Kidd Blood Group System

Number of antigens 3

Polymorphic	Jk^a, Jk^b
High prevalence	Jk3

Terminology

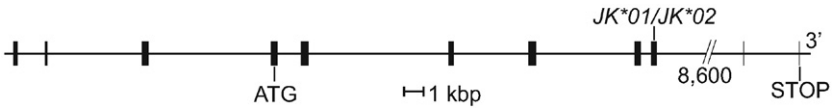
ISBT symbol (number)	JK (009)
History	Named in 1951 after the initials of the sixth child (John Kidd) of the first proband to make anti-Jk^a. John had hemolytic disease of the newborn.

Expression

Other blood cells	Not on lymphocytes, granulocytes, monocytes or platelets
Tissues	Kidney

Gene

Chromosome	18q12.3
Name	*JK (SLC14A1, HUT11A)*
Organization	11 exons distributed over 30 kbp of gDNA; exons 4 to 11 encode the mature protein
Product	Urea transporter UT-B, Kidd glycoprotein



The Blood Group Antigen (3/e). DOI:http://dx.doi.org/10.1016/B978-0-12-415849-8.00011-9

Database accession numbers

GenBank	NM_015865 (mRNA)
Entrez Gene ID	6563

Molecular basis of Kidd phenotype

The reference allele, *JK*02* or *JK*B* (Accession number NM_015865) encodes Jk^b (JK2), JK3. The nucleotide difference from this reference allele, and the amino acid affected, are given.

Allele encodes	Allele name	Exon	Nucleotide	Restriction enzyme	Amino acid	Ethnicity (prevalence)
Jk(a+b–) or JK:1,–2	*JK*01* or *JK*A*	9	838A>G	*Mnl*I+	Asn280Asp	Blacks> Whites> Asians (Common)

Molecular bases of weak or partial Kidd antigens

Homozygosity or compound herterozygosity leads to weak or partial phenotypes. Nucleotide changes from the *JK*01* or *JK*02* backgrounds, and the amino acids affected, are given. The term partial is used to classify an antigen-positive person who has the corresponding alloantibody in her/his plasma.

Allele encodes	Allele name	Exon	Nucleotide	Amino acid	Ethnicity (prevalence)	Antibody production^
Jk(a+^{w}b–)	*JK*01W.01*	4	130G>A	Glu44Lys	Many populations (Common)	−JK3 −Jk^a
Jk(a+^{w}b–)	*JK*01W.02*	7	511T>C	Trp171Arg	(Rare)	
Jk(a+^{w}b–)	*JK*01W.03*	4	28G>A	Val10Met	Black (Several)	−Jk^a
Jk(a+^{w}b–)	*JK*01W.04*	5	226G>A	Val76Ile	Black (Several)	−Jk^a
Jk(a+^{w}b–)	*JK*01W.05*	8	742G>A	Ala248Thr	Rare	
Jk(a–b+^{w})	*JK*02W.01*	7	548C>T	Ala183Val	Black (Rare)	

^Not all people with the allele have made the alloantibody.

Molecular bases of silencing of *JK*A* or *JK*B*

Homozygosity or compound heterozygosity leads to JK:–3 [Jk(a–b–)] phenotype.
Nucleotide changes from the *JK*01* or *JK*02* backgrounds, and the amino acids affected, are given.

Allele name	Exon/ intron	Nucleotide	Amino acid	Ethnicity (prevalence)
*JK*01N.01*	4 & 5	Exons 4&5 deleted	Initiation Met absent	Tunisian, English, Bosnian (Rare)
*JK*01N.02*	5	202C>T	Gln68Stop	Caucasian, American (Rare)
*JK*01N.03*	7	582C>G	Tyr194Stop	Swiss, English (Few)
*JK*01N.04*	10	956C>T	Thr319Met	African American, (Rare)
*JK*01N.05*	7	561C>A	Tyr187Stop	African American (Rare) African Brazilian (Many)
*JK*01N.06*	Intron 5	IVS5–1 g>a	Exon 6 skipped; in frame	Asian Indian (Rare)
*JK*02N.01*	Intron 5	IVS5–1 g>a	Exon 6 skipped; in frame	Polynesian, Chinese (Several)
*JK*02N.02*	Intron 5	IVS5–1 g>c	Exon 6 skipped; in frame	Chinese (Rare)
*JK*02N.03*	5	222C>A, 499A>G	Asn74Lys, Met167Val	Taiwanese (Rare)
*JK*02N.04*	Intron 7	IVS7+1 g>t	Exon 7 skipped; frameshift→ Leu223Stop	French (Rare)
*JK*02N.05*	8	723delA	Frameshift→ Ile262Stop	Hispanic American (Rare)
*JK*02N.06*	9	871T>C	Ser291Pro	Finns (Several)
*JK*02N.07*	9	896G>A	Gly299Glu	Taiwanese (Rare)
*JK*02N.08*	10	956C>T	Thr319Met	Indian, Pakistani (Rare)
*JK*02N.09*^	5	191G>A	Arg64Gln	Black (Rare)

^Non-reactive with 1 monoclonal anti-Jk^b, positive with 1 polyclonal anti-Jk^b. Absorption and elution studies where not performed; may express some Jk protein.

