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论著

肟硫磷中毒肌无力大鼠骨骼肌烟碱样乙酰胆碱受体通道特性

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摘要 目的 探讨肟硫磷引起肌无力大鼠骨骼肌烟碱样乙酰胆碱受体（nAChR）通道特性的改变，揭示其发生机制。方法 用ip 1.15 g·kg^{-1}肟硫磷制作成年大鼠肌无力模型，同时ip 16 mg·kg^{-1}阿托品以对抗毒蕈碱样症状。染毒后0.5～2 h，8只大鼠出现肌无力，7只大鼠肌力正常。将3只肌无力大鼠于肌力恢复后(染毒后12～18 h)，其余大鼠于染毒2～3 h后，断颈处死，取后肢趾短屈肌，酶解制备骨骼肌纤维，用膜片钳对肌纤维nAChR通道做单通道电流记录。结果 肌无力大鼠nAChR单通道开放频率、表观平均开放时间、平均开放时间和电导均显著低于对照组和肌力正常的染毒大鼠，且肌力恢复后，以上各参数均接近对照组。结论 肟硫磷中毒引起肌无力可能与肟硫磷导致或促进nAChR的脱敏，以及阻断nAChR通道的开放有关。

关键词 杀虫剂 肟硫磷 肌无力 受体, 烟碱 单通道电流记录

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Properties of nicotinic acetylcholine receptors on muscle fibers in phoxim-induced myasthenic rats

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Abstract

AIM To explore the changes of properties of nicotinic acetylcholine receptors(nAChR) on muscle fibers from phoxim-induced myasthenic rats and its mechanism. **METHODS** Fifteen rats were intraperitoneally intoxicated by phoxim(1.15 g·kg^{-1}) and dosed with atropine(16 mg·kg^{-1}) as antagonist to muscarinic symptoms of poisoning rats. Among them, 8 developed myasthenia, and 7 non- myasthenia within 2 h after exposure. Two to three hours after intoxication, the flexor digitorum brevis of hind foot was dissected, digested by collagenase type Ⅰ (2 g·L^{-1}), and dissociated into single muscle fibers, on which the nAChR single channel recording was performed at endplate with patch clamp in cell- attached mode. **RESULTS** The nAChR channels of myasthenic rats decreased significantly in open frequency, apparent mean open time, mean open time, and conductance in comparison with those of control and non-myasthenic rats, and became similar to those of control individuals after complete recovery from muscle weakness. **CONCLUSION** The results indicated that phoxim induced myasthenia by directly or indirectly desensitizing nAChR and blocking nAChR channels on muscle fibers.

Key words insecticides phoxim myasthenia receptors nicotinic single channel recording

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