Gerbich Blood Group System

Number of antigens 11

Low prevalence	Wb, Ls[a], An[a], Dh[a], GEIS
High prevalence	Ge2, Ge3, Ge4, GEPL, GEAT, GETI

Terminology

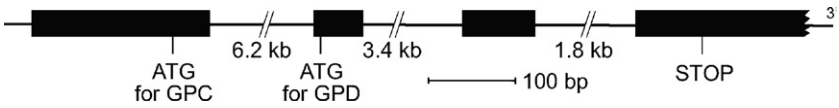
ISBT symbol (number)	GE (020)
CD number	CD236
Obsolete name	ISBT Collection 201
History	Named in 1960 after one of three mothers who were found at the same time, and whose serum contained the antibody defining Gerbich; became a System in 1990.

Expression

Other blood cells	Erythroblasts
Tissues	Fetal liver, renal endothelium

Gene

Chromosome	2q14.3
Name	*GE* (*GYPC*)
Organization	4 exons distributed over 52.7 kbp of gDNA
Product	Glycophorin C (GPC) and glycophorin D (GPD)



Database accession numbers

GenBank	M36284 (mRNA)
Entrez Gene ID	2995

The Blood Group Antigen (3/e). DOI: http://dx.doi.org/10.1016/B978-0-12-415849-8.00022-3

Molecular basis of Gerbich phenotypes

The reference allele, *GE*01* (Accession number M36284) encodes Ge2 (GE2), GE3, GE4, GE10, GE11, GE12. Nucleotide differences from this reference allele, and the amino acids affected, are given.

Allele encodes	Allele name	Exon	Nucleotide	Amino acid change	Ethnicity (prevalence)
Yus type or GE:–2,3,4	*GE*01.–02*	del Exon 2		in frame deletion→ altered GPC	Mexicans (Few) Others (Rare)
Gerbich type or GE:–2,–3,4	*GE*01.–03*	del Exon 3		in frame deletion→ altered GPC	Melanesians (up to 50%), Others (Rare)
Wb+ or GE:5	*GE*01.05*	1	23A>G	Asn8Ser in GPC	Welsh & Australians (Few), Others (Rare)
Ls(a+) or GE:6	*GE*01.06.01*	Duplicated Exon 3		in frame duplication→ altered GPC	Blacks (2%), Finns (1.6%), Others (Rare)
Ls(a+) or GE:6	*GE*01.06.02*	Triplicated Exon 3		in frame triplication→ altered GPC	Japanese (Rare)
An(a+) or GE:7	*GE*01.07*	2	67G>T	Ala23Ser in GPC Ala2Ser in GPD^	Finns (0.2%), Others (Rare)
Dh(a+) or GE:8	*GE*01.08*	1	40C>T	Leu14Phe in GPC	Scandinavians (Rare)
GEIS+ or GE:9	*GE*01.09*	2	95C>A	Thr32Asn in GPC; Thr11Asn in GPD	Japanese (Rare)
GEPL– or GE:–10	*GE*01.–10*	3	134C>T	Pro45Leu in GPC; Pro24Leu in GPD[1]	(Rare)
GEAT– or GE:–11	*GE*01.–11*	2	56A>T	Asp19Val in GPC[1]	(Rare)
GETI– or GE:–12	*GE*01.–12*	2	80C>T	Thr27Ile in GPC; Thr6Ile in GPD[1]	(Rare)
GE:2,3,4^^	*GE*01.–13*	3	173A>T	Asp58Val in GPC; Asp37Val in GPD[2]	(Rare)

^An^a is only expressed by GPD.
^^A woman with this allele *in trans* to an *GE*01.–03* made anti-Ge2[2].

Molecular bases of silencing of *GE*

Homozygosity and compound heterozygosity leads to the Gerbich$_{null}$ (GE:–2, –3,–4) phenotype.
Nucleotide differences from *GE*01* reference allele (Accession number M36284), and amino acids affected, are given.

