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E-Book

Diabetes 2 Types for Fellow an Edition _ Part 1

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"We cannot solve a problem, using the same mentality which we have created them"

-A. Einstein

Competent treatment a diabetes of 2 type at Children and Adults - psychosomatic and an infectious disease (1 part).

Clinical a BOOK (clinical cases are approved in the practice).

(Own researches and the analysis of references).

Competent treatment of the DIABETES 2 types

Prevalence and disease of a diabetes continues to increase and, on the specified data of experts the CART (2007), by 2010 in the world 4 million, and by 2025 - 380 million sick of a diabetes from which about 90% will fall to a diabetes of 2 type will be more than 239. Practically similar situation with prevalence and disease of a diabetes is observed and in the Russian Federation.

In Russia with a diabetes are ill about 8 million persons. MODY - diabetes - the hereditary form of illness also is observed at children and teenagers. LADA-diabetes - the latent form of 1 type of a diabetes also is shown at adults.

Sugar a diabetes

The most widespread endocrine the disease representing a serious problem for national services of public health services in connection with considerable cost of treatment, high frequency and weight of complications of a diabetes which are the reason early invalidation, essential reduction of life expectancy and high cases fatality.

The diabetes of 2nd type

Is disease with the heterogeneous change of a carbohydrate exchange characterized insulin resistance of peripheral fabrics and infringement of secretion of insulin β-cages of islets Langerhans's.

Derivatives Sulphonil-urea opened M Janbon in 1942, here already more than 50 years are used for treatment of a diabetes 2 types. But these preparations have not given treatment from illness (the note Autor's).

To psychosomatic illnesses carry, first of all, a diabetes, hypertireoz and Bazedov's illness

In many cases their beginning is closely connected with an emotional pressure. Always the somatic anamnesis carefully gathers. The special attention is given to studying characterogical features sick - a clinical method and by means of psychological tests. According to a number of psychiatrists, in particular (N. Eu), the anamnesis of the patient with psychosomatic frustration should represent pathography and always to be biographic - history of a life and the case record cannot be separated in this case from each other.

The signs allowing the doctor and Clinical Pharmacist at collecting anamnestic data to suspect psychosomatic disease.

1. Presence of certain personal features, first of all, in frameworks accentuation or a psychopathic warehouse.
2. The biography rich with «crisis events».
3. Presence of family predisposition to certain illnesses.
4. Development of somatic and mental frustration in the form of phases, their periodicity.
5. The obvious tendency to occurrence or strengthening of a somatic and mental pathology during the critical periods of a life.
6. Existence at an individual of sexual problems.
7. A combination at one person of some the signs set forth above.

At studying of psychosomatic illnesses paramount value gets research of the person of the sick person. The doctors focused in this plan on given psychoanalysis, since such researchers, as H. F. Dunbar (1938) and F. Alexander (1943), constantly aspired to creation of the separate psychological profiles corresponding with a certain somatic pathology. If originally described by them characterological lines of such patients and illnesses corresponding to them were treated exclusively from psychoanalysis positions in the subsequent the similar treatment has started to be supplemented with them at first concept H. Selge about an adaptable syndrome, and further - the concept about emotional stress. The researchers rejecting psychoanalysis, study characterological features of patients with psychosomatic frustration, leaning on the kliniko-descriptive method in many respects corresponding to a method of studying of psychopathic characters, to the developed I. Gannuschkin's (1933).

The diabetes 2 types - is a classic example of multifactorial complex illness

In its development play an essential role such factors, as an inactive way of life and features of a diet as the interrelation of disease with adiposity is established. Also, in pathogenesis genetic factors as level concordance the given disease among monozygotic varies twins from 35 to 60% (among dz twins he less in 2 times) participate also. Such concordance even above, then at a diabetes of type I that specifies in the big role of a genetic component in pathogenesis a diabetes 2 of type. Besides, the risk of development of a diabetes of type 2 at descendants raises more than in 2 times if both parents suffer this disease.

Additional proofs of genetic predisposition are found out during large-scale researches of gene associations of 14 specific loci.

Some typical examples of such associations

Polymorphizm's the genes connected with function of b-cages and secretion of insulin, define the raised risk of development of a diabetes of type II. The most characteristic and reproduced communication mentions the factor of a transcription 7 similar to the factor 2 (TCF7L2), on a chromosome 10q, coding synthesis of the factor of a transcription of alarm way WNT. However, unlike sugar diabetes type I - the diabetes of type 2 is not connected with the genes participating in maintenance of immune tolerance and regulation (HLA, CTLA4 etc.), and has autoimmune no basis.

The basic display of a diabetes is hyperglicemia

Restoring sugar can enter reaction without participation of enzymes (non-enzymatic glycation) with amino groups of fibers therefore astable bases Schiff'a which will be transformed to Stabler products Amadory are formed. In the course of the further reactions products Amadory turn to irreversible connections - End-products Glycation's (EPG). Presence chronic hyperglycemia's at patients with a diabetes leads to the raised formation and accumulation EPG. Them find out in circulating blood and various fabrics, including, in walls of arteries. In a number of researches communication between raised level EPG in blood at patients a diabetes and development micro-and macrovascular complications has been shown.

One of explanations of existence of communication between raised level EPG in blood and development micro-and macrovascular complications is that in the course of formation EPG there is a change of structure of the fibers which are a part of a vascular wall, leading to infringement of their function. Including, change of structure of collagen can break restoration of a vascular wall after its damage at patients with a diabetes. Changes of structure of fibers have irreversible character and do not decrease at correction hyperglicemia's. EPG can inactivate oxide nitrogen which is endothelial the factor of a relaxation and renders anti-profilerative influence on smooth muscle cells (SMC) vessel walls. Along with it, there was a set of certificates to that interaction EPG with receptors to EPG on a surface of cellular membranes can lead to infringement of function of cages and promote occurrence of the receptor to EPG - fiber of superfamily of the antibodies, belonging to the class membranes receptors, has been allocated and described for the first time in 1992, as a receptor, capable to contact the various molecules formed as a result non-enzymatic glycation of fibers. Subsequently it has been revealed, that receptors to EPG can communicate with many other things ligands. The receptor to EPG consists from extracellular the domain, transmembrane the domain and a short cytoplasmatic tail.

In normal physiological conditions biosynthesis of receptors to EPG in all fabrics low, except for a pulmonary fabric. Biosynthesis of receptors to EPG can quickly and expressed, to increase in different types of cages in reply to quantity increase ligands to them at occurrence various pathophysiological conditions.

Including, biosynthesis of receptors to EPG increases in blood cells and the cages of the vascular wall playing a key role in occurrence of inflammatory reaction and in restoration of damaged fabrics, in reply to level increase ligands to a receptor to EPG which occurs at damage of a vascular wall. Accumulation ligands to a receptor to EPG and, as consequence of it, increase in biosynthesis of receptors to EPG occurs at a diabetes. Programmed biosynthesis of

receptors to EPG find out in neutrophils, monocytes, macrophages, T-limfotsitah, eosinophils, endothelial cages, and also in (SMC). Interaction of receptors to EPG with ligands leads to various cellular answers which depend not only on what ligands contact receptors, but also from concentration ligands, and also type of the cages which are carrying out synthesis of receptors to EPG.

Receptors to EPG influence development of the inflammatory reaction arising in reply to damage of a wall of an artery, and on its subsequent restoration. Synthesis of receptors to EPG increases activated endotelia's. Their subsequent interaction with ligands's mediates adhesion and migration of leukocytes in the inflammation centre. Acknowledgement of the similar fact is research (Frommhold D., 2007) in which it has been shown, that interaction of receptors to EPG endothelia's with Intergin's the M Alpha-Beta 2 (Mac-1) leukocytes at development of sharp inflammatory reaction in a vascular wall promotes their adhesion to endotelia's. To adhesion and migration of leukocytes in the inflammation centre promotes as interaction ligands with receptors to EPG Leukocytes. The destruction of cages is accompanied by passive allocation from them in extracellular space of fiber Ampharetid's (HMGB1) which contacting receptor Mac-1 neutrophiles, promotes their adhesion and migration in the damaged fabric. In research (Orlova V.V., 2005) it has been shown, that for interaction HMGB1 with Mac-1 on a surface neutrophile presence of receptors to EPG is necessary. In other experiment it has been revealed, that Amphoteric participates as in migration monocytes, which inhibit blocking of receptors to EPG.

