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Metabolic Syndrome Of Type 2 Diabetes Mellitus

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Annotation: The subsequent development of events in the pathogenesis of DM2 includes increased insulin secretion as a response of the body to a lack of its action. However, prolonged hyperinsulinemia leads to dysfunction of pancreatic β-cells and a decrease in insulin synthesis against the background of persistent IR. As a result, the insulin-dependent transport of glucose from the bloodstream to the cells of muscle and adipose tissues is finally disrupted, glucose synthesis in the liver increases and, accordingly, its secretion into the bloodstream
Keywords: insulin resistance, diabetes mellitus, hyperglycemia, metabolic syndrome, brown adipose tissue

Despite the great progress made in understanding these mechanisms, many important details remain unclear, hindering the creation of targeted drugs.
Stable hyperglycemia develops, in which glucose and its metabolic products, carrying chemically active aldo- and keto groups, act on proteins and plasma cells, as well as on the vascular endothelium, physically modifying and disrupting their functions.
A condition long before the development of DM2, prediabetes. This concept includes such disorders of carbohydrate metabolism as impaired fasting glycemia and impaired glucose tolerance. At this stage, it is possible to prevent pathological changes and restore the sensitivity of target cells to insulin. In this regard, early diagnosis of IR is of great practical importance. The presence of IR can be determined either at the molecular level by specific IR markers, or at the systemic level using a hyperinsulinemic euglycemic clamp (HEC).
Both approaches are invasive, time-consuming, labor- and resource-intensive, and require hardware and well-established protocols. At the same time, HEC is

increasingly being used to evaluate IR, whereas molecular markers of IR have not been determined experimentally and have not been verified in the clinic. The vascular endothelium is not classically insulin-dependent, but serves as the primary target of insulin. Endothelial dysfunction mediates the association of DM2 with cardiovascular complications.

Metabolic syndrome is a pathological condition associated with increased resistance of tissues to the effects of insulin. Gradually, it causes an increase in the level of insulin in the blood plasma and a violation of glucose tolerance. If left untreated, the risk of developing type 2 diabetes is high. The consequence of the changes are abdominal obesity, arterial hypertension, hyperuricemia (increased concentration of uric acid in the blood).

Metabolic syndrome cannot be called an independent disease, since it is a set of symptoms that develop simultaneously and increase the risk of even more severe disorders. The problem of combating type 2 diabetes mellitus (DM2) is extremely relevant, but drug therapy for this disease is still not effective enough. This is largely due to the variety of risk factors and mechanisms for the development of insulin resistance (IR), the primary link in the pathogenesis of DM2, as well as the lack of means for its clinical diagnosis. IR manifests itself as the resistance of cells to the action of insulin and arises from a violation of the mechanisms of the insulin signal in them.

Under conditions of hyperglycemia, chemically active glucose and its metabolic products, such as glyoxal and methylglyoxal, have a damaging effect on plasma and blood cell proteins, as well as glycate vascular endothelial proteins. Together with malonic dialdehyde, which accumulates as a result of the peroxidation of excess lipids at earlier stages of the pathogenesis of DM2, these compounds ensure the development of oxidative and carbonyl stress that disrupt endothelial functions. Endothelial dysfunction manifests itself in a decrease in its barrier properties, the synthesis of the main vasorelaxant, nitric oxide (NO), and an increase in the synthesis of a powerful vasoconstrictor, endothelin-1 (ET-1). However, the molecular targets of glucose and the subtle mechanisms of endothelial dysfunction in DM2 remain unclear.

Molecular markers of IR should be sought among the components of the insulin cascade, which demonstrate a decrease in insulin activation under IR conditions. The target tissues of insulin (liver, muscle or adipose tissue) should be the object of analysis, which, accordingly, dictates the need to work with biopsy material and in practice significantly complicates such an analysis. The insulin cascade

includes a receptor, an insulin receptor substrate (IRS protein), a PI3 kinase cascade, and a Glut-4 glucose transporter activation system. Akt protein kinase serves as a key target of the PI3 kinase cascade. It phosphorylates the protein AS160 (Akt substrate of 160 kDa), which regulates the release of Glut-4 onto the cell membrane and the transport of glucose into the cell.