Amino acid sequence[1,2]

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MEDSPTMVRV  DSPTMVRGEN  QVSPCQGRRC  FPKALGYVTG  DMKKLANQLK   50
DKPVVLQFID  WILRGISQVV  FVNNPVSGIL  ILVGLLVQNP  WWALTGWLGT  100
VVSTLMALLL  SQDRSLIASG  LYGYNATLVG  VLMAVFSDKG  DYFWWLLLPV  150
CAMSMTCPIF  SSALNSMLSK  WDLPVFTLPF  NMALSMYLSA  TGHYNPFFPA  200
KLVIPITTAP  NISWSDLSAL  ELLKSIPVGV  GQIYGCDNPW  TGGIFLGAIL  250
LSSPLMCLHA  AIGSLLGIAA  GLSLSAPFED  IYFGLWGFNS  SLACIAMGGM  300
FMALTWQTHL  LALGCALFTA  YLGVGMANFM  AEVGLPACTW  PFCLATLLFL  350
IMTTKNSNIY  KMPLSKVTYP  EENRIFYLQA  KKRMVESPL              389
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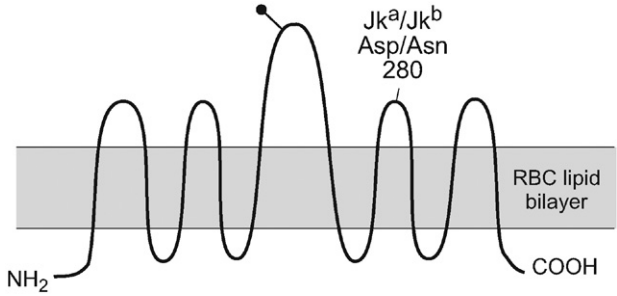
Carrier molecule

Multi-pass glycoprotein.



M_r (SDS-PAGE)	Predicted 43,000
CHO: N-glycan	2 potential sites (1 likely)
Cysteine residues	10
Copies per RBC	14,000

Function

A urea transporter in RBCs, UT-B plays a role in urea concentration by speeding up urea transport across the membrane as the RBCs pass through the descending and ascending vasa recta. Urea crosses the membrane of Jk(a–b–) RBCs about 1,000 times slower than in normal RBCs[3].

Disease association

Jk(a–b–) individuals have no clinical symptoms, but have their urine concentrating ability reduced by about one-third[3]. JK antigens may act as minor histocompatibility antigens in renal allograft rejection[4].

Phenotypes (% occurrence)

	Caucasians	Blacks	Asians
Jk(a+b–)	26.3	51.1	23.2
Jk(a–b+)	23.4	8.1	26.8
Jk(a+b+)	50.3	40.8	49.1
Jk(a–b–)	Rare	Rare	0.9 (Polynesians)

Null: Jk(a–b–)

Unusual: Jk(a–b–) [*In(Jk)*]; Several variants with altered (weakened or partial) expression of Jk^a or Jk^b (see table "Molecular bases of weak or partial Kidd phenotypes")

Comments

Jk(a–b–) RBCs resist lysis by 2 M urea[5].
Dominant type Jk(a–b–) [*In*(*Jk*)] RBCs have been found in Japanese.
Two transient Jk(a–b–) people have been described[6,7]. One was a Russian woman with myleofibrosis who made anti-Jk3 at the time her RBCs typed Jk(a–b–).

References

[1] Lucien, N., et al., 2002. Antigenic and functional properties of the human red blood cell urea transporter hUT-B1. J Biol Chem 277, 34101–34108.
[2] Olivès, B., et al., 1994. Cloning and functional expression of a urea transporter from human bone marrow cells. J Biol Chem 269, 31649–31652.
[3] Sands, J.M., et al., 1992. Urinary concentrating ability in patients with Jk(a−b−) blood type who lack carrier-mediated urea transport. J Am Soc Nephrol 2, 1689–1696.
[4] Lerut, E., et al., 2007. Duffy and Kidd blood group antigens: minor histocompatibility antigens involved in renal allograft rejection? *Transfusion*; 47, 28–40.
[5] Mougey, R., 1990. A review: the Kidd system. Immunohematology 6, 1–8.
[6] Issitt, P.D., et al., 1990. Temporary suppression of Kidd system antigen expression accompanied by transient production of anti-Jk3. Transfusion 30, 46–50.
[7] Obarski, G., et al., 1987. The Jk(a−b−) phenotype, probably occurring as a transient phenomenon [abstract]. Transfusion 27, 548.

Jk^a Antigen

Terminology

ISBT symbol (number)	JK1 (009001 or 9.1)
History	Reported in 1951. Name derived from the initials of the sixth child (John Kidd) of the antibody maker, Mrs. Kidd.

