Cromer Blood Group System

Number of antigens 16

Low prevalence	Tc^{b}, Tc^{c}, WES^{a}
High prevalence	Cr^{a}, Tc^{a}, Dr^{a}, Es^{a}, IFC, WES^{b}, UMC, GUTI, SERF, ZENA, CROV, CRAM, CROZ

Terminology

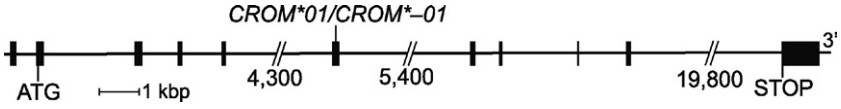
ISBT symbol (number)	CROM (021)
CD Number	CD55
Obsolete name	Collection 202
History	Named after the first antigen in this system, Cr^{a}.

Expression

Soluble form	Low levels in plasma, serum and urine
Other blood cells	Leukocytes; platelets
Tissues	Apical surfaces of trophoblasts in placenta

Gene

Chromosome	1q32.2
Name	*CROM (DAF)*
Organization	11 exons distributed over 40 kbp of gDNA
Product	Decay accelerating factor (DAF; CD55)



Database accession numbers

GenBank	NM_000573, M31516
Entrez Gene ID	1604

The Blood Group Antigen (3/e). DOI: http://dx.doi.org/10.1016/B978-0-12-415849-8.00023-5

Molecular basis of Cromer phenotypes

The reference allele is *CROM*01* or *CROM*A* (Accession number M31516); encodes Cr[a] (CROM1), CROM2, CROM5, CROM6, CROM7 (IFC), CROM9, CROM10, CROM11, CROM12, CROM13, CROM14, CROM15, CROM16. Differences from this reference allele, and the amino acids affected, are given.

Allele encodes	Allele name	Exon	Nucleotide	Restriction enzyme	Amino acid^	Ethnicity (prevalence)
Cr(a–) or CROM:–1	*CROM*–01*	6	679G>C		Ala227Pro	Blacks (Many)
Tc(a–b+) or CROM:–2,3	*CROM* 01.03*	2	155G>T	*Rsa*I–; *Stu*I+	Arg52Leu	Blacks (Rare)
Tc(a–c+) or CROM:–2,4	*CROM* 01.04*	2	155G>C	*Rsa*I–	Arg52Pro	Caucasians (Rare)
Dr(a–) or CROM:–5	*CROM* 01.–05*	5	596C>T†	*Taq*I–	Ser199Leu	Bukhara Jews (Several), Japanese (Rare)
Es(a–) or CROM:–6	*CROM* 01.–06*	2	239T>A	*Sau*3AI–	Ile80Asn	Mexicans, South Americans, Blacks (Rare)
WES(a+b–) or CROM:8	*CROM* 01.08*	2	245T>G	*Afl*1II–	Leu 82Arg	Blacks, Finns (Rare)
UMC– or CROM:–10	*CROM* 01.–10*	6	749C>T		Thr250Met	Japanese (Rare)
GUTI– or CROM:–11	*CROM* 01.–11*	6	719G>A	*Mae*II–	Arg240His	Chileans (Rare)
SERF– or CROM:–12	*CROM* 01.–12*	5	647C>T	*Bst*NI+	Pro216Leu	Thais (Rare)
ZENA– or CROM:–13	*CROM* 01.–13*	6	726T>G	*Bsr*I+	His242Gln	Syrian Turks (Rare)
CROV– or CROM:–14	*CROM* 01.–14*	3	466G>A	*Taq*I–	Glu156Lys	Croatians (Rare)
CRAM– or CROM:–15	*CROM* 01.–15*	6	740A>G		Gln247Arg	Somali (Rare)
CROZ– or CROM:–16	*CROM* 01.–16*	3	389G>A		Arg130His	Australian (Rare)

^Change from historical counting of #1 as Asp of the mature (membrane-bound protein); thus, all amino acid numbers have increased by 34.
†This transition results in two cDNA transcripts, one encoding full length DAF with the single amino acid change. The other, more abundant, transcript uses the novel branch point, which leads to use of a downstream cryptic acceptor splice site, a 44 bp deletion, and a frame-shift in exon 5 (proband KZ)[1].

