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ADIPOSE TISSUE OF THE GREATER OMENTUM: MORPHOLOGY AND FUNCTION

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Despite the fact that more and more publications about the structure and function of adipose tissue have recently appeared, further research may provide new insights into its role in metabolic diseases. Molecular and cellular mechanisms that regulate the functional activity of adipocytes, the issue of the interaction of adipose tissue with other structures of the body, as well as the role and significance of biologically active substances produced by adipocytes require in-depth research. The greater omentum, in which visceral fat accumulates and is a derivative of the peritoneum, was previously considered an inert adipose tissue, but this organ performs a large number of functions, including endocrine. White adipose tissue is an important endocrine organ that produces hormones and biologically active substances called adipokines and regulates many physiological processes. In this review, we focus on the omentum, the visceral white adipose tissue depot, as an adipocyte-rich organ. The article highlights the latest data on the morpho-functional features of the greater omentum as an endocrine organ and their significance in the development of obesity and type 2 diabetes. Changes in the greater omentum in obesity and their relationship with this disease are also described. Morphological changes in the greater omentum indicate that ischemia also plays an important role in the development of adipose tissue dysfunction in obesity.

Key words: greater omentum, white adipocytes, adipose tissue, endocrine function, obesity, diabetes.

Connection of the publication with planned research works.

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Introduction.

Adipose tissue is deposited in various anatomical sites that mediate key aspects of metabolism, including energy storage, nutrient excretion, and thermogenesis. Although adipocytes make up more than 90% of the volume of adipose tissue, they make up less than 50% of its cellular composition [1]. The greater omentum is one such organ that can accumulate a large amount of adipose tissue. There are significant biological differences between adipose tissue of the greater omentum and

subcutaneous adipose tissue. The activity of lipolysis has different metabolic pathways, for example, goblet cells are more sensitive to catecholamine than subcutaneous adipose tissue adipocytes, and there are also differences in insulin receptors and their sensitivity. In addition, adipose tissue plays a key role in the local immune response and contains a population of multipotent mesenchymal stem cells that are more conducive to the growth of endometrial tumours than subcutaneous adipose tissue adipocytes [2, 3].

Despite the fact that more and more publications about the structure and function of adipose tissue have recently appeared, further research may provide new insights into its role in metabolic diseases. Molecular and cellular mechanisms that regulate the functional activity of adipocytes, the issue of the interaction of adipose tissue with other structures of the body, as well as

the role and significance of biologically active substances produced by adipocytes require in-depth research.

The aim of the study.

Summarize the existing scientific data on the morphofunctional features of adipose tissue of the greater omentum and its role in the development of obesity and type 2 diabetes.

Main part.

The greater omentum, in which visceral fat accumulates and is a derivative of the peritoneum, was previously considered an inert adipose tissue, but this organ performs a large number of functions [4]. The greater omentum serves as a centre for inflammatory reactions, provides mechanical cushioning and insulation, and also participates in heat production to regulate body temperature. All these processes can change adaptively or maladaptively during weight loss or gain [5, 6].

The greater omentum is a visceral adipose tissue that is covered with a layer of mesothelium, which does not contribute to the formation of adipocytes in it. Adipose tissue in the greater omentum is located both in the form of complexes of adipocytes that form fat particles, separated by layers of connective tissue, and in the form of single fat cells. Blood and lymphatic vessels, nerve endings, as well as thin connective tissue trabeculae, which make up the framework of the lobule, are located inside the clusters of fat cells [7]. Adipose tissue surrounds the intracapsular blood vessels in a ring-like manner, with more adipose tissue often located near the venules than with the afferent vessels. The thickness of the layer of loose connective tissue that separates adipocytes from blood vessels varies from complete absence to 50-80 μm [8].

