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Gender difference in kidney ischemia-reperfusion injury is associated with the mitochondria quality control

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Objectives: Numbers of kidney diseases including acute kidney injury (AKI) reveal gender difference. Recent animal studies have demonstrated that the presence of male hormone, rather than the absence of female hormone, plays a critical role in high kidney susceptibility of male to AKI. However, its molecular mechanisms remain to be defined. In this study, we investigated the distribution of androgen receptor (AR) in the kidney tubules which has different vulnerability to I/R insult.

Methods: C57B6 mice were subjected to either I/R or sham-operation. AR and other protein expression were determined by immunohistochemical and western blot analysis. Mitochondrial structure was observed under transmission electron microscope (TEM).

Results: As previously reported, kidney I/R induced more severe tubular epithelial cell disruption and renal functional impairment in males than in females. Before I/R, the expression of AR was detected prominently in the distal tubules and the collecting ducts in both gender, whereas it was less detected in the proximal tubules. After I/R, those expression patterns of AR were similar to those before I/R without significant gender difference. However, the amount of AR expression in both male and female mouse kidneys decreased as time passed after I/R and this reduction of AR in males was significantly higher than that in females. When mitochondrial morphology was assessed by TEM, I/R caused severe mitochondrial disruption in both genders. This mitochondrial damage was more severe in the males than in the females. In the females, the expression of peroxisome proliferator-activated receptor γ coactivator 1-α, a transcription coactivator for mitochondrial biogenesis, was greater than males before and after I/R.

Conclusions: These results indicate that the gender difference on I/R injury is associated with the susceptibility of mitochondria to I/R and mitochondrial contents and dynamics, suggesting that tubular cell mitochondria play an important role on gender difference in AKI.