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Opalenyk S. M., Tovt-Korshynska M. I.

Educational and methodological manual

«THE VALUE OF BIOCHEMICAL ANALYSIS INDICATORS OF BLOOD FOR CLINICAL MEDICINE»

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Methodological recommendations describe the methodology for conducting biochemical blood analysis in patients; criteria for normal indicators of the biochemical blood analysis; clinical interpretation of the results of laboratory studies in various diseases of internal organs is provided; clinical and laboratory tasks for self-control and a list of recommended literature are given.

Additional studies contribute to a deeper understanding of disorders of various organs and systems. Without additional research, it is impossible to establish the correct diagnosis of the disease, the degree of its activity, the stages of development.

Editors:

Opalenyk S. M. – PhD MD, assistant of the department of internal medicine, medical faculty №2

Tovt-Korshynska M. I. – DMSc, professor, head of the department of internal medicine, medical faculty №2

Reviwers:

Ternushchak T. M. – PhD MSc, associate professor of the department of internal medicine, medical faculty №2;

Patskun S.V. – PhD MD, associate professor of the department of fundamental medical disciplines medical faculty №2;

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BIOCHEMICAL ANALYSIS OF BLOOD

PROTEIN METABOLISM

Proteins are high-molecular nitrogen-containing organic substances. Amino acids entering the blood from tissues and intestines as a result of the absorption of products of hydrolysis of food proteins are used for the synthesis of specific tissue proteins, enzymes, hormones, biogenic imines, nitrogenous compounds and other biologically active compounds. All the processes of synthesis and transformation of proteins, amino acids and other compounds associated with them are called protein metabolism. They occupy a central place in the metabolism of the human body. They perform an important structural function in the body. Parenchymal organs contain the largest amount of proteins - spleen (84%), lungs (82%), muscles (80%), kidneys (72%), heart (60%), liver (57%), etc. Proteins participate in the formation of the structural basis of cells and their organelles - membranes, mitochondria, ribosomes, nuclei, cytoplasm, as well as the walls of capillaries and other vessels. Proteins perform catalytic functions. All enzymes that provide metabolic reactions in the human body are substances of protein origin.

The functions of protein in the human body.

The total protein content in serum reflects the state of protein metabolism. They are the basic building blocks for all cells and tissues of the body.

Enzymes, many hormones, antibodies and blood coagulation factors are built from proteins. Immunoglobulins and proteins of the coagulation system perform a protective function in the body. Proteins actin, myosin, tropomyosin in the muscles of the heart, lungs, stomach and other organs provide various forms of mechanical movement.

The amount of proteins in the serum determines the osmotic pressure of the blood, due to which a balance is maintained between the water content in the tissues of the body and inside the vascular bed. Proteins are also responsible for maintaining the correct acid-base balance (pH).

Finally, it is a source of energy for malnutrition or starvation. Proteins in the body also perform a regulatory function. This is due to the protein nature of hormones (insulin, prolactin, thyroxine, etc.)

In addition, they perform the function of carriers of hormones, vitamins, minerals, fat-like substances and other metabolic components in the blood, and also provide their transport into cells. Proteins perform important transport

functions. Lipids and fat-soluble vitamins are transported by lipoproteins, oxygen and CO_2 are transported by hemoglobin, amino acids are transported by albumin, fig 1.

