KSN 2020
FULLY VIRTUAL MEETING

Abstract Type : Poster
Abstract Submission No. : PO-1515

Rg3 attenuates renal injury in ischemia reperfusion injury of mice.

Eun Ji Kim[1], Da Bi Kim[1], Jin Young Jeong[1], Jwa-Jin Kim[2], Yoon Kyung Chang[3], Young Rok Ham[2], Dae Eun Choi[2], Ki Ryang Na[2], Kang Wook Lee[2]
[1]Department of Medical Science, Chungnam National University School of Medicine, Korea, Republic of
[2]Department of Internal Medicine-Nephrology, Chungnam National University School of Medicine, Korea, Republic of
[3]Department of Internal Medicine-Nephrology, The Catholic University of Korea, Daejeon St. Mary's Hospital, Korea, Republic of

Objectives: Ginseng is a widely used herbal product and is known to exhibit various pharmacological effects. ginsenoside Rg3 is a specific ingredient that exists only in black ginseng, not in white ginseng. Rg3 has been reported to attenuate organ injury through AMPK-mediated autophagy flux. We evaluate whether ginsenoside Rg3 regulate autophagy flux through the AMPK pathway in IR-induced kidneys.

Methods: C57Bl/6 mice were divided into the following groups: sham-operated; Rg3 sham; control IR mice; Rg3 treated IR mice. Kidneys and blood were harvested 24hr after operation of mice (sham and IR operation). Functional and molecular markers of kidney injury were evaluated.

Results: The blood urea nitrogen and serum creatinine levels and the histologic renal injury scores were significantly lower in Rg3 treated IR than Control IR kidneys (all P values < .05). Rg3 treated IR mice kidney showed the decreased oxidative stress and apoptosis, compared to control IR mice kidney, WT IR mice kidney higher amounts of LC3, Beclin1, Atg7 and p62, compared to sham mice kidney. Rg3 treated IR mice kidney showed higher amounts of LC3, Beclin1 and Atg7 and lower amounts of p62 compared to WT IR mice kidney. In addition, renal cathepsin D and ATP6E were also increased in Rg3 treated IR mice kidney compared to WT IR mice kidney.

Conclusions: Rg3 attenuates the renal injury induced by Ischemia reperfusion via enhancement of autophagic flux.