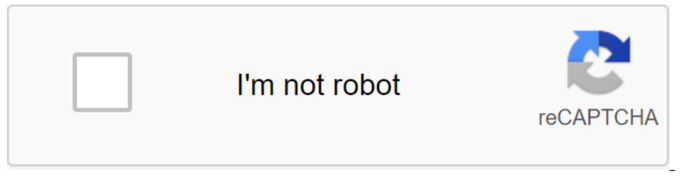

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Kidd blood group system pdf

Antigens in Kidd's blood group The Kidd antigen system (also known as Jk antigen) are proteins found in Kidd's blood group that act as antigens, i.e. antigens. Jk antigen is found on a protein responsible for urea transport in the red blood cells and kidneys. [1] They are important in transfusion medicine. Individuals with two Jk(a) antigens, for example, may form antibodies against donated blood containing two Jk (b) antigens (and thus no Jk (a) antigens). This can lead to hemolytic anemia, where the body destroys the transfused blood, leading to low red blood cells. Another disease associated with Jk antigen is hemolytic disease in the newborn, where a pregnant woman's body creates antibodies to the blood in her fetus, leading to the destruction of the fetal blood cells. Hemolytic disease in newborns associated with Jk antibodies is typically mild, although fatal cases have been reported. [2] Recoding of this protein is found on chromosome 18. [3] Three Jk alleles are Jk (a), Jk (b) and Jk3. Jk (a) was discovered by Allen et al. in 1951 and is named after a patient (Mrs Kidd delivered a child with a hemolytic disease in the newborn associated with an antibody targeting a new antigen Jk (a). While Jk (b) was discovered by Plant et al. in 1953, individuals who lack Jk antigen (Jk null) are unable to concentrate their urine at most. [4] Genetics and biochemistry Kidd consists of three antigens on a glycoprotein with 10 transmembrane spans domains, cytoplasmic N and C-termini and an extracellular N-glycosylation site. [5] The Kidd gene has 11 exons with exons 4-11 encoding the ripe protein. The Kidd gene (SLC14A1) is on chromosome 18q12.3. Kidd antigens Jka (JK1) and Jkb (JK2) Jka and Jkb are products of alleles with Asp280 and Asn280 in the fourth external loop of Kidd glycoprotein. Jka and Jkb have similar prevalences in white and Asian populations, but Jka is more common in blacks than Jkb.[5] Kidd antigens amplified by enzymes Jk (a-b-) and Jk3 Jk (a-b-) represent null phenotype and usually results from homozygosity for a silent gene on JK locus. [5] Null phenotype is rare in most populations, but has increased prevalence in Polynesians (one in 400) and Niueans (1.4%).[5] In polynesians, the null allele contains a splicing hyper mutation in intron 5, resulting in loss of exon 6 from the mRNA product. In fins (null phenotype less rare than in other European populations), null phenotype is due to a mutation coding a Ser291Pro substitution. A rare null phenotype in Japanese individuals is caused by heterozygosity for an inhibitor. I (Jk) in analogy with In(Lu) dominant inhibitor of Lutheran and other antigens. [5] Immunized individuals with Jk(a-b-) phenotype can produce anti-Jk3. Very weak expression of Jka and/or Jkb can be detected on In(Jk) red blood cells in adsorption/elution test. [5] Kidd and clinically meaning Antibody subtypes and complement fixation Anti-Jka and -Jkb are not common. They are usually hot-reacting IgG1 and IgG3, but may also include IgG2, IgG4 or IgM. About 50% of anti-Jka and -Jkb antibodies are able to bind the supplement. [6] Dosage Kidd antibodies display dosage: red blood cells from homozygotic individuals (JkaJka or Jk (a + b-)) express more antigen than heterozygode individuals (JkaJkb or Jk (a + b+)). [7] Anti-Kidd antibodies seem to react more strongly against cells that are homozygotic. Laboratory detection Kidd antibodies can be difficult to detect by direct agglutination testing and generally require the addition of antihuman globulin after a hot incubation period. Clinically meaning Kidd antibodies are dangerous as they are able to cause severe acute hemolytic transfusion reactions. They are unique in that they are able to drop to low or even undetectable levels after several months of exposure. [5] Thus, on pre-transfusion testing, an anti-Jka or -Jkb may go undetected. After transfusion, a subsequent robust antibody