AMP-activated protein kinase (AMPK) is an important metabolic sensor in eukaryotic organisms and it plays an important role in regulating energy homeostasis, at both the cells and the whole organism. AMPK controls glucose and lipid metabolism by direct stimulation of enzymes or by long term stimulation of the gene expression of energy metabolism. Skeletal muscles significantly contribute to the total body weight and metabolic rate and to the maintenance of glucose homeostasis. Due to the ability of the muscle to increase energy expenditure to 95% of whole-body energy expenditure, could be the proper development and programming of metabolism in the early postnatal period crucial for the further development of the organism in adulthood. Early postnatal development leads to substantial changes in energy requirements of the body and this suggests the significant involvement of AMPK in this period. The aim of this thesis was to study the activity and expression of isoforms of the catalytic subunit of AMPK in skeletal muscle during early postnatal development of both mouse strains A/J and C57BL/6 that differ in the development of diet-induced obesity. The next task was to analyze the expression of selected genes involved in energy metabolism - GLUT4, PGC-1α and UCP3 that AMPK regulates. It was found that the activity of AMPK changes differently during early postnatal period, depending on the genetic background of the mice. Expression of AMPK subunits α1 and α2 are different in comparison with changes in the activity of the enzyme isoforms. Profile expression levels of genes GLUT4, PGC-1α a UCP3 shows the strong influence of nutrition and development of muscle on the regulation of gene expression of energy metabolism. Significant changes in activity and the regulation of energy metabolism in skeletal muscle during the early postnatal development may contribute to the further profiling of metabolism in adulthood depending on the genetic background of the mice.