



Scientific Comment

Kidd system antigens and kidney disease[☆]



Melca Maria Oliveira Barros^{*}

Universidade Federal de São Paulo (Unifesp), São Paulo, SP, Brazil

The Kidd blood group system (ISBT009), identified in 1951, was the ninth blood group system and it is considered the ninth most clinically important. Antigens of the Kidd blood group system are expressed on type 3 glycoproteins, also known as the urea transporter B (UT-B). The JK protein is expressed on red blood cells (RBCs) and in the endothelium of the descending vasa recta and epithelial surfaces of the renal inner medulla.¹ Evidence of the function of the Kidd blood group system first came to light from the observation in 1982 that Jk (a-b-) RBCs were resistant to lysis in 2M urea in contrast to RBCs with normal Kidd antigens. RBCs having normal JK phenotypes will lyse within 30s as the urea is transported into the cells, followed by a rapid osmotic influx of water. Because of the lack of urea transport and therefore no water uptake, Jk (a-b-) cells remain intact after two minutes, which demonstrates the role of the JK protein in facilitating rapid urea transport across the RBC membrane.² The major physiologic role for urea transporters is in the urinary concentrating mechanism. However, no clinical sequela was found in two patients with the Jk null phenotype suggesting the presence of compensatory mechanisms.³

The paper by Caprioli et al.⁴ published in this issue of the *Revista Brasileira de Hematologia e Hemoterapia* investigates possible relations between the absence of Jka or Jkb antigens in patients with chronic kidney disease. This is interesting because individuals with the Jk (a-b-) phenotype had lower urine-concentrating ability, however if patients with chronic kidney disease present some difference in the Kidd phenotype, the distribution remains unknown. Moreover, the location of the JK antigens on renal cells raises fascinating questions

about the impact of the Kidd system antigens on kidney disease.

Caprioli et al.,⁴ found no difference in phenotype frequencies or the distribution of Jka and Jkb antigens when they compared patients with chronic kidney disease and controls, however the null phenotype was not observed in the groups. This suggests that the absence of Jka or Jkb does not influence the development of chronic kidney disease in these chronic kidney disease patients, although the influence of the absence of both has not been studied. Several case reports suggest a role of the Kidd System on renal graft survival in kidney transplants. Lerut et al.⁵ demonstrated in a retrospective cohort study of 370 kidney transplants that mismatch of the recipient/graft at the JK locus was associated with more interstitial inflammation than when they were matched as observed at biopsy of the transplanted kidney, although overall graft survival was not influenced.

The authors suggest that in the absence of UT-B, another transporter, such as UT-A, would not allow urea to accumulate in the renal medulla, thereby protecting against renal dysfunction, however no functional experiments were performed to affirm this conclusion. Finally, while the function of the JK protein is known, future studies would be expected to result in a more complete understanding about the impact of Kidd system antigens on kidney disease.

Conflicts of interest

The author declares no conflicts of interest.

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[☆] See paper by Caprioli et al. on pages 301–5.

^{*} Correspondence to: Departamento de Medicina, Disciplina de Hematologia, Universidade Federal de São Paulo (Unifesp), Rua Diogo de Farias, Vila Clementino, 04023-900 São Paulo, SP, Brazil.

E-mail address: melcabarros@terra.com.br

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