Acute Exercise Attenuates Cardiac Dysfunction After Ischemia/Reperfusion in Isolated Rat Heart

RYO KAKIGI*1), MAKINO WATANABE*1), HISASHI NAITO*2), TAKAMASA TSUZUKI*2), HIROTO TSUJIKAWA*2), TAKAFUMI IESAKI*1), TAKAO OKADA*1)

*1) Department of Physiology, Juntendo University Faculty of Medicine, Tokyo, Japan, *2) Department of Exercise Physiology, Graduate School of Health and Sports Science, Juntendo University, Chiba, Japan

Purpose: Regular bouts of endurance exercise can protect the heart against ischemia/reperfusion (I/R) injury. However, the effects of acute exercise immediately before I/R events on myocardial dysfunction remain unclear. This study examined (1) whether a single session of acute exercise reduced cardiac dysfunction after I/R, and (2) whether a single exercise session up-regulated the intracellular signaling pathways involved in cardioprotection in the rat heart.

Methods: Male Sprague-Dawley rats were divided into a sedentary control (CON) group and an exercise (EX) group. Rats in the EX group underwent one 30-min session of treadmill running. Following exercise, hearts were excised and subjected to Langendorff perfusion. To evaluate cardiac function during I/R, hearts from both groups were exposed to global ischemia (20 min) followed by reperfusion (45 min). Using western blotting, phosphorylation of Akt, protein kinase C-ε (PKCε) and glycogen synthase kinase 3-β (GSK-3β) in the hearts were analyzed.

Results: Cardiac function was significantly higher in the EX group compared with the CON group for 5–20 min after reperfusion (p<0.05). Phosphorylation of Akt, PKCε and GSK-3β in hearts of EX rats showed significant increases compared with CON rats (p<0.05). Exercise did not change expression levels of heat shock protein 72 in the heart.

Conclusions: Acute exercise prior to I/R attenuated cardiac dysfunction in the isolated rat heart. The attenuation might be due to exercise–induced activation of cardioprotective intracellular signaling.

Key words: running exercise, Langendorff perfusion, cardiac function, intracellular signaling, heat shock protein