

## KSN 2017 Abstract

KSN-17-P174

Bile acid cast nephropathy associated with acute hepatitis A

\*Amita VERMA, Vikas KUMAR

Pharmaceutical Sciences, Sam Higginbotham University of Agriculture,  
Technology, India

**Objectives :** Acute kidney disease (AKI) is considered as the acute renal failure disease, is linked with the enhanced mortality and morbidity. During the renal reperfusion/ischemia damage, enhanced the renal sympathetic nerve activity, which play considerable roles in the expansion of AKI. The underlying mechanism involved in the induction of AKI is not fully clear, whereas it has been reported that various factors including reactive oxygen species, neutrophil infiltration, inflammatory mediator and phospholipase activation take part in the pathogenesis of renal injury. Several researchers recommend that the Nuclear factor erythroid 2-related factor (Nrf2) play a considerable role in the amplification of oxidative stress through protein kinase C (PKC) pathway activation. Debelolactone (furano coumarin) suggest their antioxidant potential against various diseases. In the current investigation, we scrutinized the protective effect and possible underlying mechanism of debelolactone against renal reperfusion/ischemia injury in anesthetised rats.

**Methods :** Rats were divided into 4 groups to induce the renal reperfusion/ischemia injury in anesthetised rats and after 45 min the renal tissue was removed to estimate the renal ischemia/reperfusion injury. Renal injury was scrutinized via determined the blood urea nitrogen, creatinine clearance, potassium, uric acid, sodium and identified the morphological changes. Oxidative parameters including superoxide dismutase, catalase, reduced glutathione, thiobarbituric acid reactive species and myeloperoxidase, respectively. The renal expression phosphorylated-PKC, Nrf2, HO-1, Akt and inflammatory caspase-3 were also estimated, respectively.

**Results :** The renal I/R induced AKI was scrutinized via observed the significant changes in the urine and plasma parameters, oxidative stress along with the marked histopathology changes in renal tissue. The dose dependent treatment of debelolactone reduced the acute tubular necrosis in the renal tissue histopathology. Dose dependent treatment of debelolactone altered the Nrf2, p-Akt, pro-caspase-3, HO-1 and caspase-3, respectively.

**Conclusions :** We can be concluded that debelolactone attenuates the I/R renal injury rats via PI3K/Akt/Nrf2 Signaling Pathway.

**Keywords :** debelolactone, acute kidney disease, PI3K/Akt/Nrf2 pathways