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Antihypertensive responses of vasoactive androgens in an *in vivo* experimental model of preeclampsia.

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Highlights:

- *Vasorelaxation of androgens elicits antihypertensive responses.*
- *Antihypertensive responses of diverse androgens in preeclampsia syndrome.*
- *Androgens are vasoactive in the cardiovascular system.*
- *Androgen deficiency during pregnancy may trigger preeclampsia*
- *The data suggest a possible first-line therapy for an acute hypertensive emergency.*

Abstract

Dehydroepiandrosterone (DHEA), testosterone (TES) and its 5-reduced metabolites induce a nongenomic vasorelaxation in several vascular beds of mammals; similarly these hormones produce systemic hypotensive and antihypertensive responses in normotensive and hypertensive male rats. Thus, it was hypothesized that the antihypertensive response of androgens, whose levels are elevated during gestation, protect against gestational hypertension. An animal model of preeclampsia was induced in female Wistar rats using DOCA-salt-treated pregnant (PT) and normal pregnant (NP) rats. *In vivo* experiments in conscious rats revealed that bolus intravenous injections of DHEA, TES, 5 α - or 5 β -dihydrotestosterone (-DHT) log -1.0 to 2.0 $\mu\text{mol k}^{-1} \text{min}^{-1}$, produced substantial transient reductions in arterial blood pressure (BP), without significant changes in heart rate (HR). Mean arterial blood pressure (MAP) was reduced significantly in both groups. PT rats were more sensitive to the antihypertensive responses of androgens than NP. DHEA and 5 β -DHT were the most potent to reduce MAP: 66 \pm 07 and 69 \pm 2.0 mmHg in PT but only 33 \pm 0.5 and 35 \pm 1.2 mmHg in NP rats, respectively. In isolated aortas of PT and NP, the

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