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论文

左旋黄皮酰胺对冈田酸和**β**淀粉样肽₂₅₋₃₅神经毒性的保护作用

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摘要:

探讨左旋黄皮酰胺对冈田酸(okadaic acid,OA)诱导的人神经瘤细胞(SH-SY5Y)和去卵巢(ovariectomy,OVX)及单侧侧脑室注射A $oldsymbol{eta}_{25-35}$ 所致神经元损伤的保护作用。通过MTT试验、LDH释放测定试验、Hoechst 33258荧光染色试验以及SH-SY5Y细胞检测,考察左旋黄皮酰胺拮抗冈田酸诱导的细胞毒作用。通过避暗试验、电镜检测、NissI体染色及HE染色,考察左旋黄皮酰胺对去卵巢及侧脑室注射A $oldsymbol{eta}_{25-35}$ 大鼠神经元的保护作用。左旋黄皮酰胺可明显拮抗冈田酸诱导的细胞毒作用,提高去卵巢及侧脑室注射A $oldsymbol{eta}_{25-35}$ 大鼠的学习记忆能力,保护海马及皮层神经元。左旋黄皮酰胺可拮抗冈田酸及A $oldsymbol{eta}_{25-35}$ 诱导的神经毒性,具有神经保护作用。

关键词: 左旋黄皮酰胺 阿尔茨海默病 冈田酸 β 淀粉样肽 $_{25-35}$ 神经保护作用

Protective effect of (-)clausenamide against neurotoxicity induced by okadaic acid and β -amyloid peptide25-35

ZHANG Jing; CHENG Yong; ZHANG Jun-tian

Abstract:

This study is to investigate the protective effect of (-)clausenamide against the neurotoxicity of okadaic acid in SH-SY5Y cell line, and injection β -amyloid peptide $_{25-35}$ (A β_{25-35}) to the cerebral ventricle in ovariectomy (OVX) rats. MTT assay, LDH assay, and Hoechst 33258 staining were used to detect the effect of (-) clausenamide on the toxicity of okadaic acid in SH-SY5Y cell line. The animal model was induced by ovariectomized and injection of A β_{25-35} in the cerebroventricle of rats. The effect of (-)clausenamide on learning and memory deficiency was observed by step-through test. Electron microscope, Nissl body staining, and HE staining were used to examine the morphological changes in hippocampus and cerebral cortex neurons. Pretreatment of (-)clausenamide and LiCl decreased the rate of cell death from MTT, LDH release, and apoptosis from Hoechst 33258 staining in SH-SY5Y cell line. The step-through tests showed (-)clausenamide could improve the ability of learning and memory. The Nissl body staining and HE staining experiments also showed the neuroprotective effects of (-)clausenamide on the neurons of hippocampus and cerebral cortex. (-)Clausenamide has the protective effects against the neurotoxicity induced by okadaic acid and A β

Keywords: Alzheimer's disease okadaic acid β -amyloid peptide₂₅₋₃₅ neuroprotective effect (-) clausenamide

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