

## **Abstract**

The goal of this thesis was to discover the influence of adaptation to chronic hypoxia on ischemic tolerance of heart – this experiment was carried out on two different hypertension kinds of laboratory rats. Spontaneously hypertensive rats (SHR) and rats from a conplastic strain SHR/OlaIpcv-mt<sup>BN/Crl</sup>, whose mitochondrial genome of the SHR strain was replaced with a mitochondrial genome of the normotensive strain Brown Norway, were exposed to continuous normobaric hypoxia (10% O<sub>2</sub>) for a period of 3 weeks. On the other hand, the control group of rats was kept in normoxia. At the end of the adaptation period, the ischemic tolerance of heart and the mitochondrial aconitase expression were examined. In the case of both hypertensive strains, the chronic hypoxia led to a significant drop in the size of a myocardial infarction and also to a drop in the number of reperfusion arrhythmias. In the case of the SHR strain, the incidence of ischemic arrhythmias decreased. Chronic hypoxia had no impact on the aconitase expression for both analysed strains. This thesis showed that the ischemic tolerance of heart can be enhanced in the case of the SHR strain. On the other hand, the mitochondrial genome of the SHR strain does not seem to play any significant role in protection mechanism.

Key words: chronic hypoxia, hypertension, myocardial infarction, arrhythmias, oxidative stress