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1. Introduction

Brown adipose tissue (BAT) is found in fetuses and newborn. In adult humans, this type of adipose tissue is practically absent. The active brown adipose tissue in adult humans is present at discrete sites, such as in the upper trunk (in cervical, supraclavicular, paravertebral, pericardial, mediastinal, and mesenteric areas) [1]. In mammals, BAT is involved in process of thermogenesis. It produces heat metabolizing fatty acids. Its specific role is due to uniquely expressed in mitochondria of uncoupling protein 1 (UCP1). Activation of UCP1 stimulates uptake of lipids and glucose from circulation to process of thermogenesis. In women, as compared to men, functional brown adipocyte is more common. The mass of BAT depends on overweight, obesity, and age. In these people, the mass of brown adipose tissue is reduced.

White adipose tissue (WAT) store of lipids and during fasting, the release of fatty acids that in process of β-oxidation, is the source of adenosine triphosphate (ATP). ATP is necessary for all living organisms; for bacteria, fungi, plants, animals, and humans. WAT is also known as a major secretory organ and high active metabolic tissue. It secretes, for example, cholesterol, retinol, steroid hormones, prostaglandins, and proteins known as “adipokines.” Some of these molecules may be associated with pathologies such as obesity, insulin resistance. These substances may increase the risk of metabolic syndrome, cardiovascular diseases, and others. As examples of substances synthesized and released by WAT are: Leptin, tumor necrosis factor-α, adiponectin, and interleukin-6.

Leptin is a peptide hormone synthesized and released mainly by adipose tissue. Many types of human organs express leptin, such as placenta, gastric fundus mucosa, and skeletal muscle, but subcutaneous adipocytes are responsible for 80% of total leptin production [2]. It is involved in the regulation of energy balance and food intake. Leptin plays also a role in reproduction. It was observed that hypothalamic hypogonadism in humans and in rodents is due to deficiencies or insensitivity to leptin. Other roles of leptin are increase of cytokine
production and macrophage adhesion and phagocytosis, modulation of blood pressure, influence on insulin sensitivity of peripheral, hepatic and skeletal muscle, and modulation of pancreatic β-cell function [3].

Tumor necrosis factor-α is a proinflammatory cytokine. It is synthesized mainly by macrophages and lymphocytes, but in humans also in low quantities in adipocytes. This cytokine influences on inflammation, apoptosis, cytotoxicity, synthesis of interleukin-1 and interleukin-6, and on adipocyte metabolism. It alters intracellular insulin signaling and induces insulin resistance. Its expression and secretion correlate with BMI increases in obesity and decreases in weight loss.

Adiponectin is highly expressed in adipocytes. Its expression depends on the distribution of adipose tissue in the human body. The levels of this protein are higher in subcutaneous adipose tissue than in visceral adipose tissue. It was observed the negative correlation between the degree of obesity and levels of adiponectin in circulation. In patients with type 2 diabetes, obese with insulin resistance, and in patients with coronary heart disease, adiponectin is not expressed [2].

Interleukin-6 is synthesized by many cell types and different tissues, including white adipose tissue. In the absence of an acute inflammatory process, WAT synthesizes substantial amounts of IL-6. It might represent about 15–30% of circulating levels [4]. Like adiponectin, its secretion depends on the distribution. In visceral adipose tissue, its secretion is three times higher as compared to subcutaneous adipose tissue. It was observed the link between IL-6 and obesity, inflammation, and coronary heart disease.

Adipose tissue synthesize and release also many others molecules: Resistin, retinol binding protein 4 (RBP4), vaspin, visfatin, omentin, chemerin, serum amyloid A (SAA), acylation stimulating protein (ASP), plasminogen activator inhibitor-1 (PAI-1), angiotensinogen, vascular endothelial growth factor (VEGF), hepatocyte growth factor, transforming growth factor-B (TGF-B), insulin-like growth factor-1 (IGF-1), macrophage migration inhibitory factor (MIF), lipoprotein lipase, cholesterol ester transfer protein (CEPT), prostaglandins, estrogens, glucocorticoids, and apelin.

The beige adipose tissue (BeAT) is similar histologically to brown adipose tissue. Different stimuli, such as cold, exercise, thyroid hormones, bile acids, and cause differentiation of white adipose tissue into brown adipocytes. Browning of white adipose tissue is an adaptive and reversible response of WAT to stimuli. There are used different synonyms to describe the differentiation of WAT into BAT—browning, britening, and beiging. Beige (brite—from “brown in white”) adipocytes have multilocular lipid droplets in the cytoplasm, numerous mitochondria as well as several intermediate features between WAT and BAT. The process of thermogenesis in BeAT, activated by cold, and may be mediated indirectly by the sympathetic nervous system. UCP-1 protein can be also involved in process of thermogenesis in BeAT.

In humans, there are two main sites of adipose tissue accumulation: Visceral and subcutaneous. Obesity is a worldwide health problem. It is defined as a body mass index (BMI) of ≥30 kg/m², and abdominal obesity is defined as waist circumference > 102 cm for men and > 88 cm for women. Obesity increases the risk of many diseases such as diabetes mellitus,
metabolic syndrome, cardiovascular diseases, cancers, and so on. Visceral and subcutaneous adipose tissues differ not only in their distribution. They express different genes involved in insulin resistance the pattern of expression of these genes is different, as well as these tissues differ in production and secretion of adipokines. It was observed that visceral abdominal obesity reduces the life expectancy of ~ 8 years. Central abdominal fat causes insulin resistance. It is suggested that this pathology is due to release of fatty acids from visceral depot into the portal vein, increasing gluconeogenesis, and hepatic glucose output. As mentioned above, visceral adipose tissue secretes adipokines that may cause lipotoxicity in peripheral tissues. On the other hand, in male patients with diabetes, visceral fat accumulation is less correlated with insulin resistance than subcutaneous fat accumulation.

This book aims to provide an overview of adipose tissue, its types, characteristics, role in humans, and animals. There are also described processes in adipose tissue involved in human health and diseases.

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References


