

论文

慢性孵育β-淀粉样肽(25-35)对培养大鼠海马神经元外向钾电流的影响

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摘要:

目的 研究慢性孵育β-淀粉样肽(25-35) (β-AP<sub>25-35</sub>)对海马神经元上瞬时外向钾电流(I<sub>A</sub>)和延迟整流钾电流(I<sub>K</sub>)的影响。方法 在培养的大鼠海马神经元上用膜片钳全细胞记录钾通道电流。结果 β-AP<sub>25-35</sub> 3μmol·L<sup>-1</sup> 孵育细胞24h, I<sub>K</sub> 电流幅度增加(44.3±5.4)%, 电流密度由(30.4±6.4)pA·PF<sup>-1</sup> 增加至(43.8±4.7)pA·PF<sup>-1</sup>; β-AP<sub>25-35</sub> 10μmol·L<sup>-1</sup> 孵育12h, I<sub>K</sub> 电流幅度增加(69.8±4.1)%, 电流密度增加至(51.6±7.9)pA·PF<sup>-1</sup>, 呈浓度依赖性; β-AP<sub>25-35</sub> 引起的I<sub>K</sub> 增加对TEA 5mmol·L<sup>-1</sup> 敏感; β-AP<sub>25-35</sub> 上调I<sub>K</sub> 的作用主要发生在β-AP<sub>25-35</sub> 5用药后48h内。β-AP<sub>25-35</sub> 对I<sub>A</sub> 无显著性影响。结论 β-AP<sub>25-35</sub> 选择性地增加海马神经元上I<sub>K</sub>, 这一作用可能与β-AP的神经毒性有关

关键词: β-淀粉样肽(25-35) 钾通道 膜片钳技术 海马神经元

EFFECTS OF CHRONIC EXPOSURE TO BETA-AMYLOID PEPTIDE<sub>25-35</sub> ON VOLTAGE-GATED POTASSIUM OUTWARD CURRENT IN CULTURED RAT HIPPOCAMPAL NEURONS

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Abstract:

AIM To investigate the effects of chronic exposure to beta amyloid peptide<sub>25-35</sub> (β-AP<sub>25-35</sub>) on transient outward (I<sub>A</sub>) and delayed rectifying (I<sub>K</sub>) current in cultured rat hippocampal neurons. METHODS Voltage gated potassium outward current was recorded using whole cell patch clamp techniques in the cultured rat hippocampal neurons. RESULTS Cells were exposed to β-AP<sub>25-35</sub> 3 μmol·L<sup>-1</sup> for 24 h. The I<sub>K</sub> current amplitude was increased by (44±5)% (n=10, P<0.05), current density increased from (30±6) pA·PF<sup>-1</sup> to (44±5) pA·PF<sup>-1</sup> (n=10, P<0.05) at the membrane potential of 40 mV. When β-AP<sub>25-35</sub> was 10 μmol·L<sup>-1</sup>, I<sub>K</sub> was increased by (70±4)% (n=10, P<0.01), current density increased by (52±8) pA·pF<sup>-1</sup> (n=10, P<0.01), The effect of β-AP<sub>25-35</sub> was shown to be in a dosage dependent manner. The increment of I<sub>K</sub> current by β-AP<sub>25-35</sub> was sensitive to 5 mmol·L<sup>-1</sup> TEA. The increase of I<sub>K</sub> mainly happened within 48 h after exposure to β-AP<sub>25-35</sub>. I<sub>A</sub> did not change obviously after exposure to β-AP<sub>25-35</sub> (n=10, P>0.05). CONCLUSION Beta-amyloid peptide enhanced I<sub>K</sub> current selectively. This may be related to β-AP induced neurotoxicity in the hippocampal neurons.

Keywords: potassium channels patchclamp techniques hippocampal neuron beta amyloid peptide<sub>25-35</sub>

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