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K + -Channels in Smooth Muscle Cells and Their Importance

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Abstract: K + -channels of smooth muscle cells play an important role in regulating membrane potential and their functional activity. So far, 4 different K + -channels are distinguished in smooth muscle cells. These include potential-dependent K + -channels, intracellular K + -channels, calcium-activated K + -channels (KCa-channels), and ATF-sensitive K + -channels.

Key words: K + -channels, smooth muscle cells, Calcium-activated K + -channels, ATF

Introduction

Potential-dependent K + -channels are activated by changes in membrane potential during depolarization. These channels play a leading role in the regulation of smooth muscle cells and vascular tone. These channels are also involved in the effective distribution of various vasoconstrictors and vasorelaxants. In this regard, vasorelaxants are activated directly or through a sAMF-dependent pathway, while vasoconstrictors are inactivated as a result of increased intracellular calcium concentrations and activation of PKS. The classic blockers of potential-dependent K + channels are dendrotoxins and maurotoxins [Rochat et al., 1999].

Intracellular K + channels. It is detected in smooth muscle cells and they are activated as a result of increased concentrations of extracellular K + ions. These channels play an important role in regulating the membrane potential of smooth muscle cells and vascular tone. The classic blocker of these channels is tertiapine Q [Jin et al., 1999].

Calcium-activated K + -channels. Smooth muscle cells are activated during depolarization and when the concentration of calcium ions inside the cell increases. These channels move with high permeability and play an important role in the regulation of membrane potential in smooth muscle cells. Therefore, inhibition of these channels leads to vasoconstriction and depolarization. However, agents activate this channel through sGMF or sAMF cascades, leading to hyperpolarization and vasorelaxation. Selective blockers of these channels are substances such as TeA, apamine, iberitoxin and haribdotoxin [Kim et al., 2002].

ATF-sensitive K + -channels. It is activated in the presence of intracellular ATF but when it exceeds the set concentration it causes the channels to close. However, the literature suggests that the activity of these channels can also be controlled using intracellular messengers that are independent of changes in ATF concentration. Activation of ATF-sensitive K + -channels is observed during vasorelaxation of smooth muscle cells, during which their inactivation leads to vasoconstriction of smooth muscle cells. In particular, the effect of vasorelaxants such as adenosine and

prostacyclin is explained by the activation of ATF-sensitive K + -channels in the presence of PKA. At this time, the effect of some vasoconstrictors is associated with the inhibition of these channels in the presence of PKS. Selective blockers of ATF-sensitive K + -channels are glibenclamide and tolbutamide [Pelletier et all., 2000].

Conclusion

In conclusion, it has been shown that potassium ions are involved in the generalization of the action potential in smooth muscle cells, and that its concentration in the environment depends on the amplitude and duration of the action potential. Indeed, the removal of K + ions from the incubation medium or the addition of K + channel blockers affects the parameters of the action potential. In particular, the addition of K + - channel blocker tetraethylammonium (TEA) to the incubation medium causes an increase in the duration of the action potential. This indicates the involvement of K + -channels and K + ions in the formation of repolarization phases of the action potential in smooth muscle cells.

The action potential of some smooth muscle cells consists of an initial rapid peak component and an ongoing plateau. These action potentials have a more complex ionic nature than the simple action potentials discussed above. In smooth muscle cells, such as the bladder, the initial peak component of the action potential is due to a change in the nature of Ca2 +, which in turn is due to a slowly changing nature of Na +.

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