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The lncRNA profile in control and ischemically injured kidneys of old mice

Péter Hamar, Tamás Kaucsár, Pál Tod, Beáta Róka, Mária Godó, Henrietta Sárközy, Norbert Kiss, and Gábor Szénási

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Abstract

Aim

We investigated the influence of aging on the lncRNA profile after renal ischemia-reperfusion (IR) injury in old mice.

Methods

The left renal pedicle of adult (9.4±0.3 months, n=7) and old (28.5±1.2 months, n=8) C57BL/6N mice was clamped for 20 min. The right kidney was left intact. Plasma urea and urine NGAL (uNGAL) were measured prior to and 7 days after reperfusion. On day 7 tubular injury was evaluated by histology (PAS, HE) and KIM1 mRNA (qPCR). Fibrosis (FN1) and senescence (p21) was analyzed with qPCR and long non-coding RNA profile (90 lnc) with qPCR array.

Results

Older mice had higher baseline uNGAL (old: 285.7±93.8 ng/mg Crea vs. adult: 78.0±9.1 ng/mg Crea, p<0.05) and milder kidney injury (p<0.05). KIM1 (8.3x; p<0.001), FN1 (2x; p=0.05) and p21 (3.8x; p<0.05) mRNA levels were higher in their non-ischemic kidneys relative to the adult group. Baseline plasma urea levels however, were similar (old: 75.9±10.2 mg/dl vs. adult: 57.6±5.3 mg/dl). Following IR tubular injury increased in both groups (p<0.05) with KIM1 (adult: 202x, old: 19.5x, p<0.0001) and FN1 (adult: 8.5x, old: 3x, p< 0.001) mRNA compared to the contralateral kidneys. uNGAL was higher after IR compared to baseline (old: 3817±1975 ng/mg Crea, p<0.0001; adult: 2833±1201 ng/mg Crea, p<0.001). p21 mRNA increased only in the ischemic kidneys (4.5x, p<0.01), compared to the contralateral control. The plasma urea concentration increased only in the old group following 7 days of reperfusion (140.7±25.6 mg/dl, p<0.01). From the 81 measureable lncRNAs 8 increased and 1 decreased only by IR. Old age only influenced 1 lncRNA expression. Further 12 lncRNA expression was influenced by both IR and old age.

Conclusion

Our results demonstrated significant tubular damage and decreased renal function in the kidneys of old mice, in accordance with the literature. We have also found that several lncRNAs were differentially expressed in old and adult mice in both the control and ischemic kidneys.

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