomery WM, Jr, Nance SJ, Donnelly SF, Brady TW, Anderson G, Mintz PD, Moulds MK, Daniels GL, Spring FA, Molina N, de Asis EA, Olivares E: MAM: a 'new' high-incidence antigen found on multiple cell lines. *Transfusion* 2000; 40:1132–1139
- 67 Montiel M-D, Krzewinski-Recchi M-A, Delannoy P, Harduin-Lepers A: Molecular cloning, gene organization and expression of human UDP-GalNAc: Neu5Ac α 2–3Gal β -R β 1,4-*N*acetylgalactosaminyltransferase responsible for the biosynthesis of the blood group Sd^a/Cad antigen: evidence for an unusual extended cytoplasmic domain. *Biochem J* 2003; 373:369– 379
- 68 Presti LL, Cabuy E, Chiricolo M, Dall'olio F: Moleculer cloning of the human β 1, 4*N*-acetylgalactosaminyltransferase responsible for the biosynthesis of the Sol^a histo-blood group antigen: the sequence predicts a very long aytoplasmic domain. *J Biochem* 2003; 134:675–682

Appendix I

Addresses and fax numbers of Committee members

Dr G. L. Daniels	Bristol Institute for Transfusion Sciences, Southmead Road, Bristol BS10 5ND, UK. geoff.daniels@nbs.nhs.uk
Dr A. Fletcher	Growing your Knowledge, PO Box 716, Spit Junction, NSW 2088, Australia. af@growingyourknowledge.com.au
Prof. G. Garratty	American Red Cross Blood Services, Los Angeles-Orange Counties Region, 1130 South Vermont Avenue, Los Angeles, CA
	90006, USA. garratty@usa.redcross.org
Prof. S. Henry	Biotechnology Research Centre, Auckland University of Technology, Private Bag 92006, Auckland 1020, New Zealand.
	kiwi@aut.ac.nz
Dr J. Jørgensen	Regional Blood Transfusion Center, Department of Clinical Immunology, University Hospital, Skejby, DK-8200 Århus N,
	Denmark. jjo@sks.aaa.dk
Prof. W. J. Judd	Department of Pathology, University Hospitals UH-2G332, 1500 E Medical Center Drive, Ann Arbor, Michigan 48109-0054,
	USA. johnjudd@med.umich.edu
Dr C. Levene	Reference Laboratory for Immunohematology and Blood Groups, National Blood Services Centre, Magen David Adom, Tel
	Hashomer 52621, Israel. cyril@cc.huji.ac.il
Ms C. Lomas-Francis	New York Blood Center, 310 East 67th Street, New York, NY 10021, USA. clomas-francis@nybloodcenter.org
Mr J. J. Moulds	Ortho-Clinical Diagnostics, 1001 US Highway 202, Raritan, NJ 08869-0606, USA. jmoulds@ocdus.jnj.com
Dr J. M. Moulds	Drexel University College of Medicine, 2900 Queen Lane, Philadelphia, PA 19129, USA. moulds@drexel.edu
Ms M. Moulds	Gamma Biologicals Inc (subsidiary of Immunocor Inc), 3700 Mangum Road, Houston, TX 77092, USA. mmoulds@immucor.com
Dr M. A. M. Overbeeke	Sanquin Blood Supply, Diagnostic Services, Department of Immunohematology, Plasmanlaan 125, 1066 CX, Amsterdam, the Netherlands.
	m.overbeeke@sanquin.nl
Dr M. E. Reid	New York Blood Center, 310 East 67th Street, New York, NY 10021, USA. mreid@nybloodcenter.org
Dr Ph Rouger	Centre national de Référence pour les Groupes sanguines, CNTS St Antoine, 53 boulevard Diderot, F-75571 Paris Cedex 13,
	France. tcb_ints@ints.fr
Dr M. Scott	International Blood Group Reference Laboratory, Southmead Road, Bristol BS10 5ND, UK. marion.scott@nbs.nhs.uk
Dr P. Sistonen	Finnish Red Cross Blood Transfusion Service, Kivihaantie 7, SF-00310, Helsinki 31, Finland.
	pertti.sistonen@bts.redcross.fi
Mrs E. Smart	South African National Blood Service, East Coast Region, Private Bag X9044, Pinetown 3600, South Africa.
	smarte@ecr.sansb.org.za
Dr Y. Tani	Osaka Red Cross Blood Center, Morinomiya 2-4-43, Joto-ku, Osaka, 536-8505, Japan. taniy@sannet.ne.jp
Dr S. Wendel	Blood Bank, Hospital Sirio-Libanes, Rua Dona Adma Jafet 91, 29 Andar, São Paulo, Brazil. snwendel@uninet.com.br
Dr T. Zelinski	Rh Laboratory, Room P009, Pathology Building, 770 Bannatyne Avenue, Winnipeg, Manitoba R3E OW3, Canada.
	zelinski@ms.umanitoba.ca