

论文

β-内啡肽对谷氨酸神经毒性作用的影响

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摘要:

采用形态学观察、β-内啡肽(β-End)放射免疫测定及单细胞内游离钙浓度——[Ca²⁺]_i检测等方法,观察了β-End对谷氨酸单钠(MSG)诱发神经元损伤的影响,分析了可能的作用机制。结果表明,β-End可以明显加重MSG诱发的下丘脑弓状核神经元的损伤;β-End和MSG诱发的[Ca²⁺]_i增高可被维拉帕米部分逆转。另外,吗啡可以进一步加剧MSG诱导的各脑区β-End含量的变化。提示β-End可以明显地加剧MSG的神经毒性作用,其机制与MSG能诱发脑内β-End的含量的增多及β-End可进一步破坏MSG引起的胞内钙稳态失衡有关。

关键词: β-内啡肽 谷氨酸单钠 神经毒性 钙 弓状核

EFFECTS OF β-ENDORPHIN ON GLUTAMATE NEUROTOXICITY

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Abstract:

The effects of β-endorphin(β-End) on monosodium glutamate(MSG) neurotoxicity were studied via morphological observation and image analysis of neuronal areas, together with the determination of intracellular free calcium concentration ([Ca²⁺]_i) in single neuron and radioimmunoassay for β-End contents.β-End(1.0 mg·kg⁻¹, sc) was found to obviously aggravate the neuronal injury in the arcuate nucleus of hypothalamus induced by MSG(0.5 g·kg⁻¹, sc). Just as MSG increased [Ca²⁺]_i significantly, β-End(2.0 g·L⁻¹) itself also increased it, though the extent of elevation was smaller than that of MSG (17.0mg·L⁻¹). The obvious changes of [Ca²⁺]_i induced by both MSG and β-End were partially reversed after pretreatment with verapamil. On the other hand, the content of β-End in different areas of the brain were augmented following the addition of MSG and further elevated by morphine treatment. These findings suggest that the mechanisms of the enhancing effect of β-End on glutamate neurotoxicity were linked to the aggravation on the disruption of intracellular Ca²⁺ homeostasis induced by MSG.Moreover, the fact that opioids promote β-End release induced by MSG may be involved in the mechanisms as well.

Keywords: Monosodium glutamate Neurotoxicity Calcium Arcuate nucleus β-Endorphin

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