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Ginsenoside Rg3 attenuates ischemia reperfusion induced renal injury in mice via autophagy flux activation.

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Objectives : The beneficial role of Ginsenoside Rg3 (Rg3) have been demonstrated through different mechanisms. However, the specific evaluation of its renal-protective effect and the involvement of autophagy remain unclear. This study aims to examine how Rg3 induces autophagy flux and diminishes renal cell death in renal ischemia reperfusion injury (IRI).

Methods : C57Bl/6 mice were categorized into the subsequent groups: sham; sham treated with Rg3; IRI mice treated with saline; IRI mice treated with Rg3. Kidneys and blood samples were obtained 24 hours after the surgical procedure (sham and IR operation). Renal function, kidney histology, and the protein expression of autophagy markers were assessed.

Results : In IRI mice, the levels of BUN and s-Cr were increased, compared to sham. The Rg3 treatment decreased the BUN and s-Cr in IRI mice. In addition, Rg3 treatment decreased the renal injury score including the renal tubular cell detachment and necrosis in IRI mice. Rg3 treated IRI mice showed significantly less oxidative stress (greater amounts of superoxide dismutase (SOD), catalase, glutathione peroxidase (Gpx), compared to saline treated IR mice) and autophagy impairment (greater amounts of LC3 and Beclin-1; lower amounts of p62; and, higher levels of renal ATP6E, compared to saline treated IRI mice). Rg3 treatment also increased pAMPK in IRI mice kidney.

Conclusions : In conclusion, Rg3 has renoprotection against renal IR injury via autophagy flux.