Role of estrogens and age in flow-mediated outward remodeling of rat mesenteric resistance arteries

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Résumé en anglais	In resistance arteries, a chronic increase in blood flow induces hypertrophic outward remodeling. This flow-mediated remodeling (FMR) is absent in male rats aged 10 mo and more. As FMR depends on estrogens in 3-mo-old female rats, we hypothesized that it might be preserved in 12-mo-old female rats. Blood flow was increased in vivo in mesenteric resistance arteries after ligation of the side arteries in 3- and 12-mo-old male and female rats. After 2 wk, high-flow (HF) and normal-flow (NF) arteries were isolated for in vitro analysis. Arterial diameter and cross-sectional area increased in HF arteries compared with NF arteries in 3-mo-old male and female rats. In 12-mo-old rats, diameter increased only in female rats. Endothelial nitric oxide synthase expression and endothelium-mediated relaxation were higher in HF arteries than in NF arteries in all groups. ERK1/2 phosphorylation, NADPH oxidase subunit expression levels, and arterial contractility to KCl and to phenylephrine were greater in HF vessels than in NF vessels in 12-mo-old male rats only. Ovariectomy in 12-mo-old female rats induced a similar pattern with an increased contractility without diameter increase in HF arteries. Treatment of 12-mo-old male rats and ovariectomized female rats with hydralazine, the antioxidant tempol, or the angiotensin II type 1 receptor blocker candesartan restored HF remodeling and normalized arterial contractility in HF vessels. Thus, we found that FMR of resistance arteries remains efficient in 12-mo-old female rats compared with age-matched male rats. A balance between estrogens and vascular contractility might preserve FMR in mature female rats.
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