Molecular bases of silencing of *CROM*

Homozygosity or compound heterozygosity leads to $Cromer_{null}$ (CR:–7; Inab) phenotype.
Differences from *CROM*01* reference allele (Accession number M31516) are given.

Allele name	Exon	Nucleotide	Restriction enzyme	Amino acid^	Ethnicity (prevalence)
*CROM*01N.01*	2	261G>A	*Bc* II+	Trp87Stop	Japanese (Rare)
*CROM*01N.02*	2	263C>A	*Mbo* II–	Ser88Stop	(Rare)
*CROM*01N.03*	4	508C>T		Arg170Stop[2]	Japanese (Rare)
*CROM*01N.04*	3	367insA		Thr123fs; Glu128Stop[3]	Moroccan (Rare)

^Change from historical counting of #1 as Asp of the mature (membrane-bound protein); thus, all amino acid numbers have increased by 34.

Amino acid sequence[4]

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MTVARPSVPA  ALPLLGELPR  LLLLVLLCLP  AVWGDCGLPP  DVPNAQPALE   50
GRTSFPEDTV  ITYKCEESFV  KIPGEKDSVI  CLKGSQWSDI  EEFCNRSCEV  100
PTRLNSASLK  QPYITQNYFP  VGTVVEYECR  PGYRREPSLS  PKLTCLQNLK  150
WSTAVEFCKK  KSCPNPGEIR  NGQIDVPGGI  LFGATISFSC  NTGYKLFGST  200
SSFCLISGSS  VQWSDPLPEC  REIYCPAPPQ  IDNGIIQGER  DHYGYRQSVT  250
YACNKGFTMI  GEHSIYCTVN  NDEGEWSGPP  PECRGKSLTS  KVPPTVQKPT  300
TVNVPTTEVS  PTSQKTTTKT  TTPNAQATRS  TPVSRTTKHF  HETTPNKGSG  350
TTSGTTRLLS  GHTCFTLTGL  LGTLVTMGLL  T                      381
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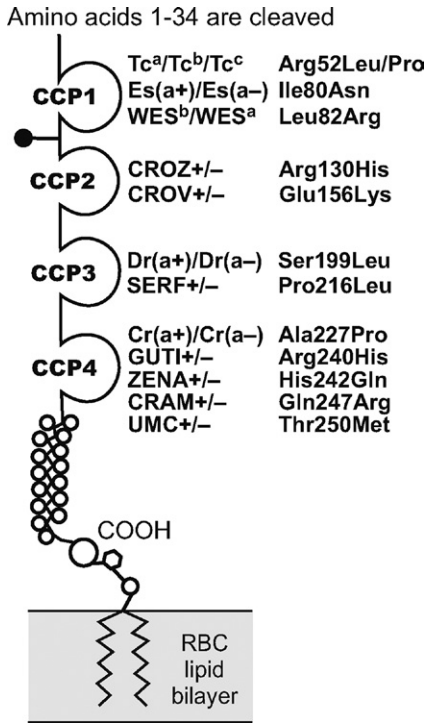
A signal peptide of 34 amino acids is cleaved from the membrane-bound protein.
The 28 carboxyl terminal amino acids are cleaved prior to attachment of DAF to its GPI-linkage.

Carrier molecule

A GPI-linked glycoprotein.



M_r (SDS-PAGE)	Reduced:	64,000–73,000
	Non-reduced:	60,000–70,000
CHO: N-glycan	1 site	
CHO: O-glycan	15 sites (32 potential)	
Cysteine residues	14	
Copies per RBC	20,000	

Function

Complement regulation: DAF inhibits assembly and accelerates decay of C3 and C5 convertases.

Disease association

Five of six known individuals with the Inab phenotype have intestinal disorders. PNH III RBCs are deficient in DAF.

Phenotypes

Null	Inab (IFC–)
Unusual	Dr(a–) RBCs weakly express inherited Cromer antigens

Comments

Antibodies in the Cromer blood group system do not cause HDFN. DAF is strongly expressed on the apical surface of placental trophoplasts[5], and will absorb antibodies in the Cromer system.
Antibodies to Cromer antigens identified early in pregnancy are often below detectable levels in late stages of pregnancy, but reappear some weeks after the birth of the baby.

References

[1] Lublin, D.M., et al., 1994. Molecular basis of reduced or absent expression of decay-accelerating factor in Cromer blood group phenotypes. Blood 84, 1276–1282.