Interaction Amphoterin's with receptors to EPG macrophages is accompanied by production them proinflammatory cytokines. Preservation of activity of receptors to EPG supports inflammatory reaction and promotes occurrence of a chronic inflammation. Damage of an arterial wall is accompanied by increase in synthesis of receptors to EPG in smooth muscle cells (SMC). Interaction of receptors to EPG with ligands stimulates proliferation and migration smooth muscle cells (SMC), and also synthesis smooth muscle cells (SMC) extracellular matrices. Receptors to EPG accept the important participation in regulation of processes of restoration of the damaged fabrics. Superfluous concentration ligands to receptors to EPG and interaction ligands with them can lead to adverse consequences, promoting occurrence of a chronic inflammation and superfluous proliferative to the answer.

One of processes is superfluous proliferation smooth muscle cells (SMC). Higher proliferation activity cultivated smooth muscle cells (SMC) patients with a diabetes in comparison with smooth muscle cells (SMC) patients without a diabetes has been revealed in research (Oikawa S., 2003). Similar law has been found out concerning cultivated smooth muscle cells (SMC) animals with a diabetes. Higher proliferation activity smooth muscle cells (SMC) at a diabetes can be connected with interaction of receptors to EPG on a surface smooth muscle cells (SMC) with EPG that has been shown in work (R. Wang, 2003).

Along with higher initial (before any interventions) the maintenance ligands to EPG in an organism of patients with a diabetes in comparison with patients without a diabetes, damage of a wall of an artery against a diabetes can lead greater to accumulation ligands and to grater to synthesis of receptors to EPG in media's and formed a neointim that has been shown in experiment on animals.

According to the authors, more expressed infiltration by leukocytes at sick of a diabetes it is connected with the raised synthesis of fiber S100A12 which is ligand's to receptors to EPG and, contacting them, mediates activation and migration of monotsitov/macrophages in the inflammation centre. Besides, in research (Kirstein M., 2003) it has been shown, that EPG are chemoattractant for monocytes and their migrations in a vascular wall promote. The subsequent interaction monocytes with containing EPG matrix is accompanied by production thrombocyte the growth factor - a stimulator proliferation smooth muscle cells (SMC) and production extracellular matrixes.

Soluble receptor to end-products Glications

There are some isoforms of a receptor to EPG which can be products alternative splicing mRNK, coding synthesis of high-grade receptors to EPG, and also to turn out as a result proteolitic rift extracellular parts of receptors to EPG under the influence of AdAm-proteaz - families albuminous peptidaz, rift an extracellular fragment membrans fibers. One of isoforms (esRAGE), formed as a result of alternative splicing and also the isoform (cRAGE), formed as a result proteolytic cleavage, are soluble isoforms and are designated by the term a soluble receptor to EPG (sRAGE). Unlike a high-grade receptor to EPG at both soluble isoforms is absent transmembrane and the cytoplasmatic domain. The mechanisms participating in regulation of formation of a soluble receptor to EPG, remain obscure. According to three-year supervision (Bower J.K. 2003) level of a soluble receptor to EPG is enough stable size, and unitary definition of level of a receptor can be used in researches according to risk of development of adverse events.

The soluble receptor to EPG can represent itself as a receptor of a trap for ligands to a receptor to EPG, thereby, blocking interaction ligands with receptors to EPG and, as consequence to it, preventing undesirable consequences of similar interaction.

The results of researches concerning as level of a soluble receptor to EPG at patients with a diabetes of 2 types - about high, and about lower level of a soluble receptor to EPG at this category of patients in comparison with patients without a diabetes. In a number of researches at sick of a diabetes find out a direct communication between receptor synthesis to EPG and level of a soluble receptor to EPG, and also between level EPG and level of a soluble receptor to EPG. While in other researches it is informed on a feedback. According to results of research (Shen Y., 2003) lower level of a soluble receptor to EPG at patients with a diabetes 2 types is interfaced to less favorable clinical results.

Proceeding from results of experimental researches on animals with a diabetes in which it has been shown, that introduction of a soluble receptor to EPG to mice with a diabetes suppresses occurrence and atherosclerosis progressing, and also formation neointims brakes. It should be used for the blessing of patients and medicine in whole (the note of the author).

Lower level of a soluble receptor to EPG should be connected with higher risk of development of adverse cardiovascular events. Similar law is noted in many researches, concerning communications between level of a soluble receptor to EPG and the forecast of chronic inflammatory diseases.

A number of researchers a direct communication between risk of development of adverse cardiovascular events and level of a soluble receptor to EPG at this category of patients try to explain to that diabetes presence is interfaced to the raised synthesis of receptors to EPG in reply to superfluous formation EPG and activity increase matrix metalloproteinase which rift an extracellular part at the increased quantity of receptors to EPG.

Accordingly, the it is more formed EPG, the it is more synthesized receptors to EPG and more level of a soluble receptor to EPG increases. However, because of the big number of receptors to EPG the quantity educated to EPG is not enough soluble receptor for neutralization of interaction of receptors to EPG from them ligands, that leads to adverse consequences.

It can be caused, as change in result non-enzymatic glication's structures of fibers of the vascular wall breaking their function, and the synthesis of receptors raised as a result of superfluous formation EPG to EPG blood cells and the vascular wall, taking part in development of an inflammation and restoration of a wall of an artery after its damage. This increase against already raised synthesis of receptors to EPG at patients a diabetes can appear so expressed, that the subsequent interaction of superfluous number of receptors with ligands to them can lead superfluous proliferation smooth muscle cells (SMC) and to superfluous synthesis extracellular matrix's.

Role thrombocytes in a hemostasis, an inflammation and restoration of fabrics

Thrombocytes are small, diameter 2-4 microns circulating in a blood-groove denuclearized cellular elements, playing the major role in hemostasis and thrombosis processes. Thrombocytes are formed in a bone brain at a fragmentation of the predecessors - megakaryocytes. Of one megakaryocyte's it is formed to 10 thousand thrombocytes. Life expectancy circulating in blood thrombocytes makes 5-9 days, then there is their recycling reticuloendothelial spleen and liver cages.

At damage of a wall of an artery as a result of intravascular intervention or rupture of the stable atherosclerotic plaque, circulating in blood thrombocytes the first of all cellular elements contact subendothelial matrix, forming the wall a blood clot. Accumulation thrombocytes in a place of damage of a wall of an artery is defined by specific interactions, such as a thrombosis-wall of a vessel (adhesion), trombotsit - trombotsit (aggregation) and a thrombosis-leukocyte (agglutination) which are regulated by a number of superficial fibers and soluble agonists. Adhesion thrombocytes is mediated by interaction glycoprotein complex Ib-IX-V (GPIb-IX-V), glycoprotein VI (GPVI) and integrin alpha-2-beta-1, which are on a surface thrombocyte, with the factor background Villebrand's and collagen from the damaged wall of a vessel. Adhesion thrombocytes to subendothelial extracellular matrix's in a damage zone consider as initial process which starts activation thrombocytes in the subsequent activated thrombocytes form platforms for the further replenishment of a place of damage thrombocytes and leukocytes. Liberation and-or production thrombocytes, leukocytes and cages of a wall of a vessel in a place of damage of soluble activators of cellular activity (adenosindiphosphat, tromboxan A2, the factor activating thrombocytes, adrenaline, serotonin and a number of other biologically active connections) promotes, strengthening activation thrombocytes to the further accumulation in a damage place thrombocytes and leukocytes. Activated thrombocytes, collecting in a damage zone, appear capable not only to participate in

blood clot formation, but also to initiate and-or accelerate inflammatory processes in an artery wall.

Not activated thrombocytes show very weak procoagulant activity and have no on the surface procoagulant phospholipids. These phospholipids settle down on an internal surface of a membrane and begin expressed on a surface activated thrombocyte thanks to the special mechanism named (Hemker H.C., 2003) membran «flip-flopom». At activation thrombocytes procoagulant phospholipids (basically phosphadilserins) by «flip-flop» are transferred from an internal surface thrombocyte on an external surface owing to what intact thrombocytes become procoagulant. Curling factors contact procoagulant phospholipids on a surface activated trombocyte's, that leads to activation X of the factor and complex formation protrombinaza's. Besides an obvious role in a hemostasis and a thrombosis, thrombocytes take part in development of an atherosclerosis, an allergy, rheumatoid an arthritis and even a cancer. It is established, that thanks to ability to liberation antimicrobic peptides and an expression a pattern-distinguishing of receptors, thrombocytes as are specialized cages of congenital immunity and modulators of the inflammatory answer.

