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Short Review

A Short Review Based on the Article Entitled "Qinggan Huoxue Recipe Protects against Experimental Alcoholic Liver Fibrosis through CXCL16 Inhibition"

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This study demonstrates that Qinggan Huoxue Recipe (QGHXR), a traditional Chinese medicinal formula, is proved to protect mice from ethanol plus CCl4-induced liver fibrosis. What's more, QGHXR-induced antifibrotic effects are dependent on CXCL16 deactivation, which also suggests that CXCL16 could serve as a novel target for the treatment of liver fibrosis.

Overview of Research Background and Purpose

Heavy alcohol consumption causes a wide spectrum of direct liver injury ranging from steatosis, alcoholic hepatitis, cirrhosis and Hepatocellular Carcinoma (HCC). Globally, 25% of cirrhosis deaths in 2019 were estimated to be associated with alcohol [1]. For each region, the highest percentage of cirrhosis deaths associated with alcohol was in Europe (42%), while the lowest was in the Eastern Mediterranean region (8%) [1]. Fibrosis is thought to initiate in the perivenular area, and continued fibrosis leads to scar tissue formation that replaces liver parenchyma, leading to cirrhosis ultimately [2]. In recent years, genetic studies have identified new genes highly associated with alcoholic liver fibrosis. Many of these genes regulate key inflammatory pathways that are dysregulated in the disease [3]. CXCL16 is a chemotactic cytokine belonging to the α-chemokine subfamily, functioning as a ligand for the CXC Chemokine Receptor 6 (CXCR6) [4]. CXCL16 plays a significant role in the progression of many diseases including liver fibrosis [4]. According to the previous study, pharmacological inhibition of CXCR6-CXCL16 interactions have been suggested as an antifibrotic treatment in mice [5]. So far, the modern medicine treatment for alcoholic liver fibrosis mainly includes alcohol cessation counselling, nutritional supplementation, delaying hepatic decompensation, treatment of complications, etc., [6]. As complementary and alternative medicine, traditional Chinese medicine (TCM) is receiving arousing attention worldwide for the prevention and treatment of various diseases [7,8]. TCMs have unique advantages and great application prospects in antifibrosis due to their diverse structures, low toxicity, and wide sources [9]. Qinggan Huoxue Recipe (QGHXR), is a TCM recipe consisting of five Chinese medicine including bupleurum, scutellaria, red sage, carapax trionycis, and radix puerariae. QGHXR was confirmed to possess prominent biological activities, including anti-inflammatory, antiapoptotic, antisteatosis and antifibrotic effects in the former research [10]. Our team have conducted a series of studies, in which QGHXR was not only proved to exert antifibrotic impact in experimental research through decreasing hepatocyte apoptosis, inducing activated HSC apoptosis, regulating some certain atrix metalloproteinases, etc., but also exhibited as an effective clinical treatment of alcoholic liver disease in improving liver function and clinical symptoms [11-13]. On the basis of evidences above, this study aimed to explore the antifibrotic role of QGHXR and its underlying mechanisms further.

Research Summary and Outlook

In our study, we determined the composition of QGHXR by ultra performance liquid chromatography-tandem mass spectrometry (UPLC-MS/MS) at first. The result showed QGHXR contained 45 compounds including salvianolic acid A, scutellarin, baicalin, rutin, and chai saponin D. Then, we found that QGHXR alleviated liver injury, hepatic steatosis and hepatic fibrosis, while it downregulated CXCL16 expression in ethanol plus CCl4-induced liver fibrosis. Conversely, pharmacological CXCL16 administration ablated the QGHXR-induced protective effects in experimental alcoholic liver fibrosis. Furthermore, it was indicated that the TLR4/NF-κB pathway is responsible for the inhibition of CXCL16 by QGHXR. The above conclusions suggest that QGHXR may have a promising prospect in clinical application of treating alcoholic liver fibrosis.

Nevertheless, there are still several limitations in the present study. First, in terms of in vivo animal experiment, the dynamic model with more varieties of alcohol concentrations and intervention time can be constructed to select a relatively more appropriate and accurate animal model. Second, QGHXR contains 45 compounds as confirmed in this study, each of which hasn't been thoroughly researched. Our team will make more contributions to investigating the specific ones that play the main roles in antifibrotic process, which may be beneficial to the further pharmacological research. It also should be noted that the animal model used in this research could not fully reflect the circumstances of actual human diseases. Thus, more detailed preclinical and clinical research is expected to be focused on improvement effect and mechanism of the QGHXR on alcoholic liver fibrosis patients. Finally, the assessment of preclinical safety is rarely documented, and

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the possible side effects and toxicity of QGHXR are still uncovered. Large-scale multicentric placebo-controlled prospective studies are required to verify the results in the future.

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