

投稿學會：	收件編號：	分類編號：
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第二十八屆生物醫學聯合學術年會 ABSTRACT FORM (正本)

在人類軟骨肉瘤細胞中瘦體素增加血管內皮生長因子表現是經由 MEK, ERK, c-Jun 訊號路徑
 Leptin increases VEGF expression in human chondrosarcoma cells through the MEK, ERK, and c-Jun signaling pathways

許凱翔¹, 譚思濉^{2, 1*}, 湯智昕^{2, 1*}
 Kai-Hsiang Hsu¹, Tan Tzu-Wei^{2, 1}, Chih-Hsin Tang^{2, 1*}

¹中國醫藥大學基礎醫學研究所 ²中國醫藥大學醫學系藥理學科

¹Graduate Institute of Basic Medical Science, China Medical University, Taichung, Taiwan

²Department of Pharmacology, School of Medicine, China Medical University, Taichung, Taiwan

Background:

Vascular endothelial growth factor (VEGF) is a signal protein produced by cells that stimulates vasculogenesis and angiogenesis. Leptin, the product of the obese gene that plays an important role in the regulation of body weight that induces neuroprotection, neurogenesis, and angiogenesis. However, the effect of leptin on VEGF expression in human chondrosarcoma cells is mostly unknown. The aim of study is try to examine the effect of leptin in VEGF expression in human chondrosarcoma cells

Materials & Methods:

The qPCR was used to examine the mRNA expression of VEGF. The MEK, ERK, c-Jun phosphorylation was examined by using Western blot method. A transient transfection protocol was used to examine AP-1 activity.

Results:

We found that leptin increased the VEGF expression in human chondrosarcoma cells. Leptin-mediated VEGF up-regulation was attenuated by MEK inhibitors (PD98059 and U0126) and AP-1 inhibitors (Curcumin and Tanshinone). Incubation of cells with leptin increased MEK, ERK, and c-Jun phosphorylation as well as AP-1 luciferase activity.

Conclusion:

Our results indicate that leptin enhances the VEGF expression in chondrosarcoma cells. One of the mechanisms underlying leptin-directed VEGF expression was through the MEK, ERK, and c-Jun signal transduction pathway.

第一作者中文姓名：許凱翔

傳真：

電話：04-220521213#7727 手機：0988739078

E-mail：arlan123@yahoo.com.tw

地址：台中市北區學士路 91 號