The greater omentum contains mainly white adipose tissue. White adipocytes usually have one large lipid droplet that occupies most of the cell and relatively few mitochondria [9]. The primary function of the white fat of the greater omentum is to store and release energy in response to changes in systemic energy levels. In addition, white adipose tissue is an important endocrine organ that produces hormones and biologically active substances called adipokines and regulates many physiological processes. Adipokines play important roles in regulating whole-body metabolism, including promoting insulin sensitivity (eg, adiponectin), insulin resistance (eg, resistin, RBP4, lipocalin), and inflammation (eg, TNF- α , IL-6, IL-1 β , IL-8, IL-18 and sFRP5) [10]. It should be noted that the level of production of these substances differs in adipocytes of subcutaneous adipose tissue and fat cells of visceral adipose tissue. For example, the expression and secretion of IL-6 and adiponectin is higher in visceral adipose tissue, and leptin is higher in subcutaneous adipose tissue. Excessive development of adipose tissue, especially in the visceral area, is directly correlated with insulin resistance, hyperglycemia, dyslipidemia, arterial hypertension, prothrombic and proinflammatory conditions [11, 12].

To date, leptin, adiponectin, tumour necrosis factor- α , IL-6, plasminogen activator inhibitor-1, resistin, proteins of the renin-angiotensin system, and to a lesser extent – adipophilin, adipsin, monobutyrin, stimulating protein acetylation, have been studied to a greater extent. Some of the adipokines have been discovered recently, such as visfatin, and a large number have not yet been discovered [11, 13]. In addition,

adipose tissue contains an arsenal of enzymes capable of activating, interconverting and inactivating sex steroids – cytochrome P450-dependent aromatase, 3- β -hydroxysteroid dehydrogenase, 5- α -reductase, etc. [11, 14, 15].

Leptin, identified in 1994, is a protein predominantly produced by mature adipocytes in response to food intake and suppresses appetite by regulating neural circuits located in the brain. It provides most of its effects by acting on the brain, namely on surface receptors in neurons of the lateral and medial hypothalamus [16, 17]. Its level in the blood reflects the state of filling of adipose tissue depots and, thus, is directly related to the energy reserves in the body. A decrease in the level of circulating leptin due to a decrease in the mass of adipose tissue causes behavioral, metabolic and endocrine reactions aimed at replenishing and preserving the body's fat reserves. Among these responses are an increase in energy intake, a decrease in energy expenditure, and a decrease or complete inhibition of energy-intensive processes such as reproduction and processes related to the immune system [18]. Leptin also promotes lipid oxidation and mitochondrial biogenesis and accelerates energy expenditure in peripheral tissues through both local signaling and regulation of brain-derived factors. Circulating blood leptin is actually increased in obesity, but hypothalamic resistance to leptin exacerbates obesity through inhibition of appetite control and lipid oxidation [17, 19].

Adiponectin is a protein that is exclusively produced by mature adipocytes and transmits a signal through specific receptors (AdipoR1 and AdipoR2) [20]. Its level in the circulating blood is closely related to the functional integrity of adipose tissue and decreases with obesity. Adiponectin functions as a potent insulin sensitizer and suppressor of cell death and inflammation, directly contributing to antidiabetic and antiatherosclerotic effects. It acts on the liver by reducing gluconeogenesis, on skeletal muscle by increasing fatty acid oxidation, and on pancreatic β -cells and cardiac muscle cells as a key antilipotoxic agent, performing many of these functions based on its effects on sphingolipids. That is, adiponectin prevents obesity and has an antidiabetic effect, alleviates insulin resistance, stimulates lipid oxidation and anti-inflammatory reaction [21]. There are two different forms of circulating adiponectin in plasma: the low molecular weight trimer and the hexamer. The trimer aggregates with the help of disulfide bonds into high-molecular multimers. These multimers are the main biologically active forms, and each of them can exhibit different biological effects. In obesity, circulating plasma adiponectin levels are reduced, and this altered adiponectin oligomeric profile is considered a reliable clinical indicator of metabolic disorders [22].

Resistin, which is mainly secreted by white adipose tissue, was thought to contribute to obesity by reducing glucose tolerance and insulin action. Given that no receptor for resistin has been identified, the molecular pathways through which resistin induces insulin resistance in obesity are still unknown. However, it has been established that in humans, despite the fact that resistin was initially recognized as a potential factor in the link between obesity and diabetes, it is not the main factor that determines insulin resistance. Indeed, human plasma resistin appears to correlate with insulin resist-

ance as a consequence of obesity itself rather than as an independent causative factor [23].

Retinol-binding protein 4, another adipokine activated in the serum of insulin-resistant individuals, impairs insulin signaling in liver and muscle. On the contrary, omentin, found in human scapular fat depot, regulates blood glucose levels by enhancing the action of insulin and decreases in obesity [24].

