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MODULATION OF AORTIC INWARD RECTIFIER POTASSIUM2.1 CHANNEL ACTIVITY BY SULFUR DIOXIDE

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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 sulfur dioxide (SO2) derivatives 1:3 M/M sodium bisulfite (NaHSO3) and sodium sulfite (Na2SO3) PowerLab tissue bath system and two-electrode voltage-clamp technique. SO2 derivatives (3–9 mM)-induced relaxation in aortic rings was inhibited by BaCl2 with IC50 from 6.21 mM to 7 mM and maximum relaxation (Emax) was reduced from 62.026 ±6.527 to 36.085±9.382%. Application of SO2 derivatives (3mM), caused a marked increment in initial and steady state KIR2.1 currents by 54.55% and 60.74%, respectively. Furthermore, addition of barium ion (Ba+2; 100 μM) significantly inhibited initial and steady state KIR2.1 currents by 51.91% and 73.19%, while, when combined with SO2 significantly caused a decrement only in KIR2.1 steady state currents by 69.2% at 5mM-K+ concentration. In the current-voltage relationship experiments, SO2 derivatives demonstrated a stronger rectification of KIR2.1 current in a testing potential -100mV, when the currents were evoked by repolarizing pulses from holding to test potentials (+60 to -100 mV) in 20 mV decrement. SO2 failed to change KIR2.1 channel chord conductance calculated from current-voltage relationship from current values of the voltages between –60 mV and -100 mV in both K+ concentrations 5 mM and 30 mM. On the other hand, application of Ba+2 and their combination with SO2 markedly attenuated KIR2.1 currents in voltages between –80 to –100mV and chord conductance. These results provide the first evidence for activation of KIR2.1 channel by SO2 derivatives, and the stimulatory effect of SO2 derivatives is voltage-dependent.