At an atherosclerosis thrombocyte by interaction with circulating leukocytes and cages-predecessors promote attraction to a place of damage of cages of an inflammation. One of displays of such intercellular interactions is formation and occurrence in a blood-groove trombocytar - leukocyte units which promote development of a local inflammation through stimulation rolling's and the subsequent capture of leukocytes by a vascular wall. Thrombocytes, expressed on the surface of the squirrel of cellular adhesion, basically P-selektin, support a binding and rolling leukocytes in places of damage of a vascular wall. Such intercellular interaction reduces speed of movement of leukocytes along an internal surface of a wall of a vessel, allowing them is strong adhesive to a surface and subsequently to migrate in a vessel wall.

Important! High level in blood osteonektin-positive nucleated the cages bearing on the surface Trombocytar antigene CD41, can be an independent indicator of presence at the patient constrictive atherosclerotic defeat of arteries that it is necessary to include 2 types (the note of the author) in standards of diagnostics of an atherosclerosis and a diabetes.

Occurrence in peripheral blood of a considerable quantity lejkocytar - thrombocyte complexes at patients with Ischemic illness of heart (IIH) can be the important component, both a system inflammation, and one of ways indication inflammatory processes in a wall of a vessel which can lead to the further development of atherosclerotic damage and aterotromboz's. It has been shown, that at a sharp coronary syndrome trombocytar-lejkocytar units appear in a blood-groove much earlier, then standard indicators of activity/activation thrombocytes or routine markers necroz's a myocardium, such as troponin and MV-fraction creatinkinaza's change. It is important for early diagnostic pathologies and appointments of competent treatment with cured (the note of the author).

Active participation thrombocytes in inflammatory reaction as is connected with their ability to regulate chouming cages-predecessors in places of damage of fabrics. Endothelial and smooth muscle cells (SMC) again formed vascular wall (including, neointims) occur exclusively from nearby cages of a wall which migrate in places of damage and there proliferate. However later works have shown participation in remodeling and restoration of the damaged sites of a wall of an artery of cages-predecessors of a kostno-brain origin. The directed migration marrowy stem cages-predecessors in the damaged fabrics is the important link of reaction of an organism in reply to damage. Ability stem cages to the directed migration in «native» body or in damage area is caused by the specific biochemical signals which are starting with the «necessary» area. One of the most studied signals for directed choumimg's stem cages is fiber SDF-1 (stromal-derived factor-1) which is produced stromal by cages of a bone brain and «keeps» stem cages in the stem to a niche. It has appeared, that at interaction with the damaged fabric, activated thrombocytes expressed on the surface and are secreted SDF-1, creating, thus, an initial gradient of it chemocine in places of damage of a wall of a vessel. It causes one of the basic mechanisms chouming's cages-predecessors of a marrowy origin in damage places, including, at formation in these zones the wall a blood clot.

Thrombocytes actively participate in creation of niches for chouming's and the subsequent directed differentiation grasped in damage places stem cages-predecessors. Thrombocytes are capable not only to participate in development thrombotic reactions and inflammatory process in a vessel wall, but also actively to participate in regeneration of the damaged sites of a wall of a vessel.

Where to find that golden mean between harm and advantage?

Molecular genetics of a diabetes 2 types

Definition of genetic predisposition to a diabetes 2 types is extremely difficult problem as there is a set of the genes involved in its development, each of which has the small contribution to dis-

ease development. As a whole genes, associated with a diabetes 2 types, can be divided on "diabetogenic" (define insulin resistance or decrease in secretion of insulin) and nonspecific genes or genes-helpers (regulate appetite, power inputs, accumulation intraabdominal fat and others). Besides, the big role in development of a diabetes 2 types play environment and way of life factors. Important value in development of adiposity and a diabetes 2 types have the features of food behaviour being under the genetic control or formed owing to family traditions, and also an inactive way of life. Psychoemotional stresses lead to failure compensator mechanisms, demonstrations and aggravate a disease current.

By present time by a method genomic the analysis of associations it is revealed more than 15 genes involved in patogenez of a diabetes of 2 types

The hereditary form - on a shoulder of 11 chromosomes, on a shoulder of 19 chromosomes - responsible for receptor synthesis to insulin. Dangerously development of blindness (diabetic retinopatia), development of a heart attack of a myocardium, development of diabetic foot with the subsequent gangrene of the bottom finite nesses, nephropathies. Daily rhythms of insulin and glucose at this type remain, and also rhythms TTG, tireoid hormones, adrenocorticotropic hormone (ACTG) and cortizol's.

These are the genes defining lowered level of secretion of insulin by b-cages of a pancreas, and the genes responsible for lowered sensitivity of peripheral fabrics to action of insulin. The Pharmacogenetic treatment is necessary (note of autor's).

The genes defining decrease of functionality of b-cages

One of the main reasons of development of a diabetes 2 types - the lowered secretion of insulin by pancreas b-cages, most likely, in the big degree is connected with defined alleles genes KCNJ11 and ABCC8, squirrels Kir6,2 coding accordingly and a receptor sulfonylurea SURL These two fibers form in a membrane of b-cages of a pancreas the adjustable channel of transport of ions calium which functioning depends on concentration ATF (adenosintriphosphat).

At low level of glucose in blood and according to low concentration ATF in b-cages calium the channel is opened, and at the expense of functioning of this channel is created membrane the potential interfering penetration in a b-cage of ions of calcium.

After increase of concentration of glucose in blood it starts to get into b-cages at the expense of passive diffusion on a gradient of concentration which amplifies a carrier of glucose of type 2.

In a cage glucose phosphorylated by glucokinase to gljukozo-6-phosphate and metabolize to ATF means glycolis's or through cycle Crebsa's in mitochondrions. Increase of concentration ATF conducts to closing calium's the channel and depolarization a cellular membrane. It, in turn, leads to opening calcium's the channel and to increase of concentration of ions of calcium in b-cages that promotes movement of the granules containing insulin, through a membrane of b-cages and insulin secretion in a blood-groove. And potassic channels play an essential role in secretion of insulin stimulated with glucose and are a point of the appendix of action hypoglicamic sulfonylurea preparations which increase insulin secretion.

High frequency of these polymorphic markers in population can be one of the reasons of high risk of development of a diabetes 2 types in the general populations. A number of activating mutations of these genes underlies development neonatal a diabetes also sensitive to action sulfonylurea of preparations.

The gene transcription the factor 7 TCF7L2 codes transcription the factor which is the basic part of way Wnt, participating in regulation of mechanisms of growth, development and functioning of various cages, including pancreas b-cages. It is supposed, that participation of this gene in development of a diabetes 2 types can be expressed in the form of direct reduction of function of b-cages or indirect influence through secretion change glycogen-like peptid-1.

Among other genes participating in decrease of function insular of the device, by present time allocate the following:

1. A gene transmembran a carrier of zinc of type 8 - SLC30A8;
2. A fiber gene, associiroval with regulator subedinitsej-1 cyclin-dependent kinase type 5 - CDKALJ;
3. genes - inhibitors cyclin-dependent kinases - CDKN2A and 2B;
4. A gene of the fiber connecting mRNA insulin-like of the factor of growth 2 - IGF2BP2;
5. A gene I HEX - code transcription the factor participating on embrional of a stage in formation of a pancreas and a liver;
6. Gene IDE - codes insulinaz's - enzyme which participates in processes of degradation of insulin and others peptid hormones.

The genes responsible for lowered sensitivity of peripheral fabrics to action of insulin

1. Gene PPARG is involved in differentiation and function adipocytes. Its polymorphic marker Pro12A1a associated with

the lowered sensitivity of peripheral fabrics to insulin action. The prepotent negative mutation of area of the fiber connected with ligand as have shown researches, has led partial lipodystrophy's, heavy insulin resistance, to a diabetes and a hypertension of the early beginning.

2. The fiber Gene adoponectin ADIPOQ codes fiber development adiponectin cages of a white fatty fabric. The lowered concentration adiponectin is one of the development reasons insulin resistance. In this connection gene A DIPOQ was considered as one of the genes-candidates defining predisposition not only to lowered sensitivity of peripheral fabrics to action of insulin, but also to development of a diabetes 2 types.
3. The Genes coding receptors to adiponectin, - AD1PORI and-2. (Researches only are begun, and reliable data is not received yet).

And, at last, a gene, associated with adiposity and increase in weight of the fatty fabric which functional role in adiposity development is not absolutely clear till now. However, gene FTO is interesting to that is a unique gene, alleles which variants contribute to development a diabetes 2 types and associated simultaneously with weight of a body. The association with an index of weight of a body has been found out both in children, and at teenagers aged is more senior 7 years.

The researches directed on studying contributing to development of a diabetes 2 of types at children and teenagers, are too small on volume to allow to draw reliable conclusions. For today, unfortunately, genes of predisposition to a diabetes 2 types described at adults, have not been studied in children's population.

