INTRODUCTION

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Biomedical interest in nitric oxide has been grown rapidly since endothelium-derived relaxing factor was reported to be indistinguishable from NO, a freely diffusible free radical that decomposes spontaneously to nitrite and nitrate ***(Monocada et al., 1991).***

Nitric oxide, which is synthesized from the amino acid L-arginine by NO synthase, is an endogenous stimulator of soluble guanylate cyclase. At least two types of NO synthase have been purified; a constitutive, calcium-and calmodulin- dependent enzyme and an inducible calcium-independent isoform that requires tetrahydrobiopterin and other cofactors ***(Yuan et al., 1993).*** Studies in several species, including human beings, indicate that normal pressure homeostasis is dependent on basal NO synthesis by the constitutive enzyme in the vascular endothelia, whereas overproduction of NO after endotoxin-or cytokine-mediated activation of the inducible isoform can lead to hypotension ***(Hegesh et al., 1993).***

Recently, NO has been implicated in the pathogenesis of sepsis especially in the mechanism by which sepsis progresses to septic shock ***(Hibbs et al., 1992).***

Endogenous NO production, as measured by serum nitrite and nitrate levels, is increased in clinical conditions characterised by immune stimulation: infection, sepsis, transplantation, and rejection, and in a clinical condition not commonly associated with immune activation as shock without sepsis ***(Hector et al., 1996).***