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cGMP compartments in cardiac myocytes

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cGMP is the common second messenger for the cardiovascular effects of nitric oxide (NO) and natriuretic peptides, such as ANP and BNP, which activate the soluble and particulate forms of guanylyl cyclase, respectively. However, natriuretic peptides and NO donors exert distinct effects on cardiac and vascular smooth muscle function. We therefore tested whether these differences are due to an intracellular compartmentation of cGMP and evaluated the role of phosphodiesterase (PDE) subtypes in this process.

Subsarcolemmal cGMP signals were monitored in adult rat cardiomyocytes by expression of the rat olfactory cyclic nucleotide-gated (CNG) channel α-subunit and recording of the associated cGMP-gated current (I_{CNG}).

Our main results indicate that: (1) the particulate cGMP pool is readily accessible at the plasma membrane, whereas the soluble pool is not; (2) PDE5 controls the soluble but not the particulate pool, whereas the latter is under the exclusive control of PDE2; (3) both pools are oppositely regulated by cGMP-dependent protein kinase (PKG), with PKG inhibiting the soluble pool (via PDE5 activation) and activating the particulate pool (in a PDE-independent manner).

These differential spatiotemporal distributions and regulations of cGMP signals may contribute to the specific effects of natriuretic peptides and NO donors on cardiac function.