论著

铅抑制急性分离的大鼠背根神经节慢失活钾电流

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摘要 目的 慢失活外向 K^+ 电流(I_D)对于长时程持续刺激中延迟动作电位的发放,调节其发放频率和复极化有重要意义,其改变对神经元的兴奋性产生重要影响,因此研究铅(Pb^{2+})对神经元细胞 I_D 的效应,并初步探讨其作用机理。方法 应用全细胞膜片钳技术,根据动力学和药理学特性分离鉴定大鼠背根神经节(DRG) I_D , 观察 Pb^{2+} 对 I_D 的抑制效应。结果 Pb^{2+} 以浓度依赖性方式抑制 I_D 。0. 1,1. 0,10. 0 和100. 0 μ mol· L^{-1} Pb^{2+} 对+60 μ mv处 I_D 的抑制率分别为(6. 9±0. 6)%、(29. 3±3. 0)%、(85. 9±5. 1)%和(99. 4±7. 0)%(I_D =15), IC_{50} 为2. 4 I_D =10. 0 I_D =10 激活具有电压依赖性, I_D =2+对 I_D =10 的抑制作用也具有电压依赖性,最大抑制作用发生在+60 I_D =2+增加了 I_D =10 的激活时程。结论 I_D =2+使得 I_D =12 电流的稳态激活曲线向去极化方向移动,并延长 I_D =16 测激活时间常数,提示 I_D =17 的激活时程。结论 I_D =17 中流,导致神经元的兴奋性增加,这可能是 I_D =2+影响神经细胞功能的作用机理之

关键词 <u>铅 大鼠 神经节, 脊</u> 钾通道 <u>膜片钳技术, 全细胞</u> 分类号 **R994**. 3

Inhibitory effect of Pb²⁺ on slow- inactivating K⁺ current inacutely isolated rat dorsal root ganglion neurons

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Abstract

AIM Slow-inactivating K⁺ current (I_D) possesses an important role in delaying the discharge of action potential after a period of long duration, modulating the frequency of the repetitive firing and repolarization. Modulation of I_D generates a powerful effect on neuronal excitability. Therefore, the inhibitory effects and mechanism of Pb²⁺ on I_D were investigated in rat dorsal root ganglion (DRG) neurons. **METHODS** With the whole cell patch clamp technique, I_D was isolated based on the kinetics and pharmacological properties, and the inhibitory effects of Pb²⁺ on I_D were investigated in rat acutely isolated DRG neurons. **RESULTS** The results showed that 0.1, 1.0, 10.0 and 100.0 μ mol·L⁻¹ Pb²⁺ inhibited the amplitude of I_D by $(6.9\pm0.6)\%$, $(29.3\pm3.0)\%$, $(85.9\pm5.1)\%$ and $(99.4\pm7.0)\%$ (n=15), respectively, IC_{50} was 2.4 μ mol·L⁻¹. It suggested that the inhibition of Pb²⁺ on I_D be dose dependent and saturated. The activation of I_D was voltage-dependent, and the inhibition of Pb²⁺ was also voltage- dependent. The strongest inhibition of Pb²⁺ was at +60 mV. The activation curve of I_D was shifted to depolarization direction with Pb²⁺. The activation time constant was enlarged with Pb²⁺, suggesting that Pb²⁺ increase the activation course of I_D . **CONCLUSION** The inhibition of Pb²⁺ on I_D significantly increase the excitation of DRG neurons, which maybe take an important part in Pb²⁺ neurotoxication.

Key words <u>lead rats ganglia spinal potassium channels patch clamp technique whole-cell</u>

扩展功能

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