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J Cardiovasc Pharmacol. 2014 Feb;63(2):144-51. doi: 10.1097/FJC.0000000000000034.

Low-dose Estrogen Is as Effective as High-dose Treatment in Rats With Postmenopausal Hypertension.

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Abstract

: This study was conducted to test the hypothesis that 17 β -estradiol therapy improves redox balance by decreasing reactive oxygen species production and increasing nitric oxide (NO) bioavailability, favoring Akt pathway activation and resulting in a better autonomic vascular control. Ovariectomized female Wistar rats were divided into 4 groups: (1) vehicle (VL) and animals treated with a pellet of 17 β -estradiol for 21 days; (2) low dose (LE; 0.05 mg); (3) medium dose (ME; 0.2 mg); and (4) high dose (HE; 0.5 mg). Arterial pressure and its sympathetic nervous system modulation were evaluated by spectral analysis. Nitric oxide synthase and NADPH oxidase (Nox) activities, H₂O₂ concentration, redox status (GSH/GSSG), protein expression of Trx-1 and p-Akt/Akt were evaluated in the aorta, whereas NO metabolites were measured in the serum. Estrogen-treated groups showed a significant decrease in arterial pressure and sympathetic vascular drive. Redox status was significantly improved and NADPH oxidase and H₂O₂ were decreased in all estrogen-treated groups. Estrogen also induced an enhancement in NO metabolites, nitric oxide synthase activity, and Akt phosphorylation. This study demonstrated that estrogen treatment to ovariectomized rats induced cardioprotection, which was evidenced by reduced blood pressure variability and vascular sympathetic drive. These effects were associated with an improved redox balance and Akt activation, resulting in an enhanced NO bioavailability.

PMID: 24157955 [PubMed - in process]

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