

Abstract

Adaptation to chronic hypoxia (CH) is characterized by a variety of functional changes in order to maintain metabolic and energy homeostasis. It has been known for many years that both humans and animals indigenous or adapted to high-altitude hypoxia are more tolerant to an acute ischemic injury of the heart. Cardioprotective mechanisms activated by adaptive responses to chronic hypoxia can be the result of altered transcriptional regulations in left ventricles. Here we report results from the gene expression profiling of adaptive responses in three models of chronically hypoxic heart. Adult male Wistar rats were exposed for 21 days to either continuous normobaric hypoxia (CCH; 10% O₂) or CCH interrupted daily by 1-hour reoxygenation (RCH) or CCH interrupted daily by 16-hour (CIH). Cardioprotective effect of CCH adaptation is abolished by brief daily reoxygenation, RCH adaptation. In the present study, we aimed to determine myocardial mRNA expression of 19 candidate genes divided into three important groups: i) Hypoxia inducible factor (HIF1 α) and its prolyl and asparaginyl hydroxylases (PHDs and FIH respectively), ii) Creatine kinase (CK) isoenzymes which play important role in energy homeostases of heart and iii) the group of main enzymatic antioxidants which maintain appropriate ROS level in the cells.

Key words: Heart, Hypoxia, Cardioprotection, mRNA, Hypoxia inducible factor, Creatine kinase, Oxidative stress, Antioxidant enzymes