Name	Allele Name	Exon	Nucleotide	Restriction Enzyme	Amino Acid	Ethnicity (Prevalence)
Leach type (PL)	*GE*01N.01*	del exons 3 & 4			Truncated protein	English (Rare)
Leach type (LN)	*GE*01N.02*	3	131G>T; 134delC	*Msp*I–	Trp44Leu; 45fs; 55Stop	North American (Rare)

Amino acid sequence[3]

Glycophorin C:

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MWSTRSPNST  AWPLSLEPDP  GMASASTTMH  TTTIAEPDPG  MSGWPDGRME   50
TSTPTIMDIV  VIAGVIAAVA  IVLVSLLFVM  LRYMYRHKGT  YHTNEAKGTE  100
FAESADAALQ  GDPALQDAGD  SSRKEYFI                           128
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Glycophorin D:

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                        MASASTTMH  TTTIAEPDPG  MSGWPDGRME    29
TSTPTIMDIV  VIAGVIAAVA  IVLVSLLFVM  LRYMYRHKGT  YHTNEAKGTE    79
FAESADAALQ  GDPALQDAGD  SSRKEYFI                           107
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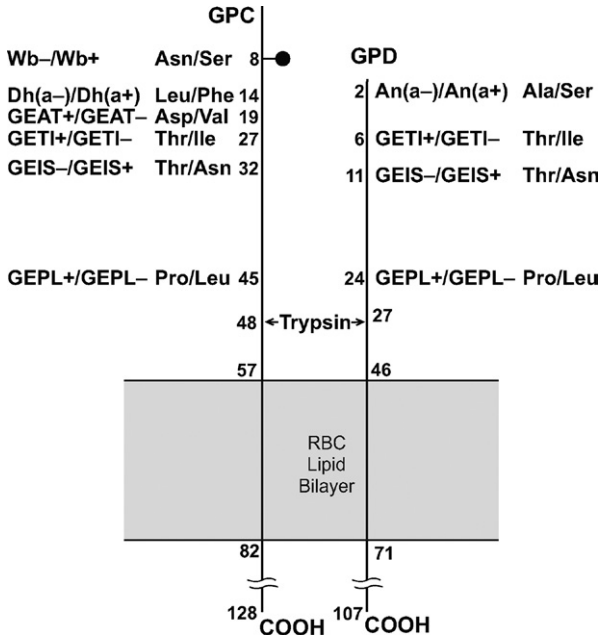
The alignment of amino acids for GPD is intended to denote that the amino acid sequence for GPD is, from the second methionine residue, identical to GPC.

Carrier molecule

Single pass (type 1) membrane protein.



	GPC	GPD
M_r (SDS-PAGE)	40,000	30,000
CHO: N-glycan	1 site	None
CHO: O-glycan	13 sites	8 sites
Copies per RBC	135,000	50,000

Function

Maintenance of RBC membrane integrity via interaction with protein 4.1 and p55. Contributes to the negatively charged glycocalyx.

Disease association

GPC and GPD are markedly reduced in protein 4.1-deficient RBCs, and as such can be associated with hereditary elliptocytosis. RBC receptors for influenza A and influenza B and *Plasmodium falciparum*.

Phenotypes (% occurrence)

	Most populations	Melanesians
GE:2,3,4 (Ge+)	>99.9	50–90
Gerbich-negative		
GE:–2,3,4 (Yus type)	Rare	Not found
GE:–2,–3,4 (Gerbich type)	Rare	10–50
GE:–2,–3,–4 (Leach type)	Rare	Not found
Null:	Leach (PL and LN types) (GE:–2,–3,–4)	

Differentiation of Gerbich-negative phenotypes using monoclonal anti-Ge4

RBCs	Normal	Yus	Gerbich	Leach
Untreated	4+	0–2+	0–2+	0
Trypsin-treated	0	0	4+	0

Comments

The majority of RBC samples with Leach or Gerbich phenotypes have a weak expression of Kell blood group system antigens. Some anti-Vel fail to react with Ge:–2,–3,4 RBCs.

Gerbich antigens are weak on protein 4.1-deficient RBCs, due to reduced levels of GPC and GPD in these membranes.

References

[1] Poole, J., et al., 2008. Novel mutations in GYPC giving rise to lack of Ge epitopes and anti-Ge production [abstract]. Vox Sang 95 (Suppl. 1), 181.