Alleles variant G3I9S of gene HNF1A has appeared the unique confirmed contributing marker, 2 types at children and teenagers in Oji-Cree, Canadians by origin are high associated with development of a diabetes. The homozygous condition given allel's a variant met at children a diabetes in 4 times more often, than at adult patients. This research supports a hypothesis, that within one population markers of predisposition to a diabetes 2 types at children will be same as at adults of the given population, however they will have higher genetic cargo.

Genetic defects B - cellular function

1. A chromosome 12, HNF-1a;
2. A chromosome 7, a gene glucocinaza's;
3. A chromosome 20, genHnF-4a;
4. Mitochondrion DNA mutation;
5. Others.

Genetical the defects causing infringements of action of insulin

1. Resistance to insulin of type and;
2. leprechaun;
3. Syndrome Rabson – Mendecholla's;
4. The Lipoatrofichesky diabetes;
5. Others.

Diseases exocrine pancreas parts

1. A pancreatitis;
2. A trauma (pancreatectomy);
3. neoplazias;
4. Kistos fibros
5. Hemochromatosis
6. Fibrocalculous pancreatopathy

Endocrinopatias

1. Acromegaly;
2. Syndrome Kuschinga's;
3. Glucagonoma;
4. Pheochromocytoma
5. Thyrotoxicosis;
6. Somatostatinoma;
7. Aldosteron;
8. Others.

The diabetes induced by pharmacological (poisonings) and chemical agents

1. Vacor;
2. Pentamidin;
3. Nicotinic acid;
4. Glucocorticoids;
5. Thireoid hormones;
6. Diasoxid;
7. Agonists an alpha - adrenoreceptors;
8. Tiazids;
9. Dilantin;
10. And - Interferon;
11. Others.

Infections

1. Congenital Rubella's;
2. A cytomegalovirus;
3. Others.

Unusual forms immune-mediated a diabetes

1. Stiff-man - a syndrome (a syndrome immobility);
2. Autobodies to insulin receptors;
3. Others.

Other genetic syndromes sometimes combined with a diabetes

1. A syndrome of Down;
2. Syndrome Clainphelter's;
3. Turner's syndrome;
4. The Tungsten syndrome;
5. Syndrome Fridrejch's;
6. Trochee Gentington'a;
7. Lawrence's syndrome - Moon – Bidl's;
8. The Miotichesky dystrophy;
9. Porphiria;
10. Syndrome Prader's - the Country house;
11. Others.

The scheme of recycling of glucose in a human body

The diabetes of 2 type shares on two groups

1. Diabetes 2 a
2. Diabetes 2 b.

The Diabetes 2 and proceeds without adiposity. Often under its mask the diabetes hidden autoimmune (as a result of infringement of function of immune system of an organism) character proceeds. The diabetes II would be characterized by adiposity presence. The streets, suffering diabetes IIa, achievement of normal level of the maintenance of glucose in blood represents certain difficulties that is observed even at application tableted hypoglycemic preparations in the maximum dose. After approximately 1-3 years after the treatment beginning tableted hypoglycemic preparations the effect from their application disappears completely. In this case resort to appointment of preparations of insulin. At a diabetes 2 type in more frequent cases develops diabetic polyneuropathic (widespread defeat of nervous system) which would progress more quickly in comparison with a diabetes 2 of type. The diabetes 2 of type is characterized by presence of hereditary predisposition. The probability of development of a diabetes of this type at the child in the presence of the same disease at one of parents makes approximately 40%. Presence of adiposity at the person promotes development of infringement of tolerance (stability) to glucose and a diabetes 2 of type. Adiposity of the first degree raises risk of development of a diabetes 2 of type three times. If there is an adiposity of average degree the probability of a diabetes increases five times. At adiposity of the third degree the probability of development of a diabetes 2 of type raises more than in 10 times.

Allocate some mechanisms of infringement of function of the cages producing insulin

1. In the absence of a pathology insulin is developed β bycages of a pancreas with certain periodicity which usually makes 10-20 minutes Thus the insulin maintenance in blood is exposed to fluctuations. In the presence of breaks in insulin development there is a sensitivity restoration to the given hormone of receptors which settle down on cages of various fabrics of a human body. The diabetes 2 of type can proceed with increase of the maintenance of insulin in a blood-groove at simultaneous absence of periodicity of its development. Thus, fluctuations of its maintenance in blood, characteristic for a normal organism, no.
2. At increase of level of glucose in blood after food intake cannot occur increases in emission of insulin a pancreas. Thus, already formed insulin is not capable to be thrown out from β-cages. Its formation proceeds in reply to increase of the maintenance of glucose in blood, despite its surplus. The glucose maintenance at the given pathology does not come to normal values.
3. Can occur premature emptying β gland-cages when active insulin was not formed yet. Proinsulin allocated thus in a blood-groove does not possess activity concerning glucose. Proinsulin can render atherogenic influence, to promote atherosclerosis development.

At increase of quantity of insulin in blood (hyperinsulinemia) surplus of glucose constantly arrives in a cage.

It leads to sensitivity decrease insulin receptors, and then to their blockade. Thus, the number insulin the receptors located on cages of bodies and fabrics of an organism, gradually decreases. Against hyperinsulinemia glucose and the fats arriving in an organism as a result of food intake, are much postponed in a fatty fabric. It leads to increase of stability of fabrics of an organism to insulin. Besides, at hyperinsulinemia disintegration of fats chokes, that, in turn, promotes adiposity progressing. Increase of the maintenance of glucose in blood has adverse influence on functional ability β gland-cages, leading to decrease in their activity. As the raised maintenance of glucose in blood is marked constantly long time insulin is produced by cages in a maximum quantity, that leads to

their exhaustion and the termination of development of insulin. For treatment use insulin preparations. In norm of 75% of consumed glucose is exposed to recycling in muscles, it is postponed in a liver in the form of spare substance – glycogen'a. As a result of stability of a muscular fabric to action of insulin formation process glicogen's from glucose in it decreases. Stability of a fabric to a hormone results from a mutation of genes in which the special fibers which are carrying out transport of glucose in a cage are coded. Besides it, at increase of level of free fat acids formation of the given fibers that leads to infringement of sensitivity of cages of a pancreas to glucose decreases. It leads to infringement of allocation of insulin by the given gland.

The mechanism of development of a diabetes of II type includes some stages

The first stage is characterized by presence at the person of congenital propensity to adiposity and the raised maintenance of glucose in blood.

The second stage includes small mobility, increase of quantity of used food in a combination to infringement of development of insulin β pancreas-cages that leads to development of stability of fabrics of an organism to influence on them of insulin.

At the third stage of development of a diabetes of 2 type there is an infringement of stability to glucose that leads to a metabolic syndrome (a syndrome of infringement of a metabolism).

The fourth stage is characterised by presence of a diabetes of II type in a combination with Hyperinsulinizm's (increase in the maintenance of insulin in blood of the person).

At the fifth stage of development of illness function of v-cages is exhausted, that leads to occurrence of requirement for insulin entered from the outside.

The leader in development of a diabetes of II type is presence of stability of fabrics of an organism to insulin. It is formed as a result of decrease in functional ability β pancreas-cages.

Generally recognized, that the diabetes of 2 type is caused insulin by resistance and insufficiency of function β-cages. An individual combination of these two principal causes feature of a current and efficiency of applied therapy speaks.

Despite it, to these two principal causes participating in patogenez of a diabetes 2 of type, it is necessary to add one more, namely – "dysregulation" insulin secretions in which mechanisms two big groups of hormones, as is known, participate: hormones of a fatty fabric (leptin, resistin, α-FNO, adiponectin, etc.) and peptide hormones enteropancreatic the systems presented by hormones of a gastroenteric path, including glucagon-like peptid-1 (GLP-1, or GLP-1), glucose-dependent insulinotropic peptide (GDIP, or GIP, earlier called gastric inhibitory polypeptide), etc.

Importance and doubtless participation of the specified third mechanism, or 3 reason, in patogenez a diabetes of 2 type proves to be true an establishment of the mechanism of physiological action Incretines (GIP-1 and GIP) on secretion of insulin and glucagon, and also introduction in clinical practice of the various medical products possessing various effects GPP-1 (analogues and agonists GPP-1) and Inhibitors dipeptidilpeptidaz 1-9 types which can be used not only as additional components hypoglicemic peroral therapies, but can be applied as monotherapy when a principal cause of a diabetes of 2nd type is "deregulation", or «dysfunction» of secretion of insulin.

