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Hypothermia attenuates Renal Fibrosis after Ischemia Reperfusion Injury via PGC 1 alpha

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Objectives : It has been known that hypothermia attenuates acute renal injury after ischemia reperfusion. We evaluated the hypothermic protection against renal ischemia reperfusion injury (IRI), and investigated the molecular mechanisms involved.

Methods : We used renal fibrosis model after ischemia reperfusion injury in mice. C57BL/6 mice were divided into the following groups: sham mice and ischemia reperfusion injury mice (37°C vs. 32°C). The kidneys were harvested at day 1, day 3, day 7 after IRI. HK2 cells, a human kidney proximal tubular cell-2, were incubated at 33°C and 37°C incubator during 24 h. We knocked down PGC-1 α by using siRNA in order to clarify the role of PGC-1 α in renal fibrosis.

Results : The blood urea nitrogen (BUN) levels, and serum creatinine (s-Cr) levels, and the histologic renal injury scores were significantly lower in the 32°C IRI groups, compared to the 37°C ischemia reperfusion injury groups. In hypothermic IR group, TGF- β and α -SMA were significantly decreased, while the PGC-1 α level was significantly increased. Cold preparation increased the PGC-1 α in HK2 cell. In TGF- β treated HK2 cell, cold preparation decreased α -SMA and type 4 collagen. In addition, siPGC-1 α in HK2 cells increased α -SMA and type 4 collagen in spite of cold preparation.

Conclusions : Hypothermia attenuates renal function deterioration and renal fibrosis in renal IRI mice kidneys. PGC-1 α may play a role in hypothermic protection in renal fibrosis after IRI.

Figure3Hypothermia.png

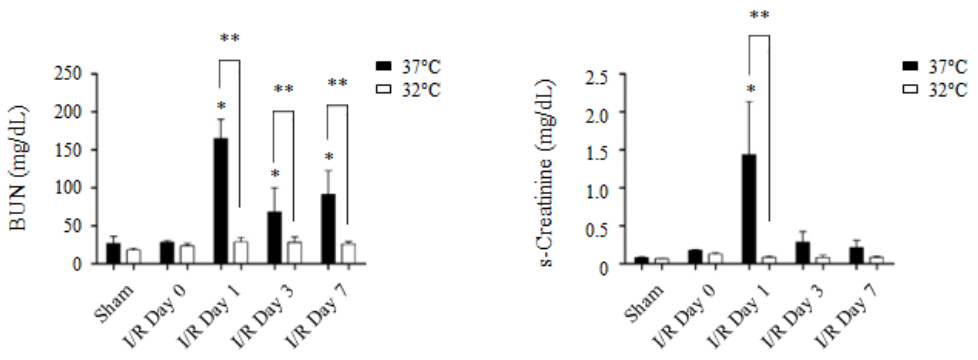


Figure3Hypothermia.png