[2] King, M-J, et al., 1997. Co-presence of a point mutation and a deletion of exon 3 in the glycophorin C gene and concomitant production of a Gerbich-related antibody. Transfusion 37, 1027–1034.

[3] Colin, Y., et al., 1986. Isolation of cDNA clones and complete amino acid sequence of human erythrocyte glycophorin C. J Biol Chem 261, 229–233.

Ge2 Antigen

Terminology

ISBT symbol (number)	GE2 (020002 or 20.2)
Obsolete name	Ge; 201002
History	Antigen lacking from all Gerbich-negative phenotypes. Originally defined by the "Yus-type" antibody found in 1961; later referred to as anti-Ge1,2, and now as anti-Ge2.

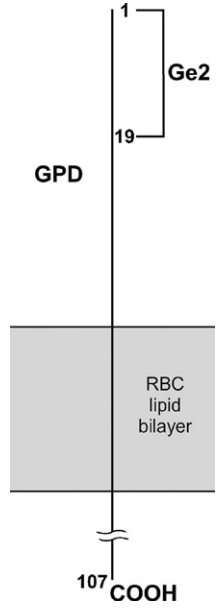
Occurrence

All populations	Greater than 99.9%

Expression

Cord RBCs	Expressed
Altered	Weak on protein 4.1-deficient RBCs Absent from Yus, Gerbich and Leach phenotype RBCs

Molecular basis associated with Ge2 antigen[1]



Ge2 as determined with alloanti-Ge2 is not expressed on GPC.

Effect of enzymes and chemicals on Ge2 antigen on intact RBCs

Ficin/Papain	Sensitive
Trypsin	Sensitive
α-Chymotrypsin	Weakened
Sialidase	Variable
DTT 200 mM	Variable (thus variable to WARM™ and ZZAP)
Acid	Resistant

In vitro characteristics of alloanti-Ge2

Immunoglobulin class	Usually IgG
Optimal technique	IAT
Complement binding	Yes; some hemolytic

Clinical significance of alloanti-Ge2

Transfusion reaction	No to moderate/immediate/delayed
HDFN	Positive DAT, but no clinical HDFN

Autoanti-Ge2

Yes; detects a determinant on GPC.

Comments

Alloanti-Ge2 can be made by individuals with Yus, Gerbich or Leach phenotypes, detects an antigen on GPD, and may be naturally-occurring.
The reciprocal gene to *GYPC.Yus* encodes two copies of amino acids encoded by exon 2.
Siblings of patients with anti-Ge2 should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.

Reference

[1] Walker, P.S., Reid, M.E., 2010. The Gerbich blood group system: a review. Immunohematology 26, 60–65.

Ge3 Antigen

Terminology

ISBT symbol (number)	GE3 (020003 or 20.3)
Obsolete name	Ge; 201003
History	Antigen originally defined by the "Ge-type" serum (identified in 1960). The defining antibody was termed anti-Ge1,2,3, and later renamed to anti-Ge3.

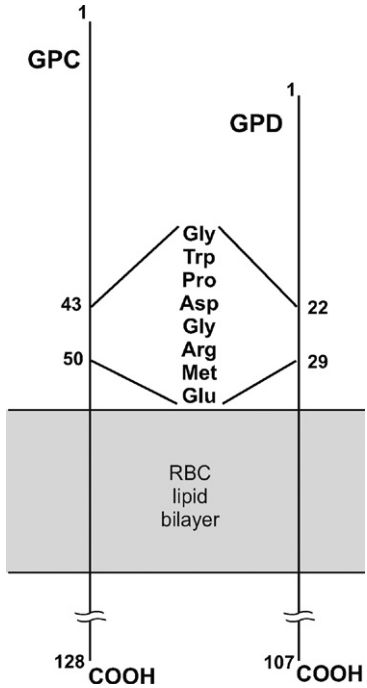
Occurrence

Most populations	>99.9%
Melanesians	50%

Expression

Cord RBCs	Expressed
Altered	Weak on protein 4.1-deficient RBCs
	Absent from Gerbich and Leach phenotype RBCs

Molecular basis associated with Ge3 antigen[1]



The Ge3 antigen amino acid sequence is encoded by exon 3 of *GYPC*.