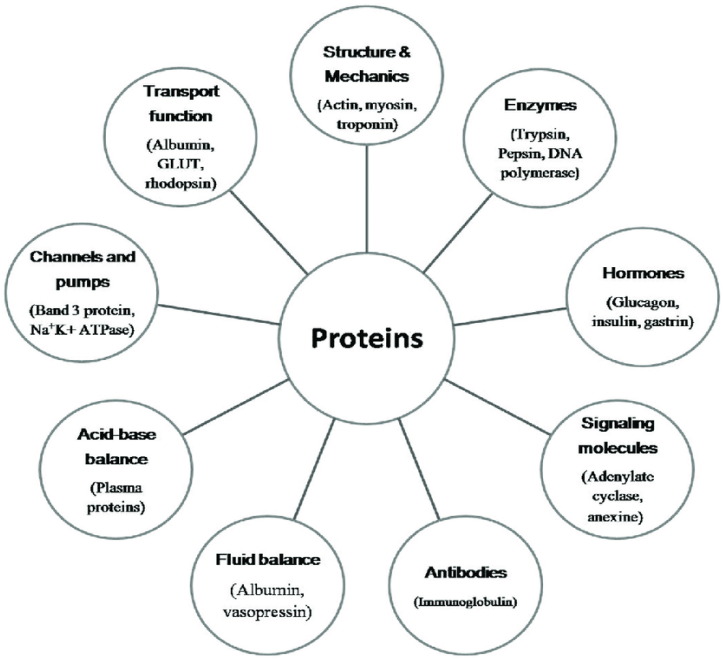

Figure 1. The main functions of protein in the body

It should be remembered that with pathology of internal organs, the first violations can be noted in protein metabolism.

The most important conditions for the normal vital activity of the body are providing it with the necessary amount of assimilated nitrogen. Its source is proteins. With general starvation, insufficient protein nutrition, or with a violation of the protein metabolism system, a state of negative nitrogen balance occurs in the body. At the same time, the amount of nitrogen that is excreted with feces and urine exceeds the amount of nitrogen that comes with food. A negative nitrogen balance indicates an increase in the intensity of tissue protein breakdown over their synthesis.

A balanced diet requires not only sufficient protein intake (100 g per 2500 kcal), but also the necessary amount of essential amino acids.

Digestion of proteins is carried out with the help of stomach enzymes (pepsin, gastrixin), pancreatic enzymes (trypsin, chymotrypsin, elastase) and intestinal enzymes (amino- and dipeptidases). Protein cleavage products (low-molecular-weight peptides, amino acids, nucleotides, and nucleosides) are absorbed into the blood.

Amino acids and proteins that have not been cleaved and not absorbed enter the large intestine, where with the participation of microorganisms, they are transformed into products toxic to the body: amines, alcohols, phenols, indole, skatole, cadaverine.

Some toxic products are neutralized in the liver (indole, skatole, cresol and phenol) in combination with glucuronic and sulfuric acids. Metabolic products - indican is determined in urine and serves as an indicator of the intensity of putrefactive processes in the intestine.

The cause of protein metabolism disorders can be a violation of the regulation of the metabolism of protein structures. Thus, the lack of anabolic hormones (insulin, sex hormones, somatotropic hormone of the pituitary gland), tissue damage of infectious origin contributes to the increase in the breakdown of protein tissues. Violation of the processes of protein biosynthesis leads to a protein deficiency, which is accompanied by a preference for the processes of protein breakdown over their synthesis - a negative nitrogen balance.

Protein fractions. The content of protein fractions is investigated in blood serum by electrophoresis. During electrophoresis, 5 protein fractions (albumins, α1, α2, β, γ-globulins) are detected on paper in a healthy person. The total amount of blood serum proteins is from 65 to 85 g/l, in blood plasma they are 2-4 g/l more due to fibrinogen. Most serum proteins are synthesized in the liver. Albumins, fibrinogen, α-globulins, β-globulins, and many components of the blood coagulation system are synthesized in hepatocytes.

Component reference values are:

- Albumin 55.8 - 66.1%
- Alpha-1-globulin 2.9 - 4.9 %
- Alpha-2-globulin 7.1 - 11.8 %
- Beta-1-globulin 4.7 - 7.2 %
- Beta-2-globulin 3.2 - 6.5 %
- Gamma globulin 11.1 - 18.8 %

Albumins support colloid-osmotic (oncotic) blood pressure, determine blood viscosity, maintain pH, combine with some substances (vitamins, hormones, cholesterol) - perform a transport function. The protective function of plasma proteins, which is related to the function of immunoglobulins, is very important.

Deviation of the level of total blood protein from the norm can be associated with a change in the water content in the circulating blood (relative deviation) and caused by changes in metabolism (absolute deviation).