In addition to peptide adipokines, lipid metabolites called "lipokines" have also been identified. In adipose tissue, C16:1n7-palmitoleate is synthesized de novo by stearoyl-CoA desaturase-1. Secreted C16:1n7-palmitoleate improves muscle sensitivity to insulin and inhibits fat accumulation in the liver [19, 25].

So, adipose tissue is an important endocrine organ, and the greater omentum plays an important role in this. Dysfunction of adipokines, lipokines and their ratio plays an important role in the development of obesity and type 2 diabetes. Obesity and type 2 diabetes are two diseases that in most cases share metabolic abnormalities; however, there are also differences. Some studies have shown that approximately 30% of obese patients have normal blood glucose and lipid levels despite the accumulation of abdominal adipose tissue. A comparison of the expression of several genes in adipose tissue associated with obesity and/or diabetes revealed that their expression is different in individuals with and without obesity and diabetes. These genes are associated with inflammation, cholesterol transport, and adipocyte differentiation [26].

The development of obesity and associated metabolic disorders is facilitated by the plasticity of adipose tissue [27]. According to some data, the growth of adipose tissue and changes in metabolic processes in it can be influenced by the level of vascularization of adipose tissue [28, 29]. However, recent studies show that in obese patients, the number of capillaries in visceral adipose tissue is reduced compared to non-obese individuals,

and the degree of vascularization was almost the same regardless of the degree of obesity or changes in the metabolic profile [30]. Such changes indicate that ischemia plays an important role in the development of adipose tissue dysfunction in obesity, which is confirmed by some studies [31, 32].

Morphologically, patients with obesity and type 2 diabetes have a significant increase in the size of adipocytes in visceral adipose tissue, but the same changes are observed in people with prediabetes. This indicates that the changes that occur in adipose tissue are not the result of prolonged hyperglycemia, and may be associated with genetic or epigenetic factors of patients [30]. However, it should be noted that the increase in the size of adipocytes is more pronounced in subcutaneous adipose tissue than in visceral adipose tissue, including in the greater omentum, and a change in the ratio between hypertrophy and hyperplasia of adipose tissue contributes to metabolic imbalance [33]. According to Belligoli A. and co-authors [30], it was established that in obesity, the increase in adipose tissue occurs due to hypertrophy and hyperplasia, which explains the relative decrease in the number of vessels in adipose tissue.

Conclusions.

Visceral adipose tissue plays an important role in the development of obesity and associated metabolic disorders. The identification of several adipokines and lipokines with different cellular actions emphasizes the central role of adipose tissue depots as endocrine organs in the dynamic regulation of homeostasis and metabolic processes. The function and morphology of adipose tissue of the greater omentum are dynamically interrelated, which requires a more detailed study.

Prospects for further research.

In the future, it is planned to investigate the proliferative activity of adipose tissue in obesity and type 2 diabetes.

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ЖИРОВА ТКАНИНА ВЕЛИКОГО ЧЕПЦЯ: МОРФОЛОГІЯ ТА ФУНКЦІЯ

Кошарний В. В., Лисаченко О. Д., Филенко Б. М., Ройко Н. В., Коноваленко С. О.

Резюме. Великий чепець є одним з органів, що може накопичувати велику кількість жирової тканини. Жирова тканина чепця відіграє ключову роль у місцевій імунній відповіді та містить популяцію мультипотентних мезенхімальних стовбурових клітин. У великому чепці розташовується переважно біла жирова тканина. Білі адипоцити зазвичай мають одну велику ліпідну краплю, яка займає більшу частину клітини, і відносно невелику кількість мітохондрій. Основною функцією білого жиру великого чепця є накопичення та вивільнення енергії у відповідь на зміни рівнів системної енергії. Крім того, біла жирова тканина є важливим ендокринним органом, що виробляє гормони та біологічно активні речовини, які називаються адипокінами та регулюють багато фізіологічних процесів.

Мета роботи – узагальнити існуючі наукові дані морфофункціональних особливостей жирової тканини великого чепця та її роль у розвитку ожиріння та цукрового діабету типу 2.

