Testosterone and Sympathetic Nerve Activity During Pregnancy

To the Editor:

I read with great interest the article by Chinnathambi et al.1 that examined the influence of testosterone on the endothelial nitric oxide (NO) system. The role of testosterone in gestational cardiovascular function has not been adequately examined, despite the fact that testosterone levels are exaggerated in preeclamptic and polycystic ovary syndrome pregnancies. Chinnathambi et al.1 used a well-controlled experimental model in which pregnant rats were injected with vehicle or testosterone propionate, with the goal of increasing plasma testosterone to mimic levels observed during preeclampsia. The authors report that increased testosterone was associated with elevated blood pressure and blunted NO-mediated vasodilation. This novel study provides critical mechanistic insight and a potential therapeutic target during gestational hypertension.

Several studies suggest that pregnancy is associated with increased levels of muscle sympathetic nerve activity, and evidence is accumulating to suggest that preeclampsia might be linked to circumstances, where this sympathoexcitation is accompanied by dysfunction of vasodilatory mechanisms.2 Moreover, Sverrisdottir et al.3 reported that polycystic ovary syndrome was associated with elevated resting muscle sympathetic nerve activity, and that the extent of sympathoexcitation was significantly related to testosterone levels. Therefore, it seems plausible that in addition to impairing NO-mediated vasodilation,1 testosterone might also augment the typical surge of muscle sympathetic nerve activity associated with pregnancy. To date, the influence of testosterone on gestational muscle sympathetic nerve activity has not been adequately examined, and it remains unclear whether abnormally high levels of testosterone worsen the sympathetic storm associated with pregnancy.

In addition, obstructive sleep apnea (OSA) is a recognized risk factor for hypertension. Evidence suggests that pregnancy increases the incidence of OSA, and that preeclampsia may exacerbate OSA severity.4 As highlighted by Chinnathambi et al.,1 testosterone is elevated ≥2-fold during preeclampsia. Recent studies suggest that OSA is also ≥5 to 10 times more prevalent in polycystic ovary syndrome women compared with weight-matched controls, and that elevated testosterone may play a key role.5 The relations among testosterone, OSA, and pregnancy remain unclear, but it seems reasonable to speculate that testosterone may be a contributing factor to the increased incidence of OSA during preeclampsia. Is it possible that the testosterone treatment in the study by Chinnathambi et al.1 increased the incidence of OSA, and that this may have contributed to the rise in arterial blood pressure and blunted NO-mediated vasodilation? Future studies might better define the role of testosterone on the presumed endothelial/cardiovascular dysfunction associated with gestational OSA.

In summary, the study by Chinnathambi et al.1 demonstrates that increased testosterone elicits a hypertensive response that is associated with blunted NO-mediated vasodilation in pregnant rats. There is a need for clinical translation of this important work, and future studies might include integrative techniques to simultaneously assess sympathetic neural activity and endothelial function.

Disclosures

None.

Jason R. Carter
Department of Kinesiology and Integrative Physiology
Michigan Technological University
Houghton, MI

Testosterone and Sympathetic Nerve Activity During Pregnancy
Jason R. Carter

Hypertension. 2013;61:e44; originally published online March 25, 2013;
doi: 10.1161/HYPERTENSIONAHA.113.01193
Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2013 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://hyper.ahajournals.org/content/61/5/e44

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Hypertension can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Hypertension is online at:
http://hyper.ahajournals.org//subscriptions/