The metabolic effects of obesity have made this disease one of the most common risk factors for diabetes, hypertension, and atherosclerosis. Adipose tissue is now recognized as an active secretory and immune organ. Chronic inflammation is a common feature of the obesity, and inflammatory signals may originate within visceral adipose tissue as this fat depot expands in response to chronic positive energy balance. Both adipocytes and macrophages within fat secrete numerous hormones and cytokines that have local effects on WAT physiology but also systemic effects on other organs and may markedly contribute to the development of pathophysiological disorders associated with metabolic syndrome. On the contrary, leanness as well as significant weight reduction in obese patients increases production and circulating levels of metabolically beneficial factors and decreases production of proinflammatory and insulin resistance-inducing factors. Endothelial dysfunction and inflammation are important signs of vascular risk and worsened prognosis in patients with metabolic syndrome and type 2 diabetes. Measures of endothelial function remain invaluable for research into disease mechanism and response to new therapies. An interesting area of ongoing investigation is the role of thiazolidinediones in improving endothelial function in patients with type 2 diabetes. Thiazolidinediones regulate the expression of numerous genes with key roles in glucose and lipid metabolism and thus are able to exert direct beneficial effect on insulin resistance. In addition, activation of PPARs could improve vascular function and inflammatory processes resulting in additional vascular effects. The aim of our study was to evaluate the effect of 5-months treatment with PPAR-γ agonist rosiglitazone on the circulating markers of endothelial dysfunction in patients with type 2 diabetes and to evaluate the role of changes in endocrine function of adipose tissue in this process. We showed



similarly to previously published studies that activation of PPAR-γ by rosiglitazone improved diabetes compensation, markedly increased circulating adiponectin levels (whereas resistin levels were not affected) and decreased circulating markers of endothelial dysfunction sVCAM-1, PAI-1 and E-selectin. It also tended to decrease circulating levels of matrix metalloproteinase-9. The beneficial effect on endothelial dysfunction is very likely explainable by the combination of glucose-lowering effect of rosiglitazone and by increased circulating levels of anti-atherogenic hormone adiponectin.

There is considerable evidence of links between increased production of some adipocyte factors and the metabolic and cardiovascular complications of obesity. On the contrary, previous studies in AN patients showed numerous metabolic and endocrine abnormalities including the perturbations of endocrine function of adipose tissue such as decreased circulating leptin levels and increased adiponectin levels. In our second study we tested the hypothesis that chronically decreased calory intake in anorexia nervosa patients enhances the mRNA expression of adiponectin and decreases expression of proinflammatory adipokines in subcutaneous adipose tissue. Surprisingly, we found significantly decreased subcutaneous fat adiponectin mRNA expression with simultaneous increase of its circulating levels. We suppose that increased production of adiponectin in other tissues

such as visceral adipose tissue or the muscle tissue could be responsible for this dissociation. Another interesting finding of the study was dissociation between unchanged circulating resistin levels in anorexia nervosa patients and its markedly up-regulated mRNA expression in subcutaneous adipose tissue, although mRNA expression of other proinflammatory factor interleukin-6 was significantly decreased relative to in the control group. We suppose that altered local adipokine production may contribute to the

development of some metabolic alterations in subcutaneous adipose tissue of patients with anorexia nervosa.

Both experimental and clinical research bring new information and shed more light on this fascinating field. However, the exact role of endocrine function of adipose tissue in humans remains to be elucidated. Understanding the endocrine function of adipose tissue will likely permit more rational approaches to treatment of the metabolic consequences of excess and deficiency of adipose tissue. The evaluation of adipose tissue and particularly its endocrine function remains an essential component of our investigations.