Tyrosine phosphorylation of the insulin receptor and its substrate IRS determines the activity of the insulin cascade, and insulin-dependent, site–specific phosphorylation of Akt and AS160 is an indicator of its activity. These parameters can be measured in cell lysates or tissue homogenates. Impaired activity of the insulin cascade is associated with serine phosphorylation of IRS under the action of a number of enzymes under conditions collectively referred to as risk factors for the development of IR.

Abdominal obesity, hypertension and metabolic disorders of carbohydrates and fats, characteristic of MS, are observed in the disease and the Itsenko-Cushing syndrome. Even the appearance of patients with MS and Itsenko-Cushing's disease is often identical, which requires differential diagnosis.

For differential diagnosis with a particular endocrine disease occurring with a clinical picture similar to MS, it is necessary to use additional research methods. Computed tomography of the adrenal glands will allow to establish or exclude their primary pathology. CT or nuclear magnetic resonance imaging of the pituitary gland will help to assess its functional and structural state and determine the presence or absence of micro- or macroadenomas. Itsenko-Cushing's disease is characterized by the presence of a pituitary tumor and bilateral adrenal hyperplasia. Itsenko-Cushing syndrome may be associated with unilateral damage to the adrenal glands (corticosteroma, adenocarcinoma of the adrenal cortex).

Hormonal research methods can provide additional assistance in differential diagnosis. For this purpose, the blood content of cortisol, aldosterone, ACTH, prolactin, thyroid-stimulating hormone, etc. is determined. Patients with MS may also experience a slight increase in cortisol, prolactin, thyroid-stimulating hormone, and ACTH levels, but with primary endocrine pathology, the excess concentration of these hormones will be tens or hundreds of times higher than normal values. If the presence of aldosteroma is suspected, the determination of the content of aldosterone and renin will help in differential diagnosis. The detection of pheochromocytoma will be facilitated, along with CT scans of the adrenal glands and paraaortic region, by the study of catecholamines in blood

and urine and vanillyl-almond acid in urine, especially in the period after a hypertensive crisis.

Type 2 diabetes mellitus (DM2) is a socially significant disease, the therapy of which is still insufficiently effective, partly due to the multifactorial development of disorders of carbohydrate metabolism, the mechanisms of which are not fully disclosed. Insulin resistance (IR) is the first link in the pathogenesis of DM2 and is associated with a decrease in the ability of insulin to enhance the uptake of glucose from the bloodstream by target cells.

Factors such as genetic predisposition, overeating and obesity, stress and chronic inflammation in conditions of impaired insulin signal transmission inside cells are involved in the development of IR. The molecular mechanisms and markers of IR are insufficiently characterized, which prevents early diagnosis and the creation of preventive drug therapy. Hyperinsulinemic euglycemic clamp remains the "gold standard" for the diagnosis of IR in a clinic setting.

Hyperglycemia is a long-term consequence of IR, in which the damaging effect of a number of molecular metabolic disorders, including oxidative and carbonyl stress of target cells, is realized. Molecular chaperones and small heat shock proteins have a protective effect in the early stages of the pathogenesis of DM2, preventing the development of endoplasmic reticulum stress and apoptosis. Vascular endothelial dysfunction is associated with DM2 and its cardiovascular complications, however, it is unclear at what stage of pathogenesis these changes occur and what their molecular inducers are. Finally, the issue of transcriptional regulation of adipogenic differentiation of progenitor cells during the formation of new fat depots, as well as the mechanisms of activation of "brown" and "beige fat", demonstrating hypolipidemic and hypoglycemic effects, is even less studied. The purpose of this work was to elucidate the molecular mechanisms of the development of IR and endothelial dysfunction, the role of transcription factor Prep1 and small heat shock proteins, to evaluate new methods for the diagnosis of IR and to identify biomishens for new antidiabetic drugs.

It is not often possible to determine the organic cause of obesity. Only 1 out of 1 thousand patients can have a disease that leads to an increase in MT. Nevertheless, a thorough examination of patients to determine the possible cause of obesity is necessary, as this greatly affects treatment tactics.

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