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论文

长期噪声暴露对海马NMDAR2B及Tau蛋白磷酸化影响

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摘要:

目的 探讨长期噪声暴露对大鼠海马的影响及其机制。方法 24只雄性SD大鼠分为对照组与噪声暴露组(100 dB,白噪声,4 h/d×30 d),检测噪声暴露组动物中海马的N-甲基-D-天冬氨酸受体2B亚基(NR2B)的表达和tau蛋白的磷酸化状态;为了探讨其可能机制,用DNA 断裂原位末端标记法检测海马神经细胞凋亡状态。结果 长期噪声暴露后,实验组与对照组相比海马中NR2B的表达下降达41%,tau蛋白发生过度磷酸化的程度升高2.1倍和海马神经细胞凋亡数量占总神经元细胞数量的0.5%~1%;免疫组织化学结果显示海马中tau蛋白发生过度磷酸化的区域主要在齿状回(DG)区和安蒙氏角(CA)1区。结论 长期噪声暴露引起大鼠神经递质系统异常及 tau蛋白过度磷酸化可能诱发神经细胞凋亡和认知障碍。

关键词: 噪声 N-甲基-D-天冬氨酸受体2B亚基(NR2B) Tau蛋白过度磷酸化 细胞凋亡

Long-term noise exposure causes abnormality of NMDAR2B expression and tau hyperphosphorylation and induction of apoptosis in rat hippocampus

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Abstract:

Objective To explore the effects and mechanism of long-term noise exposure on hippocampus of rats.Methods Twenty four male Sprague-Dawley(SD) rats were grouped as control and noise exposure group.Expression of N-methyl-D-aspartic acid receptor 2B subunit(NR2B) and the level of phosphorylation of tau in hippocampus were measured after chronic noise exposure(100 dB SPL white noise,4 hours per day for 30 days).To explore the possible mechanism,we also detected neuronal cell apoptosis in hippocampus by terminal deoxynucleotidyl transfease-mediated dUTP nick end-labeling (TUNEL) reactivity.Results After long-term noise exposure,the expression of NR2B in the hippocampus decreased by 41%.Meanwhile,the chronic noise exposure also caused tau hyperphosphorylation,which was twice higher than the normal,and neuronal cell apoptosis accounted for 0.5%-1% total neuronal cells in the hippocampus.Immunohistochemistry assaies confirmed that tau hyperphosphorylation was most prominent in dentate gyrus(DG) and CA1 region in the hippcampus.Conclusion Long-term noise exposure induces abnormality of neurotransmitter system and hyperpbosphorylation of tau and the adverse effects could lead to neuronal cell apoptosis and cognition impairment.

Keywords: noise NR2B tau hyperphosphorylation cell apoptosis

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