

عنوان مقاله:

Kangfuxin alleviates ulcerative colitis in rats by inhibiting NF- κ B p65 activation and regulating T lymphocyte subsets

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نویسندگان:

Miao He - *Yunnan Provincial Key Laboratory of Entomological Biopharmaceutical R&D, College of Pharmacy, Dali University, Dali, China*

Wan-xin Yu - *Yunnan Provincial Key Laboratory of Entomological Biopharmaceutical R&D, College of Pharmacy, Dali University, Dali, China*

Yongmei Shen - *Good Doctor Pharmaceutical Group, Chengduo, Sichuang, China*

Jing-na Zhang - *Yunnan Provincial Key Laboratory of Entomological Biopharmaceutical R&D, College of Pharmacy, Dali University, Dali, China*

Lian-li Ni - *Yunnan Provincial Key Laboratory of Entomological Biopharmaceutical R&D, College of Pharmacy, Dali University, Dali, China*

Yue Li - *Yunnan Provincial Key Laboratory of Entomological Biopharmaceutical R&D, College of Pharmacy, Dali University, Dali, China*

Heng Liu - *Yunnan Provincial Key Laboratory of Entomological Biopharmaceutical R&D, College of Pharmacy, Dali University, Dali, China*

Yu Zhao - *Yunnan Provincial Key Laboratory of Entomological Biopharmaceutical R&D, College of Pharmacy, Dali University, Dali, China*

Hairong Zhao - *Yunnan Provincial Key Laboratory of Entomological Biopharmaceutical R&D, College of Pharmacy, Dali University, Dali, China*

Chenggui Zhang - *Yunnan Provincial Key Laboratory of Entomological Biopharmaceutical R&D, College of Pharmacy, Dali University, Dali, China*

خلاصه مقاله:

Objective(s): Ulcerative colitis (UC) remains an enduring, idiopathic inflammatory bowel disease marked by persistent mucosal inflammation initiating from the rectum and extending in a proximal direction. An ethanol extract of *Periplaneta americana* L., namely Kangfuxin (KFX), has a significant historical presence in Traditional Chinese Medicine and has been broadly utilized in clinical practice for the treatment of injury. Here, we aimed to determine the effect of KFX on ۲,۴,۶-trinitro'benzene sulfonic acid (TNBS)-induced UC in Sprague-Dawley rats. Materials and

Methods: We established the UC model by TNBS/ethanol method. Then, the rats were subject to KFX (50, 100, 200 mg/kg/day) for 2 weeks by intragastric gavage. The body weight, disease activity index (DAI), colonic mucosal injury index (CMDI), and histopathological score were evaluated. The colonic tissue interleukin (IL)-1 β , IL-6, tumor necrosis factor- α (TNF- α), IL-10, transforming growth factor-1 (TGF- β 1), and epidermal growth factor (EGF) were determined by Elisa. To study T-lymphocyte subsets, flow cytometry was performed. In addition, the expression level of NF- κ B p65 was evaluated by immunohistochemistry and western blot analysis. Results: Compared with the TNBS-triggered colitis rats, the treatment of rats with KFX significantly increased the body weight, and decreased DAI, CMDI, and histopathological score. Also, KFX elicited a reduction in the secretion of colonic pro-inflammatory cytokines, namely IL-1 β , IL-6, and TNF- α , concomitant with up-regulation of IL-10, TGF- β 1, and EGF levels. Upon KFX treatment, the CD3⁺CD4⁺/CD3⁺CD8⁺ ratio in the spleen decreased, while the CD3⁺CD8⁺ subset and the CD3⁺CD4⁺CD25⁺/CD3⁺CD4⁺ ratio demonstrated an increase. In addition, the expression of NF- κ B p65 in the colon was decreased. Conclusion: KFX effectively suppresses TNBS-induced colitis by inhibiting the activation of NF- κ B p65 and regulating the ratio of CD4⁺/CD8⁺.

کلمات کلیدی:

Inflammation mediators, Inflammatory bowel disease, Kangfuxin, T lymphocyte subsets, Ulcerative colitis

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