中文題目：**血管張力素增加熱休克引致心肌受損**

英文題目：**Angiotensin Modifies Heatshock-Induced Cardiac Injuries in Spontaneously Hypertensive Rats**

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前言：Angiotensin II play a role in cardiac dysfunction and injury in hypertension. Blockade of angiotensin II AT1 receptors(ARB) in myocytes protects against cardiac damage and pathologic remodeling. We examined the protective effects of a cardiomyocyte identified ARB, Candesartan, on cardiomyocyte injury after heatshock in hypertension.

材料及方法：Spontaneously hypertensive rats (SHR) and Wistar Kyoto (WKY) controls were treated with ARB (Candesartan, 0.3mg/kg per day) via subcutaneous osmotic minipumps for 4 weeks . Heatshock was induced by exposing the rat to high blanket temperature. Heart were harvested 1-7 day after heatshock for histopathology , immune of fluoresence studies, and quantitative real-time reverse transcriptase-polymerase chain reaction analyses to assess the change in gene expression profiles of α-myosin heavy chain (∂-MHC)，atrial natriuretic factor (ANF), and transforming growth factor β_1(TGF-β_1) .We also investigated the ARB on circulating and cardiac components of rennin-angiotensin system.

結果和結論：Results were compared with those of age-matched WKY rats , and to untreated SHR with and without of heatshock . Candesartan initiated after the heatshcok lowered levels of TGF-β_1 , mRNA and elevated levels of α-MHC mRNA and AT_2 receptor mRNA in SHR . Circulating levels of rennin, angiotensin I , and angiotensin II were elevated after heatshock ,and increased gene expression were higher in SHR . In SHR , AT1 receptor mRNA and protein expression wee higher than in WKY with and without evidence of heatsock, After heatshock, Candesartan treatment in SHR significant decreased TGF-β_1 mRNA levels were higher ($P<0.05$), whereas increased AT_2 receptor and α-MHC mRNA levels were higher ($P<0.05$) than WKY . The results suggest that the anti-heatshock cardiac injury benefits of ARB in hypertension may be mediated by effects on the expression of specific genes , and local rennin-angiotensin system components.