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收录检索报告

委托内容：贾雪梅、高云霄、刘丽然、杜姚、杨倩发表的论文

委托机构：河北中医学院、河北省中医院

委托日期：2022 年 8 月 12 日

检索机构（盖章）：河北省科学技术情报研究院
（国家一级科技查新咨询单位）

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一、检索要求：

1、被检作者：贾雪梅、高云霄、刘丽然、杜姚、杨倩

2、委托机构：河北中医学院，河北省中医院

二、检索范围：

Science Citation Index Expanded (SCI-EXPANDED)2009-present

三、检索结果：

提供的待检论文中有 1 篇被 SCI 收录：

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Artemisinin Alleviates Intestinal Inflammation and Metabolic Disturbance in
Ulcerative Colitis Rats Induced by DSS

By: 贾雪梅 (Jia, Xuemei; 第一作者)、高云霄 (Gao, Yunxiao; 并列第一作者)、刘丽然 (Liu, Liran; 并列第一作者)、杜姚 (Du, Yao; 通讯作者)、杨倩 (Yang, Qian; 通讯作者)

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Artemisinin Alleviates Intestinal Inflammation and Metabolic Disturbance in Ulcerative Colitis Rats Induced by DSS

By: Jia, Xuemei; Gao, Yunxiao; Liu, Liran; Guo, Yuxi; Wang, Jie; Ma, Hongyu; Zhao, Runyuan; Li, Bolin; Du, Yao; Yang, Qian

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Abstract:

Objective. This study is aimed to reveal the possible mechanisms of artemisinin in the treatment of ulcerative colitis (UC) through bioinformatics analysis and experimental verification in UC model rats. **Methods.** Firstly, we searched two microarray data of the Gene Expression Omnibus (GEO) database to explore the differentially expressed genes (DEGs) between UC samples and normal samples. Then, we selected DEGs for gene ontology (GO) function enrichment analysis and Kyoto Encyclopedia of Genes and Genomes (KEGG) pathway enrichment analysis. The acute UC model of rats was established by using 3.5% dextran sulfate sodium (DSS) for 10 days to verify the core pathway. Finally, we evaluated the therapeutic effect of artemisinin at the molecular level and used metabolomics to study the endogenous metabolites in the rat serum. **Results.** We screened in the GEO database and selected two eligible microarray datasets, GSE36807 and GSE9452. We performed GO function and KEGG pathway enrichment analyses of DEGs and found that these DEGs were mainly enriched in the inflammatory response, immune response, and IL-17 and NF-kappa B signaling pathways. Finally, we verified the IL-17 signaling pathway and key cytokines, and ELISA and immunohistochemical results showed that artemisinin could downregulate the expression of proinflammatory cytokines such as IL-1 beta and IL-17 in the IL-17 signaling pathway and upregulate the expression of the anti-inflammatory cytokine PPAR-gamma. Metabolomics analysis showed that 33 differential metabolites were identified in the artemisinin group (AG) compared to the model group (MG). Differential metabolites were mainly involved in alanine, aspartate, and glutamate metabolism and synthesis and degradation of ketone bodies. **Conclusion.** In this study, we found that artemisinin can significantly inhibit the inflammatory response in UC rats and regulate metabolites and related metabolic pathways. This study provides a foundation for further research on the mechanism of artemisinin in the treatment of UC.

Keywords

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