

论文

组蛋白去乙酰化酶4和血管内皮生长因子受体-1对肝癌细胞株HepG2侵袭黏附的影响

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摘要:

目的 研究组蛋白去乙酰化酶4(histone deacetylase 4, HDAC4)和血管内皮生长因子受体-1(vascular endothelial growth factor receptor-1, VEGFR-1)在肝癌细胞株 HepG2中的表达, 观察组蛋白去乙酰化酶抑制剂(histone deacetylase inhibitors, HDACI) 苯丁酸钠(sodium phenylbutyrate, SPB)作用于肝癌细胞株 HepG2后, HDAC4、VEGFR-1之间的变化及对肝癌细胞株 HepG2侵袭、黏附作用的影响。方法 体外培养肝癌细胞株 HepG2, 分为对照组、实验组, 对照组常规培养, 实验组加入苯丁酸钠。分别以免疫细胞化学及Western-blotting 检测HDAC4、VEGFR-1蛋白的表达及蛋白表达变化; 应用Transwell小室观察SPB对肝癌细胞株HepG2侵袭力的影响, 应用MTT方法测定SPB对HepG2黏附作用的影响。结果 体外培养人肝癌细胞株HepG2, 免疫细胞化学试验及Western-blotting显示HepG2细胞株表达HDAC4 和VEGFR-1; Western-blotting显示, 实验组SPB抑制HDAC4、VEGFR-1的表达, 并呈现时间及剂量依赖关系; 应用Transwell小室观察经SPB作用后, 实验组、对照组细胞的穿膜细胞数分别为161.80±46.80、329.20±55.99, 抑制率达到50.85%, 有统计学意义(P<0.01); MTT方法测定黏附作用显示SPB实验组细胞的黏附率较对照组明显下降(P<0.01)。结论 HDAC4、VEGFR-1在肝癌细胞株HepG2中表达, SPB对HDAC4、VEGFR-1的表达有抑制作用; 并对 HepG2细胞株有抑制其侵袭及黏附的作用。

关键词: 组蛋白去乙酰化酶4; 血管内皮生长因子受体-1; 侵袭; 黏附

Effects of histone deacetylase 4 and vascular endothelial growth factor receptor-1 on invasion and adhesion in the human cancer cell line HepG2 in vitro

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Abstract:

Objective To investigate expression changes of histone deacetylases 4(HDAC4)and vascular endothelial growth factor receptor-1(VEGFR-1) in the human cancer cell line HepG2 treated with sodium phenylbutyrate(SPB), and its effects on invasion and adhesion in the human carcinoma cell line HepG2 in vitro. Methods The human carcinoma cell line HepG2 was cultured in vitro, and divided into the experimental group and the control group. Cells in the experimental group were cultured in the presence of SPB. Expressions of HDAC4 and VEGFR-1 were determined by Western blot and immunocytochemistry, and expression changes by Western blot. The effect on invasion was observed through transwell chambers, and the adherence ability of the HepG2 cell line was analyzed by MTT. Results Immunocytochemical and Western blot displayed that the cell line HepG2 expressed HDAC4 and VEGFR-1, and down-regulated expressions of HDAC4 and VEGFR-1 were observed in the experimental group by Western blot, in a time- and dose-dependent manner. Through Transwell chambers, the invasion cell number in the SPB-treated group was significantly decreased than that in the control group (161.80±46.80 vs 329.20±55.99, P<0.01). The restraining ratio was 50.85%, and the adhesion rate of HepG2 cells was more significantly lower than in the controls(P<0.01). Conclusion Human cancer cell line HepG2 can express HDAC4 and VEGFR-1, which can be inhibited by SPB. SPB takes an important part in the adhesion, invasion and metastasis of HepG2 cells.

Keywords: Histone deacetylases 4; Vascular endothelial growth factor receptor-1; Invasiveness; Adhesion

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