# Frequency of Kidd Blood Group in a Section of Lahore Population.

S AMJAD

*Department of Hematology, Sh. Zayed Postgraduate Medical Institute, Lahore*
*Correspondence to: Dr. Shahida Amjad Associate Professor (Hematology)*

**The prevalence of Kidd blood group was studied among 310 healthy volunteers. $Jk^{a-}$ was the most common blood group. Found in 91.29%, $JK^{b-}$ was the next most common being 87.74% followed by $JK^{b+}$ being 12.26% and $Jk^{a+}$ was the rarest group with a frequency of 8.7%. When tested for both anti-$JK^{a}$ and anti-$JK^{b+}$, the most common group was $JK^{(a-b-)}$ present in 80.09% followed by $JK^{(a-b+)}$(10)%, $JK^{(a+b-)}$(6.77%), while the least common group was $JK^{(a+b+)}$(0.96%). The proportion of individuals with $JK^{a-}$ is significantly higher (P<0.005) in our population (91.29) as is significantly higher (p<0.005 in our population(91.29% as compared to Caucasian(23.6%). Whereas about 50% have phenotype $JK^{(a+b+)}$, only 0.96% of our population had this phenotype(P<0.005).**

**Key words: Kidd blood group, frequency**

The most important system of blood groups leading to transfusion reactions and hemolytic disease of newborn (HDN) are still the ABO and Rh Systems. Since the advent of Rh prophylaxis and proper methodology of ABO and Rh grouping, some of the other minor groups have come in the forefront as being culprits to Hemolytic Disease of the Newborn (HDN) and transfusion reactions. These are Kell, Kidd and Duffy blood Groups[1].

The Kidd (JK) blood group system is clinically important in transfusion medicine. All antibodies to antigens in the system may be produced following blood transfusion or during pregnancy and can result in serious hemolytic transfusion reactions and hemolytic disease of the newborn (HDN). JK antigens on erythrocytes are carried by glycoproteins with the capacity to transport urea through cell membranes. cDNA complementary to mRNA transcribed at the JK locus was cloned in 1994[2].

The Kidd (JK) blood is carried by an integral membrane glycoprotein which transports urea through the red cell membrane and is also present on endothelial cells of the vasa recta in the Kidney. The exonintron structure of the human blood group Kidd/urea transporter gene has been determined. It is organized into 11 axons distributed over 30 Kilobase pairs. The mature protein is encoded by axons 4-11. The transcription imitation site was identified by 5 rapid amplification of cDNA ends polymerize chain reaction at 335 base pairs upstream of the translation start point located in axon 4. The 5 flanking region, from nucleotide 837 to 336, contains TATA an inverted CAAT boxes as well as GATA 1/SPI erythroid specific cis-acting regulatory elements[3]

Of the fatal hemolytic reactions reported in USA, over a 3 years period, most of the reactions were immediate in type. Of these 75%, were due to ABO incompatibility and the rest were due to a variety of the other antigens like Rh and other minor groups e.g.Kell, Kidd and Duffy[4].

A recent study from Mayo Clinic, USA[5], published in October 1999, has revealed an increasing incidence of delayed hemolytic and serologic transfusion reactions noticed since 1978. Similar observations have been made from India[6], France[7] and Sweden[8]. It is, therefore, essential that the frequency of these blood groups be known in various populations.

Since no such study existed in Pakistan, an attempt was made to fill in this gap. In a previous study[9] the frequency of Kell, group has been described. The present paper deals with the frequency of Kidd blood group in a section of Lahore population.

## Material and Methods

Three hundred and ten volunteers were studied who were well aware of the objectives of the study and willingly participated. These volunteers came from various regions of the country and had different socioeconomic back grounds.

Two ml of venous blood was drawn from each individual and immediately transferred to a glass tube containing EDTA. Each sample was tested for ABO, Rh and Kell, Duffy and Kidd blood groups. The anti-sera for ABO groups were from Biotest Diagnostic, Germany, while the anti-sera for Kell, Kidd, Duffy were from Loune Laboratory Ltd., England.

Rh grouping was done according to the technique of Dacie and Lewis[10], Kidd groups, were determined by saline, albumin and Coombs method[11]

## Results

The distribution of the Kidd blood group system is show in Table 1.

In this system the most frequent group was found to be "$Jk^{a-}$" with a frequency of 91.29%. "$Jk^{b-}$" was the next most frequent being 87.74% followed by "$Jk^{b+}$" being 12.66% "$Jk^{a+}$" was the rarest group with a frequency of 8.71%.