The diabetes represents considerable threat to health, leading early invalidism and high mortality at rather early age. Clinically the diabetes is subdivided, as is known, into 2 big groups - diabetes of 1st type and a diabetes of 2 type which differ from each other numerous laboratories and immunogenetic features. Enough of the data however collects, allowing to assume, that in mechanisms of development of a diabetes of 1st and 2nd types there are many general pathogenetic mechanisms under which influence there are changes as in quantitative and qualitative structure endocrine cages of an islet of a pancreas, and in sensitivity of peripheral fabrics to insulin. The impression is made, that speed of decrease in quantity β-cages hereditarily is programmed (hereditary predisposition), and time depends on influence external (epigenetic) factors initiation and speed of development of the pathological processes leading to a diabetes and its late vascular complications.

Chronic hyperglicemia, being the basic and objective sign of presence of a diabetes, participates in patogenez, both a diabetes per se, and vascular complications of a diabetes. This influence hyperglicemia is carried out as directly, and indirectly -initiation several biochemical processes which concern: the oxidizing stress developing as consequence of raised auto-oxidation glucose at simultaneous decrease of activity antioxidant of system, superfluous formation of end-products glycation and their interaction with the corresponding receptors, the raised activation proteinkinasa's with owing to synthesis increase diacylglicerine under the influence of surplus of glucose, Poliolove ways of an exchange of glucose, in-

flammatory processes and the broken exchange lipoproteins. Infringement of the specified processes, in turn, is accompanied by change of molecular mechanisms mediated by stress and specific alarm ways of activation transcription the nuclear factor-kappa B (NF-kB), several kinazes, the main things from which are JNK/SAPK (NH2-termmal Jun kinases/stress activated protein kinase) and r38-mitogen-activated ptoteinkinasa (MAP), and also gexozamines. It is necessary to specify, that all listed mechanisms participate not only in patogenez's vascular complications of a diabetes, but also in mechanisms insulin resistance and function infringements islet the device of the pancreas observed at patients by a diabetes of 2 type.

Clinical signs of a diabetes

In a clinical picture of a diabetes it is accepted to distinguish two groups of symptoms the basic and minor.

The basic symptoms concern

1. Polyuria - the strengthened allocation of the urine caused by increase of osmotic pressure of urine for the account of glucose dissolved in it (in norm glucose in urine is absent). It is shown speeded up plentiful urination, including at night.
2. Poledipsia - (constant unquenchable thirst) - is caused by considerable losses of water with urine and increase of osmotic pressure of blood.
3. Polyphagia - constant insatiable hunger. This symptom is caused by metabolism infringement at a diabetes, namely inability of cages to absorb and process glucose for lack of insulin (hunger among abundance).
4. A weight loss - (it is especially characteristic for a diabetes of the first type) - a frequent symptom of a diabetes which develops, despite the raised appetite of patients. The weight loss (and even an exhaustion) is caused raised catabolizm's fibers and fats because of deenergizing of glucose from a power exchange of cages.

The basic symptoms most typical for a diabetes of 1st type. They develop sharply. Patients, as a rule, can to name precisely date or the period of their occurrence.

Secondary symptoms concern low-specific the clinical signs developing slowly throughout long time.

These symptoms are characteristic for a diabetes both 1, and 2 type

1. Itch and mucous membranes (vaginal an itch),
2. Dryness in a mouth,
3. General muscular weakness (it is heavy to go - feet get tired - first sign)
4. Head a pain
5. Inflammatory the defeats of a skin which are difficultly giving in to treatment,
6. Violation sight
7. Presence acetone in urine at a diabetes of 1st type. Acetone grows out of burning of fatty stocks.

MODY-diabetes

The given disease represents non-uniform group of the autosomno-prepotent diseases caused by genetic defects, leading to deterioration secretor functions β pancreas-cages. The MODY-diabetes meets approximately at 5% sick of a diabetes. Differs the beginning at rather early age. The patient requires insulin, but, unlike patients with a diabetes of 1st type, has low insulin demand, successfully reaches indemnifications. Indicators C-peptid's correspond to norm, is absent cetoacidoz. The given disease can be carried to «intermediate» types of a diabetes conditionally: it has lines, characteristic for a diabetes of 1st and 2 types.

Gestational a diabetes

Arises during pregnancy and can disappear completely or it is considerable be facilitated after sorts. Occurrence mechanisms gestational a diabetes is similar to those in case of a diabetes of 2 type. Frequency of occurrence gestational a diabetes among pregnant women makes approximately 2-5%. In spite of the fact that after sorts this type of a diabetes can disappear completely, during pregnancy this disease does essential harm to health of mother and the child. The women suffering gestational by a diabetes during pregnancy, are subject to the big risk to be ill subsequently - a diabetes of 2 type. Influence of a diabetes on a fruit is expressed in superfluous weight of the child at the moment of a birth (macrosomia, various ugliness's and congenital developmental anomalies. Data symptom complex it is described, how diabetic phetopatia.

Diagnostics of a diabetes 2 types

1. Glucose measurement (is on an empty stomach triple).
    - The normal maintenance of glucose in blood plasma on an empty stomach amount to 6, 1 mmol/l.

- If from 6, 1 to 7, 0 mmol/l - broken glicemia on an empty stomach.
- More than 7 mmol/l - a diabetes.

2. Glucose tolerance the test. It is spent only at doubtful results, if glucose from 6, 1 to 7, 0 mmol/l. 14 hours prior to research hunger is appointed, then take blood - establish initial level of glucose, then allow to drink 75 **r** the glucose dissolved in 250 ml of water. In 2 hours take blood and look:
   - If less than 7, 8 that normal tolerance of glucose.
   - If from 7, 8-11, 1 that the broken tolerance to glucose.
   - If more than 11, 1 that is a diabetes.
3. Definition C-peptid's, it is necessary for the differentiated diagnosis. If a diabetes of 1 type, level C-peptid's should be closer to 0 (from 0-2) if from above 2 a diabetes 2 types.
4. Research glycosylated haemoglobin (an indicator of a carbohydrate exchange for last 3 months). Norm less than 6, 5% till 45 years. After 45 years to 65 - 7, 0%. After 65 years - 7, 5-8. 0%.
5. Glucose definition in urine.
6. Acetone in urine, test Lang.
7. General blood test (GBT), general urine analysis (GUA), blood biochemistry (BB), glicemic a profile.

Insulinemia - loss 1 sercetor insulin phases.

Characteristic for a diabetes of type II metabolic infringements are

1. Decrease in sensitivity of peripheral fabrics to insulin (insulin resistance);
2. Dysfunction of b-cages which is shown by inadequate secretion of insulin against insulin resistance and hyperglicemia's.

Insulin resistance precedes development hyperglicemia' s and usually at early stages diabetes it is accompanied compensator by hyperfunction of b-cages and hyperinsulinemias.

Insulin resistance is an infringement of reaction of fabrics-targets on insulin which leads to decrease in absorption of glucose by muscles, to reduction glicoliz's and oxidations of fat acids in a liver, and also to suppression infringement gluconeogenez's in a liver.

The greatest contribution in patogenez insulin resistance *In vivo* is brought by sensitivity loss hepatocyts to insulin. At insulin resistance various functional defects of an alarm way of insulin (for example, decrease phosphorilation tirozin's and increase phosphorilation serin's insulin a receptor and fibers (IRS), weakening a signal transmission are described. The most important reason insulin resistance is adiposity. The epidemiological interrelation of adiposity with a diabetes of type II has been established many years back, thus special value has visceral adiposity type (80%). Adiposity makes strong impact on sensitivity of fabrics to insulin and, as consequence, on a system homeostasis of glucose. Insulin resistance it is marked even then when adiposity is not accompanied hyperglicemia's, that testifies to fundamental infringements of a signal transmission of insulin at surplus lipids. The risk of development diabetes raises in process of increase in an index of weight of a body. At insulin resistance it is important not only absolute quantity of fat in an organism, but also character of its distribution: abdominal (central) adiposity in greater degrees associates with development insulin resistance, than visceral (peripheral, in buttock area and hypodermic).

It leads to sensitivity decrease insulin receptors, and then to their blockade. Thus, the number insulin the receptors located on cages of bodies and fabrics of an organism, gradually decreases. Against hyperinsulinemia's glucose and the fats arriving in an organism as a result of food intake, are much postponed in a fatty fabric. It leads to increase of stability of fabrics of an organism to insulin. Besides, at hyperinsulinemia's disintegration of fats chokes, that, in turn, promotes adiposity progressing. Increase of the maintenance of glucose in blood has adverse influence on functional ability β gland-cages, leading to decrease in their activity. As the raised maintenance of glucose in blood is marked constantly long time insulin is produced by cages in a maximum quantity, that, eventually, leads to their exhaustion and the termination of development of insulin. For treatment use insulin preparations. In norm of 75% of consumed glucose is exposed to recycling in muscles, it is postponed in a liver in the form of spare substance – glicogen's. As a result of stability of a muscular fabric to action of insulin formation process glicogen's from glucose in it decreases. Stability of a fabric to a hormone results from a mutation of genes in which the special fibers which are carrying out transport of glucose in a cage are coded. Besides it, at increase of level of free fat acids formation of the given fibers that leads to infringement of sensitivity of cages of a pancreas to glucose decreases. It leads to infringement of allocation of insulin by the given gland.

