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论著

没食子儿茶素没食子酸酯对1-甲基-4-苯基吡啶离子诱导的大鼠PC12细胞凋亡的作用

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摘要 目的： 探讨没食子儿茶素没食子酸酯(EGCG)抑制1-甲基-4-苯基吡啶离子（MPP+）诱导大鼠PC12细胞凋亡及其抗氧化作用、调节胞浆钙离子稳态与其抑制细胞凋亡作用之间的关系。方法: 培养大鼠肾上腺嗜铬细胞瘤细胞株PC12细胞，给予MPP+诱导细胞凋亡。EGCG（10、50及100 μmol·L-1）预处理0.5 h，再加入MPP+使其终浓度为900 μmol·L-1处理24 h后，MTT法检测细胞存活率，Annexin V-PI双染流式细胞仪检测细胞凋亡，荧光酶标仪测定细胞内活性氧，激光共聚焦荧光显微镜通过检测细胞内钙的荧光强度、检测细胞胞浆［Ca2+］i的变化，透射电镜观察凋亡细胞线粒体结构形态变化，并测定细胞内超氧化物歧化酶（SOD）和丙二醛（MDA）的含量。结果: MPP+呈剂量依赖性损伤PC12细胞，诱导细胞凋亡发生率达到31.0%。与模型组比较，EGCG处理后，明显提高细胞活力，降低凋亡细胞率，同时增强SOD活性、减少MDA和ROS的含量，降低胞浆［Ca2+］i浓度，减轻MPP+诱致的细胞线粒体改变。结论: EGCG具有抑制MPP+诱导的PC12细胞凋亡的作用，其作用机制可能与其提高细胞抗氧化能力和减少胞浆［Ca2+］i有关。

关键词 没食子儿茶素没食子酸酯； 氧化性应激； 钙； 细胞凋亡 PC12细胞

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Effects of epigallocatechin-3-gallate on 1-methyl-4-phenylpyridinium ion-induced apoptosis in rat PC12 cells

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Abstract

<FONT face=Verdana>AIM: To investigate the effects of epigallocatechin-3-gallate (EGCG) on 1-methyl-4-phenylpyridinium ion (MPP+)-induced apoptosis in rat pheochromocytoma (PC12) cells and to explore the relationships between its roles of anti-oxidation, intracellular calcium homeostasis and anti-apoptosis. METHODS: Rat PC12 cells were pretreated with vehicle control or EGCG (10, 50, and 100 μmol/L) for 30 min, then cultured with MPP+ (900 μmol/L) for 24 h. The cell viability and apoptosis were monitored by MTT assay and flow cytometry using Annexin V and PI. The activity of intracellular reactive oxygen species (ROS), contents of superoxide dismutase (SOD) and malondialdehyde (MDA), cytoplasmic Ca2+ density and apoptotic morphology of mitochondria were examined by fluorescent plate-based assays, confocal microscope, and transmission electron microscope, respectively. RESULTS: MPP+ impaired the PC12 cells in a concentration-dependent pattern and induced apoptosis of the cells (31% versus control). Compared with the control, the cells pretreated with EGCG showed markedly higher rate of viability and lower apoptosis. Meanwhile, EGCG pretreatment significantly increased the SOD activity and decreased the levels of MDA and ROS. Interestingly, EGCG also decreased the concentration of cytoplasmic Ca2+ and improved the morphology of mitochondria. CONCLUSION: EGCG exhibits inhibitory effects on MPP+-induced apoptosis in rat PC12 cells, which is possibly associated with increasing the cell ability of anti-oxidation and decreasing the concentration of cytoplasmic Ca2+.</FONT>

Key words Epigallocatechin gallate Oxidative stress Calcium Apoptosis PC12 cells

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