Endocrine Function of Adipose Tissue

Adipocytes (fat tissue cells) also have an endocrine function. They produce substances of a proteinaceous nature called **adipokines** or adipocytokines. Some of those hormones act directly in the adipose tissue (on adipocytes or macrophages and fibroblasts of the tissue), or remotely through the blood stream on organs, such as muscles, liver, brain, endothelium. Their effects affect intermediate metabolism, insulin sensitivity, hemocoagulation, immune response, etc.

Adiponectin

Increases the utilization and transport of glucose and non-esterified fatty acids (NEFA) into muscle, liver and adipose cells. These effects are provided by AMP-kinase, which restores the energy balance in the cell when cellular ATP decreases (eg β-oxidation of lipids and Glc). It also counteracts the development of atherosclerosis.

Leptin

Affects the hypothalamic **satiety center**, thereby reducing food intake and stimulating energy expenditure. Leptin receptors are on myocytes, adipocytes, hepatocytes and pancreatic β-cells. The effect on adipocyte is similar to insulin (stimulates saving of resources - glycogen synthesis, inhibition of lipolysis), in myocyte it activates AMP-kinase (increases the oxidation of triglycerides), thus protecting skeletal muscle from excess TAG. In pancreatic cells it inhibits insulin production.

Adipose tissue is composed not only of adipocytes, but also of fibroblasts, endothelium and immunocompetent cells. These cells, especially macrophages, are responsible for the production of **cytokines**. Cytokines are produced in other tissues of the body, too. That is for example interleukin 6 (IL6), that inhibits insulin receptor and enhances lipolysis during excercise. Another example of a cytokine is **TNFα** (tumor necrosis factor), which increases the level of NEFA in the blood.

Resistin

Suppresses the effect of insulin on glucose utilization and reduces glucose tolerance. These results were tested only on mice.

Visfatin

A hormone produced by adipose tissue lymphocytes, increases glucose transport in myocytes, lipogenesis, differentiation of adipocytes and reduces glucose production in hepatocytes. Increased lipogenesis and differentiation increases the deposition capacity of visceral adipocytes, so they hold more lipids that would otherwise disrupt the metabolism of other insulin-sensitive tissues.

Effect on insulin sensitivity

The adipocyte has the task of accumulating unnecessary lipids and releasing them again when there is an increased need or starvation. If a person has an excess of nutrients, the stored lipids increase the volume of the adipocyte. This cannot be done indefinitely, and therefore depends on the number of adypocytes (deposition ability) that arise from mesenchymal cells mainly in childhood and puberty. People who have a few of these cells (e.g., liposuction) have a greater risk of developing insulin resistance, since cells are overloaded by stored lipids (and thus become insulin-resistant) And it is the endocrine-active substances formed in adipose tissue that contribute to the way lipids are handled in adipocytes.

Links

Sources

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