

A biotherapy strategy for esophageal cancer in the future

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A research team from China focused on esophageal squamous cell carcinoma (ESCC) and characterized sphingosine 1-phosphate (S1P) receptor expression pattern and investigated the role of S1P receptors on ESCC cells proliferation and migration. Their results showed that ESCC cells may down-regulate the expression of S1P5 to promote proliferation and escape S1P-S1P5 induced migration inhibition.

Esophageal cancer is one of the most common malignancies worldwide. Its mortality is very high due to relatively late diagnosis and inefficient treatment. The ability to reverse the outcome of esophageal cancer is limited due to a poor understanding of its biology. Progression of esophageal cancer may be associated with sphingosine 1-phosphate (S1P) and its receptors S1P1-5, which play an important role in other cancers. A possible role for S1P and its receptors in human esophageal cells has not previously been investigated, nor has the importance of S1P and its receptors in esophageal cancer growth and metastasis been addressed.

Using semi-quantitative reverse transcription polymerase chain reaction, gene transfection, MTT assay and transwell migration assay, a research team from China investigated S1P receptor expression profile in human esophageal <u>cancer cells</u> and the effects of S1P5 on proliferation and migration. Their study will be published on April 21, 2010 in the <u>World Journal of Gastroenterology</u>.

They found S1P binding to S1P5 inhibits the proliferation and migration



of S1P5-transfected Eca109 cells.

Their results indicated that deficiency in inhibitory effect of S1P-S1P5 may be of importance in the growth and metastasis of esophageal cancer. S1P5 or its associated signaling molecules may serve as a future strategy in biotherapy for <u>esophageal cancer</u>.

More information: Hu WM, Li L, Jing BQ, Zhao YS, Wang CL, Feng L, Xie YE. Effect of S1P5 on proliferation and migration of human esophageal cancer cells. World J Gastroenterol 2010; 16(15):1859-1866. www.wignet.com/1007-9327/full/v16/i15/1859.htm

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