Obesity is associated with insulin resistance (IR) and type 2 diabetes mellitus. Among possible mechanisms leading to IR are increased plasma levels of free fatty acids and Alfred levels of adipokines secreted from adipose tissue (AT). In the first part of the work, we studied obese patients during different nutritional and physical activity interventions. Phenotypic data were related to the expression of AT genes potentially involved in the regulation of insulin sensitivity (IS) and/or low-grade inflammation. We confirmed that aerobic and dynamic strength training improved IS and demonstrated that these interventions do not promote changes in subcutaneous AT gene expression or in plasma levels of adiponectin, interleukin-6, interleukin-1 beta and tumor necrosis factor-alpha, but decrease circulating leptin level. Very low calorie diet followed by low calorie diet and weight maintenance period enhanced IS in obese women and diminished retinol-binding protein 4 (RBP4) in plasma, but RBP4 mRNA levels were reduced only after very low calorie diet. Our findings indicate that the investigated adipokines, except potentially leptin, might not be mediators of changes in IS induced by lifestyle interventions. In the second part of the work, we investigated the role of peroxisome proliferator-activated receptors (PPARs) on the protein secretion by human subcutaneous AT. We showed that PPARs regulated production of several proteins and identified new adipokines responding to activated PPARs. We demonstrated that PPARs modulate secretion of bioactive molecules from different AT cell types. These studies contribute to our understanding of the relationship between adipokines and IS.