LONG-TERM AEROBIC EXERCISE INCREASES MYOCARDIAL PPARγ EXPRESSION IN SPONTANEOUSLY HYPERTENSIVE RATS

Kun-Ru Zhang, Wei Wang, Su-Li Guo, Ai-Jing Qu, Kun-Ru Zhang. Sports College of Shaanxi Normal University

Objectives It is well known that cardiac insulin resistance exists in spontaneously hypertensive rats (SHR), which is attributable to decreased peroxisome proliferator-activated receptor-gamma (PPARγ) expression in myocardium. However, the effects of aerobic exercise (AE) on myocardial insulin sensitivity in SHR rats are largely unclear. Therefore, the present study aimed to determine the effects of 9-week swimming training on myocardial insulin sensitivity in SHR and the underlying mechanism, with the special focus on the role of exercise in myocardial PPARγ expression.

Methods 4-weeks-old SHR were randomly subjected to 9 weeks of either sedentary or freeloading swimming exercise (2 h/day, 5 d/week). Blood glucose, cardiac systolic/diastolic function and PPARγ, protein kinase B (Akt) expressions in myocardium were determined at the end of exercise.

Results Compared with Wistar-Kyoto rats (WKY), whole body and myocardial insulin sensitivity decreased in SHR as manifested by increased fasting blood glucose (6.24±0.21 vs 5.18±0.19 mmol/l, n=6, p<0.05) and decreased insulin-induced cardiac function changes especially for ±LVdp/dtmax respectively, which was partly attributable to decreased PPARγ expression in myocardium (0.72±0.08 vs 1.08±0.07, n=4, p<0.05). Moreover, 9-week swimming training not only attenuated the fasting blood glucose (5.54±0.16 vs 6.24±0.21 mmol/l, n=6, p<0.05) improved cardiac function and enhanced myocardial response to insulin in vivo in SHR, but also increased myocardial PPARγ and subsequent Akt expressions (1.18±0.12 vs 0.72±0.08, n=4, p<0.01 and 0.953±0.13 vs 0.514±0.14, n=4, p<0.05) in SHR.

Conclusions These data demonstrate that 9-week swimming training increased myocardial PPARγ and subsequent Akt expressions in SHR, which is partly involved in improved myocardial insulin sensitivity. The present findings also indicate that the decreased PPARγ expression and subsequent phosphatidylinositol 3-kinase (PI-3 kinase)/Akt signalling perhaps plays a causative role in the impaired inotropic response to insulin in SHR heart. Thus, AE merges as an important choice in future SHR preclinical and clinic investigation.