Insulin resistance is a key component of the pathogenesis of metabolic syndrome and type 2 diabetes. Skeletal muscle is an important part of energy metabolism. It recieves, stores and uses most of the glucose from blood. Insulin stimulates glucose uptake, by promoting translocation of glucose transpoters to plasma membrane. It also increases the rate of protein, glycogen and triglyceride synthesis. When muscle is unable to respond to normal levels of circulating insulin, insulin resistance occurs. Insulin resistance leads to disruption of key metabolic processes. Fatty acid transport across the membrane is upregulated, whereas the ability to oxidize fatty acids is decreased. This imbalance leads to accumulation of lipid metabolites inside the cells. Lipid intermediates may interfere with insulin signalling. Inflammatory cytokines, particularly TNF, activate kinases that may inhibit the insulin signal transmission. Insulin resistance may be treated pharmacologically, but also physical activity and decrease of energy intake are important part of therapy. Also polyunsaturated fatty acids have beneficial effects on muscle insulin sensitivity.