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新藤黄酸对**HepG2**裸小鼠移植瘤的抗肿瘤作用与**MAPK**信号转导通路的关系

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中文摘要:目的:研究新藤黄酸(neo-gambogicacid,NGA)对HepG2裸小鼠移植瘤的体内抗肿瘤作用及其机制。方法:采用HepG2裸小鼠移植瘤模型,造模7d后将模型动物随机分为5组,即荷瘤对照组,阳性对照组(5-FU10.0mg·kg^{-1}),NGA高、中、低剂量组(分别为8.0,4.0,2.0mg·kg^{-1}),隔天ip给药1次,给药7次后处死动物,分离肿瘤,测量肿瘤体积,称瘤重,计算相对肿瘤体积和抑瘤率,并将肿瘤组织切片,采用免疫组化法,检测实体瘤组织的Bax,Bcl-2,p-ERK1/2,p-MEK1/2蛋白的表达情况。结果:NGA高、中、低剂量组的平均瘤重均明显低于荷瘤对照组(1.29±0.24)g,NGA高剂量组的抑瘤率为83.75%,高于阳性对照药5-FU的抑瘤率46.54%(P<0.05);Bax表达量呈剂量依赖性升高,Bcl-2表达量呈剂量依赖性降低;NGA给药组p-ERKl/2和p-MEKl/2的表达量均明显低于荷瘤对照组(P<0.05)。结论:NGA对HepG2裸小鼠移植瘤具有确切的体内抗肿瘤作用,其作用机制与上调Bax/Bcl-2比值而诱导实体瘤细胞凋亡及下调丝裂原活化蛋白激酶(mitogen-activatedproteinkinase,MAPK)信号转导通路的磷酸化水平密切相关。

中文关键词:新藤黄酸 抗肿瘤 裸小鼠 凋亡 丝裂原活化蛋白激酶

Anti-tumor Effects of Neo-gambogic Acid on Hep G2 Xenografts in Nude Mice Correlated with MAPK Signal Transduction Pathway

Abstract:Objective: To study the anti-tumor effect of neo-gambogic acid(NGA)on Hep G2 xenograft in nude mice *in vivo* and its mechanism. **Method:**Sixty healthy BALB/c-nu mice, transplanted by Hep G2 hepatocarcinoma cells, were divided into 5 groups, including model group, 5-FU (10.0 mg·kg^{-1}) group and three groups of neo-gambogic acid in high dose, middle dose and low dose(8.0, 4.0, 2.0 mg·kg^{-1}). The mice were executed after fourteen days of treatment every other day (*qod*) and tumor tissues were excised. The size of tumor tissues was measured and tumor inhibition rate was calculated. Immunohistochemistry was used to detect the expressions of Bax, Bcl-2, p-ERK1/2 and p-MEK1/2. **Result:**the tumor weight of NGA groups was reduced remarkably compared with control group. The tumor inhibition rate of NGA high-dose (8.0 mg·kg^{-1}) group was 83.75%, which excelled the tumor inhibition rate of the 5-FU (10.0 mg·kg^{-1}) group (46.54%, P<0.05). The expression of Bax was up-regulated by NGA dose dependently while the expression of Bcl-2 was down-regulated. The expressions of p-ERKl/2 and p-MEKl/2 in NGA groups were reduced remarkably compared with control group (P<0.05). **Conclusion:**NGA exerts exact anti-tumor effect on Hep G2 xenograft in nude mice *in vivo*. Its mechanism correlates with that the cells in Hep G2 xenograft are induced apoptosis via up-regulating the Bax/Bcl-2 ratio and that the phosphorylation level of mitogen-activated protein kinase (MAPK) signal transduction pathway is down-regulated.

keywords:neo-gambogic acid anti-tumor nude mice apoptosis MAPK

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