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cGMP-dependent and -independent angiogenesis-related properties of nitric oxide

Anastasia Pyriochou*[1], Zongmin Zhou[2] and Andreas Papapetropoulos[1,2]

Address: [1]Laboratory of Molecular Pharmacology, Department of Pharmacy, University of Patras, Patras, Greece and [2]G.P. Livanos and M. Simou Laboratories, Evangelismos Hospital, Critical Care Department, University of Athens School of Medicine, Athens, Greece

Email: Anastasia Pyriochou* - anastassouli@yahoo.com

* Corresponding author

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Nitric oxide exerts a stimulatory role during postnatal angiogenesis. Although soluble guanylyl cyclase (sGC) mediates many of the effects of nitric oxide (NO) in the vascular system, the contribution of cGMP-dependent vs cGMP-independent pathways in NO-induced angiogenesis remains unclear. Herein, we determined the effects of a NO donor (sodium nitroprusside; SNP) and a NO-independent sGC activator (BAY 41–2272) in the growth and migration of endothelial cells devoid of sGC. Lack of enzymatically active sGC in rat aortic EC (RAEC) was demonstrated through the inability of these cells to accumulate cGMP upon exposure to SNP. However, treatment of RAEC with SNP promoted a modest increase in their proliferation and migration that was dependent on extracellular signal regulated kinase1/2 activation. Moreover, when RAEC were exposed to vascular endothelial growth factor we observed a 5-fold increase in migration that was inhibited by NO synthase, but not sGC, inhibition. Infection of cells with adenoviruses containing sGC greatly increased the efficacy of SNP as a mitogenic and migratory stimulus. We conclude that NO is capable of stimulating EC proliferation and mobility in the absence of sGC; however, increased intracellular levels of cGMP following sGC activation greatly amplify the angiogenic potential of NO.

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