Relaxing action of adrenergic β2-agonists on guinea-pig skinned tracheal muscle

http://www.firstlight.cn 2006-03-29

Although adrenergic β2-agonist-induced smooth muscle relaxation has been attributed to increased intracellular cyclic AMP (cAMP), a relaxation response has been observed at low β2-agonist concentrations that do not cause increased cAMP. To elucidate the mechanism of tracheal muscle relaxation induced by low concentrations of β2-agonists, we used a guinea-pig skinned tracheal smooth muscle preparation to examine the effects on the contractile protein system. The isotonic contraction of β-escin-treated skinned tracheal muscle from guinea-pig was measured. When the intracellular Ca2+ concentration was maintained at 1 μmol/L in the presence of guanosine 5'-triphosphate (GTP; 100 μmol/L), neither isoproterenol (10 nmol/L) nor salbutamol (60 nmol/L) affected Ca2+ sensitivity, but a significant decrease in Ca2+ sensitivity was observed in the presence of okadaic acid (1 μmol/L). The decrease in Ca2+ sensitivity was a slow response and was blocked by pretreatment with propranolol (1 μmol/L). Forskolin (1 μmol/L) did not affect Ca2+ sensitivity. These results suggest that adrenergic β2-agonists may activate protein phosphatase through an unknown pathway involving the β2-receptor, which enhances dephosphorylation of the myosin light chain and/or thin filament proteins, resulting in relaxation of the tracheal smooth muscle.

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