

## Effects of Tempol on Blood Pressure and Tissue Oxidative Stress in DOCA-salt and L-NAME-induced Hypertension

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**Background :** Effects of oxidative stress on the development of hypertension were examined in deoxycorticosterone acetate (DOCA)-salt or NG-nitro-L-arginine (L-NAME)-treated rats.

**Methods :** Sprague-Dawley rats were treated with DOCA (200 mg/kg, subcutaneous)-salt or L-NAME (40 mg/L in drinking water) for 4 weeks. To reduce the tissue oxidative stress, 4-hydroxyl-2,2,6,6-tetramethylpiperidine-1-oxyl (Tempol, 3 mM/L in drinking water) was cotreated. The expression of endothelial nitric oxide synthase (eNOS) and nitrotyrosine was determined in the renal cortex and aorta.

**Results :** Tempol prevented the development of hypertension in DOCA-salt rats, whereas it was without effect in L-NAME-treated rats. In DOCA-salt rats, the eNOS expression in the renal cortex was increased, the degree of which was decreased by Tempol. In the aorta, the eNOS expression was significantly decreased, however, which was not affected by Tempol. The expression of nitrotyrosine was markedly decreased both in the renal cortex and the aorta, the degree of which was not affected by Tempol in the aorta but was more pronounced in the renal cortex. In L-NAME-treated rats, the expression of eNOS in the renal cortex was significantly increased, which was blocked by Tempol. The expression of eNOS was slightly decreased in the aorta, which was not affected by Tempol. The expression of nitrotyrosine was not significantly affected in the renal cortex. It was markedly decreased in the aorta, which was more pronounced when Tempol was cotreated.

**Conclusion :** The blockade of oxidative stress may attenuate the development of hypertension and provide tissue protection in DOCA-salt rats. The blockade of oxidative stress may also provide tissue protection in L-NAME-induced hypertension, although it was without effect on the blood pressure.