Decrease in the first phase of secretion of insulin conducts to more to increase postprandial glucose level in blood. It stimulates a pancreas to secret surplus of insulin on the second phase. Any

time allows to constrain this mechanism insulin resistance in norm (Yalow Berson, 1930). It occurs at patients to the raised weight. Adiposity and insulin resistance promote occurrence hyperinsulinemia's. Presence hyperinsulinemia's on an empty stomach says that pancreas loses 50% of the activity. Disease progressing conducts to loss of functions of B-cages. At level glicemia's 140 mg of% and above amplify liberation of glucose by a liver. Insulin resistance a fatty fabric it is characterised by increase in plasma of blood of level of free fat acids.

Pathogenetic representations underlie recommendations about treatment of a diabetes 2 types

I consider, that all these preparations exhaust sooner or later concentration of insulin and raise glucose level in blood.

The author adheres etiologic to the theory and treatment practice (the note of the author)

1. Decrease Insulin resistance - weight reduction, playing sports, Metformin, Tiozolidions, Normaglicemia (glucose toxicity).
2. Increase of the first phase of secretion of insulin – Meglitinids, Preparations Sulfanilurea fast action - Lizpro or Aspart.
3. Liberation increase bazal insulin or the second phase of secretion of insulin - Preparations Sulfanilurea long action - Insulin of average or long duration of action.
4. Suction carbohydrates - treatment postprandial glycemia's - Acarboza, Miglitol (class Miglitinids).

And patients with a full exhaustion pancreas at constant increase of sugar in plasma of blood above 250 mg of% it is possible to simulate the first phase of secretion of insulin analogues of insulin of fast action – Lizpro or Aspart. Basal insulin level can be added by preparations Sulfanilurea or introduction of insulin of average or long duration of action. Introduction basal insulin and insulin of the short action simulating the first phase of secretion of insulin is possible.

4 stages of a diabetes 2 types are known - is characterised by increase in duration of disease and increase in plasma level of glucose.

As the first and second phases of secretion of insulin decrease and liberation of glucose by a liver increases, insulin resistance increases because of glucose toxicity.

Preventive maintenance of a diabetes 2 types - is connected with power and hormonal activity of an organism and reached by reception of power natural preparations.

Plants - activators of hormonal activity

1. Licorice, a train, pollen of plants, bean, cereals;
2. Accomulations power groups - a gooseberry, grapes, apples, a rhubarb;
3. Antihypoxants - honey, an onion (the red onions are stronger), garlic, bitterness and spice in any kind.

Power medicines - a horse-radish, tomatoes, a radish, daikon. Are power natural medicines, which preventive sugar deabetes 2 types (the note of the author) easier.

Sinteticheesky medicines

1. Koenzym Q10 - optimizes all oxidizing processes, improves power of all cages of an organism, including heart cages.
2. Actovegin - improves a metabolism of cages, promotes glucose and oxygen accumulation in organism cages.
3. L-Karnitin - promotes activation of a cellular metabolism, helps to cope with oxygen insufficiency, restores the damaged cages.
4. Piracetam - improves power processes at cellular level, an excellent antioxidant.
5. Mexidol (Mexiprim) - does not suppose oxidation lipids cellular membranes of all cages of an organism. Helps to avoid oxygen starvation of cages.

The author writes the huge book on Antioxidants and Antihypoxants (it is not published yet).

Daikon - preventive maintenance of a diabetes 2 types.

Daikon it can widely be used at preventive treatment of patients with a diabetes and at an irradiation. There is an opinion, that Daikon is capable to deduce radiation if it to eat crude.

Daikon it is useful to preventive maintenance and treatment of an atherosclerosis, various cardiovascular diseases as deduce from an organism superfluous cholesterol. The medical effect is shown at long consumption Daikon.

Usually daikon use in a crude kind, but it can and … be preserved. As roots Daikon's contain calium, calcium, Vitamin C with

a combination to carrots and a beet daikon remarkably helps at an anaemia. The mix of this juice (vegetables to grate) follows 2 3 hours let it sit in an oven, and then to accept on 1 table spoon three times a day 15-20 minutes prior to meal. The mix for children (a dose to reduce to 1 teaspoon) is especially good, and course of treatment (or preventive maintenance) makes 2 3 months.

The medicine can be very pleasant on taste: for this purpose, it is necessary to cut Daikon with thin circles, plentifully to sugar and allow to lie down some hours (until sugar will not extend all juice and itself it will be dissolved in it).

Contra-indications. Daikon, as well as a radish, it is impossible to include in the diet at following diseases: at a stomach ulcer, hyperacid gastritises, diseases of kidneys and a liver, a gout and the diseases connected with a metabolism.

Daikon today is not exotic vegetable and widely is on sale in the markets, in shops and supermarkets.

Important! At the elderly it is often observed hyperglicemia at normal level of insulin in blood.

At them resistance to insulin raises. And more often, in the autumn and in the winter. Risk factors of vascular complications (lipids low and very low density, glycolized fibers, mucopolysacharids - it conducts all to an atherosclerosis of large vessels of heart, a brain, kidneys, finite nesses), to change of capillaries with deformation basal membranes, that conducts to infringement from microcirculation (development of a hypertension and diabetic foot) collect. To appoint it is necessary vegetative Immunomodulators, a diet, antioxidants (the note of the author).

Attention! At women in postmenopause with a normal metabolism of glucose, postprandial glucose levels can be the best marker of risk of an atherosclerosis, then levels trio-acylglicerids or results peroral the tolerance test to glucose. Means, that it is necessary to treat an original cause a-atherosclerosis, instead of to reduce sugar of blood and pressure (the note of the author). I have a Book for treatment an atherosclerosis, but while it unpublished (the note of the author).

Attention to the Doctor and the Pharmacist! Chemical changes in an organism which lead to occurrence of a diabetes of the second type, begin some years prior to occurrence of first signs of disease.

Researchers have precisely defined some changes of level of glucose in blood and sensitivity to a hormone to insulin. Participants of experiment with steady indicators of a sugar curve throughout all research have been least subject to occurrence of a diabetes of the second type. However, those who has faced sharp changes in level of glucose and sensitivity to insulin, after some years, had a diabetes. The obtained data can help considerably at revealing of the people who are in group of risk of occurrence of a diabetes of the second type (the note of the author).

Attention! In occurrence of a pathology of vessels the reed white cleared sugar and glycated haemoglobin which is formed through a chain of biochemical reactions and cause damages endotelium vessels is guilty. It causes formation of cracks on endotelia's, which are filled with cholesterol. Or the food-processing industry needs to forbid to make and sell it, or it is necessary to do sugar by another - with other structure (the note of the author)!

Signs 2 types which were hiding-proceeding (latent) diabetes

Signs it is hidden proceeding disease - furunculosis, long not healing wounds, an itch crotch, various rash on a skin, a periodontosis. At display of any of the specified symptoms it is better, not postponing to address to the Doctor and the Clinical Pharmacist.

Should guard - fast fatigue, weakness, thirst, plentiful drink and uritation, growing thin. If necessary treatment in due time is not spent, the patient can have the complications at times menacing to a life (a diabetic coma). The started illness quite often leads to early defeat of vessels of eyes, kidneys, hearts, the bottom finitenesses, nervous system. Defeat of these bodies demands further long treatment and brings not only to time, but also in proof disability.

Hunger provokes hyperactivity

Especially, it is characteristic for children. It is necessary to check sugar of blood at hyperactive children and to include this research in diagnostics standards (the note of the author). Hyperactivity helps to find meal, and such reaction is connected with ensym activated proteinkinaza (AMP-kinaza). This enzym also stimulates development of a hormone which operates glucose liberation. If to learn to reduce level of it enzym's it is possible to operate sugar level in blood and desire to search for food. It will help to lower risk of disease by a diabetes and to supervise an excess weight set (the note of the author).

