肿瘤防治研究
ZHONGLIU FANGZHI YANJIU
Cancer Research on Prevention and Treatment
中华人民共和国卫生部主管
中国抗癌协会系列杂志

肿瘤防治研究 2012, Vol. 39 Issue (1): 23-27 DOI: 10.3971/j.issn.1000-8578.2012.01.006
基础研究

香加皮三萜类化合物对实验性大鼠食管癌的阻断作用及机制

王丽芳1,卢安2,孟凡茹1,曹青1,纪昕1,单保恩3

1.050000 石家庄，河北医科大学第二医院检验科；2.河北省疾病预防控制中心；3.河北医科大学第四医院科研中心

Inhibitory Effects of Triterpenes Compound of Cortex Periplocae on N-nitrosomethyl-benzylamine- induced Rat Esophageal Tumorigenesis

Wang Lifang[1],Lu An[2],Meng Fanru[1],Cao Qing[1],Ji Xin[1],Shan Baoen[3]

1.Laboratory Department,The Second Hospital of Hebei Medical University,Shijiazhuang 050000,China;2.Research Center,Hebei Province Center for Disease Prevention and Control;3.Research Center,The Fourth Hospital of Hebei Medical University

- 摘要
- 参考文献
- 相关文章

全文: PDF (1775 KB) HTML (0 KB) 输出: BibTeX | EndNote (RIS) 背景资料

摘要 目的

探讨中药香加皮提取物三萜类化合物（TCCP）对甲基苄基亚硝胺（NMBA）诱导F344大鼠食管癌前病变形成的阻断作用及其机制。方法健康雄性F344大鼠90只，随机分为模型组、TCCP干预组、大豆油对照组和正常对照组。模型组大鼠皮下注射0.5 mg/kg NMBA，TCCP干预组大鼠同时给予0.5 mg/kg NMBA皮下注射及香加皮三萜类化合物20 mg/kg肌肉注每周给药3次，连续5周；大豆油对照组大鼠肌注大豆油1 ml/kg，正常对照组大鼠常规饲养。分别在给药后第9、15和25周，麻醉后解剖大鼠，HE染色后光镜下观察食管上皮组织病理学变化；用Western blot法检测GSK-3β和β-catenin蛋白表达水平；用RT-PCR法检测c-myc mRNA表达水平。结果(1)正常对照组及大豆油对照组在整个实验过程中未发现食管异常变化，模型组大鼠随诱癌时间延长食管病变逐渐加重。TCCP干预可缓解食管上皮的病变。(2)正常大鼠食管上皮中有少量β-catenin蛋白表达，模型组大鼠食管上皮β-catenin蛋白表达显著增强（P<0.05）；正常大鼠食管上皮GSK-3β的蛋白表达丰富，随着诱癌时间延长表达水平逐渐降低；与模型组相比，在诱癌9、15和25周时TCCP干预组大鼠食管上皮组织中β-catenin蛋白表达水平均显著下降（P<0.05）；而GSK3β蛋白表达显著升高（P<0.05）。(3)与正常对照组相比，各时间点模型组大鼠食管上皮c-myc mRNA表达水平均显著升高。与模型组相比，在诱癌9、15周时TCCP干预组大鼠食管上皮组织中c-myc mRNA表达水平均显著下降，而诱癌25周时差异无统计学意义（P>0.05）。结论TCCP可能通过促进Wnt信号分子GSK-3β的表达，抑制β-catenin蛋白的表达，进而下调下游靶基因c-myc的转录，干扰细胞周期转化，逆转细胞的分化，可能是其抑制食管癌变细胞生长机制之一。

关键词： 食管癌 甲基苄基亚硝胺 香加皮三萜类化合物 β-catenin

Abstract： Objective

To investigate the inhibitory effects of triterpenes compound of cortex periplocae (TCCP) on N-nitrosomethylbenzylamine (NMBA) -induced esophageal tumorigenesis in F344 rats and its mechanism.Methods Ninety male F344 rats (5~6 weeks of age) were randomly divided into four groups:rats were treated with 0.5 mg/kg NMBA only as model group and rats received NMBA 0.5 mg/kg s.c.plus TCCP 20 mg/kg i.m.as TCCP treatment groups and those treated with soya oil 1 ml/kg intramuscularily(i.m.) acted as soya oil control and normal control groups.The administration of drugs was scheduled as below:three times a week for 5 weeks.At 9th,15th and 25th week,5 rats from normal group and soya oil control group and 10 rats from model group and TCCP treatment group were euthanized by pentobarbital sodium and subjected to gross necropsy,respectively.The esophagus of each rat was excised and opened longitudinally.Then esophagus were fixed in 10% phosphate-buffer formalin solution,and routinely embedded in paraffin for HE staining to observe the pathological changes of esophageal tissues,while the expression of c-myc mRNA was detected by RT-PCR.Western blot was used to investigate the protein expression of GSK-3β and β-catenin in the esophageal epithelium.ResultsThere were no abnormal change in normal and soya oil groups among bioassay.It was observed that the expression of β-catenin was slightly present in normal mucosa of esophagi of the

