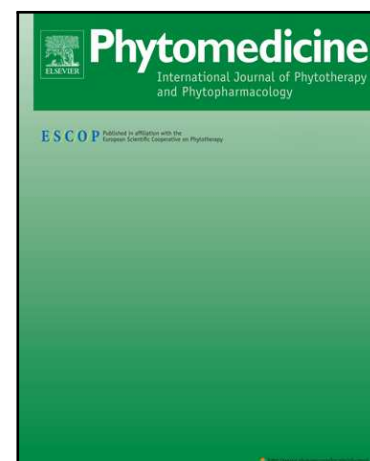


Accepted Manuscript

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PII: S0944-7113(18)30069-2
DOI: [10.1016/j.phymed.2018.03.034](https://doi.org/10.1016/j.phymed.2018.03.034)
Reference: PHYMED 52408



To appear in: *Phytomedicine*

Received date: 8 November 2017
Revised date: 31 January 2018
Accepted date: 17 March 2018

Please cite this article as: Hua Chen , Tian Yang , Min-Chang Wang , Dan-Qian Chen , Yang Yang , Ying-Yong Zhao , Novel RAS inhibitor 25-O-methylalisol F attenuates epithelial-to-mesenchymal transition and tubulo-interstitial fibrosis by selectively inhibiting TGF- β -mediated Smad3 phosphorylation, *Phytomedicine* (2018), doi: [10.1016/j.phymed.2018.03.034](https://doi.org/10.1016/j.phymed.2018.03.034)

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Novel RAS inhibitor 25-O-methylalisol F attenuates epithelial-to-mesenchymal transition and tubulo-interstitial fibrosis by selectively inhibiting TGF- β -mediated Smad3 phosphorylation

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Scientific Reports, American Journal of Nephrology, Editorial Board

Abstract

Background: Tubulo-interstitial fibrosis (TIF) is the common pathway in the chronic kidney disease. Epithelial-to-mesenchymal transition (EMT) is a major contributor to the TIF by the increased myofibroblasts. Renin-angiotensin system (RAS) is critical mediator on EMT in progressive CKD. Angiotensin II (ANG) mediates EMT and causes TIF by stimulating transforming growth factor- β 1 (TGF- β 1). RAS activation could further activate TGF- β 1. Inhibition of the RAS is one of the most powerful therapies for progressive CKD. 25-O-methylalisol F (MAF) is a new tetracyclic triterpenoid compound isolated from the *Alismatis rhizoma*, which is extensively used for anti-hypertensive, diuretic and anti-hyperlipidemic effects.

Methods: Inhibitory effect of MAF on EMT is investigated in both TGF- β 1- and ANG-induced tubular epithelial cells (NRK-52E) and fibroblasts (NRK-49F). Western blot analyses, qRT-PCR, siRNA, immunofluorescence

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