response in the patient may occur (anamnestic response), resulting in hemolysis of the transfused red blood cells. Kidd antibodies are often able to bind supplement and cause intravascular hemolysis. More often, however, Kidd antibodies cause acute extravascular hemolysis. [7] They are a notorious cause of delayed hemolytic transfusion reactions, and can occur up to a week after transfusion in some cases. Kidd antibodies rarely cause hemolytic disease in the fetus and newborns. [5] Kidd glycoprotein as urea transporter Kidd antigens are placed on a red blood cell urea transporter (human urea transporter 11-HUT11 or UT-B1). [8] When red blood cells approach the renal medulla (where there is a high concentration of urea), the urea conveyor allows for rapid uptake of urea and prevents cell shrinkage in medulla's hypertonic environment. [5] When the red cell leaves the medulla, the urea is transported back out of the cell, preventing cellular swelling and preventing urea from being torn away from the kidneys. [5] HUT11 was detected on endothelial cells in vasa recta (vascular delivery of kidney medulla), but it is not present in kidney tubules. [5] Due to the absence of urea conveyor, Jk (a-b-) cells are not hemolyzed by 2M urea. This can be used as a screening test for Jk(a-b-) donors. Jk(a-b-) phenotype has no clinical defect, although two people with this phenotype have been reported to have mild urine-concentrated defects. [4] Kidd antibodies in transplant patients Kidd antibodies are able to behave as histocompatibility antigens in kidney transplants and may be responsible for allograft rejection in some cases. [9] References Online Mendelian Inheritance in Man (OMIM): 111000 - OMIM page on Kidd antigen system protein ^ Olive B, Mattei MG, Huet M, Neau Pedersen, S, Cartron JP, Bailly P. Kidd blodgruppe og urea urea function of human erythrocytes is carried by the same protein. Journal of Biological Chemistry. 1995 Jun 30.270(26):15607-10. doi:10.1074/jbc.270.26.15607 PMID 7797558 ^ Kim WD, Lee YH. A fatal case of severe hemolytic disease in newborns associated with anti-Jk(b). Journal of Korean Medical Science. 2006 Feb;21(1):151-4. PMID 16479082 ^ Geitvik GA, Hoyheim B, Gedde-Dahl T, Grzeschik KH, Lothe Rasmussen, Tomter H, Olaisen B. The Kidd (JK) blood group locus assigned chromosome 18 by close connection to a DNA RPFL. 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External links Kidd on the BGMUT Blood Group Antigen Gene Mutation Database at NCBI, NIH Retrieved from The Kidd (JK) glycoprotein is the red blood cell (RBC) urea transporter. Located in the membrane it quickly transports urea in and out of RBC, maintaining osmotic stability and form of RBC in the process. Kidd glycoprotein is also expressed in the kidneys, where it allows the kidneys to build up a high concentration of urea, which is necessary for the kidneys to produce concentrated urine. People who do not produce Kidd glycoprotein tend not to be able to maximum concentrate urine, but despite this, they are healthy and their RBCs have a normal shape and longevity. Antibodies that target Kidd antigens are a major cause of delayed hemolytic transfusion reactions. Anti-Kidd antibodies are also a cause of hemolytic disease in the newborn (HDN), the severity of the disease varies, but tends to be mild in nature. In 1951, a patient called Mrs. Kidd was found to have produced antibodies targeting a then unknown red cell antigen during her pregnancy. The cursor was present at RBCs of her fetus, and the mother's antibodies targeting it caused the fatal hemolytic disease in her newborn baby. The protein was named Jka and was the first antigen to be detected in the Kidd blood group system. Since then, two other antigens, Jkb and Jk3, have been found. In 1959 it was example of null phenotype, i.e. was found in a woman who had become jaundice after a blood transfusion. Her serum turned out to contain an antibody that recognized both Jka and Jkb. This antibody was subsequently named anti-Jk3.NomenclatureNumber of Kidd