The protein provoking development of a diabetes of 2 type and an atherosclerosis, can cause development of a bronchial asthma

According to scientists, the protein under the name a-P2 can become a missing link in a chain of the biochemical processes con-

necting such diseases, as adiposity, a diabetes, an atherosclerosis and an asthma. In norm a-P2 does not carry out any function in an organism of mammals, however it becomes more active at the diseases connected with infringement of a metabolism, making negative impact on a metabolism of glucose and fat acids.

Scientists have found out the raised levels a-P2 in the lungs, experimental mice suffering by adiposity. Earlier this protein was considered as a specific marker of fatty cages. At the same time the mouse with a mutant gene a-P2, considerably reducing development of this protein in an organism of an animal, it has appeared not an asthma subject to attacks (Bennet Shum, 2005).

Results of the spent experiments say that protein blocking a-P2 can become an effective remedy of treatment of an asthma and of some other inflammatory diseases.

About the important role which plays a protein in variety of pathological processes, says also that fact, that genetically modified mouse with the lowered levels a-P2 has appeared steady not only to an asthma, but also to adiposity, a diabetes both a fatty dystrophy of a liver and an atherosclerosis.

Importance of open properties a-P2 is estimated and have started working out capable to block its preparations (Gokhan S. Hotamisligil, 2005). As he said, laboratory researches of new medicines have passed successfully, and the clinical stage of researches is planned to begin the next years.

While to the people predisposed to an asthma to give it is more than attention to struggle against excess weight. Level a-P2 increases in process of increase in weight and change of an exchange of glucose if the asthma at you is combined with excess weight, you can improve the condition, having dumped superfluous kgs.

The allergy on red meat is connected with cardiovascular diseases

Allergen is the newest risk factor of a coronary atherosclerosis potentially giving in to change. Researchers have revealed, that 26% of patients had antibodies to α-Gal, that specifies in sensitivity to red meat. At the given patients considerably, above indicators of number of atherosclerotic plaques (on 30% above in comparison with the patients not sensitive to red meat). The structure of the given plaques also tends to be astable, that raises probability of a heart attack of a myocardium and a stroke. After updating on a floor, a diabetes, a hypertension, use Statines!!! and the general IgE the interrelation between α-Gal-specific IgE and atheroma's remains considerable. This research says that for atherosclerosis treatment other preparations (phytopreparations) are necessary, and it is necessary to be engaged in atherosclerosis preventive maintenance from 5 years (there is a book, it is unpublished yet - the note of the author).

The short dream promotes resistance of insulin and decrease in shipping of glucose, that in turn raises risk of development of a diabetes

People who are accustomed to burn the midnight oil, do not observe a normal mode of a dream, and test shortage of night rest, risk to face serious risk of a diabetes.

The majority of the people living in the western countries, have short duration of a dream, in addition to absence of physical activity and an overheating. At the same time quality of a dream directly influences probability of development of a diabetes.

Interrelation between smoking and tolerance infringement to glucose

Tolerance infringement to glucose is an early risk factor of development of a diabetes 2 types (insulin-independent) (Thomas Houston, 2010).

To all participants twice (in the beginning of research and in 15 years) did the test for tolerance to glucose - blood researches on sugar through certain intervals of time after glucose intake (loading test with glucose). Results of research have shown, that is the most frequent - in 22% of cases - the broken tolerance to glucose arose at smokers. At the people who were exposed to passive smoking, the broken tolerance to glucose developed in 17% of cases.

For comparison, at the non-smoking people who did not have contacts to a tobacco smoke, the broken tolerance to glucose arose in 12% of cases.

Toxins of a tobacco smoke can operate on a pancreas - body in which insulin (a hormone regulating level of sugar in blood) is developed. Passive smokers are treated to action of the same toxins, as active smokers, and concentration of some toxins at them even above.

Inhalation of a tobacco smoke by non-smoking people is a new risk factor of the broken tolerance to glucose

The diabetes arising owing to absolute or relative insufficiency of insulin, can cause development of dangerous complications. Surplus of glucose in blood negatively affects a condition of cardiovascular system, can conduct to blindness and chronic nephritic insufficiency.

Credit card use - a risk factor of a set of excess weight

Visiting of shop with a credit card leads to purchase more quantities of sweets and other high-calorie food.

Scientists have made experiment in which course have found out, that the campaign in shop with a credit card pushes women on rash purchases, and, first of all, it concerns purchases of unhealthy food. Card presence dulls feeling of economy of money as the woman physically does not see them.

Experts have informed, that girls who go to shop with cash and the concrete list, for the clear reasons afford impulsive purchases less often. It occurs that is less temptation to buy of something from them harmful to their figure.

Essential risk factor of occurrence of the diabetes 2 types

Scientists have found two hereditary factors which influence weight of the child at a birth. One of them has appeared is connected with diabetes occurrence in the future - low weight at a birth.

According to experts, results of research are the first convincing proofs of that between insufficient weight at a birth and diabetes occurrence further there is a communication.

Excess weight negatively influences health from first months of a life

The problems connected with excess weight at the child, involve negative consequences much earlier, than it was supposed earlier.

Scientists have carried out researches and have found out, that at babies suffering from adiposity the risk of occurrence of delays in development, an asthma and various problems with breath is raised. In most cases parents do not realize, that at their children of a problem with weight.

Though it is considered to be, that excess weight consequences come later, but doctor Ron Shaoul, (2007) asserts, that it not so, and consequences, as he said, come much earlier, then thought earlier.

Results of the first research have shown, that children happen to excess weight are hospitalised much more often, than their coevals with normal weight. In turn, the second research has shown, that at children, suffering excess weight, the asthma and other problems with breath, and also delays in development and a snore are more often observed. Only 32% of interrogated mothers realised a problem of adiposity at the child.

Parents should concern more attentively a problem of excess weight at children as it can lead to set of serious problems with health at kids. Parents should try to supervise weight of the child in borders of norm since the earliest childhood.

Attention! High cholesterol in the childhood causes presenilation of vessels!

Coffee promotes decrease in risk of disease by a diabetes

Is better effect it is reached at the use decaffeinated coffee.

During proceeding 10 years of research physicians conducted supervision over thousand women which at the moment of the beginning of experiment did not test problems with cardiovascular system and were not ill with a diabetes. All of them shared on groups, representatives of one of which at all did not drink coffee.

In other group there were those who drank on one cup of coffee in day, in the third - from one to three cups, in the fourth - from four to five cups and in the fifth - those who used more than six cups of coffee in day.

When have been summed up, it was found out, that from 1400 persons who were taking part in research, the diabetes has been diagnosed? Also, it has been established, that among those who drank many coffees which were ill with a diabetes, was on 22% less, than among those who did not take this drink.

It was found out, that the lowest indicators of disease by a diabetes were among those who drank decaffeinated coffee, and irrespective of age and weight of a body.

However, physicians are at a loss to tell, which of a part coffee of elements reduces risk of this disease rather extended in the USA.

In any case, authors of research confirm, first of all, not to be ill with a diabetes, it is necessary to keep to a diet, to be engaged in physical exercises and to watch the weight. And coffee becomes here the good assistant.

Neutrofiles - a new target in struggle against a diabetes of the second type.

Neutrofiles - a principal view of leukocytes of blood of the person, play absolutely unexpected role of modulators in insulin resistance - the main characteristic of a diabetes of the second type.

Neutrofiles are cages of immune system which answer the with the first an inflammation of fabrics; they are capable to make its chronic, having called to a place of events other white blood cages - macrofages. At the same time, it is established, that the insignificant chronic inflammations meeting in a fatty fabric, are one of principal causes regular insulin resistance.

Scientists (Jerrold M. Olefsky, 2012) used in research a liver and fatty cage of mice and people, and also live experimental rats. It was revealed, that the enzyme allocated nejtrofiles (nejtrofil-elastaza, NE), breaks insulin alarm ways, urging on resistance, inexplicable decrease in affinity of receptors on insulin presence is observed.

And here removal NE at suffering adiposity, but thus mice all the same sitting on a fat diet led to sensitivity increase to insulin.

Earlier was considered, that such «time» cages, as nejtrofiles (five days living only), simply are not capable to support a small chronic inflammation. Now it was necessary to recognise, that nejtrofiles possess rather powerful immimodulator's effect. They use enzyme NE for activation of an alarm way which forces to allocate patogen-devouring macrophages inflammatory molecules - cytocines. At the same time same enzyme NE causes degradation of fiber IRS1, a key protein in insulin an alarm way, both in a liver, and in fatty cages.

The activity inhibition immunomodulator's enzyme NE is quite capable to turn (or at least to weaken) insulin resistance. Here only the stick, as always, about two ends turns out … (the note of the author).

