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基金项目: 黑龙江省自然科学基金项目(D200618); 国家自然科学基金青年基金项目(30901516)

DOI:

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摘要:

目的:研究肝细胞生长因子(HGF)能否阻抑TGF-β1诱导的大鼠内侧副韧带(MCL)成纤维细胞?琢-SMA过表达及其可能涉及的信号传导通路。方法:采用组织块培养法培养大鼠MCL成纤维细胞,培养液中加入TGF-β1(5ng/ml)及HGF(10—40 ng/ml)。培养72h后,用RT-PCR检测各组?琢-SMA mRNA及Smad3 mRNA的变化;细胞免疫组化检测?琢-SMA蛋白的表达。结果: TGF-β1能显著诱导?琢-SMA及Smad3的表达(P<0.01),而HGF则可以有效地阻抑其表达,其效应呈剂量依赖性(P<0.05)。结论:HGF可以通过下调Smad3的表达来阻抑TGF-β1诱导的?琢-SMA过表达。这为利用HGF预防和治疗MCL损伤后瘢痕及纤维化在细胞和分子水平提供了依据。

关键词: \underline{H} 细胞生长因子 转化生长因子- β 内侧副韧带 成纤维细胞 ?琢-平滑肌肌动蛋白

Hepatocyte growth factor suppresses the overproduction of α -SMA induced by TGF- β 1 in rat medial collateral ligament fibroblasts — Download Fulltext

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Abstract:

Objective: To examine the effectiveness of HGF in blocking TGF- β 1-induced ?琢-SMA production in rat medial collateral ligament (MCL) fibroblasts. Method: Fibroblasts were obtained from rat MCL. Cell culture was supplemented with 5ng/ml of TGF- β 1 along with increasing doses of HGF (10-40 ng/ml). After 72 hours incubation, the productions of ?琢-SMA and Smad3 mRNA were assayed by RT-PCR. Expression of ?琢-SMA protein was assessed by immunostaining. Result: Treatment with TGF- β 1 significantly stimulated ?琢-SMA and Smad3 mRNA production in MCL fibroblasts (P<0.01). Remarkably, the addition of HGF reduced productions of all components induced by TGF- β 1 in a dose-dependent manner (P<0.05). Conclusion: HGF antagonizes TGF- β 1 induced ?琢-SMA production in MCL fibroblasts by down regulating Smad3. The findings provide a cellular and molecular basis for HGF's acting as a therapeutic agent for MCL scar and fibrosis formation.

Keywords: hepatocyte growth factor transforming growth factor-β medial collateral ligament fibroblast ?塚-smooth muscle actin

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