In various pathological conditions, the content of total protein in blood plasma fluctuates both in the direction of increase (hyperproteinemia) and decrease (hypoproteinemia).

The causes of absolute and relative hypoproteinemia:

- Physiological absolute hypoproteinemia can occur with prolonged bed rest, in women during pregnancy (especially in its last third) and breastfeeding, in children at an early age, that is, in conditions of insufficient intake of protein from food or an increased need for it. In these cases, the total protein in the blood decreases.
- The development of physiological relative is associated with excess fluid intake.
- Pathological relative hypoproteinemia is observed with excessive fluid retention in the circulating blood (impaired renal function, deterioration of the heart, some hormonal disorders, etc.).

The causes of absolute and relative hyperproteinemia:

- An absolute increase in total blood protein can occur in acute and chronic infectious diseases due to increased production of immune globulins, in some rare health disorders characterized by intensive synthesis of abnormal proteins (paraproteins), in liver diseases, etc.
- Relative hyperproteinemia (increased levels of total protein in the blood) can be caused by excess water loss, such as excessive sweating.
- Relative pathological (associated with a disease) hyperproteinemia is caused by a significant loss of fluid and thickening of the blood (with profuse vomiting, diarrhea or chronic nephritis).

Albumin. The amount of albumin in blood serum is 35-45 g/l. Serum albumin is a protein that makes up 60% of the total amount of proteins in blood plasma. They are synthesized in the liver (about 15 g per day). Diseases associated with a decrease in the amount of blood albumin include some disorders in the liver (decreased protein synthesis in it), kidneys (loss of albumin in the urine as a result of a violation of the blood filtration mechanism in the kidneys), certain endocrine disorders (disorders of hormonal regulation of protein metabolism).

Causes of decreased albumin levels:

• kidney disease (nephrotic syndrome);

• protein-synthetic deficiency in severe liver damage: (cirrhosis, terminal stages of hepatitis);

• burns;

• extensive soft tissue injuries;

• sepsis;

• oncological diseases;

• thyrotoxicosis;

• long-term treatment with corticosteroids;

• rheumatic diseases.

Globulins (α1, α2, β, γ), like albumins, belong to simple proteins. In blood serum, there is a ratio of the number of albumins to globulins - the albumin-globulin coefficient.

The amount of α1-globulins is 1-4 g/l, α2-globulins - 4-12 g/l.

The amount of α1-globulins is 1-4 g/l, α2-globulins - 4-12 g/l.

A decrease in the number of α-globulins is rarely observed: with severe dystrophic processes in the liver, with lymphocytic leukemia, myeloma.

An increase in α1 and α2-globulins is characteristic of all acute inflammatory processes, exacerbation of chronic hepatitis, liver tumors, acute attacks of rheumatism, connective tissue diseases. The concentration of α2-globulin increases in the early stages of a myocardial infarction.

A decrease in the number of α-globulins is rarely observed: with severe dystrophic processes in the liver, with lymphocytic leukemia, myeloma.

The reasons for the increase in the fraction of alpha-1-globulin:

- acute or chronic inflammatory diseases;
- lymphogranulomatosis;
- cirrhosis of the liver;
- peptic ulcer;
- pregnancy;
- stress;
- taking oral contraceptives.

The reasons for the decrease in the fraction of alpha-1-globulin:

- deficiency of alpha-1-antitrypsin;
- acute viral hepatitis.

The reasons for the increase in the fraction of alpha-2-globulin:

- acute rheumatic fever and rheumatoid arthritis;

- chronic glomerulonephritis;
- cirrhosis of the liver;
- diabetes mellitus;
- lymphogranulomatosis;
- osteomyelitis;
- pneumonia;
- polyarteritis nodosum;
- sarcoidosis;
- systemic lupus erythematosus.

The reasons for the decrease in the fraction of alpha-2-globulin:

- acute viral hepatitis;
- hypogaptoglobinemia;
- intravascular hemolysis;
- hyperthyroidism;
- malabsorption;

The content of β-globulins is 5-11 g/l. This indicator has no independent value. It increases immediately with α2- or γ-globulins in chronic infections, liver cirrhosis, connective tissue diseases, and malignant tumors.

