Emodin. Propriedades antitumorais e apoptóticas do emodim, antraquinona derivada do Rheum officinale Baill contra o câncer de pâncreas via inibição da ativação do Akt


Rheum officinale


Antitumor and apoptosis-promoting properties of emodin, an anthraquinone derivative from Rheum officinale Baill, against pancreatic cancer in mice via inhibition of Akt activation.

Wei WT, Chen H, Ni ZL, Liu HB, Tong HF, Fan L, Liu A, Qiu MX, Liu DL, Guo HC, Wang ZH, Lin SZ.
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Source

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Abstract

Pancreatic adenocarcinoma is one of the most common malignancies worldwide. Gemcitabine is currently the standard first-line chemotherapeutic agent for pancreatic cancer. However, gemcitabine can induce activation of Akt and nuclear factor-κB (NF-κB), which is associated with its chemoresistance. It has been reported that gemcitabine combination therapies result in improved survival outcomes in pancreatic cancer. Therefore, agents that can either enhance the effects of gemcitabine or overcome chemoresistance to the drug are needed for the treatment of pancreatic cancer. Emodin is an active component of Chinese medicinal herbs and can inhibit the activation of Akt and NF-κB. In this study, we investigated whether emodin could enhance the anticancer

effect of gemcitabine on pancreatic cancer in vivo. We demonstrated that treatment of gemcitabine combined with emodin efficiently suppressed tumor growth in mice inoculated with pancreatic tumor cells. This treatment paradigm promoted apoptotic cell death and mitochondrial fragmentation. Furthermore, it reduced phosphorylated-Akt (p-Akt) level, NF-κB activation and Bcl-2/Bax ratio, increased caspase-9 and -3 activation, Cytochrome C (CytC) release occurred in combination therapy. Collectively, emodin enhanced the activity of gemcitabine in tumor growth suppression via inhibition of Akt and NF-κB activation, thus promoting the mitochondrial-dependent apoptotic pathway. Therefore, our findings may provide new insights into understanding the pharmacological regulation of emodin on gemcitabine-mediated proapoptosis in pancreatic cancer and may aid in the design of new therapeutic strategies for the intervention of human pancreatic cancers.

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