Occurrence

Caucasians	77%
Blacks	92%
Asians	72%

Antithetical antigen

Jk^b (**JK2**)

Expression

Cord RBCs	Expressed
Altered	See table: "Molecular basis of weak and partial Kidd phenotypes." When an altered allele is present, less Kidd glycoprotein is incorporated into the RBC membrane or epitope expression detected by monoclonal antibodies is reduced

Molecular basis associated with Jk^a antigen

Amino acid	Asp280
Nucleotide	G at bp 838 in exon 9

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

Ficin/Papain	Resistant (enhanced)
Trypsin	Resistant (enhanced)
α-Chymotrypsin	Resistant (enhanced)
Sialidase	Resistant
DTT 200 mM	Resistant
Acid	Resistant

In vitro characteristics of alloanti-Jk^a

Immunoglobulin class	IgG; many IgG plus IgM; IgM
Optimal technique	IAT; enzymes; PEG; CAT (gel)
Complement binding	Yes, provided that IgM is present; some hemolytic[1]

Clinical significance of alloanti-Jk^a

Transfusion reaction	No to severe; immediate or delayed/hemolytic
HDFN	Mild to moderate (rare)

Autoanti-Jk^a

Autoanti-Jk^a have been reported. With the discovery of weak or partial Jk^a phenotypes[2] it is possible some autoantibodies were actually alloantibodies.

Comments

Anti-Jk^a deteriorate *in vitro* and *in vivo*. Often found in multispecific sera. Anti-Jk^a may react more strongly with Jk(a+b–) than Jk(a+b+) RBCs (i.e., show dosage).

References

[1] Yates, J., et al., 1998. IgG anti-Jk^a/Jk^b antibodies are unlikely to fix complement. Transfus Med 8, 133–140.

[2] Wester, E.S., et al., 2011. Characterization of Jk(a+^{weak}): a new blood group phenotype associated with an altered JK*01 allele. Transfusion 51, 380–392.

Jk^b Antigen

Terminology

ISBT symbol (number)	JK2 (009002 or 9.2)
History	Found in 1953, and named for its antithetical relationship to Jk^a.

Occurrence

Caucasians	74%
Blacks	49%
Asians	76%

Antithetical antigen

Jk^a (**JK1**)

Expression

Cord RBCs	Expressed
Altered	See table: "Molecular basis of weak and partial Kidd phenotypes"

Molecular basis associated with Jk^b antigen

Amino acid	Asn280
Nucleotide	A at bp 838 in exon 9

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

Ficin/Papain	Resistant (enhanced)
Trypsin	Resistant (enhanced)
α-Chymotrypsin	Resistant (enhanced)
Sialidase	Resistant
DTT 200 mM	Resistant
Acid	Resistant

In vitro characteristics of alloanti-Jk^b

Immunoglobulin class	IgG; many IgG plus IgM; IgM
Optimal technique	IAT; enzymes; PEG; CAT
Complement binding	Yes; provided that IgM is present; some hemolytic[1]

Clinical significance of alloanti-Jk^b

Transfusion reaction	No to severe; immediate or delayed/hemolytic
HDFN	No to mild (rare)

Autoanti-Jk^b

Autoanti-Jk^b have been reported. With the discovery of weak or partial Jk^b phenotypes it is possible that some autoantibodies were actually alloantibodies.

Comments

Anti-Jk^b deteriorate *in vitro* and *in vivo*. Often found in multispecific sera.

Reference

[1] Yates, J., et al., 1998. IgG anti-Jk^a/Jk^b antibodies are unlikely to fix complement. Transfus Med 8, 133–140.

Jk3 Antigen

Terminology

ISBT symbol (number)	JK3 (009003 or 9.3)
Obsolete names	Jk^{ab}; Jk^{a}Jk^b
History	Anti-Jk^{a}Jk^b was identified in 1959 and renamed anti-Jk3 by ISBT when numbers became popular.

Occurrence

Most populations	100%
Polynesians, Finns	>99%

Expression

Cord RBCs	Expressed
Altered	Very weak on Jk(a–b–) of the *In*(*Jk*) type (detected by absorption/elution); weak expression in the presence of certain alleles (see table: "Molecular basis of weak and partial Kidd phenotypes")

Molecular bases associated with Jk3 antigen

See System pages for molecular basis of Jk(a–b–) phenotype.

Effect of enzymes and chemicals on Jk3 antigen on intact RBCs

Ficin/Papain	Resistant (enhanced)
Trypsin	Resistant (enhanced)
α-Chymotrypsin	Resistant (enhanced)
Sialidase	Resistant
DTT 200 mM	Resistant
Acid	Resistant

In vitro characteristics of alloanti-Jk3

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

Clinical significance of alloanti-Jk3

Transfusion reaction	No to severe/immediate or delayed
HDFN	No to mild

Autoanti-Jk3

Rare

Comments

Anti-Jk3 has been found in a non-transfused male.
People with *In*(*Jk*) do not make anti-Jk3, and the presence of Jk antigens can be detected by absorption and elution.
Siblings of patients with anti-Jk3 should be tested for compatibility, and the patient urged to donate blood for cryogenic storage when his/her clinical state permits.