[2] Hue-Roye, K., et al., 2005. Novel molecular basis of an Inab phenotype. Immunohematology 21, 53–55.

[3] Karamatic Crew, V., et al., 2010. Two unusual cases within the Cromer blood group system: (I) A novel high incidence antigen CROZ; and (II) A novel molecular basis of Inab phenotype. Transfus Med 20 (Suppl. 1): 12.

[4] Lublin, D.M., Atkinson, J.P., 1989. Decay-accelerating factor: biochemistry, molecular biology and function. Ann Rev Immunol 7, 35–58.

[5] Holmes, C.H., et al., 1990. Preferential expression of the complement regulatory protein decay accelerating factor at the fetomaternal interface during human pregnancy. J Immunol 144, 3099–3105.

Cr^a Antigen

Terminology

ISBT symbol (number)	CROM1 (021001 or 21.1)
Obsolete names	Go^b; 202001; 900013
History	Named in 1975 after Mrs. Cromer, a black antenatal patient who made the antibody. Cr^a was originally thought to be antithetical to Go^a.

Occurrence

Most populations	100%
Blacks	Greater than 99%
Hispanics	One Cr(a–) found

Expression

Cord RBCs	Expressed
Altered	Weak on Dr(a–) and negative on PNH III RBCs

Molecular basis associated with Cr^a antigen[1]

Amino acid	Ala227 (previously reported as 193) in CCP4
Nucleotide	G at bp 679 in exon 6
Cr(a–)	Pro227 and C at bp 679

Effect of enzymes and chemicals on Cr^a antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Resistant
α-Chymotrypsin	Sensitive
Pronase	Sensitive
DTT 200 mM/50 mM	Weakened/resistant (thus weakened by WARM™ and ZZAP)
Acid	Resistant

In vitro characteristics of alloanti-Cr^a

Immunoglobulin class	IgG
Optimal technique	IAT
Neutralization	With concentrated plasma/serum/urine

Clinical significance of alloanti-Cr^a

Transfusion reaction	No to moderate
HDFN	No, because DAF on apical surface of trophoblasts in placenta absorbs maternal antibody

Comments

Siblings of patients with anti-Cr^a should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.

Reference

[1] Lublin, D.M., et al., 2000. Molecular basis of Cromer blood group antigens. Transfusion 40, 208–213.

Tc^a Antigen

Terminology

ISBT symbol (number)	CROM2 (021002 or 21.2)
Obsolete names	202002; 900020
History	Named in 1980, and placed in the Cromer system when the antibody was shown to be non-reactive with Inab RBCs. The initials of the first two probands to have the antibody were GT and DLC, hence Tc^a.

Occurrence

Most populations	100%
Blacks	>99%

Antithetical antigen

Tc^b (**CROM3**); Tc^c (**CROM4**)

Expression

Cord RBCs	Expressed
Altered	Weak on Dr(a–) and negative on PNH III RBCs

Molecular basis associated with Tc^a antigen[1]

Amino acid	Arg52 (previously reported as 18) in CCP1
Nucleotide	G at bp 155 in exon 2

Effect of enzymes and chemicals on Tc^a antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Resistant
α-Chymotrypsin	Sensitive
Pronase	Sensitive
DTT 200 mM/50 mM	Weakened/resistant (thus weakened by WARM™ and ZZAP)
Acid	Resistant

In vitro characteristics of alloanti-Tc^a

Immunoglobulin class	IgG
Optimal technique	IAT
Neutralization	With concentrated serum/plasma/urine

Clinical significance of alloanti-Tc^a

Transfusion reaction	No to severe[2]
HDFN	No, because DAF on apical surface of trophoblasts in placenta absorbs maternal antibody

Comments

Siblings of patients with anti-Tc^a should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.

Only three examples of anti-Tc^a have been reported but a few others have been found. All Tc(a–) Blacks are Tc(b+); Tc(a–) Caucasians are Tc(c+).

References

[1] Lublin, D.M., et al., 2000. Molecular basis of Cromer blood group antigens. Transfusion 40, 208–213.

[2] Kowalski, M.A., et al., 1999. Hemolytic transfusion reaction due to anti-Tc(a). Transfusion 39, 948–950.

