Role of angiotensin II in the remodeling induced by a chronic increase in flow in rat mesenteric resistance arteries

Titre	Role of angiotensin II in the remodeling induced by a chronic increase in flow in rat mesenteric resistance arteries
Type de publication	Article de revue
Auteur	Cousin, M. [1], Custaud, Marc-Antoine [2], Baron-Menguy, Céline [3], Toutain, Bertrand [4], Dumont, O. [5], Guihot, Anne-Laure [6], Vessieres, Emilie [7], Subra, Jean-François [8], Henrion, Daniel [9], Loufrani, Laurent [10]
Editeur	American Heart Association
Type	Article scientifique dans une revue à comité de lecture
Année	2010
Langue	Anglais
Date	2010
Numéro	1
Pagination	109 - 15
Volume	55
Titre de la revue	Hypertension
ISSN	1524-4563
Mots-clés	Angiotensin II Type 1 Receptor Blockers/pharmacology [11], Angiotensin II/metabolism/physiology [12], Angiotensin-Converting Enzyme Inhibitors/pharmacology [13], Animals [14], Antioxidants/pharmacology [15], Benzimidazoles/pharmacology [16], Blood Flow Velocity [17], Blotting, Western [18], Butadienes/pharmacology [19], Cyclic N-Oxides/pharmacology [20], Extracellular Signal-Regulated MAP Kinases/antagonists & inhibitors/metabolism [21], Hydralazine/pharmacology [22], Male [23], Mesenteric Arteries/metabolism/pathology/physiopathology [24], Mice [25], Mice, Knockout [26], Microscopy, Confocal [27], NG-Nitroarginine Methyl Ester/pharmacology [28], Nitric Oxide Synthase Type III/antagonists & inhibitors/genetics/metabolism [29], Nitriles/pharmacology [30], Perindopril/pharmacology [31], Rats [32], Rats, Wistar [33], Reactive Oxygen Species/metabolism [34], Spin Labels [35], Tetrazoles/pharmacology [36], Vascular Resistance/drug effects/physiology [37], Vasodilator Agents/pharmacology [38]

Résumé en anglais	Angiotensin II is a potent growth factor involved in arterial wall homeostasis. In resistance arteries, chronic increases in blood flow induce a rise in diameter associated with arterial wall hypertrophy. Nevertheless, the role of angiotensin II in this remodeling is unknown. We investigated the effect of blocking angiotensin II production or receptor activation on flow-induced remodeling of mesenteric resistance arteries. Arteries were ligated in vivo to generate high-flow arteries compared with normal flow (control) vessels located at a distance. Arteries were isolated after 1 week for in vitro analysis. Arterial diameter, media surface, endothelial NO synthase expression, superoxide production, and extracellular signal-regulated kinase 1/2 phosphorylation were higher in high-flow than in control arteries. Angiotensin-converting enzyme inhibition (perindopril) and angiotensin II type 1 receptor blockade (candesartan) prevented arterial wall hypertrophy without affecting diameter enlargement. The nonselective vasodilator hydralazine had no effect on remodeling. Although perindopril and candesartan increased endothelial NO synthase expression in high-flow arteries, hypertrophy remained in rats treated with N(G)-nitro-l-arginine methyl ester and mice lacking endothelial NO synthase. Perindopril and candesartan reduced oxidative stress in high-flow arteries, but superoxide scavenging did not prevent hypertrophy. Both Tempol and the absence of endothelial NO synthase prevented the rise in diameter in high-flow vessels. Extracellular signal-regulated kinase 1/2 activation in high-flow arteries was prevented by perindopril and candesartan and not by hydralazine. Extracellular signal-regulated kinase 1/2 inhibition in vivo (U0126) prevented hypertrophy in high-flow arteries. Thus, a chronic rise in blood flow in resistance arteries induces a diameter enlargement involving NO and superoxide, whereas hypertrophy was associated with extracellular signal-regulated kinase 1/2 activation by angiotensin II.
URL de la notice	http://okina.univ-angers.fr/publications/ua8397 [39]
DOI	10.1161/HYPERTENSIONAHA.108.127456 [40]
Lien vers le document	http://dx.doi.org/10.1161/HYPERTENSIONAHA.108.127456 [40]
Titre abrégé	Hypertension

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