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cGMP/cGKI signaling in peripheral vascular smooth muscle does not involve TrpC3 or TrpC6 channels

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Background

Signaling by intracellular cGMP and cGMP-dependent protein kinase I (cGKI) is the major pathway in vascular smooth muscle, by which endothelial NO regulates vascular tone. The most important targets of cGKI include the myosin-interacting subunit of myosin phosphatase 1, the regulator of G-protein signaling 2, the inositol receptor associated cGKI-substrate, and the BK channel. Recent evidence suggest that TrpC channels are also targets of cGKI in smooth muscle and mediate, at least partially, the relaxant effects of cGMP.

Results

We tested this new concept by investigating the role of cGMP/cGKI signaling on vascular tone and peripheral resistance using cGKI-, TrpC6-, and TrpC3-knock-out mice. We found no differences in the response to alpha-adrenergic stimulation with respect to the contractility of thoracic aorta or to the increase in peripheral resistance using preparations from the knock-out models. Activation of cGKI by 8-Br-cGMP diminished aortic tone and peripheral resistance to a similar extent in control, TrpC6-/-, and TrpC3-/- mice. No effect of 8-Br-cGMP was observed in preparations from smooth-muscle specific cGKI-/- mice.

Conclusion

The results suggest that cGMP/cGKI signaling in aorta and peripheral vessels from mice does not require TrpC6 or TrpC3 channels.

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