Abstract

Endoplasmic reticulum (ER) is a cellular organelle responsible for folding of proteins that are then transported to the various places in the cell or secreted. It is also crucial for the synthesis of triglycerides. Metabolic imbalance leads to ER stress and consequently triggers signaling pathway, which is called unfolded-protein response (UPR). The aim of this pathway is to alleviate ER stress, restore natural homeostasis and prevent death of cell. At the same time however, it activates stress kinases and other factors that may perturb insulin signaling and increase expression of proinflammatory cytokines. These signs are characteristic for human obesity, which is associated with reduced tissue's sensitivity to insulin and is considered as a disease with low level of inflammation. Recent studies have suggested that the source of proinflammatory cytokines in obesity are stressed adipocytes and macrophage infiltrated into adipose tissue. Indeed, it has been demonstrated that stress of endoplasmic reticulum is significantly increased in adipose tissue of obese individuals. Weight loss associated with reduction of adipose tissue mass decreases stress while lowers both, the production of proinflammatory cytokines and insulin resistance.

This work aims to collect and discuss these new findings, which suggest that ER stress might by crucial for the development of diseases associated with obesity.