Effect of enzymes and chemicals on Ge3 antigen on intact RBCs

Ficin/Papain	Resistant
Trypsin	Variable
α-Chymotrypsin	Resistant
Pronase	Sensitive
Sialidase	Resistant
DTT 200 mM	Resistant
Acid	Resistant

In vitro characteristics of alloanti-Ge3

Immunoglobulin class	IgG more common than IgM
Optimal technique	IAT
Complement binding	Yes; some hemolytic

Clinical significance of alloanti-Ge3

Transfusion reaction	No to moderate, immediate or delayed
HDFN	Positive DAT to severe[2] (see Comments)

Autoanti-Ge3

Yes; can be clinically insignificant or cause severe *in vivo* hemolysis.

Comments

Similar to the mechanism of erythroid suppression described in HDFN caused by anti-K, anti-Ge3 has been associated with antibody-dependent hemolysis, as well as suppression of erythroid progenitor cell growth in the infant. In these cases, the affected infants may require initial treatment at delivery, followed by monitoring for signs of anemia for several weeks after birth.
Alloanti-Ge3 and autoanti-Ge3 detect the antigen on both GPC and GPD[3,4].
Alloanti-Ge3 can be made by individuals with either Gerbich or Leach phenotypes.
Siblings of patients with anti-Ge3 should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.

References

[1] Walker, P.S., Reid, M.E., 2010. The Gerbich blood group system: a review. Immunohematology 26, 60–65.
[2] Arndt, P., et al., 2002. First example of anti-Ge associated with severe hemolytic disease of the newborn [abstract]. Transfusion 42 (Suppl.), 19S.
[3] Blackall, D.P., et al., 2008. Hemolytic disease of the fetus and newborn due to anti-Ge3: combined antibody-dependent hemolysis and erythroid precursor cell growth inhibition. Am J Perinatol 25, 541–545.
[4] Denomme, G.A., et al., 2006. Inhibition of erythroid progenitor cell growth by anti-Ge3. Br J Haematol 133, 443–444.

Ge4 Antigen

Terminology

ISBT symbol (number)	GE4 (020004 or 20.4)
Obsolete name	201004

History	Ge4 was given the next number when an antibody was found that agglutinated Gerbich-positive and Gerbich-negative (both Yus and Gerbich type) RBCs, but not RBCs with the Leach (Ge_{null}) phenotype.

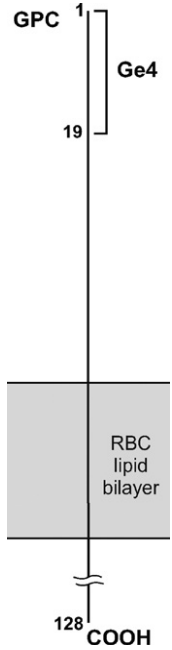
Occurrence

All populations	100%

Expression

Cord RBCs	Expressed
Altered	Weak on protein 4.1-deficient RBCs Absent from Leach phenotype RBCs

Molecular basis associated with Ge4 antigen[1]



Effect of enzymes and chemicals on Ge4 antigen on intact RBCs

Ficin/Papain	Sensitive
Trypsin	Sensitive
α-Chymotrypsin	Resistant
Sialidase	Sensitive
DTT 200 mM	Resistant
Acid	Resistant

In vitro characteristics of alloanti-Ge4

Immunoglobulin class	IgG
Optimal technique	IAT

Clinical significance of alloanti-Ge4

No information because only one alloanti-Ge4 has been described.

Comments

Ge4 is expressed on the N-terminal domain of normal and all variants of GPC (GPC.Yus, GPC.Gerbich, GPC.Wb, GPC.Ls^{a}, etc.).
Siblings of patients with anti-Ge4 should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.

Reference

[1] Walker, P.S., Reid, M.E., 2010. The Gerbich blood group system: a review. Immunohematology 26, 60–65.

Wb Antigen

Terminology

ISBT symbol (number)	GE5 (020005 or 20.5)
Obsolete names	Webb; 201005 or 700009
History	Found in 1963 and named after the donor whose group O RBCs were agglutinated by a high-titer ABO typing serum. Shown to be on a variant form of GPC in 1986.

