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第二十八屆生物醫學聯合學術年會 ABSTRACT FORM (正本)

鹼性細胞生長因子在人類軟骨肉瘤細胞增加血管內皮細胞生長因子經由 p38, c-Src, NF-κB 訊號路徑

bFGF increases VEGF expression in human chondrosarcoma cells through the p38, c-Src, NF-κB signaling pathway

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Background:

Vascular endothelial growth factor (VEGF) is a signal protein produced by cells that stimulates vasculogenesis and angiogenesis. Solid cancers cannot grow beyond a limited size without an adequate blood supply; cancers that can express VEGF are able to grow and metastasize. In normal tissue, basic fibroblast growth factor (bFGF) is present in basement membranes and in the subendothelial extracellular matrix of blood vessels. It has been found that bFGF increased the formation of new blood vessels and angiogenesis during tumor growth and metastasis. However, the effect of bFGF in VEGF expression in human chondrosarcoma is largely unknown. In this study, we examine the effect of bFGF in VEGF expression in human chondrosarcoma cells.

Materials & Methods:

The qPCR and ELISA was used to examine the mRNA expression of VEGF. The p38, c-Src, and NF-κB phosphorylation was examined by using Western blot method. A transient transfection protocol was used to examine NF-κB activity.

Results:

We found that bFGF increased mRNA and protein expression of VEGF in a time- and concentration-dependent manner in human chondrosarcoma cells by using qPCR and western blotting. Pretreatment of cells with p38, c-Src, or NF-κB inhibitor antagonized bFGF-induced production of VEGF. Incubation with bFGF induced p38, c-Src and NF-κB phosphorylation.

Conclusion:

Our results indicated that bFGF enhances the VEGF expression of chondrosarcoma cells. One of the mechanisms underlying bFGF-directed VEGF expression was through the p38/ c-Src/NF-κB signal transduction pathway.

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