Abstract

One of the leading causes of death worldwide is cardiovascular diseases. Researchers are, therefore, dealing with the mechanisms that induce a cardioprotection. Cardioprotection is a general pathophysiological term under which we understand myocytes protection against damage by ischemia and subsequent reperfusion impairment, inflammation, hypertension, and toxic and degenerative changes, including some types of apoptosis. One of the less common ways of cardioprotection is a cold adaptation. Adaptive thermogenesis is an important part of energy homeostasis and protection against obesity, metabolic disorder threatening heart. The PGC family of proteins plays a very important role in adaptive thermogenesis. This thesis summarizes the current state of literature in cold adaptation issues, especially the role of PGC1α and its effects at the cellular and tissue level. mRNA expression of PGC-1α is strongly induced in brown fat and skeletal muscles of mice exposed to cold. PGC-1α also increases the transcriptional activity of PPAR-γ and thyroid hormone receptor protein on UCP-1 (uncoupling protein). UCPs (uncoupling proteins) are small proteins localized to the inner side of the mitochondrial membrane to facilitate the transport of protons, which they release into concentration gradient without ATP synthesis and thereby generate heat.