BK_{Ca} current and BK_{Ca} peak current density in aortic VSMC increased significantly as the time of exposure to cold went until the 6th week exposure to cold. Furthermore, the expression of L-type Cav1.2 channelα_1C subunit mRNA in the cold-treated rats were higher than those in the control rats.

Conclusions Cold stress increased expression of L-type Cav1.2 channelα_1C subunit mRNA, indicate that cytoplasmic Ca^{2+} concentration increased with cold-treated extended at 6 weeks in the beginning, resulting in increasing blood pressure. BK_{Ca} channel currents increased in this progress as the negative-feedback regulators of vascular tone. Our results indicated that activation of BK_{Ca} channel could reduce blood pressure (7th and 8th week) in hypertensive subjects.

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BKCA CHANNEL ACT AS A NEGATIVE-FEEDBACK REGULATOR IN THE DEVELOPMENT OF COLD-INDUCED HYPERTENSION

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Objectives Cold is an important factor contributing to the high incidence of hypertension. In order to explore the mechanisms of cold-induced hypertension. This study observed whether Large conductance calcium-activated potassium channel (BK_{Ca}) on vascular smooth muscle cells (VSMC) played a role in the development of hypertension during chronic exposure to cold.

Methods Forty-eight rats were divided into cold-treated group and control group randomly, then, one subgroup of the cold-treated and one subgroup of control rats were sacrificed at 2, 4, 6 and 8 weeks.

Results Systolic blood pressure started to rise after 2 weeks of cold exposure and continued to increase, reaching a maximal level by the 6th week of exposure to cold, and then pressure begin to decrease slowly throughout the remainder of the experiment. The