**Abstract**

Cardiovascular disease, including ischemic heart disease, its acute form of myocardial infarction and subsequent heart failure, is still the most common cause of death in the world. For decades, scientists have been aiming to find clinically usable cardioprotective interventions that effectively prevented this disease or reduced the consequences of this disease and improved the quality of life of patients. It is known that a healthy lifestyle, which includes long-term hardening, contributes to heart protection. The beneficial effects of hardening on personality have been known for two hundred years, but no one before us has investigated the effect of a cold environment on the magnitude of ischemia-reperfusion (I/R) damage 10 years ago, we introduced a model of mild cold exposure (8 ± 1 °C) (MCE) which was protective on the size of the myocardial infarction and at the same time no negative side effects such as hypertension and myocardial hypertrophy were demonstrated. Our aim was to investigate the mechanism of cardioprotection induced by MCE. We asked the following questions: What is the metabolic profile of rats exposed to MCE? What is the effect of MCE on the magnitude of I/R damage in the short and long term? When does brown adipose tissue mature during MCE? Which signalling pathways are involved in MCE-induced cardioprotection? Our work showed that MCE temporarily began to protect the metabolism. We were also the first to demonstrate that after 10 days of MCE there is proliferation and maturation of brown adipose tissue. Likewise, after 10 days there was a reduction in the magnitude of I/R damage, the beneficial effect of cold deepened after 5 weeks of continuous cold exposure and persisted even after two weeks returning to thermoneutral temperature (24 ± 1 °C). Using selective beta-blockers, we confirmed some signalling pathways which may apply in long-term cardioprotective effect of MCE and after return to thermoneutral conditions.

Key words: rat, myocardium, brown adipose tissue, mild cold exposure, ischemia-reperfusion injury, cardioprotection