The angiotensin II type 2 receptor activates flow-mediated outward remodelling through T cells-dependent interleukin-17 production

Titre	The angiotensin II type 2 receptor activates flow-mediated outward remodelling through T cells-dependent interleukin-17 production
Type de publication	Article de revue
Auteur	Caillon, Antoine [1], Grenier, Céline [2], Grimaud, Linda [3], Vessieres, Emilie [4], Guihot, Anne-Laure [5], Blanchard, Simon [6], Lelièvre, Eric [7], Chabbert, Marie [8], Foucher, Etienne D. [9], Jeannin, Pascale [10], Beauvillain, Céline [11], Abraham, Pierre [12], Loufrani, Laurent [13], Delneste, Yves [14], Henrion, Daniel [15]
Pays	Royaume-Uni
Editeur	Oxford University Press (OUP)
Ville	Oxford
Type	Article scientifique dans une revue à comité de lecture
Année	2016
Langue	Anglais
Date	21 Juin 2016
Numéro	1
Pagination	515-25
Volume	112
Titre de la revue	Cardiovascular Research
ISSN	0008-6363
Mots-clés	Angiotensin II type 2 receptor [16], Blood flow [17], Collateral artery growth [18], Interleukin-17 [19]

Résumé en anglais	AIMS: The angiotensin II type 1 receptor (AT1R) through the activation of immune cells plays a key role in arterial inward remodelling and reduced blood flow in cardiovascular disorders. On the other side, flow (shear stress)-mediated outward remodelling (FMR), involved in collateral arteries growth in ischaemic diseases, allows revascularization. We hypothesized that the type 2 receptor (AT2R), described as opposing the effects of AT1R, could be involved in FMR. METHODS AND RESULTS: We studied FMR using a model of ligation of feed arteries supplying collateral pathways in the mouse mesenteric arterial bed in vivo. Seven days after ligation, diameter increased by 30% in high flow (HF) arteries compared with normal flow vessels. FMR was absent in mice lacking AT2R. At Day 2, T lymphocytes expressing AT2R were present preferentially around HF arteries. FMR did not occur in athymic (nude) mice lacking T cells and in mice treated with anti-CD3ε antibodies. AT2R activation induced interleukin-17 production by memory T cells. Treatment of nude mice or AT2R-deficient mice with interleukin-17 restored diameter enlargement in HF arteries. Interleukin-17 increased NO-dependent relaxation and matrix metalloproteinases activity, both important in FMR. Remodelling of feeding arteries in the skin flap model of ischaemia was also absent in AT2R-deficient mice and in anti-interleukin-17-treated mice. Finally, remodelling, absent in 12-month-old mice, was restored by a treatment with the AT2R non-peptidic agonist C21. CONCLUSION:AT2R-dependent interleukin-17 production by T lymphocyte is necessary for collateral artery growth and could represent a new therapeutic target in ischaemic disorders.
URL de la notice	http://okina.univ-angers.fr/publications/ua15168 [20]
DOI	10.1093/cvr/cvw172 [21]
Lien vers le document	http://cardiovascres.oxfordjournals.org/content/112/1/515 [22]
Titre abrégé	Cardiovasc Res

Liens

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