Tc^b Antigen

Terminology

ISBT symbol (number)	CROM3 (021003 or 21.3)
Obsolete names	202003; 700035
History	Original antibody found in a serum containing anti-Go^a; named in 1985 when it was recognized to be antithetical to Tc^a.

Occurrence

Caucasians	None found
Blacks	6%

Antithetical antigen

Tc^a (**CROM2**); Tc^c (**CROM4**)

Expression

Cord RBCs	Expressed

Molecular basis associated with Tc^b antigen[1]

Amino acid	Leu52 (previously reported as 18) in CCP1
Nucleotide	T at bp 155 in exon 2

Effect of enzymes and chemicals on Tc^b antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Resistant
α-Chymotrypsin	Sensitive
Pronase	Sensitive
DTT 200 mM/50 mM	Weakened/resistant (thus weakened by WARM™ and ZZAP)
Acid	Resistant

In vitro characteristics of alloanti-Tc^b

Immunoglobulin class	IgG
Optimal technique	IAT

Clinical significance of alloanti-Tc^b

No data because antigen and antibody are rare.

Reference

[1] Lublin, D.M., et al., 2000. Molecular basis of Cromer blood group antigens. Transfusion 40, 208–213.

Tc^c Antigen

Terminology

ISBT symbol (number)	CROM4 (021004 or 21.4)
Obsolete names	202004; 700036
History	Described in 1982, and named when it was recognized to be antithetical to Tc^a.

Occurrence

Less than 0.01%; two Tc(a–b–c+) have only been found in two Caucasian families.

Antithetical antigen

Tc^a (**CROM2**); Tc^b (**CROM3**)

Expression

Cord RBCs	Expressed

Molecular basis associated with Tc^c antigen[1]

Amino acid	Pro52 (previously reported as 18) in CCP1
Nucleotide	C at bp 155 in exon 2

Effect of enzymes and chemicals on Tc^c antigen on intact RBCs

Ficin/Papain	Presumed resistant
Trypsin	Presumed resistant
α-Chymotrypsin	Presumed sensitive
DTT 200 mM/50 mM	Presumed weakened/resistant (thus weakened by WARM™ and ZZAP)

In vitro characteristics of alloanti-Tc^c

Immunoglobulin class	IgG
Optimal technique	IAT

Clinical significance of alloanti-Tc^c

Transfusion reaction	No to mild
HDFN	No, because DAF on apical surface of trophoblasts in placenta absorbs maternal antibody

Comments

A female with the rare Tc(a–b–c+) phenotype made an antibody that appears to be an inseparable anti-Tc^{a}Tc^b.

Reference

[1] Lublin, D.M., et al., 2000. Molecular basis of Cromer blood group antigens. Transfusion 40, 208–213.

Dr^a Antigen

Terminology

ISBT symbol (number)	CROM5 (021005 or 21.5)
Obsolete names	202005; 900021
History	Reported in 1984, and named after the Israeli Dr(a–) proband, Mrs. Drori.

Occurrence

Dr(a–) phenotype has been reported only in Jews from Bukhara and in Japanese.

Expression

Cord RBCs	Expressed
Altered	Absent from PNH III RBCs

Molecular basis associated with Dr^a antigen[1]

Amino acid	Ser199 (previously reported as 165) in CCP3
Nucleotide	C at bp 596 in exon 5
Dr(a–)	Leu199; the T at bp 596 introduces a branch point that leads to use of a downstream cryptic acceptor splice site, deletion of 44 bp, and a frame-shift

Effect of enzymes and chemicals on Dr^a antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Resistant
α-Chymotrypsin	Sensitive
Pronase	Sensitive
DTT 200 mM/50 mM	Weakened/resistant (thus weakened by WARM™ and ZZAP)

In vitro characteristics of alloanti-Dr^a

Immunoglobulin class	IgG
Optimal technique	IAT
Neutralization	With concentrated serum/plasma/urine

Clinical significance of alloanti-Dr^a

Transfusion reaction	No to mild
HDFN	No, because DAF on apical surface of trophoblasts in placenta absorbs maternal antibody

Comments

All inherited Cromer antigens are expressed weakly on Dr(a–) RBCs, due to a markedly reduced copy number of DAF[1].
Siblings of patients with anti-Dr^a should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.
Dr^a is the receptor for uropathogenic *E. coli*[2].

