Estrogens are needed for the improvement in endothelium-mediated dilation induced by a chronic increase in blood flow in rat mesenteric arteries

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Résumé en anglais	Resistance arteries play a key role in the control of local blood flow. They undergo outward remodeling in response to a chronic increase in blood flow as seen in collateral artery growth in ischemic disorders. We have previously shown that mesenteric artery outward remodeling depends on the endothelial estrogen receptor alpha. As outward arterial remodeling is associated with improved endothelium-dependent dilation, we hypothesized that estrogens might also play a role in flow-mediated improvement of endothelium-dependent dilation. Local increase in blood flow in first order mesenteric arteries was obtained after ligation of adjacent arteries in three-month old ovariectomized female rats treated with 17-beta-estradiol (OVX+E2) or vehicle (OVX). After 2 weeks, diameter was equivalent in high flow (HF) than in normal flow (NF) arteries with a greater wall to lumen ratio in HF vessels in OVX rats. Acetylcholine-mediated relaxation was lower in HF than in NF vessels. eNOS and caveolin-1 expression level was equivalent in HF and NF arteries. By contrast, arterial diameter was 30% greater in HF than in NF arteries and the wall to lumen ratio was not changed in OVX+E2 rats. Acetylcholine-mediated relaxation was higher in HF than in NF arteries. The expression level of eNOS was higher and that of caveolin-1 was lower in HF than in NF arteries. Acetylcholine (NO-dependent)-mediated relaxation was partly inhibited by the NO-synthesis blocker L-NAME in OVX rats whereas L-NAME blocked totally the relaxation in OVX+E2 rats. Endothelium-independent relaxation (sodium nitroprusside) was equivalent in OXV and OVX+E2 rats. Similarly, serotonin- and phenylephrine-mediated contractions were higher in HF than in NF arteries in both OVX and OVX+E2 rats in association with high ratio of phosphorylated ERK1/2 to ERK1/2. Thus, we demonstrated the essential role of endogenous E2 in flow-mediated improvement of endothelium (NO)-mediated dilatation in rat mesenteric arteries
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Liens

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