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METABOLIC DISORDERS — THE BASIS OF AGE-RELATED DISEASES OR AGING OF THE ORGANISM - STATE OF THE PROBLEM

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Abstract: Metabolic syndrome (MS) is a symptom complex that combines lipid and carbohydrate metabolism disorders and their pathological manifestations. The concept of MS was developed in recent years, summing up the results of studies of the causes of the high frequency of cardiovascular complications and atherosclerotic manifestations in type II diabetes. At the end of the 20th century, interaction between specialists studying the problem of atherosclerosis and diabetologists began to emerge. Attention was drawn to such a risk factor as the level of triglycerides in the blood, and to such objects of study as fatty acids and adipose tissue. Research in this area was also initiated by the obesity epidemic that engulfed the United States. As a result, the role of visceral adipose tissue in the development of insulin resistance was determined. Epidemiological studies have shown that the incidence of MS, visceral obesity and insulin resistance depends on ethnicity, lifestyle and age: it increases gradually as the body ages, sharply increasing in people over 50. Age is currently considered an independent risk factor for type II diabetes. The phenomenon of insulin resistance has not been adequately studied at present, the causes of visceral tissue growth have not been clarified, and the factors in the development of the pathological process have not been identified. Nevertheless, it is becoming increasingly clear that metabolic disorders underlying atherosclerosis and type II diabetes can be considered a subject of gerontology. In this regard, we have developed and presented a program of scientific research to study the causes and consequences of glucose, fatty acid and cholesterol metabolism disorders and their relationship with aging.

Key words: insulin resistance, adipose tissue, metabolic disorders, age.

Atherosclerosis is a disease characterized by the deposition of lipids, primarily cholesterol and its esters, in the intima of blood vessels. Experimentally, the etiology of atherosclerosis was determined first by feeding rabbits meat (Ignatovsky A. I., 1908), then egg yolks (Stukkey N. V., 1910), and finally, directly cholesterol (Anichkov N. N. and Khalatov S. S., 1912). Thus, it was shown for the first time that: 1) atherosclerosis can develop in response to the consumption of food of a certain composition; 2) cholesterol is the "culprit" of atherosclerosis; 3) atherosclerosis is not necessarily a phenotypic manifestation of the aging of the body. The conclusions that followed from the observations of St. Petersburg scientists formed the basis of the cholesterol theory of atherosclerosis, subsequently developed by N. Anichkov, and meat and eggs were classified as atherogenic food products for many years. In 1947, N. Anichkov argued that "atherosclerosis is not an expression of the natural senile wear and tear of arteries, but a special disease of them, which only reaches a sharp development in old age, but begins at a much earlier age." This phrase contains two ideas related to gerontology: the first is that as the body ages, its tissues "wear out"; the second is that aging is accompanied by a continuous (throughout ontogenesis) accumulation in the body of certain metabolic products (for example, cholesterol) or disorders that lead to vascular pathology in old age. N. Anichkov's

cholesterol theory becomes the main working hypothesis explaining the etiology of atherosclerosis, which will celebrate its centenary in 2019. No matter how much this theory is criticized today, and no matter how much the priority of Russian scientists in its creation is hushed up, it gave a powerful impetus to research in the field of lipid and cholesterol metabolism. Over the past years, it has been found that in the blood there is a system for transporting hydrophobic lipids in an aqueous environment, consisting of a complex of lipids with certain proteins (lipoproteins). These proteins (apoproteins) not only carry out passive transport, but also participate in the synthesis of new lipids (cholesterol esters), in the hydrolysis of blood fats (triglycerides), in the distribution of flows of certain classes of lipids to those organs and tissues where they are the main substrates.

Thus, cholesterol is transported by lipoproteins to the liver, where bile is synthesized from it, and to steroidogenic tissues, where sex hormones are synthesized. The system of distribution of cholesterol flows to the liver and steroidogenic tissues has gender differences, which causes different susceptibility to the development of atherosclerosis in young and middle-aged men and women. The cause of hereditary (familial) hypercholesterolemia, leading to early myocardial infarctions, has been found. It lies in the genetic modification of a specific receptor for low-density lipoproteins (LDL), expressed in hepatocytes. Reduced sensitivity of the receptor blocks the flow of cholesterol to the liver. LDL "gets stuck" in the blood, is modified by specific enzymes, is recognized by macrophages as "foreign elements" and is captured by them in large quantities. Recently, a hypothesis has been put forward that vascular intimal macrophages are part of the reticuloendothelial system. These macrophages express the non-specific protein CD36, which by its properties is a "scavenger" receptor. After the general outline of the cholesterol transport system in the bloodstream was clarified, LDL began to be considered the main "culprit" of atherosclerosis, and their elevated blood levels are still considered a diagnostic sign of this disease.

LDL transport esterified cholesterol in the blood, which is synthesized in high-density lipoproteins (HDL). An increase in the number of LDL particles leads to a compensatory decrease in the content of HDL particles. The latter receive their cholesterol partly from macrophages, freeing them from its deposits. This property (reverse cholesterol transport) allowed them to be called "antiatherogenic". A decrease in the concentration of HDL cholesterol in the blood is considered the second most important diagnostic criterion for the development of atherosclerosis. It turned out that women have a higher HDL content than men. This explains why women suffer from atherosclerosis less often than men. HDL has another function, which was discovered after the discovery of new class B "scavenger" receptors (scavenger receptors B1 - SRB1). These receptors are located in steroidogenic tissues and are specific to HDL. Reproductive function depends on the activity of SRB1, the degree of their expression, and the amount of HDL.