antigens: 3Gene name: Solute carrier family 14, member 1 There are three common Kidd phenotypes: JK (a + b-), JK (a-b+), and JK (a+ b+). Jk-null phenotype, JK (a-b-), is rare in most populations. People with this blood type are often detected after they have been vaccinated for Kidd antigens during a previous blood transfusion or pregnancy. After immunisation, JK(a-b-) individuals form anti-Jk3, which can cause HDN in subsequent pregnancies and haemolysis donor blood containing Jka and/or Jkb antigens during a subsequent blood transfusion. The expression of Kidd antigens is limited to RBC and the kidneys (in vasa recta). Kidd protein is a great urea transporter in RBCs. It quickly transports urea in and out of RBCs and in the process helps maintain osmotic stability. Urea transport across kidd null RBC membranes is ~1000 times slower than across normal RBC membranes (2, 3). The transport of urea by Kidd glycoprotein in the kidneys allows the kidney medulla to maintain a high concentration of urea, which in turn allows the kidneys to produce concentrated urine. However, the absence of Kidd glycoprotein is not associated with disease. RBCs in Kidd null individuals have a normal shape and longevity (3). People with Jk(a-b-) phenotype are unable to concentrate urine to the maximum, but this does not cause other health problems (4). Kidd antibodies are often difficult to detect, making them dangerous in transfusion medications where they are suspected to be a common cause of delayed hemolytic transfusion reactions (DHTRs) (5). Anti-Jka can cause severe and deadly hemolytic transfusion reactions (6), but is more commonly associated with less severe DHTRs. It has been estimated that over a third of DHTRs are caused by anti-Jka (7, 8). Case studies have also pointed to anti-Jkb as being responsible for severe DHTR (9, 10). Anti-Jk3 has also been responsible for causing severe hemolytic transfusion reactions, both immediate and delayed (5). During pregnancy, fetal Kidd antigens are able to cause alloimmunization of the mother (11). However, unlike the hemolytic activity of Kidd antibodies in incompatible blood transfusions, anti-Jka and anti-Jkb are only rarely responsible for severe HDN (12). Similarly, anti-Jk3 is a rare cause of HDN, but the first documented case in Ms Kidd's newborn was fatal. The SLC14A1 gene (Solute carrier family 14, member 1) is a member of the urea carrier gene family and is located on chromosome 18 (18q12-q21). The gene is organized into 11 exons spread over 30kb of DNA. The first three exons and part of the fourth are not translated; exons 4-11 encode the mature Kidd protein. and Jkb antigens are products from two alleles inherited a co-dominant way. Jka/Jkb polymorphism is the result of an 838G→A transition resulting in a D280N substitution (13). Based on this, several investigators have suggested different methods of JK genotyping (13-15). Jk(a-b-) phenotype is generally inherited as a recessive trait-a number of different mutations have been shown to be responsible 16). In the Polynesian population, where null phenotype is less rare, a splicing site mutation causes loss of exon 6 from mRNA prints and it is unlikely that the truncated Kidd protein produced is transported to the RBC membrane (17). A similar situation applies in the Finnish population, where a different genetic explanation causes the same phenotype (17,18). Watch sequences of Kidd alleles on thedbRBC Sequence Alignment ViewerThe Kidd protein urea transporter is an integrated protein of RBC membrane. It is a transmembrane protein containing 389 amino acid esters. The protein is expected to spread over the membrane 10 times, where both the N endpoint and the C endpoint are intracellular. This membrane topology is shared by the anion exchanger who carries Diego blood group antigens. The Kidd protein consists of two hydrophobic domains, each extending across the membrane five times, and they are connected by a large glycosylated extracellular loop. 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