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甜橙黄酮对人胃癌AGS细胞增殖和凋亡的作用及其机制研究

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作者中文名	作者英文名	单位中文名	单位英文名	E-Mail
董杨	DONG Yang	上海中医药大学 中药研究所 上海市复方中药重点实验室,上海 201203	Institute of Chinese Materia Medica, Shanghai Key Laboratory of Complex Prescription, Shanghai University of Traditional Chinese Medicine, Shanghai 201203, China	
季光	Ji Guang	上海中医药大学 脾胃病研究所,上海 200032	Institute of Digestive Diseases, Shanghai University of Traditional Chinese Medicine, Shanghai 200032, China	
曹爱丽	CAO Aili	上海中医药大学 中药研究所 上海市复方中药重点实验室,上海 201203	Institute of Chinese Materia Medica, Shanghai Key Laboratory of Complex Prescription, Shanghai University of Traditional Chinese Medicine, Shanghai 201203, China	
施建群	SHI Jianrong	上海中医药大学 教学实验中心,上海201203	Experimental Teaching Centre, Shanghai University of Traditional Chinese Medicine, Shanghai 201203, China	
石海燕	SHI Haiyan	上海中医药大学 中药研究所 上海市复方中药重点实验室,上海 201203	Institute of Chinese Materia Medica, Shanghai Key Laboratory of Complex Prescription, Shanghai University of Traditional Chinese Medicine, Shanghai 201203, China	
谢建群	XIE Jianqun	上海中医药大学 脾胃病研究所,上海 200032	Institute of Digestive Diseases, Shanghai University of Traditional Chinese Medicine, Shanghai 200032, China	wdazheng@hotmail.com
吴大正	WU Dazheng	上海中医药大学 中药研究所 上海市复方中药重点实验室,上海 201203	Institute of Chinese Materia Medica, Shanghai Key Laboratory of Complex Prescription, Shanghai University of Traditional Chinese Medicine, Shanghai 201203, China	wdazheng@hotmail.com

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中文摘要:目的:探讨甜橙黄酮对人胃癌AGS细胞增殖和凋亡的作用及其机制。方法:采用MTT法观察甜橙黄酮对AGS细胞生长的抑制作用;流式细胞仪检测甜橙黄酮对细胞周期的影响;Annexin V-FITC/PI标记定量检测细胞凋亡率;DNA片段化分析进一步验证甜橙黄酮对细胞凋亡的作用;Hoechst 33342染色后倒置荧光显微镜观察凋亡细胞形态;Western blot检测对p21和p53蛋白表达的影响。结果:甜橙黄酮可明显抑制AGS细胞的增殖,呈时间和剂量依赖性;甜橙黄酮可阻滞细胞于G₂/M期并增加AGS细胞凋亡率,并随剂量增大作用增强;60 μmol·L⁻¹甜橙黄酮作用48 h产生凋亡特有的梯形条带;Hoechst 33342染色观察到甜橙黄酮给药组出现细胞核浓缩、边缘化以及凋亡小体等细胞凋亡的形态特征;甜橙黄酮可呈剂量依赖地增强p21和p53蛋白表达。结论:甜橙黄酮可显著抑制人AGS胃癌细胞的增殖,使细胞阻滞于G₂/M期,并可诱导细胞凋亡,其机制可能与上调p21和p53蛋白有关。

中文关键词:甜橙黄酮 胃癌 增殖 凋亡

Effects of sinensetin on proliferation and apoptosis of human gastric cancer AGS cells

Abstract:Objective: To study the effects and mechanisms of sinensetin on proliferation and apoptosis of human AGS gastric cancer cells. Method: MTT assay was used to detect the growth inhibition rates of human AGS gastric cancer cells treated with sinensetin in different concentrations and times. The cell cycle distribution was measured by flow cytometry. The apoptosis was examined by Annexin-FITC/PI staining and DNA fragment analysis. The apoptosis morphology was observed by inverted fluorescence microscope after Hoechst 33342 staining. The protein expressions of p21 and p53 were detected by western blot. Result: MTT assay showed that sinensetin inhibited the growth of AGS gastric cancer cells in a dose- and time-dependent manner. Sinensetin blocked AGS cells in G₂/M and increased the apoptosis rates of AGS cells in a dose-dependent manner. DNA ladder was observed in cells treated with 60 μmol·L⁻¹ sinensetin for 48 h. The typical apoptotic morphological changes including cell nucleus shrinkage, chromatin condensation and apoptotic bodies were observed when treated with different dose of sinensetin. Western blot showed that sinensetin increased expressions of p53 and p21 in a dose-dependent manner. Conclusion: Sinensetin could inhibit human AGS gastric cancer cells proliferation and induce cell cycle block in G₂/M phase and apoptosis. The up regulation of p53 and p21 protein might be one of the mechanisms.

keywords: sinensetin gastric cancer proliferation apoptosis

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