

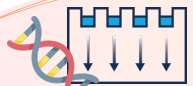
Cellular Prion Protein Alleviates Acute Kidney Injury by Interacting with TSPAN7 to Induce Formation of Migrasome



Methods

WT and Prnp^{-/-} mice


Renal I/R injury model   Bilateral renal pedicle clamping for 35 min


Prp^c in the kidney

Gene and protein expression 

Localization  Source 

Prnp^{hi}Fib and HK-2 cells


Co-culture 










H/R injury model 

Migrasome  


- Number
- Markers
- Morphology


Results


Post I/R injury 

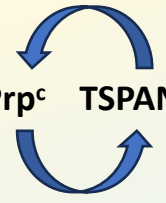
<p>WT mice</p> <p> α-SMA, Vimentin 12 hrs after I/R Prp^c expression peak at 48 hrs</p>	<p>Prnp^{-/-} mice</p> <p>  kidney damage   Damaged</p> <p>  Serum creatinine   I/R induced TSPAN7 overexpression</p>
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Transwell co-cultured


HK-2 cells  Apoptosis of HK-2 cells


Prnp^{hi}Fib  Formation of migrasome


Prp^c TSPAN7 


 Prp^c bind to TSPAN7 directly, co-located in the migrasome


TSPAN7 

 H/R induced migrasome formation

 Damage to renal tubular epithelial cells

The Prion protein 

 Formation of migrasome

 Damage to renal tubular epithelial cells

Conclusion Fibroblasts are the main source of endogenous Prp^c in the kidney. After injury, rapidly activated fibroblasts secrete Prp^c, which acts on the TSPAN7 protein in renal tubular epithelial cells, mediates the formation of migrasome, promotes the repair of renal tubular epithelial cells, and reduces AKI.

Weize Chen, Nana Song et al.
VA by Ahmed AKL
@aiakl2001