Abstract

Endogenous cardiac protection against acute ischemia/reperfusion injury can by increased by cardiac adaptation to various forms of chronic hypoxia. Chronic hypoxia induces a large variety of adaptive changes in the myocardium that could be considered as protective, but the exact mechanism of increased ischemic tolerance is unknown. Different studies suggest that catecholamine release and their effect on β-adrenergic signaling after adaptation to chronic hypoxia contributes to cardioprotection.

In this study we focused on characterization of β-adrenergic receptors (β-ARs) in the myocardium of rats after adaptation to three different hypoxic conditions: 1. intermittent normobaric hypoxia - INH/R (23 h hypoxia, 1 h reoxygenation), 2. intermittent normobaric hypoxia - INH (8 h hypoxia, 16 h normoxia), 3. continuous normobaric hypoxia - CNH (24 h hypoxia). We compared how each hypoxic model affects the total number of β-adrenergic receptors and proportion of individual subtypes (β_1-and β_2-ARs) in the left and right ventricles compared control normoxic rats.

The INH model had apparently no effect on β-ARs in either ventricles. On the other hand, adaptation to INH/R and CNH was accompanied by a significant decrease (by about 25%) in the total number of β-adrenergic receptors in the right ventricles. Our present result demonstrated that these models could lead to downregulation of β_1-ARs in right ventricles, which may represent one of the mechanisms involved in the development of a cardioprotective phenotype. However, due to not very significant differences in the determined values it is not possible to decide which of these two models would be more beneficial.

Key words: β-adrenergic receptors, ischemia/reperfusion injury, chronic hypoxia, cardioprotection