Response to Roles of Sex Steroid Hormones and Nitric Oxide in the Regulation of Sympathetic Nerve Activity in Women

We thank Dr Tsuda1 for recognizing our work and bringing to light additional aspects of the complex effect of estrogen on sympathetic nerve activity and NO. Indeed, NO-synthase is well known to be upregulated by estrogen, and endothelial function (via flow-mediated dilation) is known to be improved during the high-estrogen phases of the menstrual cycle.2,3 We have recently shown that transdermal estrogen in young women improves flow-mediated dilation, but that this improvement is antagonized by progesterone.4 Unfortunately, simultaneous measurements of sympathetic activity were not performed in those studies. However, data are forthcoming using the same model as that study in which muscle sympathetic nerve activity was measured during independent and combined estrogen and progesterone administration (data from Minson laboratory to be submitted). If NO acts centrally to inhibit sympathoexcitation as suggested, it is plausible that the decline in estrogen associated with menopause could contribute to the greater age-related increase in sympathetic activity in women compared with men.5 Furthermore, the decline in NO in the vasculature to buffer the sympathetic nerve activity could also contribute to the greater increase in arterial pressure for a given rise in muscle sympathetic nerve activity in older women.6 To our knowledge, no study has blocked endogenous NO in the presence and absence of estrogen or progesterone during simultaneous measurement of muscle sympathetic nerve activity. Although the integrative nature of the vascular, neural, and endocrine systems could make interpretation of such data challenging, it would represent a further, and important, step in our understanding of the roles of the sex steroids in women.

Disclosures

None.

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