**STRESZCZENIE W JĘZYKU ANGIELSKIM**

**Objective:** In obesity, adipose tissue (AT) undergoes dynamic remodeling, including an alternation in adipogenesis, AT-resident cell content, angiogenesis, and turnover of extracellular matrix (ECM) components. Studies of AT in humans have been carried out mostly in people with severe metabolic abnormalities, like type 2 diabetes or morbid obesity.

**Aim:** The purpose of this study was to investigate subcutaneous AT gene expression of markers of adipogenesis, ECM remodeling, and inflammation in young, healthy, overweight or obese subjects.

**Design:** The study group comprised 83 normal-weight, 48 overweight, and 19 obese subjects. Euglycemic hyperinsulinemic clamp, biopsy of subcutaneous AT, and isolation of peripheral blood mononuclear cells (PBMCs) were performed. Gene expression was measured with real-time polymerase chain reaction.

**Results:** Overweight/obese subjects had lower AT expression of markers of adipogenesis, insulin signaling, and angiogenesis; higher expression of markers of ECM remodeling; altered expression of genes of the nuclear factor-k-B (NF-κB), but not c-Jun NH2-terminal kinase, pathway; and higher expression of macrophage markers but not markers of other immune cells. In multiple regression analysis, the expression of *CEBPA*, *ADIPOQ*, *IRS1*, *IRS2*, *SLC2A4*, and *MMP9* was associated with insulin sensitivity independently of body mass index. No differences were found in inflammatory-gene PBMC expression.

**Conclusion:** Overweight/obesity is associated with altered expression of genes of adipogenesis, insulin signaling, ECM remodeling, and inflammation. NF-κB seems to be the earliest inflammatory pathway altered at the transcriptional level in AT. Macrophages seem to be the first immune cells to infiltrate AT. Adipogenesis and ECM remodeling are the initial processes in AT that are independently associated with insulin sensitivity.