Renal fibrosis progression is halted by delayed removal of the intact kidney after unilateral renal ischemia in mice

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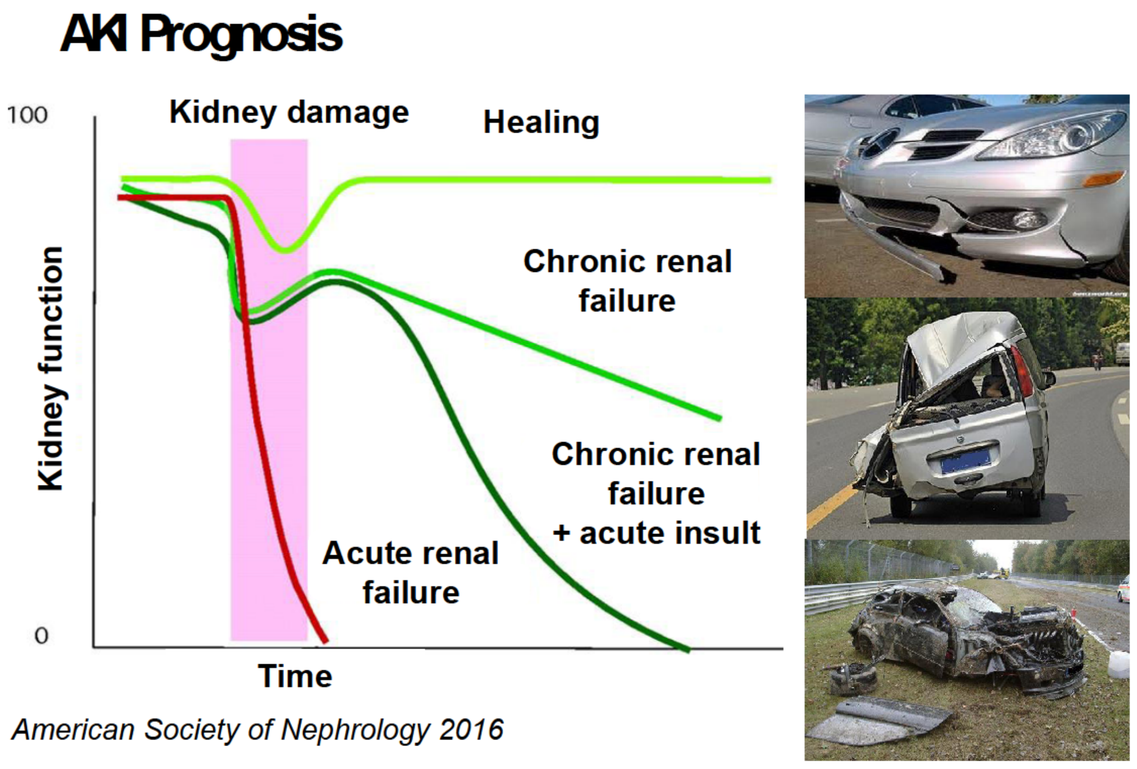
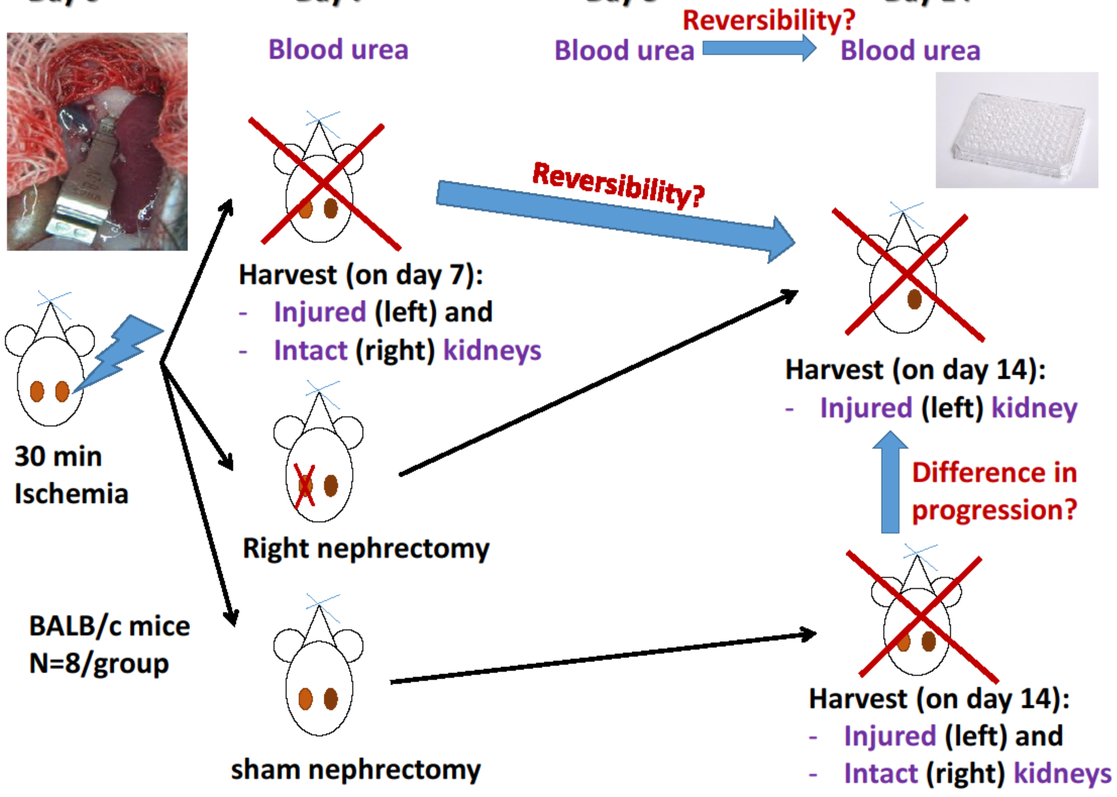
BACKGROUND