An approach to solving this problem may be developed when another direction of searching for the etiology of atherosclerosis, which emerged at the end of the 20th century, i.e. only some 20 years ago, is finally formed. It would seem quite unexpected to look for the origins of atherosclerosis in carbohydrate metabolism disorders. However, the results of prospective studies show that with diabetes, the risk of developing cardio- and cerebrovascular complications increases by 3-5 times. Diabetes is a long-known medical problem. Despite the fact that this disease can also occur at a young age, the main group of the population that it affects are elderly people. At the beginning of the 20th century, the theory of aging was popular, according to which every person suffers from diabetes in a latent form to one degree or another, and it manifests itself as a disease towards the end of life. This led to the conclusion that the metabolic disturbances underlying diabetes are either the cause of human aging or closely associated with aging. Indeed, diabetes is becoming an important social problem as the world's population ages. Almost half of people with diabetes are over 60 years of age, but the highest incidence of diabetes in a country such as the United States is observed in the over 80 age group. By 2050, the number of people with diabetes in the United States is expected to increase to 40 million. Each year, up to 1.3 million new cases of diabetes are

registered in the United States, and only 5-10% of these are type I diabetes, while 90-95% are type II diabetes. More than 90% of elderly people with type II diabetes are characterized by insulin resistance and relative insulin deficiency. More than 80% of people diagnosed with type II diabetes die from cardiovascular disease.

Obesity is accompanied by an increase in the blood content of triglycerides, which are in a bound form with apoprotein B-100, lipoproteins of very low density. The latter are secreted into the blood by the liver, and the triglycerides contained in them are hydrolyzed in the blood by the enzyme lipoprotein lipase, which is secreted by adipose tissue. Freed from triglycerides, apoprotein B-100 is loaded with cholesterol esters, which come from HDL, and is converted into LDL. Fatty acids released during the hydrolysis of triglycerides enter the cells. There are no specific fatty acid carrier proteins on the plasma membrane of cells, since fatty acids freely overcome the lipid bilayer. Insulin promotes faster penetration of fatty acids into the cell, especially in adipose tissue, where it stimulates lipogenesis by suppressing hormone-sensitive lipase. Triglycerides are becoming a focus of attention among atherosclerosis experts, and are being called the "forgotten risk factor."

Atherosclerosis, type II diabetes, cardio- and cerebrovascular diseases that manifest in the older age group are the subject of geriatrics. In gerontology, however, the situation has historically developed in which the metabolic processes that underlie the functioning of a multicellular organism as a system have fallen outside the scope of its interests. Research in gerontology has traditionally been devoted to identifying aging genes, cellular aging and death, failures in the central regulatory system (pineal gland, hypothalamus), oxidative stress, tumor growth, limitation of the energy value of nutrition, and so on, that is, in fact, to individual components of the functioning of the organism as a system [27]. Of the above list, only the limitation of the energy value of nutrition seems to be related to metabolic processes. Meanwhile, genes, one way or another associated with aging, regulatory systems of cells and the body, oxidative stress and tissue malignancy can be combined within the framework of a systemic process of functioning of the body as a whole, in which energy flows play an important role. In our opinion, the time has come to make another aspect of the existence of a living organism — systemic energy metabolism — the subject of gerontology, that is, to combine achievements in the field of the etiology of atherosclerosis and type II diabetes with the knowledge that gerontology has accumulated.

This program is based on the idea of the functioning of the metabolic node. It is through this node that the energy flow passes, and its role is to maintain the stationarity of this flow in order to create homeostatic conditions, or constancy of the internal environment. The internal environment of the body is external in relation to an individual cell. A cell can perform its functions under optimal environmental conditions, one of the factors of which is the steady-state flow of energy substrates. In the metabolic node, homeostasis of glucose and fatty acids is ensured by dumping excess substrates into adipose tissue. The fact that the mass of adipose tissue continuously increases after the end of the growth period of a multicellular organism indicates that the energy flow into the body exceeds its expenditure. The growth of adipose tissue has its physiological limits, which are determined by the size of the adipocyte. However, the question of whether the increase in the mass of adipose tissue is a consequence of erythrocyte hypertrophy or hyperplasia remains open. It is also necessary to find out why visceral adipose tissue, which is not characterized by accumulating fat, mainly grows. Fatty acids from visceral adipose tissue enter the liver, that is, it, in fact, controls the level of triglycerides in the blood. In turn, blood triglycerides through the lipoprotein system are closely linked to cholesterol metabolism, the production of bile and sex hormones. Thus, the function of visceral adipose tissue is to regulate the flow of energy into the body (via bile through the intestine) and reproductive function. If we take into account that sex hormones regulate the proliferation of subcutaneous adipose tissue and muscle tissue, the role of visceral adipose tissue significantly expands.

CONCLUSION. The growth of visceral adipose tissue is the trigger that starts the pathological process. The pathology develops both in response to an increase in the mass of the tissue itself and to an increase in the content of circulating fatty acids. An increase in the mass of adipose tissue initiates inflammation, and an increase in the concentration of fatty acids leads to the accumulation of fat in lean tissues, which has consequences such as insulin resistance, apoptosis, oxidative stress, cell differentiation according to the adipocyte-like phenotype and a number of other phenomena, among which one can name the structural and functional reorganization of mitochondria. Insulin resistance, caused by the accumulation of fatty acids in the cell, induces an increase in the concentration of glucose in the cell. Excess fatty acids and glucose in the blood and pericellular space make possible the manifestation of glucose and lipotoxicity. The proposed research program is aimed at studying these phenomena and clarifying their connection with the aging of the body.

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