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Renoprotective and antioxidant enhancing effects for Cinnamaldehyde in a rat model of chronic kidney disease

Rohit Kumar, Neeraj Tiwari, Manvender Singh
Department of Pharmaceutical Sciences, Memorial Institute of Engineering and Technology, India

Objectives: More than one fifth of people over ages of 65 years have some degrees of chronic kidney disease (CKD). Cinnamaldehyde (CA) is a diterpene with a wide range of anti-inflammatory, cognitive enhancer, anticancer, antileukemic and immunomodulatory actions, anti-diabetic effects thus may be advantageous in the treatment of metabolic disease. This study was carried out to examine the effects of CA in adenine-induced tubular epithelial apoptosis, signaling pathway and renal damage in CKD rat model.

Methods: A rat model of renal damage was created by adenine. Rats in normal and vehicle groups received distilled water. Rats were given aqueous extract of CA dose (100, 200 and 500 mg/body weight and allopurinol. Proteinuria; urinary N-acetyl-β-D-glucosaminidase (NAG) levels; the blood biochemical parameters; renal histopathology damage; transferase-mediated dUTP nick-end labeling (TUNEL)-staining; the key molecular protein expressions in mitochondrial and transforming growth factor (TGF)-β1-c-JunNH2-terminal kinase (JNK) pathways were examined, respectively.

Results: Adenine administration induced severe renal damages, as indicated by the mass proteinuria, the heavy urinary NAG, and the marked histopathological injury in tubules and interstitium. This was associated with the activation of TGF-β1-JNK signaling pathway and tubular epithelial apoptosis. CA treatment, however, significantly prevented proteinuria and urinary NAG elevation, and attenuated tubular epithelial apoptosis in dose dependent manner. CA (500mg/body) is most effective to normalize renal damage and restoration. It suppressed the protein expressions of Bax and cleaved caspase-3, whereas it enhanced the protein expression of Bcl-2. Furthermore, it also suppressed the protein levels of TGF-β1 as well as phosphorylated-JNK (p-JNK).

Conclusions: It can be concluded that CAN alleviate adenine-induced tubular epithelial apoptosis and renal damage in vivo, presumably through the suppression of TGF-β1-JNK pathway activation.