References

[1] Lublin, D.M., et al., 1994. Molecular basis of reduced or absent expression of decay-accelerating factor in Cromer blood group phenotypes. Blood 84, 1276–1282.
[2] Hasan, R.J., et al., 2002. Structure-function analysis of decay-accelerating factor: identification of residues important for binding of the *Escherichia coli* Dr adhesin and complement regulation. Infect Immun 70, 4485–4493.

Es^a Antigen

Terminology

ISBT symbol (number)	CROM6 (021006 or 21.6)
Obsolete names	202006; 900022
History	Named in 1984 after Mrs. Escandon, whose Mexican parents were cousins.

Occurrence

Three Es(a–) probands are known: one Mexican; one South American; and one Black[1].

Expression

Cord RBCs	Expressed
Altered	Weak on Dr(a–), WES(a+b–), and negative on PNH III RBCs

Molecular basis associated with Es^a antigen[2]

Amino acid	Ile80 (previously reported as 46) in CCP1
Nucleotide	T at bp 239 in exon 2
Es(a–)	Asn80 and A at bp 239

Effect of enzymes and chemicals on Es^a antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Resistant
α-Chymotrypsin	Sensitive
Pronase	Sensitive
DTT 200 mM/50 mM	Weakened/resistant (thus weakened by WARM™ and ZZAP)

In vitro characteristics of alloanti-Es^a

Immunoglobulin class	IgG
Optimal technique	IAT

Clinical significance of alloanti-Es^a

Transfusion reaction	One report of a mild transfusion reaction
HDFN	No, because DAF on apical surface of trophoblasts in placenta absorbs maternal antibody

Comments

Siblings of patients with anti-Es^a should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.
Es(a–) RBCs have a weak expression of WES^b.

References

[1] Reid, M.E., et al., 1996. A second example of anti-Es^a, an antibody to a high-incidence Cromer antigen. Immunohematology 12, 112–114.
[2] Lublin, D.M., et al., 2000. Molecular basis of Cromer blood group antigens. Transfusion 40, 208–213.

IFC Antigen

Terminology

ISBT symbol (number)	CROM7 (021007 or 21.7)
Obsolete name	202007
History	Anti-IFC is made by people with the Inab phenotype. Named in 1986 from the names of the first three IFC– probands.

Occurrence

Rare IFC– (Inab phenotype) people have been mostly from Japan, but Caucasians (one was Swedish; a brother and sister were Italian American), an African American, and a Moroccan have been reported.

Expression

Cord RBCs	Expressed
Altered	Weak on Dr(a–) and absent from PNH III RBCs

Molecular bases associated with IFC antigen

For molecular bases associated with an absence of IFC refer to System pages.

Effect of enzymes and chemicals on IFC antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Resistant
α-Chymotrypsin	Sensitive
Pronase	Sensitive
DTT 200 mM/50 mM	Weakened/resistant (thus weakened by WARM™ and ZZAP)

In vitro characteristics of alloanti-IFC

Immunoglobulin class	IgG
Optimal technique	IAT
Neutralization	With concentrated serum/plasma/urine

Clinical significance of alloanti-IFC

Transfusion reaction	No to mild
HDFN	No, because DAF on apical surface of trophoblasts in placenta absorbs maternal antibody

Comments

The only phenotype that lacks IFC is the Inab phenotype, because the RBCs do not express DAF[1].

Siblings of patients with anti-IFC should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.

A few patients with an acquired (transient) form of the Inab phenotype who made anti-IFC have been reported including: one with thalassemia who had splenic infarctions[2]; another with chronic CLL[3]; and a young child with multiple medical problems[4].

References

[1] Lublin, D.M., et al., 1994. Molecular basis of reduced or absent expression of decay-accelerating factor in Cromer blood group phenotypes. Blood 84, 1276–1282.

[2] Matthes, T., et al., 2002. Acquired and transient RBC CD55 deficiency (Inab phenotype) and anti-IFC. Transfusion 42, 1448–1457.

[3] Banks, J., et al., 2004. Transient loss of Cromer antigens and anti-IFC in a patient with chronic lymphatic leukaemia [abstract]. Vox Sang 87 (Suppl. 3), 37.

[4] Yazer, M.H., et al., 2006. Case report and literature review: transient Inab phenotype and an agglutinating anti-IFC in a patient with a gastrointestinal problem. Transfusion 46, 1537–1542.

