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The Role of Matrine and Mitogen-Ativated Protein Kinase/Extracellular Signal-Regulated Kinase Signal Transduction in the Inhibition of the Proliferation and Migration of Human Umbilical Veins Endothelial Cells Induced by Lung Cancer cells

Jinchang LU, Qiang LUO, Ping CHENG, Xianjun LIU, Ming BAI, Mingli TU

摘要

Background and objective Matrine, one of the major alkaloid components of the traditional Chinese medicine Sophora roots, has a wide range of pharmacological effects including anti-inflammatory activities, growth inhibition and induction of cell differentiation and apoptosis. Motigen-activated protein kinase (MAPK)/extracellular signal-regulated kinase (ERK) has found to be a crucial signaling pathway in endothelial cells. The aim of this study is to investigate the role of Matrine and MAPK/ERK signal transduction in the inhibition of the proliferation and migration of human umbilical veins endothelial cells (HUVECs) induced by lung cancer cells. Methods HUVECs were cultured with A549CM. Mat or PD98059 (i.e PD), specific inhibitor of MAPK/ERK, was added into the A549CM. The proliferation of the HUVECs was measured by cell counting. The migration of the HUVECs was observed by wound healing assay. The expression levels of ERK and p-ERK protein were detected by Western Blot analysis. Results On 24 hours after intervention, the A549CM significantly stimulated the proliferation, migration and expression of p-ERK of HUVECs. Compared with the A549CM group, Mat significantly inhibited the proliferation, migration and p-ERK expression of HUVECs induced by A549CM. While PD only decreased the proliferation and p-ERK expression of HUVECs induced by A549CM. PD had no effect in the migration of HUVECs. Conclusion The results demonstrated that Mat and PD98059 can effectively decrease proliferation and expression of p-ERK of HUVECs induced by A549CM. Furthermore Mat can also inhibit migration of HUVECs induced by A549CM that did not changed by PD98059. These data implied that suppressing MAPK/ERK signal transduction may play the crucial role in resisting lung cacinoma angiogenesis with Mat.

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Jinchang LU

Qiang LUO

Ping CHENG

Xianjun LIU

Ming BAI

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