Occurrence

Most populations	<0.01%
Wales and Australia	<0.1%

Expression

Cord RBCs	Presumed expressed

Molecular basis associated with Wb antigen[1]

Amino acid	Ser8 of GPC. This substitution results in a loss of the N-glycan, and possibly a gain of an O-glycan[2]. Thus, GPC.Wb has an M_r of approximately 2,700 less than GPC.
Nucleotide	G at bp 23 in exon 1
Wb– (wild type)	Asn8 and A at bp 23

Effect of enzymes and chemicals on Wb antigen on intact RBCs

Ficin/Papain	Sensitive
Trypsin	Sensitive
α-Chymotrypsin	Resistant
Sialidase	Sensitive
DTT 200 mM	Resistant
Acid	Resistant

In vitro characteristics of alloanti-Wb

Immunoglobulin class	IgM and IgG
Optimal technique	RT; IAT

Clinical significance of alloanti-Wb

Transfusion reaction	No
HDFN	No

Comments

Anti-Wb are usually naturally-occurring[3].

References

[1] Walker, P.S., Reid, M.E., 2010. The Gerbich blood group system: a review. Immunohematology 26, 60–65.

[2] Reid, M.E., et al., 1987. Structural relationships between human erythrocyte sialoglycoproteins beta and gamma and abnormal sialoglycoproteins found in certain rare human erythrocyte variants lacking the Gerbich blood-group antigen(s). Biochem J 244, 123–128.

[3] Bloomfield L., et al., The Webb (Wb) antigen in South Wales donors. Hum. Hered, 36, 352–356.

Ls^a Antigen

Terminology

ISBT symbol (number)	GE6 (020006 or 20.6)
Obsolete names	Lewis II; Rl^a (Rosenlund); 700007; 700024; 201006
History	Anti-Ls^a identified in an anti-B typing serum in 1963. Originally called Lewis II after the antigen-positive donor, but later renamed Ls^a to avoid confusion with the established Lewis antigens. Associated with Gerbich in 1990.

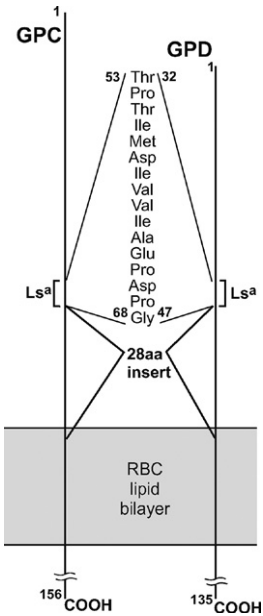
Occurrence

Most populations	<0.01%
Blacks	2%
Finns	1.6%

Expression

Cord RBCs	Presumed expressed
Altered	Increased on RBCs with three copies of amino acids encoded by exon 3

Molecular basis associated with Ls^a antigen[1,2]



Ls^a antigen is located within an amino acid sequence encoded by nucleotides at the junction of the replicated exon 3 to exon 3.

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

Ficin/Papain	Sensitive
Trypsin	Sensitive
α-Chymotrypsin	Resistant
Sialidase	Resistant
DTT 200 mM	Resistant

In vitro characteristics of alloanti-Ls^a

Immunoglobulin class	IgM and IgG
Optimal technique	RT; IAT

Clinical significance of alloanti-Ls^a

Transfusion reaction	No data because antibody and antigen are rare
HDFN	No

Comments

Anti-Ls^a is naturally-occurring.

References

[1] Reid, M.E., et al., 1994. Duplication of exon 3 in the glycoprotein C gene gives rise to the Ls^a blood group antigen. Transfusion 34, 966–969.

[2] Walker, P.S., Reid, M.E., 2010. The Gerbich blood group system: a review. Immunohematology 26, 60–65.

An^a Antigen

Terminology

ISBT symbol (number)	GE7 (020007 or 20.7)
Obsolete names	Ahonen; 700020
History	Identified in 1972 and named after the donor (Ahonen), whose RBCs were agglutinated by a patient's serum. Joined Gerbich in 1990 when the antigen was located on a variant of GPD.