The reasons for the increased fraction of beta globulin:

- acute inflammatory diseases;
- diabetes mellitus;
- glomerulonephritis;
- hypercholesterolemia;
- iron deficiency anemia;
- subhepatic jaundice;
- pregnancy;
- rheumatoid arthritis;
- sarcoidosis;
- taking oral contraceptives.

The reasons for the decrease in the fraction of beta globulin:

- autoimmune diseases;
- leukemia;
- lymphoma;

- nephrotic syndrome;
- systemic scleroderma;
- systemic lupus erythematosus;
- cirrhosis of the liver.

The concentration of γ-globulins is 5-16 g/l of the total amount of globulins. This fraction contains the main amount of antibodies — immunoglobulins (Ig), IgG, IgM, IgA, IgE.

The reasons for the increase in the fraction of gamma globulin:

- amyloidosis;
- cirrhosis of the liver;
- chronic lymphocytic leukemia;
- cystic fibrosis;
- Hashimoto's thyroiditis;
- rheumatoid arthritis;
- sarcoidosis;
- systemic scleroderma;
- systemic lupus erythematosus;

The reasons for the decrease in the fraction of gamma globulin:

- acute viral hepatitis;
- glomerulonephritis;
- leukemia;
- lymphoma;
- nephrotic syndrome;
- malabsorption;
- scleroderma.

C-reactive protein (CRP), or the protein of the acute phase of inflammation, is a product of tissue breakdown in various inflammatory and necrotic processes, especially with a pronounced exudative component

A healthy person has a negative reaction. A positive reaction to CRP is determined in rheumatism, septic endocarditis, myocardial infarction, collagenosis, tuberculosis, cancer, peritonitis.

NON-PROTEIN COMPONENTS OF BLOOD

After precipitation of blood proteins, organic and inorganic compounds of nitrogen remain in the solution.

The normal concentration of nitrogen in the blood is 14-28 mmol/l, or 0.2-0.4 g/l. About 50 % of residual nitrogen is urea nitrogen; 25 % - nitrogen of amino acids; 7.5 %-creatinine and creatine; 0.5% - ammonium salts and indican, 13 % - other nitrogenous substances.

An increase in the concentration of residual nitrogen in the blood is called hyperazotemia. It has a different nature of occurrence: retention and production.

Retention occurs as a result of insufficient excretion of nitrogen-containing products with urine when they enter the blood normally. It, in turn, can be renal or extrarenal. Renal retention hyperazotemia is a consequence of a decrease in the excretory function of the kidneys. At the same time, the level of residual nitrogen increases due to urea, uric acid, creatine (glomerulonephritis, pyelonephritis, kidney amyloidosis). The degree of increase in urea level reflects the depth of organ damage.

Extrarenal retention hyperazotemia is a consequence of impaired urine outflow due to compression of the urethra in tumors, prostate adenoma, cardiovascular decompensation, shock, diabetes, etc.

Productive hyperazotemia occurs with an excessive influx of nitrogenous substances into the blood due to the breakdown of tissue proteins (starvation, cachexia, chronic infections, leukemia, radiation sickness).

Normal blood concentrations:

- creatinine - 50-115 μmol/l; in urine - 4.42-17.6 mmol/day;
- blood urea - 4.2-8.3 mmol/l, urine - 330-580 mmol/l;
- uric acid - men's blood 214-458 μmol/l, women's blood - 149-404 μmol/l, urine - 2.4-6.0 mmol/l.

ENZYME COMPOSITION OF BLOOD

Enzymes are contained in all cells of the body, but blood serum is most often used as an object for research, the enzyme composition of which is relatively constant and has a diverse origin. Three groups of enzymes are isolated in blood serum: cellular, secretory and excretory. Cellular enzymes can be non-specific (common to all tissues) and organ-specific (marker) - arginase in liver parenchyma, creatine phosphokinase in muscle tissue.

