

Pimaric acid inhibits contraction of pulmonary artery via BKCa channel activation

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Large-conductance Ca^{2+} -activated K^{+} (BKCa) channels are expressed in vascular smooth muscle cells and regulate the membrane excitability. Activation of BKCa channels following membrane depolarization and/or cytosolic $[\text{Ca}^{2+}]$ increase causes membrane hyperpolarization and subsequent cytosolic $[\text{Ca}^{2+}]$ decrease. Therefore, BKCa channels are recognized as a key factor for the negative feedback regulation of vascular tone. Pimaric acid is a common resin acid naturally contained in pine rosin. We previously reported that pimaric acid activated BKCa channels and slightly blocked voltage-dependent Ca^{2+} channels. However, the effects of pimaric acid on contractile response of vascular smooth muscles are still unclear. In the present study, we examined the effects of pimaric acid on contraction of pulmonary artery. Pulmonary arteries were isolated from male Sprague-Dawley rats and contracted with high K^{+} solution. The high K^{+} -induced contraction was reduced by pimaric acid in a concentration-dependent manner (1-100 μM). Quantitative real-time PCR data revealed that the α and $\beta 1$ subunits of BKCa channel were highly expressed in human pulmonary arterial smooth muscle cells. These results indicate that pimaric acid enhances the activity of BKCa channels and results in the relaxation of pulmonary arterial smooth muscles.