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Astilbin attenuates hyperuricemia and ameliorates nephropathy in fructose-induced hyperuricemic rats.

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Abstract

Astilbin is a flavonoid compound isolated from the rhizome of Smilax china L. The effects and possible mechanisms of astilbin on hyperuricemia and nephropathy rats were elucidated in this study. Different dosages of astilbin (1.25, 2.5, and 5.0 mg/kg) were administered to 10% fructose-induced hyperuricemic rats. The results demonstrated that astilbin significantly decreased the serum uric acid (Sur) level by increasing the urinary uric acid (Uur) level and fractional excretion of urate (FEUA) but not inhibiting the xanthine oxidase (XOD) activity. In addition, kidney function parameters such as serum creatinine (Scr) and blood urea nitrogen (BUN) were recovered in astilbin-treated hyperuricemic rats. Further investigation indicated that astilbin prevented the renal damage against the expression of transforming growth factor- β1 (TGF-β1) and connective tissue growth factor (CTGF) and also exerted a renal protective role by inhibiting formation of monosodium urate (MSU) and production of prostaglandin E_2 (PGE_2) and interleukin-1 (IL-1). These findings provide potent evidence for astilbin as a safe and promising lead compound in the development of a disease-modifying drug to prevent hyperuricemia and nephropathy.