The distribution of the Kidd blood group system according to test done by both anti-$Jk^{a}$ and anti-$Jk^{b}$ is shown in Table 2. The commonest group was $Jk^{(a-b-)}$ which was present in 80.97% individuals. The next most common group was $Jk^{(a-b+)}$ being 10.32% $Jk^{(a+b-)}$ was 6.78% while the least common group encountered was $Jk^{(a+b+)}$ which was only 1.93%.

The distribution of Kidd blood group system according to sex is shown in Table 3. The most common Kidd blood

group observed in females was $Jk^{a-}$ which was 38.38% $Jk^{b-}$ was the next most common being 35.8% $Jk^{b+}$ was the next being 6.12% while the least common was $Jk^{a+}$ being 3.22% only.

Among the males, again the $Jk^{a-}$ was the most common blood group being 52.9% $Jk^{b-}$ was next most common being 51.93% $Jk^{a+}$ was the next being 6.12% while the least common blood group was $Jk^{a+}$ being 5.48%.

The distribution of the Kidd blod group system among different sexes according to test done with both anti-$Jk^a$ and anti-$Jk^b$ is shown in Table 4. The commonest blood group in the females was found to be $Jk^{(a-b-)}$ being 33.55%. The next most common group was $Jk^{(a-b+)}$ being 4.52% $Jk^{(a+b-)}$ was 2.58, while the least common blood group was Jk(a+b+) being 0.64 % only.

Among males, the most common blood group was found to be $Jk^{(a-b-)}$ being 47.42%. The next most common group was $Jk^{(a-b+)}$ being 5.81% $Jk^{(a+b-)}$ was 4.19%, while the rarest group encountered among the males was again $Jk^{(a+b+)}$ like the females being 1.29 only.

**Discussion**

The incidence of the antigen $Jk^a$ in Caucasians, as reported by Race and Sangers[12] is shown in Table 5. In these studies out of 4,275 Caucasians tested with anti-$Jk^a$, 76.40% were $Jk^{(a+)}$ while 23.60% were $Jk^{(a-)}$ which is very different from the results of the present study. The commonest blood group observed in the present study was $Jk^{a-}$ with a frequency of 91.29%. $Jk^{b-}$ was the next commonest with a frequency of 87.74%, $Jk^{b+}$ was 2.26% and the rarest blood group in the kidd blood group system was $Jk^{a+}$ which was 8.7%.

The studies done on unselected English population[12] with anti-$Jk^a$ and anti-$Jk^b$ are shown in Table 6. From this Table, it is obvious that in one group of 275 individuals the frequency of $Jk^{(a-b+)}$ was 27.27%, that of $Jk^{(a+b+)}$ was 50.55% and that of $Jk^{(a-b+)}$ was 22.18%. In the next group of 955 individuals, 23.66% were of the group $Jk^{a+b-}$, 53.30% were of $Jk^{(a+b+)}$ and 23.04% were $Jk^{(a-b+)}$. No case of $Jk^{(a-b-)}$ was found in these individuals. Similar results have been reported by Mollison[13]. According to him about 76% of English people posses the $Jk^a$ antigen, 26% have the genotyype $Jk^a$ $JK^a$ Phenotype $Jk^{(a+b+)}$. The remaining 24% have the genotype $Jk^b$ $Jk^b$ phenotype $JK^{(a-b+)}$ However in the present study $Jk^{(a-b-)}$ was the most common and $Jk^{(a+b+)}$ the least common, which is exactly the opposite of the Caucasian figures. In the present study, $Jk^{(a-b-)}$ group was the most common being 80.97. The next most common group was $Jk^{(a-b+)}$ being 10.32.% $Jk^{(a+b-)}$ was 6.78% while the least common group encountered was $Jk^{(a+b+)}$ with a frequencies of 1.93% only.

Sussman et al. (14), studied 305 New York Negroes and 93% were found to be $Jk^{a+}$. A small series of 67 unrelated New York Negroes were tested in 1955; with both anti-$Jk^a$ and anti-$Jk^b$: In this study $Jk^{(a+b+)}$ were 57%, 34 were $Jk^{(a+b-)}$ and 9% were $JK^{(a-b+)}$ According to Wintrobe[15] about half the population has the phenotype $Jk^{(a+b+)}$, a quarter are $Jk^{a-b+}$ another quarter are $JK^{a+b-}$ but it also has a silent allele of infrequent occurrence that gives rise to the phenotype $Jk^{(a-b-)}$.

