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EGCG对人肝癌细胞的抑制作用及其可能的机制 点此下载全文

张勇 沈筱芸 冯雁 谢裕安 张力图 利基林 罗小玲

广西医科大学附属肿瘤医院 实验研究部，广西 南宁 530021;广西医科大学附属肿瘤医院 实验研究部，广西 南宁 530021;广西医科大学附属肿瘤医院 实验研究部，广西 南宁 530021;广西医科大学附属肿瘤医院 实验研究部，广西 南宁 530021;广西医科大学附属肿瘤医院 实验研究部，广西 南宁 530021;广西医科大学附属肿瘤医院 实验研究部，广西 南宁 530021

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摘要：

目的：观察表没食子儿茶素没食子酸酯\[(-)-epigallocatechin-3-gallate， EGCG\]对体外培养的人肝癌细胞株生物学特性的影响，研究其作用效果与血红素氧合酶-1（hemeoxygenase-1， HO-1）及相关信号分子的关系，探讨其作用机制。方法：利用MTT法检测EGCG对HepG2、Sk-hep1、SMMC7721等肝癌细胞增殖的影响，并用吖啶橙/溴化乙锭（AO/EB）双染法观察肝癌细胞的形态学变化，流式细胞术检测EGCG作用后Sk-hep1细胞周期的变化，Real-time PCR和Western blotting法检测EGCG作用后Sk-hep1细胞中HO-1、IL-10及TNF-α等信号分子表达的变化。结果：EGCG作用后，3株肝癌细胞贴壁细胞数量显著少于对照组，凋亡细胞数增多\[HepG2:（16.33±3.51） vs （3.67±1.15)个， P <0.01），Sk-hep1:（18.33±2.31） vs （2.33±2 08)个， P <0.01），SMMC7721:（15.33±3.06） vs （3.33±2.08)个， P <0.01）\]。实验组Sk-hep1细胞 G 2/M期比例明显高于对照组\[（ 34.33±8.09）% vs （3.07±2.32）%， P <0.01\]。设对照组基准值为1.00，实验组Sk-hep1细胞中HO-1、IL-10、及TNF-α的mRNA相对表达水平依次为（0.58±0.15）、（5.91±1.11）、（5.29±1.14），差别均有统计学意义(P <0 01）；与对照组相比，实验组HO-1蛋白表达水平明显下调（0.16±0.04 vs 0.33±0.08， P <0.05），IL-10（0.42±0.06 vs 0.24±0.08， P =0 034， P <0.05）和TNF-α蛋白（0.95±0.17 vs 0.58±0 08， P <0.05）表达水平明显上调。结论：EGCG可抑制肝癌细胞增殖及诱导细胞凋亡，并将Sk-hep1细胞阻滞在G 2/M期，其机制可能与HO-1、IL-10、TNF-α等炎症信号分子表达的变化有关。

关键词：没食子儿茶素没食子酸酯 肝癌细胞 血红素氧合酶-1

Epigallocatechin-3-gallate-induced growth inhibition and the underlying mechanisms in human hepatocellular carcinoma cells Download Fulltext

Zhang Yong Shen Xiaoyun Feng Yan Xie Yu' an Zhang Litu Li Jilin Luo Xiaoling

Department of Research, Affiliated Cancer Hospital of Guangxi Medical University, Nanning 530021, Guangxi, China;Department of Research, Affiliated Cancer Hospital of Guangxi Medical University, Nanning 530021, Guangxi, China;Department of Research, Affiliated Cancer Hospital of Guangxi Medical University, Nanning 530021, Guangxi, China;Department of Research, Affiliated Cancer Hospital of Guangxi Medical University, Nanning 530021, Guangxi, China;Department of Research, Affiliated Cancer Hospital of Guangxi Medical University, Nanning 530021, Guangxi, China;Department of Research, Affiliated Cancer Hospital of Guangxi Medical University, Nanning 530021, Guangxi, China

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Abstract:

Objective :To investigate the effect of Epigallocatechin-3-gallate (EGCG) on hepatocellular carcinoma cell growth and the molecular mechanisms underlying the effect in vitro . Methods: Three human hepatocellular carcinoma cell lines (i.e., HepG2, Sk-hep1 and SMMC7721) were used in this study. Cells were cultured in the presence of 0, 40, 80 or 120 μg/ml EGCG. At 24, 48 and 72 h after EGCG treatment, cell viability was assessed by MTT assay, apoptosis by AO/EB staining, cell cycle progression by flow cytometer, and mRNA and protein levels of HO11, IL-10 and TNF-α by Realtime PCR and Western blotting respectively. Results: EGCG treatment significantly induced cell attachment (P <0 05), increased the proportion of apoptotic cells (P <0.01), and induced G 2/M arrest (P <0.01) in all three cell lines tested as compared with the control. HO-1, IL-10 and TNF-α mRNA levels were 0.58±0.15, 5.91±1.11 and 5 29±1.14 in EGCG-treated Sk-hep1 cells, significantly different from the levels in the control cells (P =0.008, P =0 002, P =0.003). EGCG resulted in a significant decrease in HO-1 protein content as compared with the control (0.16±0.04 vs 0.33±0.08, P <0.05). In contrast, EGCG significantly increased levels of IL-10 protein (0.42± 0 06 vs 0.24±0.08, P <0.05) and TNF-α protein (0.95±0.17 vs 0.58± 0.08, P <0.05). Conclusions: EGCG may inhibit proliferation and block cell cycle progression and induce apoptosis in hepatocellular carcinoma cells. The mechanism(s) underlying these effects of EGCG may involve modulation of HO-1, IL-10 and TNF-α expression.

Keywords:(-)-epigallocatechin-3-gallate hepatocellular carcinoma hemeoxygenase-1(HO-1)

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