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论著

没食子儿茶素没食子酸酯对Cr^{6+}诱导的Vero及786-0肾细胞凋亡的影响

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摘要 目的 探讨没食子儿茶素没食子酸酯（EGCG）对肾脏受活性氧应激损伤的保护作用。方法 以Cr^{6+}应激诱导Vero和786-0肾细胞凋亡为实验模型，用吖啶橙染色、流式细胞仪检测和DNA凝胶电泳等方法研究了EGCG对两种肾细胞凋亡的影响。结果 Cr^{6+}浓度依赖地降低Vero和786-0细胞存活率，IC_{50}分别为9.8和8.6 mg·L^{-1}；其中400 mg·L^{-1}/2 h Cr^{6+}处理可诱导Vero和786-0细胞凋亡。20～60 mg·L^{-1} EGCG有效抑制Cr^{6+}引起的Vero活细胞数下降，且40 mg·L^{-1} EGCG显著抑制该细胞凋亡；但EGCG对786-0细胞没有相应的保护作用，相反促进786-0细胞凋亡。结论 EGCG对正常肾细胞的氧化应激损伤有保护作用，但对肿瘤细胞的氧化损伤没有保护作用。EGCG对正常肾细胞和肿瘤肾细胞的选择性作用具有积极意义。

关键词 没食子儿茶素没食子酸酯 铬 应激 凋亡

分类号 R983

Effect of epigallocatechin-3-gallate on the apotosis of Vero and 786-0 renal cells stressed by chromium *in vitro*

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Abstract

AIM This study was conducted to identify the protection of epigallocatechin-3-gallate(EGCG) against the renal damage by reactive oxygen species(ROS）. **METHODS** Cr^{6+} was used to induce apotosis of renal cells(Vero cells and 786-0 cells) *in vitro*. The effect of EGCG on apotosis was investigated by fluorescent staining, DNA electrophoresis and flow cytometry. **RESULTS** Cr^{6+} decreased the viability of Vero and 786-0 cells in a concentration-dependent manner, and the IC_{50} was 9.8 and 8.6 mg·L^{-1}, respectively. Treatment with Cr^{6+} 400 mg·L^{-1} for 2 h caused the apotosis of both Vero and 786-0 cells. EGCG 40 mg·L^{-1} markedly inhibited the apotosis of Vero cells, but no effect was seen on 786-0. **CONCLUSION** EGCG protected Vero cells from damage by ROS, but promoted the apotosis of 786-0 cells. The selective effect of EGCG on the apotosis of normal and tumor cells induced by chromium is valuable in clinic.

Key words epigallocatechin-3-gallate chromium stress apotosis sis

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