**MODULATION OF AORTIC INWARD RECTIFIER POTASSIUM CHANNELS ACTIVITY BY HYDROGEN SULFIDE**

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The aim of the present work was to study the role of inward rectifier potassium (KIR) channels in mediating relaxation of rat thoracic aorta, and the activity of KIR2.1 channel expressed in Xenopus oocytes in response to H2S donor “sodium sulfide (Na2S)” using PowerLab tissue bath system and two-electrode voltage-clamp technique. Na2S (1-6 mM)-induced relaxation in aortic rings was inhibited by BaCl2 with IC50 from 2.368 to 2.387mM and percentage of relaxation was reduced from 53.05±3.212% to 41.22±7.317%. Application of Na2S (1mM), caused a marked increment in instantaneous and steady state KIR2.1 currents by 46.45% and 54.97%, respectively. Furthermore, addition of barium ion (Ba+2) significantly inhibited instantaneous and steady state KIR2.1 currents by 73% and 65.9%. Combination of Ba+2 with Na2S, decreased instantaneous and steady state KIR2.1 currents by 79.2% and 83.8% at 5mM-K+ concentration. In the current-voltage relationship experiments, Na2S demonstrated a stronger rectification of KIR2.1 current in a testing potential -100mV, when the currents were evoked by repolarizing pulses from holding (0 mV) to test potentials (+60 to -100 mV) in 20mV decrement. Na2S failed to change KIR2.1 channel chord conductance calculated from current-voltage relationship from current values of the voltages between -60mV and -100mV in both K+ concentrations 5mM and 30mM. On the other hand, application of Ba+2 and their combination with Na2S markedly attenuated KIR2.1 currents in voltages between -80 to -100mV and chord conductance