WES^{a} Antigen

Terminology

ISBT symbol (number)	CROM8 (021008 or 21.8)
Obsolete names	202008; 700042
History	Named in 1987 after the first antibody producer.

Occurrence

Most populations	<0.01%
Blacks (America)	0.48%
Blacks (N. London)	2.04%
Finns	0.56%

Antithetical antigen

WES^{b} (**CROM9**)

Expression

Cord RBCs	Expressed

Molecular basis associated with WES^{a} antigen[1]

Amino acid	Arg82 (previously reported as 48) in CCP1
Nucleotide	G at bp 245 in exon 2

Effect of enzymes and chemicals on WES^{a} antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Resistant
α-Chymotrypsin	Sensitive
Pronase	Sensitive
DTT 200 mM/50 mM	Weak/resistant (thus weakened by WARM™ and ZZAP)

In vitro characteristics of alloanti-WES[a]

Immunoglobulin class	IgG
Optimal technique	IAT
Neutralization	With concentrated serum/plasma/urine

Clinical significance of alloanti-WES[a]

Transfusion reaction	No to mild
HDFN	No, because DAF on apical surface of trophoblasts in placenta absorbs maternal antibody

Comments

WES(a+b–) RBCs have a weak expression of Es[a].

Reference

[1] Lublin, D.M., et al., 2000. Molecular basis of Cromer blood group antigens. Transfusion 40, 208–213.

WES[b] Antigen

Terminology

ISBT symbol (number)	CROM9 (021009 or 21.9)
Obsolete names	202004; 900033
History	Named in 1987 when it was recognized to be antithetical to WES[a].

Occurrence

WES(a+b–) probands have been found in people of African ancestry and in Finns.

Antithetical antigen

WES[b] (**CROM8**)

Expression

Cord RBCs	Expressed
Altered	Weak on Dr(a–) and Es(a–), and negative on PNH III RBCs

Molecular basis associated with WES[b] antigen[1]

Amino acid	Leu80 (previously reported as 48) in CCP1
Nucleotide	T at bp 245 in exon 2

Effect of enzymes and chemicals on WES^b antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Resistant
α-Chymotrypsin	Sensitive
Pronase	Sensitive
DTT 200 mM/50 mM	Weakened/resistant (thus weakened by WARM™ and ZZAP)

In vitro characteristics of alloanti-WES^b

Immunoglobulin class	IgG
Optimal technique	IAT
Neutralization	Concentrated serum/plasma/urine

Clinical significance of alloanti-WES^b

Transfusion reaction	No data
HDFN	Few examples of anti-WES^b, produced as a result of pregnancy, are described. The baby's RBCs had a positive DAT, but there were no clinical signs of HDFN. HDFN is unlikely because DAF on apical surface of trophoblasts in placenta absorbs maternal antibody.

Comments

WES(a+b–) RBCs have a weak expression of Es^a antigen.
Siblings of patients with anti-WES^b should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.

Reference

[1] Lublin, D.M., et al., 2000. Molecular basis of Cromer blood group antigens. Transfusion 40, 208–213.

UMC Antigen

Terminology

ISBT symbol (number)	CROM10 (021010 or 21.10)
Obsolete name	202010
History	Named in 1989, from the name of the first producer of the antibody.

Occurrence

Only one UMC– proband and her UMC– brother (Japanese) have been described.

Expression

Cord RBCs	Expressed
Altered	Weak on Dr(a–) and absent from PNH III RBCs

Molecular basis associated with UMC antigen[1]

Amino acid	Thr250 (previously reported as 216) in CCP4
Nucleotide	C at bp 749 in exon 6
UMC–	Met250 and T at bp 479

Effect of enzymes and chemicals on UMC antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Resistant
α-Chymotrypsin	Sensitive
Pronase	Sensitive
DTT 200 mM/50 mM	Weakened/resistant (thus weakened by WARM™ and ZZAP)

In vitro characteristics of alloanti-UMC

Immunoglobulin class	IgG
Optimal technique	IAT
Neutralization	With concentrated serum/plasma/urine

Clinical significance of alloanti-UMC

Transfusion reaction	No data
HDFN	The proband had three children with no signs or symptoms of HDFN. HDFN is unlikely, because DAF on apical surface of trophoblasts in placenta absorbs maternal antibody.

