The endothelial mineralocorticoid receptor: mediator of the switch from vascular health to disease

Resumo
Purpose of review Endothelial dysfunction is an early feature of vascular disease induced by cardiovascular risk factors (CRFs). In growing populations with obesity, diabetes, hypertension, and heart failure, mineralocorticoid receptor antagonism improves endothelial function. This review summarizes recent advances in our understanding of the specific role of endothelial cell mineralocorticoid receptor in vascular function in health and disease. Recent findings Using transgenic mice with mineralocorticoid receptor expression specifically modulated in endothelial cells, recent studies support the emerging concept that while endothelial cell mineralocorticoid receptor may be protective in health, in the presence of CRFs, endothelial cell mineralocorticoid receptor activity contributes to endothelial dysfunction and progression of vascular disease. Proposed mechanisms include a role for endothelial cell mineralocorticoid receptor in decreased nitric oxide production and bioavailability, increased vascular oxidative stress, regulation of epithelial sodium channels that enhance vascular stiffness, and increased endothelial cell adhesion molecules promoting inflammation. The role of endothelial cell mineralocorticoid receptor may also depend on the sex, race, or vascular bed involved. Summary Recent advances support the idea that endothelial cell mineralocorticoid receptor is a mediator of the switch from vascular health to disease in response to CRFs. Further investigation of the molecular mechanism is underway to identify therapeutic interventions that will limit the detrimental effects of endothelial cell mineralocorticoid receptor in patients at cardiovascular risk. (AU)