Abstract

Currently, it is growing evidence that autophagy is involved in the prevention of various diseases, which of course also includes heart diseases. This thesis is therefore aimed at clarifying the role of autophagy in the heart, especially during ischemia and subsequent reperfusion. Autophagy is a physiological cellular process by which the cell maintains homeostasis by eliminating long-lived proteins and damaged organelles. The role of autophagy during ischemia/reperfusion in the heart is complex. Predominantly it functions as a pro-survival pathway, because it protects the heart from ischemia or hypoxia. However, when triggered over, which happens during reperfusion, it may lead to cell death. In the heart autophagy is activated in response to various stimuli, such as decrease in ATP and subsequent activation of AMPK, protein Bnip3, reactive oxygen and nitrogen species, the opening of mitochondrial permeability transition pore, endoplasmic reticulum stress or unfolded protein response.