The endothelial mineralocorticoid receptor regulates vasoconstrictor tone and blood pressure

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Résumé en anglais	Pathophysiological aldosterone (aldo)/mineralocorticoid receptor (MR) signaling has significant effects on the cardiovascular system, resulting in hypertension and cardiovascular remodeling; however, the specific contribution of the vascular MR to blood pressure regulation remains to be established. To address this question, we generated a mouse model with conditional overexpression of the MR in endothelial cells (MR-EC). In basal conditions, MR-EC mice developed moderate hypertension that could be reversed by canrenoate, a pharmacological MR antagonist. MR-EC mice presented increased contractile response of resistance arteries to vasoconstrictors (phenylephrine, thromboxane A(2) analog, angiotensin II, and endothelin 1) in the absence of vascular morphological alterations. The acute blood pressure response to angiotensin II or endothelin 1 infusion was increased in MR-EC mice compared with that in littermate controls. These observations demonstrate that enhanced MR activation in the endothelium generates an increase in blood pressure, independent of stimulation of renal tubular Na(+) transport by aldo/MR or direct activation of smooth muscle MR and establish one mechanism by which endothelial MR activation per se may contribute to impaired vascular reactivity.
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