Comments

Siblings of patients with anti-UMC should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.

Reference

[1] Lublin, D.M., et al., 2000. Molecular basis of Cromer blood group antigens. Transfusion 40, 208–213.

GUTI Antigen

Terminology

ISBT symbol (number)	CROM11 (021011 or 21.11)
History	Named in 2002 after the first producer of the antibody. The immunogen was a transfusion following a motorcycle accident.

Occurrence

Only one GUTI– proband (Chilean) and his sister have been reported. 15% of Mapuche Indians are heterozygotes for *CROM*01.–11*.

Expression

Cord RBCs	Expressed
Altered	Weak on Dr(a–) and negative on PNH III RBCs

Molecular basis associated with GUTI antigen[1]

Amino acid	Arg240 (previously reported as 206) in CCP4
Nucleotide	G at bp 719 in exon 6
GUTI–	His240 and A at bp 719

Effect of enzymes and chemicals on GUTI antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Resistant
α-Chymotrypsin	Sensitive
Pronase	Sensitive
DTT 200 mM/50 mM	Weakened/resistant (thus weakened by WARM™ and ZZAP)
Acid	Resistant

In vitro characteristics of alloanti-GUTI

Immunoglobulin class	IgG
Optimal technique	IAT

Clinical significance of alloanti-GUTI

No data because anti-GUTI has only been found in one male.
HDFN is unlikely because DAF on the apical surface of trophoblasts in placenta absorbs maternal antibody.

Comments

Siblings of patients with anti-GUTI should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.

Reference

[1] Storry, J.R., et al., 2003. GUTI: a new antigen in the Cromer blood group system. Transfusion 43, 340–344.

SERF Antigen

Terminology

ISBT symbol (number)	CROM12 (021012 or 21.12)
History	Named in 2004 after the first producer of the antibody.

Occurrence

The only two SERF– probands reported (and the sister of one) were Thai[1].

Expression

Cord RBCs	Expressed
Altered	Weak on Dr(a–) and negative on PNH III RBCs

Molecular basis associated with SERF antigen[2]

Amino acid	Pro216 (previously reported as 182) in CCP3
Nucleotide	C at bp 647 in exon 5
SERF–	Leu216 and T at bp 647

Effect of enzymes and chemicals on SERF antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Resistant
α-Chymotrypsin	Sensitive
DTT 200 mM/50 mM	Weakened/resistant (thus weakened by WARM™ and ZZAP)

In vitro characteristics of alloanti-SERF

Immunoglobulin class	IgG
Optimal technique	IAT

Clinical significance of alloanti-SERF

No data because only one anti-SERF has been described.
HDFN is unlikely because DAF on apical surface of trophoblasts in placenta absorbs maternal antibody.

Comments

Siblings of patients with anti-SERF should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.

References

[1] Palacajornsuk, P., et al., 2005. Analysis of SERF in Thai blood donors. Immunohematology 21, 66–69.

[2] Banks, J., et al., 2004. SERF: a new antigen in the Cromer blood group system. Transfus Med 14, 313–318.

ZENA Antigen

Terminology

ISBT symbol (number)	CROM13 (021013 or 21.13)
History	Named in 2004; derived from the given name of the ZENA– proband.

Occurrence

The only reported ZENA– proband was a Syrian Turk.

Expression

Cord RBCs	Expressed
Altered	Weak on Dr(a–) and absent from PNH III RBCs

Molecular basis associated with ZENA antigen[1]

Amino acid	His242 (previously reported as 208) in CCP4
Nucleotide	T at bp 726 in exon 6
ZENA–	Gln242 and G at bp 726

Effect of enzymes and chemicals on ZENA antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Resistant
α-Chymotrypsin	Sensitive
DTT 200 mM/50 mM	Weakened/resistant (thus weakened by WARM™ and ZZAP)

In vitro characteristics of alloanti-ZENA

Immunoglobulin class	IgG
Optimal technique	IAT

Clinical significance of alloanti-ZENA

No data because only one example of anti-ZENA has been described. The proband's baby was born with a normal hemoglobin level and no clinical evidence of HDFN. The baby's RBCs were negative in the direct antiglobulin test.

Comments

Siblings of patients with anti-ZENA should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.

