Subject 優秀論文分享

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題目：Desmodium caudatum (Thunb.) DC. extract attenuates hyperuricemia-induced renal fibrosis via modulating TGF-β1 pathway and uric acid transporters: Evidence from in vitro and in vivo studies

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摘要：

Ethnopharmacological relevance

Desmodium caudatum (Thunb.) DC., a traditional Chinese medicinal herb, has been used to treat conditions such as rheumatic back pain, diarrhea, jaundice-related hepatitis, and abscesses; it also serves as an anthelmintic. The extract of Desmodium caudatum (Thunb.) DC. (DCE) is also known for its antioxidant and anti-inflammatory properties. However, its impact on kidney fibrosis remains unclear.

Aim of the study

This study investigated whether DCE can alleviate hyperuricemia-induced kidney fibrosis by modulating the transforming growth factor-β1 (TGF-β1) pathway, activating epithelial-mesenchymal transition (EMT), and regulating uric acid transporters.

Materials and methods

NRK52E cells were exposed to uric acid (UA) followed by DCE and isovitexin (IV) for 24 h. Cell damage was assessed using an Oxidative Stress Kit, ELISA, Gelatin Zymography, and Western blotting. In parallel, adenine-induced C57BL/6 mice received DCE and IV treatment for 11 weeks. After sacrifice, renal injury was assessed through histopathological examination and protein expression analysis of fibrosis markers, EMT indicators, and uric acid transporters.

Results

DCE reduced reactive oxygen species (ROS) accumulation in uric acid-induced NRK52E cells and inhibited EMT by suppressing TGF-β1 and Slug while restoring E-cadherin expression. DCE treatment reduced fibrosis-related proteins (CTGF, collagen I, fibronectin, and α-SMA) in UA-treated cells and modulated uric acid transporters by increasing ABCG2 and OAT3 while decreasing URAT1 and GLUT9. In adenine-induced hyperuricemic C57BL/6 mice, DCE administration reduced serum uric acid levels and xanthine oxidase activity. Histological analysis showed that DCE attenuated renal fibrosis through decreased glomerular atrophy, reduced collagen deposition, and diminished α-SMA and fibronectin expression.

Conclusion

Our study demonstrates that DCE exerts protective benefits against hyperuricemia-induced renal fibrosis. The potential mechanism may involve suppressing the TGF-β1 signaling pathway and regulating the uric acid transporter, thereby mitigating kidney injury.