Limit: 300 words

TITLE:

Role of Nitric Oxide on Small Conductance Calcium- Activated Potassium Channels

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Introduction

Small conductance calcium-activated potassium channels (SK channels) are channels that conduct potassium ions across the cell membrane in response to an increase in the intracellular calcium concentration. By doing so, SK channels play a key role in determining the firing pattern in neurons and also modify how neurons respond to excitatory inputs. Nitric oxide (NO), a gaseous neuromodulator, is a critical component in the regulation of various channels, including SK channels. However, the mechanism by which NO regulates SK channels is unknown.

Purpose

The purpose of this experiment is to investigate the direct role of NO on SK channels through their heterologous expression in transfected HEK (Human Embryonic Kidney) cells.

Methods

A vector expressing both the SK channel from the gastropod *Helisoma trivolis* and green fluorescent protein (GFP) were transfected into HEK cells. Whole cell recordings were performed on the transfected HEK cells in the presence and absence of NO. A voltage ramp from -60 to +60 mV (1s) was used to characterize and quantify SK channel-mediated currents.

Results

SK channels were inhibited by NO under physiological conditions.

Discussion

NO can modify proteins via phosphorylation or nitrosylation. Our preliminary data suggest that NO does not affect SK channels by phosphorylation. Therefore, NO may act by nitrosylating critical cysteine residues in SK channels.

Future Experiment

A critical cysteine residue within the pore region (active site) of the SK channel will be mutated to an alanine residue, which cannot be nitrosylated. If NO has no effect on the mutated channel, I will conclude that the critical cysteine is important in mediating NO's effect on the SK channel.