A heretical view on the role of NO and cGMP in vascular proliferative diseases.

Abstract: Endogenous nitric oxide (NO), and possibly NO-releasing drugs, can both inhibit and promote vascular proliferative disorders, such as atherosclerosis and restenosis. The cell types and signaling pathways that mediate these opposing effects are controversial. It is widely assumed that the NO-mediated synthesis of the second messenger cGMP and the activation of cGMP-dependent protein kinase type I (cGKI) inhibits the proliferation of vascular smooth muscle cells and, thus, vascular remodeling. However, recent data from transgenic mouse models challenge this view. Here, we propose that cGMP signaling through cGKI might promote vasculoproliferative processes and their clinical complications. This new concept has important implications for the use of cGMP-elevating drugs in humans and might help to identify novel therapeutic strategies for vascular proliferative diseases.

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