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Effect of N-acetyl cysteine on conductance of KATP channels in thoracic aorta rings

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N-acetyl cysteine (NAC) is a precursor in the synthesis of glutathione and acts as an antioxidant, anti-inflammatory and vasodilating substance [1, 2]. However, the signaling pathway involved in NAC effects is not entirely elucidated. In the present study, we aimed to investigate the effect of N-acetyl cysteine on conductance of KATP channels in thoracic aorta rings by using single channel recordings.

Cultured thoracic aorta cells were used to record KATP channel currents. Cells were divided into four groups as control, 2 mM NAC, 5 mM NAC, and 10 mM NAC. The patch-clamp method was used to record single-channel currents from the cell membrane in the cell-attached mode. Pipette voltage was set to +50 mV, and the experiments were carried out at 37°C. Single-channel currents were recorded. KATP channel conductance was calculated from single channel recordings.

The mean conductance were 49.45±10.35, 39.65 ± 10.45, 61.84 ± 8.31, and 61.29 ± 6.37 pS in the control, 2mM NAC, 5 mM NAC, and 10 mM NAC groups, respectively. In all treatment groups, single channel conductances were significantly larger than that in the control group (*p*<0.05). There were no statistically significant differences between mean current amplitudes in different NAC groups.

In conclusion, N-acetyl cysteine causes hyperpolarization by increasing the outward K⁺ conductance through KATP channels. This hyperpolarization may be related to vasodilator, antioxidant and anti-inflammatory effects of N-acetyl-cysteine in thoracic aorta smooth muscle cells.

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References

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Keywords: N-acetyl cysteine, KATP channels, patch-clamp, vascular smooth muscle

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