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论文

缺氧缺糖条件下大鼠脑皮质神经元内游离钙浓度的变化及神经生长因子的作用

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摘要:

以Fura-2/AM为细胞内钙离子的荧光指示剂,用双波长荧光分光光度计测定了缺氧缺糖时体外培养的大鼠胎鼠神经细胞内游离钙([Ca^{2+}]i)的变化,并观察了神经生长因子(NGF)的影响。结果表明,脑皮质细胞缺氧缺糖培养16～24h时,细胞大量死亡。NGF剂量依赖地减少神经元缺氧缺糖培养24h时乳酸脱氢酶(LDH)的释放,提高细胞生存力。细胞缺氧缺糖早期引起[Ca^{2+}]i减少,而后期引起[Ca^{2+}]i显著升高,导致细胞损害。NGF50μg·L^{-1}在缺氧缺糖早期提高[Ca^{2+}]i到正常水平,降低后期[Ca^{2+}]i的升高。提示,NGF通过稳定[Ca^{2+}]i或降低后期的胞内钙升高保护了脑皮质神经元免受缺氧缺糖的损害。

关键词: 神经生长因子 细胞内游离钙 新型钙离子荧光指示剂(Fura-2) 神经细胞 乳酸脱氢酶

EFFECTS OF NERVE GROWTH FACTOR ON INTRACELLULAR FREE Ca^{2+} IN OXYGEN/GLUCOSE-DEPRIVED CULTURES FROM CEREBRAL CORTEX OF FETAL RATS

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Abstract:

Oxygen/glucose deprived cortical cell cultures were used to explore the involvement of calcium in the mechanism of hypoxic/hypoglycemic insults and protection of nerve growth factor(NGF). Neuron viability and lactate dehydrogenase(LDH) efflux in the bathing medium of cerebral cortex cultures of fetal rats were measured as indication of the NGF effect. Calcium fluorescence indicator Fura-2/AM was used to measure free intracellular calcium([Ca^{2+}]i). When cultures were deprived of oxygen/glucose, massive neuronal death occurred 16～24 h following the onset of hypoxia/hypoglycemia. NGF(3～100 μg·L^{-1}) dose dependenly attenuated the 24 h hypoxia/hypoglycemia induced efflux of LDH and elevated the number of surviving neurons. Hypoxia/hypoglycemia induced a reduction in [Ca^{2+}]i in early stage and then a large elevation of [Ca^{2+}]i in 12～24 h. NGF 50μg·L^{-1} brought [Ca^{2+}]i to normal limits during the early stage of hypoxia/hypoglycemia and prevented the later elevation in [Ca^{2+}]i. The [Ca^{2+}]i elevation may be involved in the cell damage in hypoxia/hypoglycemia deprived cultures. It is belived that NGF protect cerebral cortical neurons against hypoxic/hypoglycemic insults via 'stabilizing' [Ca^{2+}]i level or preventing the late rise in [Ca^{2+}]i.

Keywords: Intracellular free Ca^{2+} Fura-2 Neurons Lactate dehydrogenase Nerve growth factor (NGF)

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