服务
把本文推荐给朋友
加入我的书架
加入引用管理器
E-mail Alert
RSS
作者相关文章
王丽芳
卢安
孟凡茹
曹青
纪昕
单保恩

rats which were untreated with NMBA,and then was significantly increased by administering NMBA from week 9 to 25 (P<0.05).The up-regulated β-catenin expression was decreased significantly by TCCP treatment at each check-point (P<0.05).The expression of GSK-3β was rich in normal mucosa of esophagus,but was decreased significantly by administering NMBA,and was lowest at 25th week (P<0.05).The decreased protein level of GSK3β was significantly elevated by TCCP treatment at each check-point (P<0.05).The gene expression of c-myc of esophageal epithelium in NMBA control group was significantly increased at 9th,15th,25th week compared with normal control (P<0.05).TCCP suppressed the mRNA expression of c-myc at both 9th and 15th week (P<0.05),but not at 25th week.ConclusionTCCP inhibited NMBA-induced rat esophageal carcinogenesis probably via activating GSK-3β expression and suppressing β-catenin and c-myc expression.

Key words： Esophageal neoplasms N-nitrosomethylbenzylamine Triterpenes compound of cortex periplocae β-catenin

收稿日期: 2011-04-28;

引用本文:

王丽芳,卢安,孟凡茹等. 香加皮三萜类化合物对实验性大鼠食管癌的阻断作用及机制[J]. 肿瘤防治研究, 2012, 39(1): 23-27.

Wang Lifang,Lu An,Meng Fanru et al. Inhibitory Effects of Triterpenes Compound of Cortex Periplocae on N-nitrosomethyl-benzylamine- induced Rat Esophageal Tumorigenesis [J]. CHINA RESEARCH ON PREVENTION AND TREATMENT, 2012, 39(1): 23-27.

没有本文参考文献

[1] 刘丽华;;孟君;张璁;段玉青;王士杰;单保恩 . 运用MALDI-TOF MS方法建立食管癌患者血清蛋白指纹图谱诊断模型[J]. 肿瘤防治研究, 2012, 39(2): 169-172.

[2] 侯向生;万清廉;郑慧禹;常国涛 . 大网膜环包术预防食管胃吻合口瘘临床应用体会[J]. 肿瘤防治研究, 2012, 39(1): 118-119.

[3] 陈正言. 食管黏膜癌变过程中组织细胞增殖、凋亡和p53表达的变化 [J]. 肿瘤防治研究, 2011, 38(8): 918-920.

[4] 张振华;吴敬波 . 脂质体阿霉素热化疗对食管癌细胞的毒性实验研究[J]. 肿瘤防治研究, 2011, 38(7): 736-739.

[5] 林宏伟;白桦;栗敏;肖鹏;陈奎生;张红新 . 间隙连接蛋白Cx26和Cx43的表达及与食管鳞癌浸润和转移关系[J]. 肿瘤防治研究, 2011, 38(7): 809-813.

[6] 吴正国;龚家权 . 机械吻合和手工吻合在国人食管癌术后吻合口瘘发生率的Meta分析[J]. 肿瘤防治研究, 2011, 38(7): 823-826.

[7] 巩合义;和劲光;李宝生 . 18F-FDG PET/CT在食管癌中的应用 [J]. 肿瘤防治研究, 2011, 38(7): 840-843.

[8] 孙晓宏;庞作良;罗洞波. 转录水平环氧合酶-2在食管癌中的表达及临床意义 [J]. 肿瘤防治研究, 2011, 38(7): 830-831.

[9] 梅家转;刘桂举;李瑞君;栗敏;张晓娟 . IL-15上调NKG2D表达对CIK细胞杀伤活性的增强效应 [J]. 肿瘤防治研究, 2011, 38(5): 495-497.

[10] 吕鹏;胡志坚. 乙醇脱氢酶2基因多态性与食管癌发病风险的Meta分析[J]. 肿瘤防治研究, 2011, 38(5): 579-583.

[11] 许林平;庞雅青;买玲. 食管癌血管形成相关因子的表达及意义 [J]. 肿瘤防治研究, 2011, 38(3): 286-290.

[12] 韩向春;康金旺;郑力强;左连富. Wnt1、β-catenin、APC和cyclin D1蛋白在胃癌中的表达[J]. 肿瘤防治研究, 2011, 38(2): 163-166.

[13] 曾同霞;马彦清;徐莉;蔡金凤;李锋;何玲;秦江梅. 免疫相关基因Tap2、HLA-DR9与新疆哈萨 克族食管癌的交互作用[J]. 肿瘤防治研究, 2011, 38(2): 210-213.

[14] 石国庆;吴会超;徐刚 . 超声内镜在食管癌TN分期中的应用 [J]. 肿瘤防治研究, 2011, 38(12): 1413-1414.

[15] 傅玲;王明玉;曾洪生. 洛铂联合替加氟治疗晚期食管癌的临床观察[J]. 肿瘤防治研究, 2011, 38(12): 1426-1428.