中文題目: PPAR 作用劑在熱休克中改變壓力蛋白的表現

英文題目: Relevance of PPAR Receptor Stimulator in Heatshock Induction of Cardiac Heat Shock Protein Expression

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前言: Experiments were carried out to ascertain whether the expression of leptin and peroxisome proliferator-activated nuclear receptor (PPAR) involved myocardial damage in the heatstroke-induced circulatory shock .We validate the hypothesis that PPAR receptor stimulator pioglitazone and fenofibrate may confer myocardial protection in diabetes against heatstroke.

<u>材料及方法</u>: To deal with matter, we assessed the effects of heatstroke on mean arterial pressure(SAP), heart rate, cardiac output(CO) and stroke volume(SV), total peripheral vascular resistance(TPR), colonic temperature, blood gases, and serum levels of leptin and tumor necrosis factor-alpha (TNF-a) in urethane-anesthetized rats pretreated without and with pioglitazone and fenofibrate for 4 wks. In addition, heat shock protein (HSP) and injury markers expression in the heart was determined in different groups.

結果和結論: Mean arterial pressure, CO and SV, blood pH, onset time of heatstroke and survival time after beat stress were all lower in diabetics. However, blood lactate concentrations, TPR, levels of leptin and TNF- α were greater in diabetics. Diabetic rats pretreated with pioglitazone and fenofibrate, when exposed to same heat stress, were longer onset and survival times, greater CO and SV, longer latency times for onset of cardiac arrhythmia, higher leptin level and lower TNF-a level. After the onset of heatstroke, HSP and injury markers in the heart were found to be significantly higher and lower, respectively in pioglitazone and fenofibrate pretreated diabetic rats.