- Despite an almost full functional recovery, an ischemic insult to the kidney (acute kidney injury, AKI) initiates a slow fibrotic process



- Severe unilateral renal ischemia/reperfusion (I/R) induces renal fibrosis
- Delayed removal of the intact contralateral kidney may alter progression

METHODS



- Histology: Masson's trichrome stain
  - 10 connected HRF of renal cortex / sample were scaned for Ki67+ tubular cells
- Tubular injury: NGAL mRNA expression
- Fibrosis: FN1, TGFβ mRNA expression
- Oxidative stress: NRF2 mRNA expression
- Inflammation: TNFα mRNA expression
  - RNA from whole kidney homogenates
  - Real-time PCR after cDNA synthesis

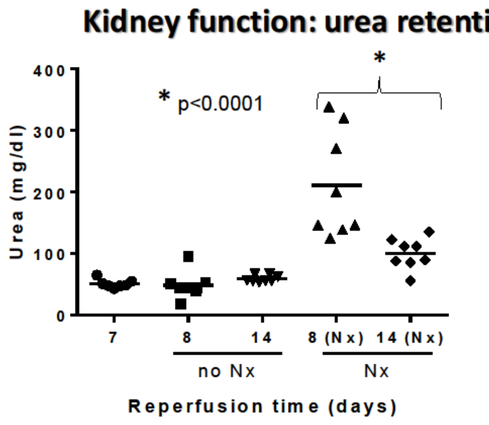
BioRad CFX96 real-time PCR

CONCLUSIONS

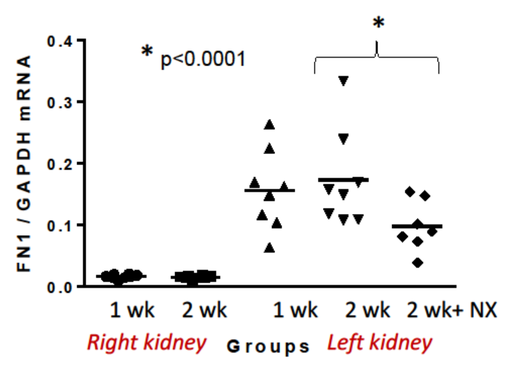
- Post ischemic kidney had a reduced function
- Function of the postischemic kidney improved significantly after removal of the intact right kidney.
- Fibrotic processes diminished (TGFb, FN1 production)
- Possible mechanism: Unilateral nephrectomy -> hemodynamic regulation, glomerular hyperfiltration and hypertrophy

