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The role of Ginsenoside Rg3 in Ischemia Reperfusion renal Injury Mice via AMPK and autophagy.

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Objectives : The specific renal-protective mechanisms of Ginsenoside Rg3 (Rg3) have been demonstrated through various beneficial roles. However, the precise evaluation of its renal-protective effect and its involvement in autophagy are not yet clearly understood. The primary objective of this research is to investigate how Rg3 induces autophagy flux and mitigates renal cell death in the context of renal ischemia-reperfusion injury (IRI).
Methods : C57Bl/6 mice were divided into the following groups: sham, sham treated with Rg3; IRI mice treated with saline; and IRI mice treated with Rg3. Kidneys and blood samples were collected 24 hours post-surgical procedure, which included both sham and ischemia-reperfusion (IR) operations. The assessment included evaluations of renal function, kidney histology, and the protein expression of autophagy markers.
Results : In the IRI mice group, there was an observed elevation in BUN and s-Cr levels compared to the sham group. However, administration of Rg3 resulted in a significant reduction of BUN and s-Cr levels in the IRI mice. Furthermore, Rg3 treatment exhibited a mitigating effect on renal injury, evident in lower scores for renal tubular cell detachment and necrosis in comparison to the IRI mice receiving saline. The Rg3-treated IRI mice also demonstrated a reduction in oxidative stress and improvement in autophagy. This was characterized by increased levels of LC3 and Beclin-1, decreased levels of p62, and elevated levels of renal ATP6E when compared to IRI mice treated with saline. Moreover, Rg3 treatment facilitated the phosphorylation of AMPK in the kidneys of IRI mice.
Conclusions : Ginsenoside Rg3 (Rg3) induces activation AMPK-induced-autophagy flux and diminishes renal cell death in acute renal renal injury via ischemia reperfusion.