Epidermal growth factor receptor mediates the vascular dysfunction but not the remodeling induced by aldosterone/salt

Titre	Epidermal growth factor receptor mediates the vascular dysfunction but not the remodeling induced by aldosterone/salt
Type de publication	Article de revue
Auteur	Griol-Charhbili, V. [1], Fassot, Céline [2], Messaoudi, S. [3], Perret, C. [4], Agrapart, V. [5], Jaisser, F. [6]
Editeur	American Heart Association
Type	Article scientifique dans une revue à comité de lecture
Année	2011
Langue	Anglais
Date	2011
Numéro	2
Pagination	238 - 44
Volume	57
Titre de la revue	Hypertension
ISSN	1524-4563
Mots-clés	Acetylcholine/pharmacology [7], Aldosterone/pharmacology [8], Angiotensin II/pharmacology [9], Animals [10], Aorta/drug effects/metabolism/physiopathology [11], Blotting, Western [12], Carotid Arteries/drug effects/physiopathology [13], Dose-Response Relationship, Drug [14], Gene Expression/drug effects [15], Genotype [16], Hemodynamics/drug effects [17], Male [18], Mice [19], Mice, Mutant Strains [20], NADPH Oxidase/genetics/metabolism [21], Nephrectomy [22], Nitric Oxide Synthase Type III/metabolism [23], Nitroprusside/pharmacology [24], Phenylephrine/pharmacology [25], Receptor, Epidermal Growth Factor/genetics/metabolism/physiology [26], Reverse Transcriptase Polymerase Chain Reaction [27], Signal Transduction/drug effects [28], Vasoconstriction/drug effects [29], Vasoconstrictor Agents/pharmacology [30], Vasodilator Agents/pharmacology [31]

Résumé en anglais	Pathophysiological aldosterone (aldo)/mineralocorticoid receptor signaling has a major impact on the cardiovascular system, resulting in hypertension and vascular remodeling. Mineralocorticoids induce endothelial dysfunction, decreasing vasorelaxation in response to acetylcholine and increasing the response to vasoconstrictors. Activation of the epidermal growth factor receptor (EGFR) is thought to mediate the vascular effects of aldo, but this has yet to be demonstrated in vivo. In this study, we analyzed the molecular and functional vascular consequences of aldo-salt challenge in the waved 2 mouse, a genetic model with a partial loss of EGFR tyrosine kinase activity. Deficient EGFR activity is associated with global oxidative stress and endothelial dysfunction. A decrease in EGFR activity did not affect the arterial wall remodeling process induced by aldo-salt. By contrast, normal EGFR activity was required for the aldo-induced enhancement of phenylephrine- and angiotensin II-mediated vasoconstriction. In conclusion, this in vivo study demonstrates that EGFR plays a key role in aldosterone-mediated vascular reactivity.
URL de la notice	http://okina.univ-angers.fr/publications/ua8238 [32]
DOI	10.1161/HYPERTENSIONAHA.110.153619 [33]
Lien vers le document	http://dx.doi.org/10.1161/HYPERTENSIONAHA.110.153619 [33]
Titre abrégé	Hypertension

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