中国药理学与毒理学杂志 2011 25 (1): 52-56 ISSN: 1000-3002 CN: 11-1115/R

论著

表没食子儿茶素没食子酸酯对鱼藤酮诱导PC12细胞损伤的保护作用

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收稿日期 2010-5-6 **修回日期** **网络版发布日期** 2011-1-25 **接受日期** 2010-8-19

摘要 **目的** 探讨表没食子儿茶素没食子酸酯(EGCG)对鱼藤酮诱导的PC12细胞损伤的保护作用及其可能机制。**方法** 将培养的大鼠PC12细胞分别加入EGCG 1，5和10 μmol·L^{-1}预处理30 min后加入鱼藤酮250 nmol·L^{-1}继续作用24 h。MTT比色法检测细胞存活率，Hoechest33258染色观察细胞核形态的变化，流式细胞仪检测细胞凋亡率，JC-1染色检测线粒体膜电位变化。**结果** 与鱼藤酮模型组存活率(77.0±1.3)%相比，EGCG 1，5和10 μmol·L^{-1}组细胞存活率显著增加(P<0.05)，分别为(79.8±2.3)%，(82.4±2.2)%和(88.3±2.0)%。Hochest33258染色发现，EGCG组细胞核形态明显改善。与正常对照组比较，模型组细胞凋亡率增加8.2倍；与鱼藤酮模型组比较，EGCG组细胞凋亡率分别下降46%，25% 和63%，差异具有统计学意义(P<0.01）。流式细胞仪检测鱼藤酮组凋亡率为33.8%，EGCG 1，5和10 μmol·L^{-1}组的凋亡率下降至30.6%，14%和15.7%。与模型组相比，EGCG 1，5 和10 μmol·L^{-1}组线粒体膜分别增高了2.48，3.96和4.04倍，差异具有统计学意义(P<0.01)。**结论** EGCG具有抑制鱼藤酮诱导的细胞凋亡作用，其机制可能与其稳定线粒体膜电位有关。

关键词 表没食子儿茶素没食子酸酯 鱼藤酮 PC12细胞 细胞凋亡

分类号 R285

Effects of epigallocatechin gallate on rotenone-induced injury in rat PC12 cells

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Abstract

OBJECTIVE To investigate protective effects of epigallocatechin gallate (EGCG) on rotenone- induced injury in rat pheochromocytoma (PC12) cells and to explore potential mechanisms. **METHODS** EGCG 1, 5 and 10 μmol·L^{-1} were added to cultivated rat PC12 cells, and 30 min later, rotenone 25 nmol·L^{-1} was added and then coincubated for 24 h. The cell viability was assessed by MTT assay. Hoechst33258 was employed to observe morphological changes of cell nucleus. Apoptosis rate was detected by flow cytometry using Annexin Ⅴ and PI. Mitochondrial membrane potential was measured by JC-1 staining. **RESULTS** Compared with (77.0±2.9)% in rotenone- treated group, cell survival in EGCG 1, 5 and 10 μmol·L^{-1} groups were significantly higher (79.8±2.3)%, (82.4±2.2)% and (88.3±2.0)%, respectively (P<0.05). Hochest33258 staining demonstrated that EGCG improved the nuclear changes morphologically. Compared with control group, the apoptosis rate was significantly increased in rotenone- model cells by 8.2 folds which was significantly reduced by 46%, 25% and 63% in EGCG 1, 5 and 10 μmol·L^{-1} groups (P<0.01). Comparsion with 30.6% in rotenone group, in EGCG 1, 5 and 10 μmol·L^{-1} apoptosis rate reduced to 30.6%, 14%, 15.7% respectively by FCM analysis. Meanwhile, the mitochondrial membrane potential of EGCG 1, 5 and 10 μmol·L^{-1} groups increased 2.48-, 3.96- and 4.04-fold compared with rotenone- treated group (P<0.01). **CONCLUSION** EGCG exhibits inhibitory effect on rotenone induced apoptosis in rat PC12 cells, which is possibly due to its ability to stabilize mitochondrial membrane potential.

Key words epigallocatechin-3-gallate rotenone PC12 cells apoptosis

DOI: 10.3867/j.issn.1000-3002.2011.01.010

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