

Obesity and associated disorders are becoming one of the most serious problems for healthcare systems in the developed countries. Possible treatment for obesity and the associated disorders would be to increase energy expenditure. It is known that leptin regulates food intake and energy expenditure, both among rodents and humans. Leptin acts directly on tissues and also indirectly by affecting hypothalamus and stimulation of sympathetic nervous system, involving $\alpha 1$ -adrenergic receptors. The aim of the study was to establish if leptin and $\alpha 1$ -adrenergic stimulation of peripheral tissues are important for resistance of A/J mice strain to obesity induced by high-fat diet. Unlike in mice of B6 strain, which are prone to obesity, in A/J mice, which are resistant to obesity, high-fat diet feeding during two weeks after weaning led to increased leptin levels in blood. In A/J mice, phenylephrine, the agonist of $\alpha 1$ -adrenergic receptors, induced increase of energy expenditure as measured by indirect calorimetry. Circadian rhythm of leptin levels in blood changed in dependence on diet in A/J mice strain. The results support a hypothesis that leptin-dependent adrenergic stimulation is important for control of energy expenditure, and hence for susceptibility to obesity.