Abstract

Metabolic pathways in adipose tissue affect the whole-body energy homeostasis. *De novo* lipogenesis and futile metabolic cycling based on lipolysis and fatty acid re-esterification which is engaged in regulation of fatty acid level in bloodstream are occuring there. These processes are partly regulated by nuclear receptor PPARγ. Mitochondrial biogenesis and oxidative phosphorylation in adipocytes are controlled by interacting of PPARγ with transcriptional coactivators PGC-1α and PGC-1β. The aims of this thesis were to find out whether PGC-1β is connected with regulation of futile cycling and *de novo* lipogenesis in white adipose tissue and also how specific inactivation of PGC-1β gene in adipose tissue affects phenotype of mice during short-term cold exposure or treatment based on high fat diet enriched by *n*-3 polyunsaturated fatty acids in combination with mild calorie restriction.

The results show that inactivation of PGC-1β probably does not affect futile cycling based on lipolysis and fatty acid re-esterification. In mice with PGC-1β ablation compensation in weight of brown adipose tissue was observed as well as increase in the gene expresion of nuclear receptors PPAR, transcriptional coactivator PGC-1α and UCP1 during cold exposure. Even though the inactivation of PGC-1β in brown adipose tissue is compensated by upregulation of gene expression and also by increase of UCP1 protein, tissue does not appear to be fully functional.