Abstract

The aim of the present study was to find out whether adaption to chronic hypoxia affects the expresion of TNF-α and IL-10 in rat myocardium. TNF-α is a proinflammatory cytokine, which amplifies inflammatory reaction, while IL-10 has opposite antiinflammatory effect. We also measured concentration of nitrotyrosine as a marker of nitrosative stress. We used male Wistar rats divided into four groups: 1) normoxic controls; 2) exposed to continous normobaric hypoxia (10% O_2) for three days or 3) for three weeks and 4) exposed to intermittent normobaric hypoxia (10% O_2) for three weeks with one hour daily reoxygenation. Cytosolic and membrane proteins (cytosolic and particulate fractions) were obtained from the left ventricle, right ventricle and interventricular septum. Concentrations of TNF-α and IL-10 in both fractions were measured by ELISA. Continous hypoxia increased TNF-α production in particulate fractions from all ventricular parts and decreased the ratio of IL-10/TNF-α in particulate and cytosolic fractions. Intermittent hypoxia redistributed TNF-α from cytosol into the particulate fraction and prevented the drop of IL-10/TNF-α ratio in the cytosolic fraction. The highest concentration of nitrotyrosine was found in the particulate fraction from the right ventricle after three days of hypoxia. Our data suggest that short periodic reoxygenation significantly modulates the proinflammatory effect of chronic hypoxia on the expresion and localization of TNF-α and IL-10 in rat myocardium. (In Czech)

Key words: myocardium, hypoxia, cytokines, TNF-α, IL-10, nitrotyrosine