There are only two major antigens in the Kidd system: $Jk^a$ and $JK^b$. These are determined by allelic genes, each having an average frequency of blood 0.50. The major phenotypes are $Jk^{(a+b-)}$, $Jk^{(a-b+)}$, and $Jk^{(a+b+)}$. Nothing is known of their biochemical structure, but these antigens (especially Jka) are extremely important in blood transfusion. Their respective antibodies are characteristically transient and difficult to demonstrate serologically, even though they are capable of severe transfusion reactions associated with complement fixation, intravascular hemolysis, and serious renal damage. In some cases, the antibodies react in vitro only with cells from homozygotes, but heterozygote cells are destroyed rapidly after transfusion.. There would certainly be some justification for employing $Jk^a$ typing as a routine procedure in blood transfusion laboratories if sufficient antisera were available. Unfortunately. Most examples of anti- $Jk^a$ either react weakly in vitro or lose their potency after relatively brief storage.

Several recent studies have further substantiated the clinical importance of Kidd group. Several hemolytic reactions due to anti $Jk^3$ has been reported by Marshal et al in a1999 study from U.S.A[16]. A 35 year old gravida 3, para 3 Fillipino woman with negative antibody screen, no prior history of transfusion, and no hemolytic disease of the newborn in her children suffered a massive postpartum hemorrhage requiring transfusion. A severe hemolytic transfusion reaction occurred 5 days after delivery. Subsequently, a panagglutinin on a routine antibody identification panel was identified as anti-$Jk^3$. The patients red blood cell phenotype was $Jk^{(a-b-)}$ and all of her children were $Jk^{(a-b+)}$, yet the antibody that formed reacted with equal strength against all $Jk^{(a)}$ or $Jk^{(b)}$ positive cells. Nanu and Thapliyal from New Delhi, India[6] studied 6334 north Indian blood donors in 1997 for genetic frequencies and found the presence of allele $Jk^{(a-b-)}$ in 0.54% cases. In a another study conducted in Spain during 1998, Christoph et al[17] has shown that heterozygous mothers with Kidd genetic system have a reproductive disadvantage. Mothers with heterozygous loci had a higher frequency of malformed children.

Whenever a patients has a severe transfusion reaction with intravascular hemolysis not due to anti-A or anti-B, the first suspect should be anti $Jk^a$ particularly if the compatibility test is negative or only weakly positive.

Table 1 Distribution of the Kidd blood group system among 310 volunteers.

| Groups | Numbers observed | Percentage |
|---|---|---|
| $Jk^{a+}$ | 27 | 8.71 |
| $Jk^{a-}$ | 283 | 91.29 |
| Total | 310 | 100.0 |
| $Jk^{b+}$ | 38 | 12.26 |
| $Jk^{b-}$ | 272 | 87.74 |
| Total | 310 | 100.0 |

Table:2 Distribution of the Kidd blood group system according to tests done with both anti-Jk$^a$ and anti-Jk$^b$.

| Groups | Numbers observed | %age |
|---|---|---|
| Jk$^{(a+b-)}$ | 21 | 6.78 |
| Jk$^{(a-b+)}$ | 32 | 10.32 |
| Jk$^{(a+b+)}$ | 6 | 1.93 |
| Jk$^{(a-b-)}$ | 251 | 80.97 |
| Total | 310 | 100.0 |

Table:3 Distribution of the Kidd blood group system according to sex.

| Groups | Sex | Numbers observed | %age |
|---|---|---|---|
| Jk$^{a+}$ | M | 17 | 5.48 |
| | F | 10 | 3.22 |
| Jk$^{a-}$ | M | 164 | 52.9 |
| | F | 119 | 38.38 |
| Total | M+F | 310 | 100.0 |
| Jk$^{b+}$ | M | 19 | 6.12 |
| | F | 19 | 6.12 |
| Jk$^{b-}$ | M | 161 | 51.93 |
| | F | 111 | 35.8 |
| Total | M+F | 310 | 100.0 |

Table:4 Distribution of the Kidd blood group system among males and females according to test with anti-Jka and anti Jkb

| Groups | Sex | Numbers observed | %age |
|---|---|---|---|
| Jk$^{(a-b-)}$ | M | 147 | 47.42 |
| | F | 104 | 33.55 |
| Jk$^{(a+b-)}$ | M | 13 | 4.19 |
| | F | 8 | 2.58 |
| Jk$^{(a-b+)}$ | M | 18 | 5.81 |
| F | 14 | 4.52 | |
| Jk$^{(a+b+)}$ | M | 4 | 1.29 |
| | F | 2 | 0.64 |
| | Total | 310 | 100.0 |

