Abstract

Acute cold exposure is a significant stressor activating heat production by shivering after the prolonged exposure cellular oxidative stress increases. Chronic exposure to cold lasting at least 2 weeks leads to the development of cold acclimatization. The main thermogenic role is taken over by non-shivering thermogenesis taking place in brown adipose tissue, which significantly increases its weight due to cold. Cardiac hypertrophy, hypertension and impaired renal function are frequently observed pathologies of acclimatization at 4-5 °C. Our laboratory recently introduced a model of mild chronic cold acclimatization at 8 °C, during which no damage to the heart or kidneys occurs and has proven cardioprotective effect on reducing infarct size. Hence, the influence of this cold acclimatization model on the other cellular and molecular processes needs to be investigated. The cardioprotective effect of cold acclimatization includes changes in β-AR signaling, activation of anti-apoptotic pathways or augmentation of the antioxidant system. The aim of this thesis was to investigate the effect of cold acclimation and subsequent reacclimation on proteins regulating Ca^{2+} levels in the rat heart (SERCA2 and phospholamban) and on the stimulation of regulatory proteins β-arrestin 1/2 and protein kinase PDPK1. The results showed slight changes in these proteins, which modulate the flow of Ca^{2+} in the heart of the cold acclimated rat.

Key words: heart, cold acclimation, β-adrenergic signaling, SERCA2