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论文

锂-匹罗卡品致痫大鼠海马结构nNOS、iNOS及活化Caspase-3蛋白表达的动态变化

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摘要：

目的 探讨大鼠癫痫持续状态（SE）后海马结构神经型一氧化氮合酶（nNOS）、诱导型一氧化氮合酶（iNOS）及活化Caspase-3蛋白表达的动态变化及其相互关系。 方法 采用锂-匹罗卡品腹腔注射建立大鼠SE模型，用免疫组织化学及免疫印迹法在2h、6h、3d及7d不同时相点检测大鼠海马nNOS、iNOS 及活化Caspase-3蛋白的表达。 结果 大鼠海马nNOS的蛋白表达在SE后2h开始迅速增高，在6h时达到高峰，之后逐渐降低，但在3、7d时仍显著高于对照组（P均<0.01）。iNOS蛋白的表达在SE后6h时开始持续增高（P<0.01），并在7d时达到高峰（P<0.01）。活化Caspase-3蛋白的表达在3d开始增高（P<0.01），7d时达到高峰（P<0.01）。结论 大鼠在SE后海马的nNOS、iNOS及活化Caspase-3的表达均有不同程度的增高，提示一氧化氮合酶的反应产物一氧化氮可能与癫痫发作后海马结构内的神经元受损有关，而神经元受损与凋亡之间的关系亦密不可分。

关键词： 癫痫持续状态；模型，动物；海马；Caspase蛋白；一氧化氮合酶;细胞凋亡；大鼠，Wistar

Dynamic changes of nNOS, iNOS and activated Caspase-3 protein expressions in the hippocampus of rats with lithium-pilocarpine-induced epilepsy

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Abstract:

Objective To explore the correlation and dynamic changes of protein expressions of nNOS, iNOS and activated Caspase-3 after status epilepticus (SE). Methods The animal model was established by lithium-pilocarine induction in rats. 2h, 6h, 3d and 7d after SE, variations of expressions of nNOS, iNOS and activated Caspase-3 proteins were determined by immunohistochemistry and immunoblot, respectively. Results Expression of the nNOS protein significantly increased 2h after SE(P<0.01), reached its peak at 6h (P<0.01), and decreased thereafter. However, expressions of the nNOS protein on 3d and 7d were still significantly higher compared with the control group (P<0.01). Expression of the iNOS protein was significantly up-regulated 6h after SE(P<0.01), and reached its peak on 3d (P<0.01). Expression of the activated Caspase-3 protein significantly increased 3d after SE(P<0.01), and reached its peak on 7d (P<0.01). Conclusion Expressions of nNOS, iNOS and activated Caspase-3 proteins are up-regulated at different levels in the rat hippocampus at different times after SE, suggesting that the activation of Caspase-3 may be involved in the excitotoxic effect induced by nitric oxide synthase (NOS) and apoptosis could be closely related to neuron injury.

Keywords: Status epilepticus; Models, animal; Hippocampus; Caspase protein; Nitric oxide synthase; Apoptosis; Rat, Wistar

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