中文題目：**血管張力素參與心肌細胞的壞死及凋亡**

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

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前言：Although the benefit of angiotensin II AT_1 receptor blocker (ARB) treatment during and after myocardial injury had been found in hypertension, the molecular basis of these actions remain unclear . in this study, we tested the hypothesis that ARB in hypertension would prevent stress-associated changes in necrotic and apoptotic myocyte cell death in the heart .

材料及方法：Spontaneously hypertensive rats (SHR) and Wistar Kyoto rats (WKY) controls were treated with ARB (Candesartan, 0.3mg/kg per day) for 4 weeks. Heatshock was induced by exposing the rat to high blanket temperature. To determine the effects of heatshock on myocyte cell death, rat were injected with myosin monoclonal antibody for the localization and quantification of necrotic myocyte cell death in the left ventricle (LV). Conversely, the present of DNA strand breaks in myocyte nuclei, indicative of programmed cell death, was evaluated by the terminal deoxy-nucleotidyl transferase assay and confirmed by DNA laddering.

結果和結論：Myocyte necrosis, progressively increased after heatshock. Programmed cell death was restricted to LV and increased at the subsequent time after heatshock. The combination of necrosis and apoptosis in the LV free wall were significantly increased in the SHR. Candesartan decreased LV necrotic and apoptotic expression in SHR and WKY. There were no significant difference in Bcl-2 protein expressed by hearts among the groups. WAF-1 mRNA level were increased in both heatshock without ARB groups; in SHR-heatshock with ARB, the density of WAF-1 mRNA was lower than in SHR-heatshock without ARB. The results, suggesting the Candesartan is associated with a reduction in exaggerated myocyte cell death in hypertension that accompanies heatshock.