FP273

AKT1 IS INVOLVED IN RENAL DAMAGE AND APOPTOSIS AFTER RENAL ISCHEMIA REPERFUSION INJURY IN MICE MODEL

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INTRODUCTION: Renal ischemia-reperfusion injury (IRI) is one of the major causes of acute kidney injury. The Akt has been reported to be involved in renal IRI. However, which one of Akt isoforms plays important role in renal IRI were not fully understood. In this study, we investigated the role of Akt1 in renal IRI.

METHODS: Fifteen male C57BL/6J mice were allocated to three groups (n = 5 per group): the sham group, the IRI group, and Akt1 knockout (KO) IRI group. Renal IRI was induced by clamping the left renal artery for 30 min followed by reperfusion. At the 48 h of reperfusion, the renal damage was assessed by histologic grading. Apoptosis was assessed by the TUNEL method and morphological criteria. Expression of cleaved caspase-3, -9, and bcl-2 was also evaluated.

RESULTS: The IRI caused an increase in kidney volume. The Akt1 KO alleviated this change. Semi-quantitative assessment of the histological lesions showed that the IRI group developed marked structural damage (tubular necrosis, hemorrhage, cast formation, and inflammation) whereas significantly less damage was observed in the Akt1 KO IRI group. The IRI group showed an increase in TUNEL-positive cells that was accompanied by morphological evidence of apoptosis. In the Akt1 KO IRI group, only a

few scattered TUNEL-positive cells were observed. The Akt1 KO IRI group showed attenuated expression of cleaved caspase-3, -9, and bcl-2 compared with IRI group.

CONCLUSIONS: Akt1 KO attenuated the renal damage and apoptosis after renal IRI in mice model. Our finding suggests that inhibition of Akt1 may provide a novel approach to prevent the renal IRI.