New data about the mechanism of influence of insulin on glucose development in cages of a liver and about development of a diabetes of the second type are obtained

The research which results will help to understand the mechanism breaking suppression mediated by insulin of development of glucose in a liver, and the reasons leading to development insulin resistance and a diabetes of 2 types has been carried out.

In the given work the hypothesis has been stated, that insulin suppresses development of glucose at the expense of inhibition of processes of splitting of fats, that in turn reduces level coensym's KoA which is a key molecule in adjustment of synthesis of glucose from amino acids and lactat's. Infringement of this process is started at inflammatory reactions in a fatty fabric that leads to the raised level of glucose of blood. These results have been received in experiments on the rodents receiving food rich with fats and confirmed at inspection of teenagers with superfluous weight of a body and insulin resistance.

Opening will help to create new methods of therapy of a diabetes 2 types as any of preparations used now does not influence original causes of development of excessive hepatic production of glucose (the note of the author).

Risk factors on the diabetes 2 types

Attention! 14 new DNA of breakages are found at development of a diabetes which will open all secrets of occurrence of a diabetes 2 types.

Jatropatichesky pathology (it is necessary to carry out differential diagnostics poisonings - should be engaged in it Clinical Pharmacists).

Statines in a high dose increase risk of a diabetes 2 types

Thus the risk of cardiovascular outcomes at treatment by high doses Statines has decreased on 16%. The risk of a diabetes was 80 mg comparable at treatment Atorvastatin's and Simvastatin's 80 mg, however in efficiency in preventive maintenance of cardiovascular outcomes Atorvastatin surpassed Simvastatin (decrease in risk of cardiovascular outcomes on 22% and 5%, accordingly). The mechanism of development of a diabetes remains to unknown persons (Preiss D, Seshasai SR, Welsh P., *et al*. 2011).

Attention! The preparations lowering cholesterol, raise risk of occurrence of a diabetes (Statines, Fibrats).

Reception of preparations for cholesterol fall in high doses can raise risk of occurrence a diabetes 2 type.

Scientists have carried out research in which have analysed five previous researches with total of participants more than 32 thousand persons.

Patients regularly accepted the preparations blocking cholesterol, and have been divided by researchers on two conditional groups depending on a daily dose - from 20 to 40 mg and 80 mg. It was

found out, that reception of the big doses of preparations raised risk of occurrence of a diabetes of 2nd type on 12%.

Experts have informed, that only to the Great Britain, which population makes 62 million inhabitants, preparations for cholesterol fall accept more than 7 million the persons, every third adult at the age from 40 years and is more senior. Physicians advise not to abuse preparations lowering cholesterol as it is negatively can effect on health.

Attention! Presence at one of members of a family of a diabetes 2 types increases risk of occurrence of disease at representatives of a female twice.

Excess weight and preferences in meal are the important risk factors of occurrence of a diabetes 2 types, however experts have decided to find out, what role is played by the hereditary factor. Within 20 years scientists observed of women. Excess weight and a way of life were considered. For this period 2 types were ill with a diabetes of 5102 women. Having analysed all data, experts have found out, that if in a family one of near relations was ill with a diabetes, the risk increased more, than twice. Also, it was possible to establish, that the women having family history of a diabetes, on 20% have been more subject to excess weight occurrence.

Scientists consider, that the hereditary factor plays the important role, but the healthy way of life and eutrophy can render protective effect, therefore it is not necessary to neglect it.

Attention! A risk factor on occurrence of a diabetes 2 types - the big use of milk.

Attention! Surplus of iron in an organism - a diabetes risk factor! Such patients with chemochromatosis and a diabetes the author met (the note of the author).

Attention! According to research FIELD, at patients with a diabetes 2 types traditional parities lipids have appeared same exact concerning forecasting of risk of cardiovascular diseases, as well as the relation апоВ/апоА-1. There is no necessity to replace traditional indicators of levels lipids and their parities on indicators апо-В, апоА-1 and their parity (the note of the author).

Attention! Even the small increase in weight leads to dysfunction development endotelum's at persons with normal weight of a body and normal pressure (Virend Somers, 2010). Have carried out research with participation of 43 healthy volunteers with initial normal weight of a body. Then the randamization in groups of increase in weight of a body and stable weight of a body. The group of increase in weight of a body within 8 weeks received on 1000 calories more than it is necessary, and then the period of observance of a diet for normalisation of weight of a body followed. In group of a set of weight of a body the weight has increased by 4, 1 kg that was accompanied by decrease a stream-dependent dilation vessel about 9, 1% to 7, 8%.

Consumption of a foodstuff and the drinks containing sugar, in particular fructose, 2 types are a major factor of sharp growth of disease of a diabetes. According to statistics, about every tenth adult person in the world suffers from a diabetes. In total for the last some decades the number of diabetics has grown more than twice. So, in 1980 from a diabetes suffered 153 million people, and in 2008 - 347 million.

Fructose is a banal principal cause of distribution of a diabetes

Researchers have estimated data of experiments on animals and with the assistance of people. Last researches have shown, that replacement of glucose by fructose leads to adverse consequences.

The fructose containing in table sugar and a corn syrup (sweetening, most often used in the food-processing industry) is especially dangerous. Scientists have explained, that the fructose which is a part of natural products, such as fruit and vegetables, is harmless to an organism. In Russia doctors forbid to eat fruit at a diabetes (the note of the author).

Though, consumption of fruit and vegetables, on the contrary, protects from a diabetes and other metabolic infringements. Researchers urgently recommend to people to replace the products containing added sugar, with fruit and vegetables.

Attention! All diabetic products are made on fructose and on fat (the note of the author).

In no event for diabetics it is impossible to eat products to healthy people. And products with fructose too are harmful to diabetics!

Deficiency of vitamin A can matter at type 2 diabetes

The lack of Vitamin A at mice leads апоптозу beta cages of a pancreas and to infringement of development of insulin.

Development of a diabetes of type 2 is connected with steady decrease in quantity of beta cages. The reasons and the mechanism, an aetiology and patogenez, diseases continue to be investigated. In

model on animals value in these processes of deficiency of vitamin A has been proved.

Scientists fed mice with food with the insufficient maintenance of Vitamin A. The similar diet led scale apoptoz's pancreas beta cages. At animals vitamin A and insulin levels in a pancreas decreased, developed hyperglicemia. Deficiency of this substance in a diet of adult animals led remodeling endocrine to a pancreas part, reduction of volume of islets Langergans's, reduction of quantity of beta-cages and increase in quantity of alpha cages, also was observed hyperglucagonemia. In beta cages changes of level of the fibers involved in a metabolism of vitamin A were marked. As a whole, penotipic changes were similar to the infringements observed at the developed diabetes of type 2.

Return to a usual diet raised vitamin A level in a pancreas, improved glicemic the control, normalised structure of body and a parity of quantity of beta cages and alpha cages. Thus, it was not observed proliferation or neogenez beta cages.

Authors count, that their results will lead to creation of the pharmacological means changing level of vitamin A in cages of a pancreas and facilitating a current of a diabetes of type 2. It is pharmagenetic treatment. It's to Dependently Clinical Pharmacists (the note of the author).

Important! In venous blood Vitamin A transitor, therefore the author has developed diagnostics about a lack of excess Vitamin A (the note of the author). But it is a theme of the separate book.

Pollutants are connected with a diabetes

People with higher levels of pesticides and other pollutants in blood have higher probability of development of a diabetes 2 types. Pollutants, including pesticides and polychlorinated biphenils, or PCBs, are substantially found out in meat and fat fish. Some of them, are forbidden and not used any more in many countries.

In the spent research (Duk-Hee Lee, 2011) and colleagues have continued earlier researches which have connected these chemical substances with a diabetes of 2 types.

At group of elderly adults without a diabetes were samples of blood are taken to estimate their levels pollutants. The subsequent supervision proceeded five years.

At 36 from participants of research the diabetes 2 types during this time has been diagnosed. After the account of other risk factors of a diabetes, such as the weight, physical activity and smoking, subjects which had high levels PCBs, had to nine times higher probability of development of a diabetes, than patients with very low levels pollutants in blood.

Attention! The women actively using autosunburn and a hairspray, are subject to the raised risk of development of a diabetes.

Scientists have analysed given more than 2 thousand women at the age from 20 till 80 years. There was, that a high level ftalats, getting to an organism with cosmetic means, increased risk of development of a diabetes by 200%. Also, it was marked insulin resistance. If concentration was concerning moderated the risk raised approximately on 70%.

Experts have explained, that ftalats it is possible to meet practically everywhere (in nail polish, shampoo, soap, plastic packings). On properties they are similar to the hormones developed in a body of the person. Most likely, connections interfere with a metabolism of a fatty fabric and lead to infringements.

To be continued…

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