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## Mechanism of Chemotactic Factor CCL5/RANTES in Diabetic Patients with Hepatic Carcinoma

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摘要

目的 观察不同浓度葡萄糖培养的人肝癌细胞系HepG2和鼠肝癌细胞系H22中趋化因子CCL5 mRNA的表达水平,并比较H22细胞系在糖尿病小鼠和正常小鼠中的成瘤性,以及探讨CCL5在糖尿病肝癌患者中的机制。方法 将人肝癌HepG2细胞和鼠肝癌H22细胞在体外不同浓度葡萄糖(5.5和25 mmol/L)中培养,通过划痕实验检测细胞迁移。分别提取HepG2和H22细胞在不同浓度葡萄糖培养后的总RNA,用RT-PCR方法检测CCL5 mRNA的表达水平。建立糖尿病小鼠模型后,比较糖尿病小鼠和正常小鼠的成瘤性,并用免疫组化检测肿瘤组织中CCL5的表达水平。结果 随着培养基中葡萄糖浓度的增加,HepG2细胞的迁移能力增强。HepG2和H22细胞在高浓度葡萄糖培养时CCL5 mRNA的表达水平均高于低浓度葡萄糖培养时。小鼠成瘤实验显示,糖尿病小鼠中H22细胞的成瘤速度明显快于正常小鼠。糖尿病小鼠肿瘤组织中CCL5的表达水平明显高于正常小鼠(P<0.05)。结论 高浓度葡萄糖培养HepG2和H22细胞可增加CCL5的表达水平,高血糖可能增加肝癌发生和加速肿瘤生长。趋化因子CCL5可能在糖尿病引起的肝癌发生和迁移中起作用。

关键词: CCL5, 趋化因子, BALB/c

### Abstract

Objective To observe the mRNA expression level of chemotactic factor CCL5 in human hepatoma cell line HepG2 and murine cell line H22 cultured with different concentrations of glucose, and to compare the tumorigenicity of H22 cell line in diabetic mice and normal mice, as well as to explore the mechanisms of CCL5 in diabetic patients with hepatic carcinoma. Methods Human hepatoma HepG2 cells and murine hepatoma H22 cells were cultured in vitro with different concentrations of glucose, 5.5 and 25 mmol/L. The migration of cells was determined by scratch assay. The total RNA of HepG2 and H22 cells cultured with different concentrations of glucose were extracted respectively. The mRNA expression level of chemotactic factor CCL5 was detected by RT-PCR method. After the mouse model of diabetes mellitus was established, we compared the tumorigenicity between diabetic mice and normal mice, and detect the expression levels of CCL5 in tumor tissues by immunohistochemistry assay. Results The migration of HepG2 cells was enhanced with the increase of glucose concentration in the medium. The mRNA expression levels of CCL5 in HepG2 and H22 cells cultured with high concentration of glucose were relatively higher than that with low concentration of glucose. The tumorigenicity speed of murine cells H22 in diabetic mice was faster than that with normal mice. The expression level of CCL5 in tumor tissues of diabetic mice was more than that of mice with normal glucose (P<0.05). Conclusion HepG2 and H22 cells cultured with high concentration of glucose could increase the expression level of CCL5. High blood glucose could cause an increased risk of hepatic carcinoma and accelerate the tumor growth. The chemotactic factor CCL5 might play a role in the growth and migration of hepatic carcinoma caused by diabetes mellitus.

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