Role of Estrogens in the Regulation of Membrane Microviscosity

To the Editor:

We read with great interest the recent article by Lew and colleagues1 dealing with the relationship between estrogens and endothelial function in men. The results of their study demonstrated that the suppression of endogenous estrogens with an aromatase inhibitor, anastrozole, resulted in impairment of flow-mediated dilatation of the brachial artery in healthy young men. The authors proposed that estrogens might play a direct regulatory role in endothelial function not only in women but also in men.

There is evidence that estrogens might actively participate in the regulation of membrane function. Whiting et al2 demonstrated the nongenomic effects of steroid hormones on phosphatidylcholine liposome, synaptosomal plasma membranes, and sarcoplasmic reticulum membranes. In the studies we presented earlier, a relationship between membrane fluidity (a reciprocal value of membrane microviscosity) of erythrocytes and estrogens was investigated by means of an electron paramagnetic resonance method. The decreased membrane fluidity of erythrocytes might cause a disturbance in the blood rheologic behavior and the microcirculation, which could contribute, at least in part, to the pathophysiology of circulatory disorders. We demonstrated that hormone (estrogen) replacement therapy significantly increased the membrane fluidity of erythrocytes with a concomitant increase in plasma nitric oxide (NO) metabolite level in postmenopausal women.3 In an in vitro study, we showed that 17β-estradiol, estrone, and estriol increased the membrane fluidity of erythrocytes and improved the rigidity of cell membranes via the NO- and cGMP-dependent mechanism not only in women but also in men.4–6 One hypothesis is that the membrane action of estrogens could be one of the mechanisms responsible for their beneficial effects on the rheologic properties of erythrocyte membranes. In this context, we speculate that estrogen-induced NO production might modulate the membrane microviscosity both in men and women, which could partially explain the preventing effect of estrogens against the vascular complications. It would be necessary to assess more precisely the functional interactions between estrogens and NO in the regulation of membrane microviscosity and their contribution to the vascular function both in men and women.

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