Endoplasmic reticulum stress is involved in cardiac damage and vascular endothelial dysfunction in hypertensive mice

Titre	Endoplasmic reticulum stress is involved in cardiac damage and vascular endothelial dysfunction in hypertensive mice
Type de publication	Article de revue
Auteur	Kassan, M. [1], Galan, M. [2], Partyka, M. [3], Saifudeen, Z. [4], Henrion, Daniel [5], Trebak, M. [6], Matrougui, K. [7]
Editeur	American Heart Association
Type	Article scientifique dans une revue à comité de lecture
Année	2012
Langue	Anglais
Date	2012
Numéro	7
Pagination	1652 - 61
Volume	32
Titre de la revue	Arteriosclerosis, Thrombosis, and Vascular Biology
ISSN	1524-4636
Mots-clés	Animals [8], Endoplasmic Reticulum Stress/physiology [9], Endothelium, Vascular/physiopathology [10], Fibrosis [11], Hypertension/pathology/physiopathology [12], Male [13], Mesenteric Arteries/physiopathology [14], Mice [15], Mice, Inbred C57BL [16], Myocardium/pathology [17], Nitric Oxide Synthase Type III/physiology [18], Oxidative Stress [19], RNA, Messenger/analysis [20], Transforming Growth Factor beta1/genetics [21], Vascular Resistance [22], Vasodilation [23]

Résumé en anglais	OBJECTIVE: Cardiac damage and vascular dysfunction are major causes of morbidity and mortality in hypertension. In the present study, we explored the beneficial therapeutic effect of endoplasmic reticulum (ER) stress inhibition on cardiac damage and vascular dysfunction in hypertension. METHODS AND RESULTS: Mice were infused with angiotensin II (400 ng/kg per minute) with or without ER stress inhibitors (taurine-conjugated ursodeoxycholic acid and 4-phenylbutyric acid) for 2 weeks. Mice infused with angiotensin II displayed an increase in blood pressure, cardiac hypertrophy and fibrosis associated with enhanced collagen I content, transforming growth factor-beta1 (TGF-beta1) activity, and ER stress markers, which were blunted after ER stress inhibition. Hypertension induced ER stress in aorta and mesenteric resistance arteries (MRA), enhanced TGF-beta1 activity in aorta but not in MRA, and reduced endothelial NO synthase phosphorylation and endothelium-dependent relaxation (EDR) in aorta and MRA. The inhibition of ER stress significantly reduced TGF-beta1 activity, enhanced endothelial NO synthase phosphorylation, and improved EDR. The inhibition of TGF-beta1 pathway improved EDR in aorta but not in MRA, whereas the reduction in reactive oxygen species levels ameliorated EDR in MRA only. Infusion of tunicamycin in control mice induced ER stress in aorta and MRA, and reduced EDR by a TGF-beta1-dependent mechanism in aorta and reactive oxygen species-dependent mechanism in MRA. CONCLUSIONS: ER stress inhibition reduces cardiac damage and improves vascular function in hypertension. Therefore, ER stress could be a potential target for cardiovascular diseases.
URL de la notice	http://okina.univ-angers.fr/publications/ua8236 [24]
DOI	10.1161/ATVBAHA.112.249318 [25]
Lien vers le document	http://dx.doi.org/10.1161/ATVBAHA.112.249318 [25]
Titre abrégé	Arterioscler Thromb Vasc Biol

Liens

[1] http://okina.univ-angers.fr/publications?f[author]=13501
[2] http://okina.univ-angers.fr/publications?f[author]=13500
[3] http://okina.univ-angers.fr/publications?f[author]=2838
[4] http://okina.univ-angers.fr/publications?f[author]=13875
[5] http://okina.univ-angers.fr/d.henrion/publications
[6] http://okina.univ-angers.fr/publications?f[author]=13503
[7] http://okina.univ-angers.fr/publications?f[author]=13504
[8] http://okina.univ-angers.fr/publications?f[keyword]=964
[9] http://okina.univ-angers.fr/publications?f[keyword]=13170
[10] http://okina.univ-angers.fr/publications?f[keyword]=13128
[11] http://okina.univ-angers.fr/publications?f[keyword]=7752
[12] http://okina.univ-angers.fr/publications?f[keyword]=13171
[13] http://okina.univ-angers.fr/publications?f[keyword]=968
[14] http://okina.univ-angers.fr/publications?f[keyword]=13172
[15] http://okina.univ-angers.fr/publications?f[keyword]=1102
[16] http://okina.univ-angers.fr/publications?f[keyword]=1478
[17] http://okina.univ-angers.fr/publications?f[keyword]=13173
[18] http://okina.univ-angers.fr/publications?f[keyword]=13174
[19] http://okina.univ-angers.fr/publications?f[keyword]=6203
[20] http://okina.univ-angers.fr/publications?f[keyword]=13175
[21] http://okina.univ-angers.fr/publications?f[keyword]=13176

[22] http://okina.univ-angers.fr/publications?f[keyword]=8463
[23] http://okina.univ-angers.fr/publications?f[keyword]=1232
[24] http://okina.univ-angers.fr/publications/ua8236
[25] http://dx.doi.org/10.1161/ATVBAHA.112.249318