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Reversal of Endotoxin-Induced Hypotension by Inhibition of Inducible Nitric Oxide Synthase Activity is Associated with Improved Oxidative Status in Rat Heart, Aorta and Mesenteric Artery

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Abstract: Overproduction of reactive oxygen and nitrogen species leads to oxidative stress and decreased total antioxidant capacity, which is responsible for high mortality from several diseases such as endotoxic shock. Nitric oxide (NO) produced by inducible NO synthase (iNOS) during endotoxemia is the major cause of vascular hyporeactivity, hypotension and multiple organ failure. We investigated whether NO-mediated oxidative stress in rat heart, aorta and mesenteric artery is involved in the attenuation of endotoxin-induced hypotension by inhibition of iNOS. In conscious male Wistar rats, injection of endotoxin (10 mg/kg, i.p.) caused a gradual fall in mean arterial pressure (MAP) for 4 hours and increased serum and tissue nitrite levels. These effects of endotoxin were prevented by selective inhibition of iNOS with phenylene-1,3-bis[ethane-2-isothiourea] dihydrobromide (1,3-PBIT) (10 mg/kg, i.p.; 1 hour after endotoxin). Myeloperoxidase (MPO) activity was increased in the heart and aorta and decreased in the mesenteric artery by endotoxin which was reversed by 1,3-PBIT. Endotoxin caused a decrease in products of lipid peroxidation in the tissues, which is prevented by 1,3-PBIT. These data suggest that NO-mediated decrease in MAP during endotoxemia is associated with decreased oxidative stress in the heart, aorta and mesenteric artery and that the beneficial effects of iNOS inhibitors on the endotoxin-induced hypotension may be due to restoration of total antioxidant capacity.

Key Words: Rat, endotoxin, hypotension, inducible nitric oxide synthase, lipid peroxidation

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