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Potential effect of Naringenin against ischemia/reperfusion induced acute kidney injury in rats via Nrf2/HO-1 signaling pathway

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Objectives: Renal ischemia/reperfusion (I/R) is well-accepted model for acute kidney injury (AKI) that alters kidney function test responsible for high morbity and mortality rates. In clinical practice AKI remains a high troublesome condition to deal with. Researchers are still clueless for exact mechanism how to decipher the role and I/R working character, however toxic effects are due to apoptosis, inflammation and oxidative stress. Naringenin flavonoid from can alter the cellular apoptosis, inflammation and oxidative. The current study comforts the potential role of naringenin on renal injury of animal via Nrf2/HO-1 signaling pathway.

Methods: Wistar rats randomly divided into 5 groups. The rats received the naringenin (2.5, 5 and 10 mg/kg) or I/R control before ischemia. Group I rats received vehicle without ischemia to serve as negative control. After the reperfusion (24 h), serum parameter and renal tissues harvested to scrutinize the renal function and histopathologic examination. Additionally, the inflammation related proteins expression, antioxidant enzymes and apoptotic molecules were also analyzed.

Results: As compared with I/R control group, the naringenin tested group significantly altered the renal function such as bloodurea nitrogen (67%), serum creatinine (54%) and renal tube architecture and reduced the inflammatory response such as IL-1β (32%), IL-6 (44.5%), TNF-α (48.2%) and altered the renal tubule apoptosis. Additionally, naringenin treated group rats showed the marked increase in the antioxidant protein expression such as Nrf2 (74%), HO-1 (70%), andNQO-1 (69%) as compared with I/R group rats.

Conclusions: Naringenin has significant potential as a therapeutic intervention to ameliorate renal injury after renal I/R via reduction of mitochondrial apoptosis pathway and nuclear factor-kB, as well as activation of nuclear factor erythroid 2erelated factor 2/heme oxygenase-1 pathway.