Occurrence

Most populations	0.01%
Finns	0.2%

Expression

Cord RBCs	Presumed expressed

Molecular basis associated with An^a antigen[1,2]

Amino acid	Ser2 of GPD. The altered GPC (Ser23) does not express An^a
Nucleotide	T at bp 67 in exon 2 of *GYPC*
An(a–) (wild type)	GPC has Ala23 and GPD has Ala2, and G at bp 67

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

Ficin/Papain	Sensitive
Trypsin	Sensitive
α-Chymotrypsin	Weakened
Sialidase	Sensitive
DTT 200 mM	Resistant

In vitro characteristics of alloanti-An^a

Immunoglobulin class	IgM and IgG
Optimal technique	RT; IAT

Clinical significance of alloanti-An^a

Transfusion reaction	No data because antibody and antigen are rare
HDFN	No

Comments

Anti-An^a may be naturally-occurring.

References

[1] Daniels, G., et al., 1993. A point mutation in the *GYPC* gene results in the expression of the blood group An^a antigen on glycophorin D but not on glycophorin C: further evidence that glycophorin D is a product of the *GYPC* gene. Blood 82, 3198–3203.

[2] Walker, P.S., Reid, M.E., 2010. The Gerbich blood group system: a review. Immunohematology 26, 60–65.

Dh^a Antigen

Terminology

ISBT symbol (number)	GE8 (020008 or 20.8)
Obsolete names	Duch; 700031
History	Identified in 1968 during pretransfusion testing, and named after the antigen-positive Danish blood donor. Joined Gerbich in 1990 when Dh^a was located on a variant of GPC.

Occurrence

All populations	<0.01%

Expression

Cord RBCs	Presumed expressed

Molecular basis associated with Dh^a antigen[1,2]

Amino acid	Phe14 of GPC
Nucleotide	T at bp 40 in exon 1
Dh(a–) (wild type)	Leu14 and C at bp 40

Effect of enzymes and chemicals on Dh[a] antigen on intact RBCs

Ficin/Papain	Sensitive
Trypsin	Sensitive
α-Chymotrypsin	Resistant
Sialidase	Sensitive
DTT 200 mM	Resistant

In vitro characteristics of alloanti-Dh[a]

Immunoglobulin class	IgM and IgG
Optimal technique	RT and IAT

Clinical significance of alloanti-Dh[a]

Transfusion reaction	No data because antibody and antigen are rare
HDFN	No

Comments

Anti-Dh[a] may be naturally-occurring.

References

[1] King, M.J., et al., 1992. Point mutation in the glycophorin C gene results in the expression of the blood group antigen Dh[a]. Vox Sang 63, 56–58.

[2] Walker, P.S., Reid, M.E., 2010. The Gerbich blood group system: a review. Immunohematology 26, 60–65.

GEIS Antigen

Terminology

ISBT symbol (number)	GE9 (020009 or 20.9)
History	Found and named in 2004, "GE" from Gerbich and "IS" from the name of the index case.

Occurrence

Japanese	Only three GEIS+ probands have been reported

Expression

Cord RBCs	Presumed expressed

Molecular basis associated with GEIS antigen[1]

Amino acid	Asn32 in GPC and Asn11 in GPD
Nucleotide	A at bp 95 in exon 2
GEIS– (wild type)	Thr32 in GPC and Thr11 in GPD, and C at bp 95

Effect of enzymes and chemicals on GEIS antigen on intact RBCs

Ficin/Papain	Sensitive
Trypsin	Resistant
α-Chymotrypsin	Sensitive
Sialidase	Sensitive
DTT 200 mM	Presumed resistant

In vitro characteristics of alloanti-GEIS

Immunoglobulin class	IgG
Optimal technique	IAT

Clinical significance of alloanti-GEIS

No data because antibody and antigen are rare

Reference

[1] Yabe, R., et al., 2004. Is a new Gerbich blood group antigen located on the GPC and GPD [abstract]? Vox Sang 87 (Suppl. 3), 79.

GEPL Antigen

Terminology

ISBT symbol (number)	GE10 (020010 or 20.10)
History	Reported in 2008 and named in 2010, "GE" from Gerbich, "P" from proline and "L" from leucine.