Reference

[1] Hue-Roye, K., et al., 2007. Three new high-prevalence antigens in the Cromer blood group system. Transfusion 47, 1621–1629.

CROV Antigen

Terminology

ISBT symbol (number)	CROM14 (021014 or 21.14)
History	Named in 2005 from "CRO" from Croatia (and the Cromer blood group system), and the first initial of the town (Vinkovci) from whence the CROV– proband hailed.

Occurrence

The only reported CROV– proband was from Croatia.

Expression

Cord RBCs	Expressed
Altered	Weak on Dr(a–) and absent from PNH III RBCs

Molecular basis associated with CROV antigen[1]

Amino acid	Glu156 (previously reported as 122) in CCP2
Nucleotide	G at bp 466 in exon 3
CROV–	Lys156 and A at bp 466

Effect of enzymes and chemicals on CROV antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Resistant
α-Chymotrypsin	Sensitive
DTT 200 mM/50 mM	Weakened/resistant (thus weakened by WARM™ and ZZAP)

In vitro characteristics of alloanti-CROV

Immunoglobulin class	IgG
Optimal technique	IAT

Clinical significance of alloanti-CROV

No data because only one example of anti-CROV has been described.
HDFN is unlikely because DAF on apical surface of trophoblasts in placenta absorbs maternal antibody.

Comments

Siblings of patients with anti-CROV should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.

Reference

[1] Hue-Roye, K., et al., 2007. Three new high-prevalence antigens in the Cromer blood group system. Transfusion 47, 1621–1629.

CRAM Antigen

Terminology

ISBT symbol (number)	CROM15 (021015 or 21.15)
History	Named in 2006 from "CR" for the system and "AM" from the CRAM– proband's name.

Occurrence

The only reported CRAM– proband was from Somalia.

Expression

Cord RBCs	Expressed
Altered	Weak on Dr(a–) and absent from PNH III RBCs

Molecular basis associated with CRAM antigen[1]

Amino acid	Gln247 (previously reported as 213) in CCP4
Nucleotide	A at bp 740 in exon 6
CRAM–	Arg247 and G at bp 740

Effect of enzymes and chemicals on CRAM antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Resistant
α-Chymotrypsin	Sensitive
DTT 200 mM/50 mM	Weakened/resistant (thus weakened by WARM™ and ZZAP)

In vitro characteristics of alloanti-CRAM

Immunoglobulin class	IgG
Optimal technique	IAT

Clinical significance of alloanti-CRAM

No data because only one anti-CRAM has been described.
HDFN is unlikely because DAF on apical surface of trophoblasts in placenta absorbs maternal antibody.

Comments

Siblings of patients with anti-CRAM should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.

Reference

[1] Hue-Roye, K., et al., 2007. Three new high-prevalence antigens in the Cromer blood group system. Transfusion 47, 1621–1629.

CROZ Antigen

Terminology

ISBT symbol (number)	CROM16 (021016 or 21.16)
History	Named in 2010 from "CR" for the system, and "OZ" for "Australia," from whence the proband hailed.

Occurrence

The only reported CROZ– proband was from Australia and may be of Italian descent.

Expression

Cord RBCs	Expressed
Altered	Weak on Dr(a–) and absent from PNH III RBCs

Molecular basis associated with CROZ antigen[1]

Amino acid	Arg130 (previously reported as 96) in CCP2
Nucleotide	G at bp 389 in exon 3
CROZ–	His130 and A at bp 389

Effect of enzymes and chemicals on CROZ antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Resistant
α-Chymotrypsin	Sensitive
DTT 200 mM/50 mM	Weakened/resistant (thus weakened by WARM™ and ZZAP)

In vitro characteristics of alloanti-CROZ

Immunoglobulin class	IgG
Optimal technique	IAT

Clinical significance of alloanti-CROZ

No data because only one example of anti-CROZ has been reported.
HDFN is unlikely because DAF on apical surface of trophoblasts in placenta absorbs maternal antibody.

Comments

Siblings of patients with anti-CROZ should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.

Reference

[1] Karamatic Crew, V., et al., 2010. Two unusual cases within the Cromer blood group system: (I) A novel high incidence antigen CROZ; and (II) A novel molecular basis of Inab phenotype [abstract]. Transfus Med 20 (Suppl. 1), 12.