Secretory enzymes are sometimes called "proper blood enzymes" because they perform specific functions in the bloodstream (for example, ceruloplasmin provides copper transport). Excretory enzymes are produced by some organs of the digestive system (pancreas, intestinal mucosa, biliary tract endothelium). The appearance of these enzymes (alkaline phosphatase, amylase, trypsin, etc.) in blood serum is explained by the natural destruction of cellular structures in which they are formed.

It should be noted that enzyme dysfunction can be hereditary (primary) and acquired (secondary). Primary enzymopathies arise as a result of changes in the

genetic code of enzyme synthesis. A set of laboratory tests is required for their diagnosis.

Secondary, acquired, enzymopathies develop as a result of tissue damage by various agents (viruses, bacteria, protozoa, poisons, etc.). The etiological factor leads to a malfunction of one or more enzyme systems and a change in the corresponding metabolic processes, resulting in a disease with its characteristic symptoms. Therefore, enzymopathies include all types of human pathology (nosological forms).

There are several options for changes in the activity of enzymes in pathology: hyperfermentemia — an increase, hypofermentemia — a decrease in the activity of enzymes compared to the norm, dysfermentemia — the appearance of enzymes in the blood that are not normally detected. Enzyme activity is measured by the rate of the enzymatic reaction, which is proportional to the concentration of the enzyme.

The unit of activity of any enzyme is the amount of it that, under optimal conditions, catalyzes the conversion of 1 μmol of substrate in 1 minute (μmol/min).

α-Amylase - hydrolyzes starch and dextrins. In the body it is synthesized mainly in the salivary and pancreatic glands, found in the liver, kidneys, intestines, etc. 2 forms of amylase are detected in blood serum, one of which (type I), not bound to serum globulins, easily enters the urine, the other (type II) is contained in the fraction of 7S-globulins.

An increase in the activity of α-amylase is most significantly determined in acute pancreatitis, peritonitis, thrombosis of mesenteric vessels, rupture of the fallopian tube in case of ectopic pregnancy, contraction of the sphincter of Oddi, administration of morphine, codeine. A decrease in activity is found in mental states accompanied by depression, anacid disorders. The normal range for amylase in a blood sample for an adult is 30 to 110 units per liter (U/L).

Aminotransferases, or transaminases, catalyze the intermolecular transfer of an amino group from amino acids to keto acids. An increase in the activity of aminotransferases (the most significant) is found in myocardial infarction (pronounced cyclicity), viral hepatitis.

Aspartate transaminase (AST) is an enzyme the liver makes. Other organs, like heart, kidneys, brain, and muscles, also make smaller amounts. AST is also called SGOT (serum glutamic-oxaloacetic transaminase). Normally, AST levels in the blood are low.

A high AST level is a sign of liver damage, but it can also mean your patient has damage to another organ that makes it, like heart or kidneys. That's why you should do the AST test together with tests of other liver enzymes.

Normal ranges are:

- Males: 10 to 40 units/L
- Females: 9 to 32 units/L

Higher-than-normal AST levels (more than 3-4 times the normal value) can be caused by:

- Chronic (ongoing) hepatitis;
- Cirrhosis (long-term damage and scarring of the liver);
- Blockage in the bile ducts that carry digestive fluid from the liver to the gallbladder and intestine;
- Liver cancer.

Very high AST levels (more than 10 times the normal value) can be caused by:

- Acute viral hepatitis;
- Damage to the liver from drugs or other toxic substances;
- A blockage in blood flow to the liver;

These are other conditions that can also raise AST level:

- Burns;
- Heart attack;
- Intense exercise;
- Muscle injury;
- Pregnancy;
- Pancreatitis;
- Seizures;
- Surgery;
- Some diseases or medicines your patient takes can cause a "false positive" result on the AST test, for example diabetic ketoacidosis, some antibiotics, such as erythromycin estolate or para-aminosalicylic acid;
- Increased concentrations of serum aspartate transaminase and alanine transaminase are common in COVID-19 patients.

