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The Role of Antioxidants in Nitric-Oxide Induced Apoptosis in Vascular Smooth Muscle Cells

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THE ROLE OF ANTIOXIDANTS IN NITRIC-OXIDE INDUCED APOPTOSIS IN VASCULAR SMOOTH MUSCLE CELLS

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Apoptosis of vascular smooth muscle cells (VSMC) has been identified as having an important role in the prevention of neointimal hyperplasia. This is due to the ability of NO to induce programmed cell death, or apoptosis, of VSMC following injury. While the regulation of VSMC apoptosis is complex and not fully understood, it has been shown that upon VSMC stimulation with NO there is upregulation of the tumor suppressor gene p53. Previous studies indicate that down-regulation of p53 results in an increase in cellular ROS. Since p53 is a transcription factor known to affect the transcription of antioxidant proteins, it has been speculated that increased ROS levels are a result of the absence of p53 transcribed antioxidant proteins. ROS levels in VSMC were measured using flow cytometry both at baseline and following NO exposure. At baseline, ROS levels were significantly higher in p53-/- than p53+/+ VSMC. In response to NO treatment, the ROS increase was more pronounced in p53-/- than p53+/+ VSMC. Western blot analysis was performed to identify which pro- and anti-oxidant proteins were affected. The antioxidant protein most differentially expressed between p53-/- and p53+/+ VSMC was peroxiredoxin (PRX)-III. Appropriately, PRX-III is a mitochondrion specific H2O2 scavenging enzyme and has been cited as a critical regulator of mitochondrial H2O2. The prospect of PRX-III providing a p53 mediated protective effect in p53+/+ VSMC matches well with the current understanding of PRX-III's role in mitochondria as well as the model of NO-induced apoptosis.