**ABSTRACT:**

Cardiovascular diseases (CVDs) are the most widely spread diseases of modern civilization. Mechanisms involved in the protection of myocardial tissue are for that very reason in the focus of cardiovascular research. The adaptation to chronic hypoxia has been studied for many years in the context of its positive effects on heart function and its increased tolerance to ischemia-reperfusion (I/R) injury. This Master thesis describes the role of Akt kinase in the mechanisms leading to myocardial protection against I/R injury using the model of adaptation to chronic normobaric hypoxia (CNH). The hearts from male Wistar rats, that were kept in normoxic or hypoxic conditions ($O_2$ 0.1) for the period of 3 weeks, were retrogradely perfused by oxygenated Krebs-Henseleit solution and then subjected to 10 min of ischemia and 10 min of reperfusion. Samples prepared from left ventricles (LV) of experimental hearts were later used for protein analyses. The adaptation to CNH leads to increased phosphorylation of Akt kinase on $Ser^{473}$, but it did not affect the phosphorylation on $Thr^{308}$ nor the total protein level of Akt. A significant increase in Bcl-2/Bax ratio was also observed in hearts adapted to CNH. This Master thesis further elucidates, how Akt signaling pathway and its activation are affected by short periods of ischemia in the context of adaptation to CNH.