RESULTS

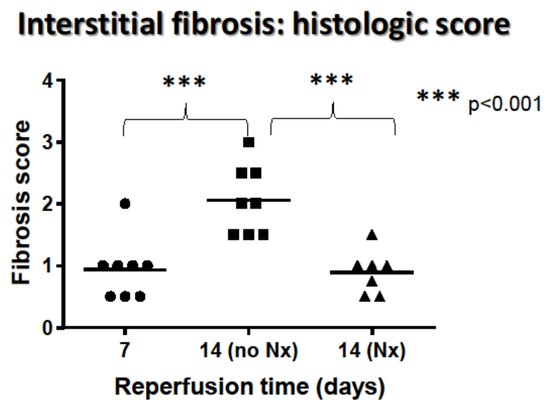
Kidney function: urea retention



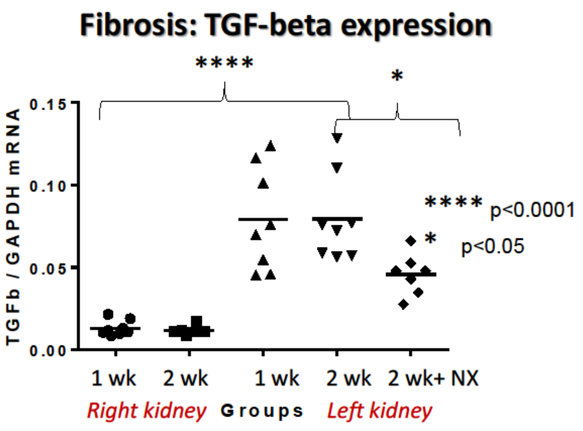
Fibrosis: Fibronectin expression



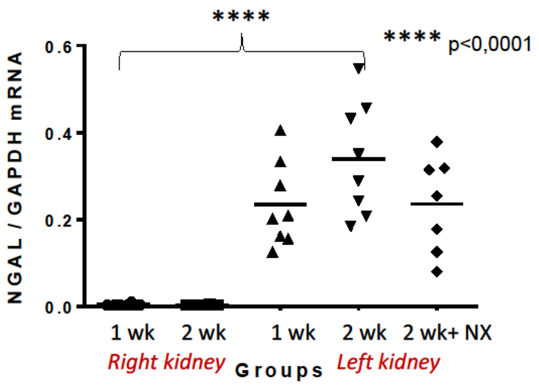
Interstitial fibrosis: histologic score



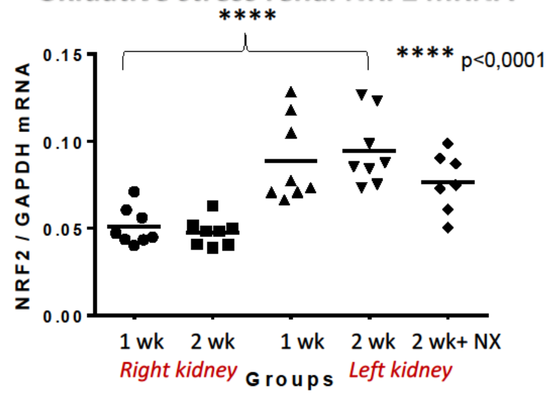
Fibrosis: TGF-beta expression



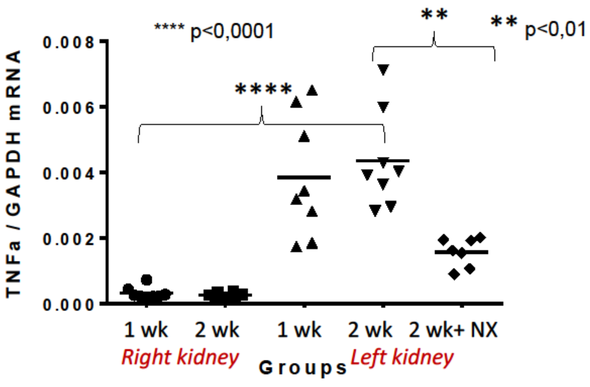
Tubular injury: renal NGAL mRNA



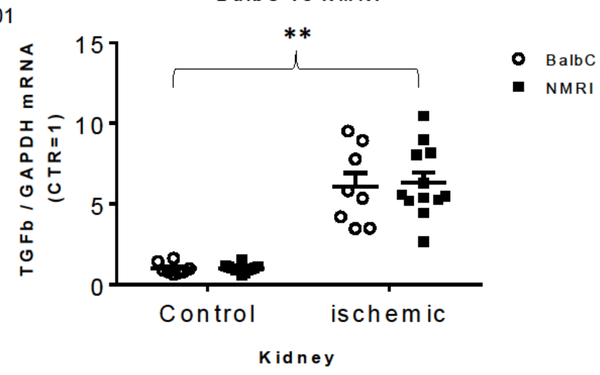
Oxidative stress renal NRF2 mRNA



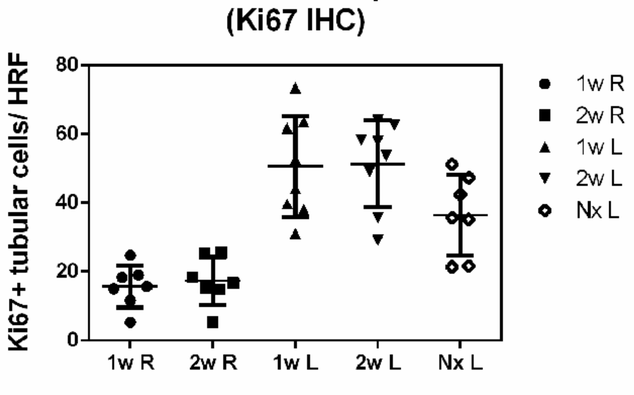
Inflammation: renal TNFa mRNA



BalbC vs NMRI



Proliferation of tubular epithelial cells (Ki67 IHC)



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