Occurrence

Only one GEPL– proband has been described.

Expression

Cord RBCs	Presumed expressed

Molecular basis associated with GEPL antigen[1]

Amino acid	Pro45 in GPC and Pro24 in GPD
Nucleotide	C at bp 134 in exon 3
GEPL–	Leu45 in GPC and Leu24 in GPD, and T at bp 134

Effect of enzymes and chemicals on GEPL antigen on intact RBCs

Ficin/Papain	Sensitive
Trypsin	Presumed sensitive
α-Chymotrypsin	Presumed resistant
DTT 200 mM	Presumed resistant

In vitro characteristics of alloanti-GEPL

Immunoglobulin class	IgG
Optimal technique	IAT

Clinical significance of alloanti-GEPL

No data because antibody is rare.

Comments

The plasma of the GEPL– proband appeared to contain anti-Ge3, whereas the patient's RBCs were GE:2,3,4 with aberrant expression of Ge3.
Siblings of patients with anti-GEPL should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.

Reference

[1] Poole, J., et al., 2008. Novel mutations in GYPC giving rise to lack of Ge epitopes and anti-Ge production [abstract]. Vox Sang 95 (Suppl. 1), 181.

GEAT Antigen

Terminology

ISBT symbol (number)	GE11 (020011 or 20.11)
History	Reported in 2008 and named in 2010, "GE" from Gerbich, and "A" and "T" from the nucleotides involved.

Occurrence

Only one GEAT– proband has been reported.

Expression

Cord RBCs	Presumed expressed

Molecular basis associated with GETI antigen[1]

Amino acid	Asp19 in GPC
Nucleotide	A at bp 56 in exon 2
GEAT–	Val19 in GPC and T at bp 36

Effect of enzymes and chemicals on GEAT antigen on intact RBCs

Ficin/Papain	Sensitive
Trypsin	Sensitive
α-Chymotrypsin	Resistant
DTT 200 mM	Resistant
Acid	Resistant

In vitro characteristics of alloanti-GEAT

Immunoglobulin class	IgG
Optimal technique	IAT

Clinical significance of alloanti-GEAT

No data because antibody is rare.

Comments

Plasma from the GEAT– proband did not react with GE:–2,–3,4 or GE:–2,–3,–4 RBC samples, and gave variable/weak reactions with GE:–2,3,4 RBC samples. The GEAT– proband had the GE:2,3,4 phenotype, but some antisera reacted weakly.

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

Reference

[1] Poole, J., et al., 2008. Novel mutations in GYPC giving rise to lack of Ge epitopes and anti-Ge production [abstract]. Vox Sang 95 (Suppl. 1), 181.

GETI Antigen

Terminology

ISBT symbol (number)	GE12 (020012 or 20.12)
History	Reported in 2008 and named in 2010, “GE” from Gerbich, “T” from threonine, and “I” from isoleucine.

Occurrence

Only one GETI– proband has been reported.

Expression

Cord RBCs	Presumed expressed

Molecular basis associated with GETI antigen[1]

Amino acid	Thr27 in GPC and Thr6 in GPD
Nucleotide	C at bp 80 in exon 2
GETI–	Ile27 in GPC and Ile6 in GPD, and T at bp 80

Effect of enzymes and chemicals on GETI antigen on intact RBCs

Ficin/Papain	Sensitive
Trypsin	Presumed sensitive
α-Chymotrypsin	Presumed resistant
DTT 200 mM	Presumed resistant

In vitro characteristics of alloanti-GETI

Immunoglobulin class	IgG
Optimal technique	IAT

Clinical significance of alloanti-GETI

No data because antibody is rare.

Comments

Initially the antibody made by a GETI– patient, by her GETI– brother, and by another patient appeared to be anti-Ge2. The red cells from these people were GE:–2,3,4 (except that one autoanti-Ge2 reacted), and there was marginal weakening of Ge3 and Ge4.

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

Reference

[1] Poole, J., et al., 2008. Novel mutations in GYPC giving rise to lack of Ge epitopes and anti-Ge production [abstract]. Vox Sang 95 (Suppl. 1), 181.