Адипокіни відіграють важливу роль у регулюванні метаболізму всього організму, включаючи сприяння чутливості до інсуліну, інсулінорезистентність і запалення. Порушення функції адипокінів, ліпокінів та їх співвідношення відіграє важливу роль у розвитку ожиріння, цукрового діабету 2 типу. Ожиріння та цукровий діабет 2 типу є двома захворюваннями, які в більшості випадків мають спільні метаболічні порушення; однак є й відмінності. Розвитку ожиріння та пов'язаних з ним метаболічних порушень сприяє пластичність жирової тканини. За деякими даними на ріст жирової тканини і зміни метаболічних процесів у ній можуть впливати рівень васкуляризації жирової тканини. Проте, недавні дослідження показують, що у пацієнтів з ожирінням кількість капілярів у тканині вісцерального жиру зменшується порівняно з людьми без ожиріння, а ступінь васкуляризації був майже однаковим незалежно від ступеня ожиріння або зміни метаболічного профілю. Морфологічно у пацієнтів на ожиріння та цукровий діабет типу 2 у вісцеральній жировій тканині спостерігається значне збільшення розмірів адипоцитів, але зміни, які виникають у жировій тканині, не є результатом тривалої гіперглікемії, а можуть бути пов'язані з генетичними або епігенетичними факторами пацієнтів. При ожирінні збільшення жирової тканини відбувається за рахунок гіпертрофії і гіперплазії, а це пояснює відносно зменшення кількості судин у жировій тканині.

Ключові слова: великий чепець, білі адипоцити, жирова тканина, ендокринна функція, ожиріння, цукровий діабет.

ADIPOSE TISSUE OF THE GREATER OMENTUM: MORPHOLOGY AND FUNCTION

Kosharnyi V. V., Lysachenko O. D., Fylenko B. M., Roiko N. V., Konovalenko S. O.

Abstract. The greater omentum is one organ that can accumulate a large amount of adipose tissue. Adipose tissue plays a key role in the local immune response and contains a population of multipotent mesenchymal stem cells. The greater omentum contains mainly white adipose tissue. White adipocytes usually have one large lipid droplet that occupies most of the cell and relatively few mitochondria. The primary function of the white fat of the greater omentum is to store and release energy in response to changes in systemic energy levels. In addition, white adipose tissue is an important endocrine organ that produces hormones and biologically active substances called adipokines and regulates many physiological processes.

The aim of the work – summarize the existing scientific data on the morphofunctional features of adipose tissue of the greater omentum and its role in the development of obesity and type 2 diabetes.

Adipokines play important roles in regulating whole-body metabolism, including promoting insulin sensitivity, insulin resistance, and inflammation. Dysfunction of adipokines, lipokines and their ratio plays an important role in the development of obesity and type 2 diabetes. Obesity and type 2 diabetes are two diseases that in most cases share metabolic abnormalities; however, there are also differences. The development of obesity and associated metabolic disorders is facilitated by the plasticity of adipose tissue. According to some data, the growth of adipose tissue and changes in metabolic processes in it can be influenced by the level of vascularization of adipose tissue. However, recent studies show that in obese patients, the number of capillaries in visceral adipose tissue is reduced compared to non-obese individuals, and the degree of vascularization was almost the same regardless of the degree of obesity or changes in the metabolic profile. Morphologically, patients with obesity and type 2 diabetes have a significant increase in the size of adipocytes in visceral adipose tissue, but the changes that occur in adipose tissue are not the result of prolonged hyperglycemia and may be associated with genetic or epigenetic factors of patients. In obesity, the increase in adipose tissue occurs due to hypertrophy and hyperplasia, which explains the relative decrease in the number of vessels in adipose tissue.

Key words: greater omentum, white adipocytes, adipose tissue, endocrine function, obesity, diabetes.

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ADAPTIVE CHANGES OF THE CIRCULATORY SYSTEM IN STRENGTH SPORTS ATHLETES

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Several factors determine high performance in sports of higher achievements, the main ones of which are the peculiarities of physical load, the construction of the training process, the functional reserves of the athletes' bodies, etc. The effectiveness of sports activity is reflected in the form of certain reactive and adaptive changes in functional systems that make the most significant contribution to achieving sports results, including the blood circulation system. It is known from the literature that the long-term adaptation of the executive organs of the circulatory system after regular exposure to sports loads manifests itself in the form of changes in the morphological and physiological parameters of its executive organs, which, together with the adaptation of the mechanisms of regulation of the