Involvement of angiotensin II in the remodeling induced by a chronic decrease in blood flow in rat mesenteric resistance arteries

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Résumé en anglais	Blood flow reduction induces inward remodeling of resistance arteries (RAs). This remodeling occurs in ischemic diseases, diabetes and hypertension. Nonetheless, the effect of flow reduction per se, independent of the effect of pressure or metabolic influences, is not well understood in RA. As angiotensin II is involved in the response to flow in RA, we hypothesized that angiotensin II may also be involved in the remodeling induced by a chronic flow reduction. We analyzed the effect of angiotensin I-converting enzyme inhibition (perindopril) and angiotensin II type 1 receptor blockade (candesartan) on inward remodeling induced by blood flow reduction in vivo in rat mesenteric RAs (low flow (LF) arteries). After 1 week, diameter reduction in LF arteries was associated with reduced endothelium-dependent relaxation and lower levels of eNOS expression. Superoxide production and extracellular signal-regulated kinases 1/2 (ERK1/2 phosphorylation were higher in LF than in normal flow arteries. Nevertheless, the absence of eNOS or superoxide level reduction (tempol or apocynin) did not prevent LF remodeling. Perindopril and candesartan prevented inward remodeling in LF arteries. Contractility to angiotensin II was reduced in LF vessels by perindopril, candesartan and the ERK1/2 blocker PD98059. ERK1/2 activation (ratio phospho-ERK/ERK) was higher in LF arteries, and this activation was prevented by perindopril and candesartan. ERK1/2 inhibition in vivo (U0126) prevented LF-induced diameter reduction. Thus, inward remodeling because of blood flow reduction in mesenteric RA depends on unopposed angiotensin II-induced contraction and ERK1/2 activation, independent of superoxide production. These findings might be of importance in the treatment of vascular disorders.
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