ABSTRACT

Heart is a highly oxidative tissue with the ability to modulate its energetic metabolism according to prevailing conditions of the organism. Stress conditions such as exercise and prolonged staying at high altitude induce a fetal gene programme leading to increased carbohydrate and decreased fatty acid utilization. It seems that mitochondrial fuction and the changes in redox state of the cell play a key role in activation of various transcriptional factors, mainly HIF-1α modulating expression of numerous signalling and metabolic pathways as well as changes in structural proteins in the heart. Activation of these mechanisms results in cardioprotective phenotype which increased the tolerance of the heart to acute lack of oxygen. Understanding the mechanisms of these adaptive changes is important for the development of tools that would help reduce the effects of acute myocardial infarction in humans.