

综述

TGF-β1与 IL-13在血吸虫病肝纤维化细胞信号转导中的作用

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摘要

肝纤维化是由于胶原纤维的产生和分解失衡,肝组织细胞外基质(ECM)过度沉积的结果。研究表明,转化生长因子β1(TGF-β1)、替代激活的巨噬细胞(aaM)和白细胞介素-13(IL-13)在纤维化过程中起关键作用。其中TGF-β1和IL-13为近年来的研究热点,前者通过肝星状细胞(HSC)的TGF-β1-Smads细胞信号转导通路起促进作用,而后者通过JAK-STAT6信号途径促纤维化,作用似乎更加关键。此外,替代激活的巨噬细胞为TGF-β1的重要来源,其本身又受IL-13刺激。因此,本文就IL-13、TGF-β1与血吸虫病肝纤维化细胞信号转导进行综述,以探讨血吸虫病肝纤维化的新药作用靶点。

关键词

肝纤维化 IL-13 TGF-β1 信号转导

分类号

The Role of TGFβ1 and IL-13 in Cellular Signal Transduction of Hepatic Fibrosis of Schistosomiasis

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Abstract

Liver fibrosis is characterized by an abnormal hepatic accumulation of extracellular matrix (ECM) that results from both increased deposition and reduced degradation of collagen fibres. Some studies show that transforming growth factor β1 (TGF-β1), alternatively activated macrophage (aaM) and interleukin 13 (IL-13) play a key role in the evolution of fibrosis, of which TGF-β1 and IL-13 become research hotspots. TGF-β1 mainly activates hepatic stellate cells (HSC) through TGF-β1/Smad signal pathway, while IL-13 seems to play a rather crucial role through JAK-STAT6 signal pathway. aaM is an important source of TGF-β1 and activated with IL-13. This paper reviews the role of those signaling molecules in cellular signal transduction of hepatic fibrosis of schistosomiasis japonica, and provides some targets for future drug development.

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