Ii Blood Group Collection

Number of antigens 1

High prevalence i

Terminology

ISBT symbol (number)	I (207)
History	I and i antigens were placed in the Ii Blood Group Collection in 1990. In 2002 the I antigen was promoted to a blood group system, leaving i alone in Blood Group Collection 207.

Gene

The genetic basis of i expression is unknown.

Carrier molecule

The i antigen is on unbranched carbohydrate chains of repeating *N*-acetyllactosamine units on glycolipids and glycoproteins on RBCs, and on proteins in plasma. With the action of the branching enzyme, β6GlcNAc-transferase, these i antigen-carrying chains become the I antigen (see I Blood Group System [**027**])[1], i.e. i is the precursor structure for I.

Disease association

Enhanced expression of i antigens is associated with leukemia, Tk polyagglutination, thalassemia, sickle cell disease, HEMPAS, Diamond Blackfan anemia, myeloblastic erythropoiesis, sideroblastic erythropoiesis, and any condition that results in stress hemopoiesis.
Anti-i is associated with infectious mononucleosis and other lymphoproliferative disorders (e.g., Hodgkins disease), and occasionally with CHAD.

The Blood Group Antigen (3/e). DOI: http://dx.doi.org/10.1016/B978-0-12-415849-8.00037-5

Phenotypes associated with i antigen and the reciprocal I antigen

See I Blood Group System [**027**].

Molecular basis of adult i phenotype

See I Blood Group System [**027**].

Comments

The i antigen occurs on unbranched A-, B-, and H-active oligosaccharide chains.

Reference

[1] Cooling, L., 2010. Polyactosamines, there's more than meets the "Ii": a review of the I system. Immunohematology 26, 133–155.

i Antigen

Terminology

ISBT symbol (number)	I2 (207002 or 207.2)
Obsolete name	900027
History	Named in 1960 because of its reciprocal, but not classical antithetical, association with the I antigen.

Occurrence

All RBCs of adults have at least trace amounts of i antigen. The adult i phenotype is rare.

Reciprocal antigen

I (See I Blood Group System [**027**]).

Expression

Cord RBCs	Strong
Altered	Enhanced on CDA II RBCs and RBCs produced under hemopoietic stress

Molecular basis associated with i antigen[1]

Linear type 2 chains	Galβ1-4(GlcNAcβ1-3Galβ1-4)$_n$-Glc-Cer, but also as part of unbranched glycans on glycoproteins

See I Blood Group System (**027**) pages for molecular basis associated with adult i phenotype.

Effect of enzymes and chemicals on i antigen on intact RBCs

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

In vitro characteristics of anti-i

Immunoglobulin class	IgM (rarely IgG)
Optimal technique	RT or 4°C
Complement binding	Yes; some hemolytic

Clinical significance of anti-i

Transfusion reaction	No
HDFN	Rare

Autoanti-i

Anti-i are considered to be autoantibodies. Transient autoanti-i can occur in infectious mononucleosis and some lymphoproliferative disorders.

Comments

So-called compound antigens have been described: iH, iP1, iHLe^{b}.
RBCs with the dominant Lu(a–b–) phenotype have a depressed expression of i antigen, whereas RBCs with the X-linked form of the Lu(a–b–) phenotype have enhanced expression of i antigen. The i antigen expression is often enhanced on RBCs from patients with hemopoietic stress, due to the rapid transit through ER and Golgi. Horse RBCs have a strong expression of i antigen, and can be used as a diagnostic tool for infectious mononucleosis.

Reference

[1] Roelcke, D., 1995. Serology, biochemistry, and pathology of antigens defined by cold agglutinins. In: Cartron, J-P, Rouger, P (Eds.), Molecular Basis of Human Blood Group Antigens. Plenum Press, New York, NY, pp. 117–152.