SIGNIFICANCE STATEMENT

Mitochondrial dysfunction plays an important role in the pathogenesis of acute kidney injury (AKI), but therapeutic approaches to improve mitochondrial function are still limited. microRNAs are receiving increased attention as possible targets for therapy of AKI. In the present study, in both the cisplatin-induced AKI mouse model and human AKI kidney biopsies, miR-709 was found to be upregulated in the proximal tubular cells (PTCs); expression of miR-709 was correlated with severity of kidney injury. Furthermore, inhibition of miR-709 ameliorated cisplatin-induced mitochondrial dysfunction and cell injury. Further analyses showed that TFAM (mitochondrial transcription factor A) is a target gene of miR-709. The results support a detrimental role of miR-709 in mediating cisplatin-induced AKI possibly via targeting mitochondrial gene TFAM and subsequent mitochondrial dysfunction.