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Titel des Beitrags:  
cGMP-dependent protein kinase mediates NO- but not acetylcholine-induced dilations in resistance vessels in vivo.

Abstract:  
cGMP and cGMP-dependent protein kinase type I (cGKI) mediate the dilation of large vessels in response to NO and acetylcholine (ACh). However, the physiological significance of the NO/cGMP/cGKI pathway in resistance vessels is controversial. Here, we analyzed NO- and ACh-induced dilations of arterioles in cGKI-deficient (cGKI-/-) or endothelial NO synthase-deficient (eNOS-/-) mice. Mean arterial pressure was similar in cGKI-/- and wild-type mice (105 mm Hg). Pressure drops in response to intracarotid bolus application of the NO donor sodium nitroprusside (SNP) were almost abolished in cGKI-/- mice, whereas ACh-induced pressure decreases remained intact in cGKI-/- and eNOS-/- mice. The direct observation of arterioles in the cremaster muscle by intravital microscopy showed impaired SNP-induced dilations in cGKI-/- mice (by 80%) and normal ACh-induced dilations in cGKI-/- and eNOS-/- mice. ACh-induced dilations in eNOS-/- mice were attenuated by iberiotoxin (by 50%), indicating that they were mediated in part by Ca2+-activated K+ channels, but not by inhibitors of cyclooxygenase or p450-monoxygenases. We conclude that cGMP and cGKI are the major effectors of NO to induce acute dilations of murine resistance vessels. However, the NO/cGMP/cGKI pathway is not essential for ACh-induced dilation of arterioles and for basal blood pressure regulation in
mice.

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