Table:5 Incidence of the Antigen Jk$^a$ in Caucasians.

| Authors/ Country | Year | No Tested | Jk$^{a+}$ No | %age | Jk$^{a-}$ No. | %age |
|---|---|---|---|---|---|---|
| Allen et al. USA | 1951 | 189 | 146 | 77.25 | 43 | 22.75 |
| Race et al UK | 1951 | 201 | 154 | 76.62 | 47 | 23.38 |
| Sanger & Race Uk | 1653 | 343 | 253 | 73.76 | 93 | 26.24 |
| Rosenfield et al. USA | 1953 | 726 | 557 | 76.72 | 169 | 23.28 |
| Lundeval Norway | 1955 | 1,816 | 1,373 | 75.61 | 443 | 24.39 |
| Halle et al.Switzerland | 1955 | 1,000 | 783 | 73.30 | 217 | 21.70 |
| Total | | 4,275 | 3,266 | 76.40 | 1,009 | 23.60 |

Table:6 Distribution of the Kidd groups among unselected English population tested with anti-Jk$^a$ and anti-Jk$^b$

| Source/ Authors | Total | ka+b No. | %age | Jk (a+b$^+$) No. | %age | Jk (a-b+) No | %age |
|---|---|---|---|---|---|---|---|
| BGRS | 275 | 75 | 27.27 | 139 | 50.55 | 61 | 22.18 |
| Cleghorn | 955 | 226 | 23.66 | 509 | 53.30 | 220 | 23.04 |
| Total | 1,230 | 301 | 24.47 | 648 | 52.68 | 281 | 22.85 |

BGRS=Blood group Research Unit

## References


1. Myhre BA. Fatalities from blood transfusion. JAMA 1980;244:1333-1336.
2. Irshaid NM, Thureason B, Olsson ML. Genomic typing of Kidd blood group by a single tube allele specific primer PCR technique. Br J Haematol 1998. 102; 1010-1014
3. Lucien N,Sidoux-Walter F, Oliver B, Moulds J, Le Pennec PY, Cartron JP, Bailly P Characterization of the gene encoding the human Kidd blood group/urea transporter protein. Evidence for splice site mutations in Jk null individuals. J Biol Chem 1998; 273: 12-73-80.
4. Waheed A, Kendedy MS. Delayed Hemolytic transfusion reaction caused by anti Jsb in a Js(a+b+) patient. Transfusion 1982; 22:161-162.
5. Pineda A, Vamvakas EC, Gorden LD, Winters JL, Moore SB. Trends in the incidence of delayed hemolytic and delayed serologic transfusion reactions. Transfusion 1999;39:1097-103.
6. Nanu A, Thapliyal RM. Blood group gene frequency in a selected north Indian population. Indian J Med Res 1997; 106:242-6.
7. Oliver B, Mattel MG, Huet M, Neau P, Martial S, Cartron JP, Bailly 1. Kidd blood group and urea transport function of human erythrocytes are carried by the same protein. J Biol Chem 1995;270:15607.
8. Irshad NM, Henry SM, Olsson ML. Genomic characterization of the Kidd blood group gene: different basis of the JK$^{(a-b-)}$ phenotype in Polynesians and Finns. Transfusion 2000;40:69-74.
9. Shahida ARS. Frequency of Kell and ABO blood groups in a section of Lahore population. Pak J Med Res 1980;29:134-137.
10. Dacie JV, Lewsi SM. Practical hematology. Edinburgh: Churchill Livingstone, 1975.
11. Simmos A. Techincal hematology. Philadelphia: JB Lippincott, 1980; 454-460.
12. Race RR, Sanger R. Blood groups in Man. Oxford: Blackwell, 1975.
13. Mollison PL, Blood transfusion in clinical medicine. Oxford:blackwell, 1983.
14. Sussman LN, Solomon R, Grogan S. The Kidd minus-minus phenotypes in a Causcasian. Transfusion 1975;15:356-358.
15. Wintrobe MM, Lee GR, Boggs DR, et al. Clinical hematology. Philadelphia: Lea and Febiger, 1981;553-554.
16. Marshall CS, Dwyre D, Echert R, Russell L. Severe hemoloytic reaction due to anti-JK$^3$.Arch Pathol Lab Med 1999;123:949-51.
17. Christoph CG, Thumala ML, Nazer J, Armanet L Cifuentes L, Association between maternal heterozygosity for five blood groups and reproductive success. Res Med Chil 1998; 126: 27-31.