Alanine aminotransferase (ALT) is an enzyme that catalyzes the reversible transfer of the amino group (NH2) from the amino acid (alanine) α-ketoglutarate, leading to the formation of glutamate and pyruvic acid.

ALT is found mainly in the liver, at the level of hepatic cells (it is an intracellular liver enzyme, that located in the cytosol) and also in low concentrations may be in the kidneys, myocardium, muscles and pancreas. When cells are damaged or destroyed, this leads to the passage of cytoplasmic

components into the serum (cytolysis), causing an increase in the activity of ALT in the blood.

Normally, the blood contains a small amount of the enzyme. The degree of the increase in enzyme activity indicates the cytolytic syndrome. ALT is more specific for liver disorders than AST. High levels of ALT may indicate liver damage from hepatitis, infection,cirrhosis, liver cancer or other liver diseases.

When ALT levels are very high, it may be a sign of an acute liver problem. Mild or moderate elevation, especially if it persists on several tests over time, can be an indicator of a chronic disease.

Reasons for increased ALT activity:

- liver diseases (acute viral and toxic hepatitis, liver steatosis (2-3 times increase), liver cirrhosis (normal or 1-5 times higher than normal), alcoholic hepatitis, obstructive jaundice, liver metastases);
- shock, severe burns;
- Infectious mononucleosis;
- acute lymphoblastic leukemia;
- cardiovascular pathology: myocardial infarction, heart failure, myocarditis;
- acute pancreatitis;
- severe gestosis of the second trimester of pregnancy;
- myositis, myodystrophy, muscle injury, rhabdomyolysis, polymyositis and dermatomyositis (combined with a high level of creatine kinase);
- intramuscular injections;
- obesity;
- uses some substances, for example cholestatics, anabolic steroids, nicotinic acid, oral contraceptives, ethanol (in excess), iron salts, MAO inhibitors, mercaptopurine, methotrexate, methohifuoran, methyldopa, sulfonamides, aminoglycosides, azithromycin, cephalosporin, larithromycin, clindamycin, clofibrate, clotrimazole, cyclosporine, fluoroquinolones.

There are some factors that can influence normal level of ALT:

- exercise: intense or extreme exercise can cause an increase in ALT levels;
- medications: some medications and supplements can alter ALT measurements;
- gender: males typically have higher levels of ALT, which is believed to be related to hormonal differences;
- menstruation: ALT levels can go up or down during the course of the menstrual cycle;

- age: there is a tendency for ALT levels to decrease with older age;
- body mass index: several research studies have found an association between ALT levels and body mass index, which may change the interpretation of test results in people with obesity.

Reasons for decreased ALT activity:

- genitourinary system infections;
- tumors of various origins;
- deficiency of pyridoxal phosphate (malnutrition, alcohol consumption);
- liver disease (alcoholic hepatitis, fatty liver);
- pregnancy;
- uses some substances, for example aspirin, phenothiazines, interferon.

A normal ALT test result can range from 7 to 55 units per liter (U/L). Levels are normally higher in men. Slightly high ALT levels may be caused by: alcohol abuse.

Reference values:

- 0 - 1 year old; 56 U / l
- 1 - 4 years old; 29 U / l
- 4 - 7 years old; 29 U / l
- 7 - 13 years old; 37 U / l
- 13 - 18 years old; 37 U / l
- adults: men - 41 U / l, women - 33 U / l

A decrease in the activity of aminotransferases is observed in pregnant women whose pregnancy is at risk of hypertension or diabetes, with insufficient development of the placenta in pregnant women.

A normal AST:ALT ratio should be <1. In patients with alcoholic liver disease, the AST:ALT ratio is >1 and can be >2, which strongly suggestive of alcoholic liver disease. And scores <1 more suggestive of NAFLD/NASH. In most liver diseases, the ALT activity in the blood is higher than the AST activity, so the AST / ALT ratio (de Ritis coefficient) will be low. However, there are a few exceptions: alcoholic hepatitis, cirrhosis, and muscle damage.

Gamma-glutamyltransferase (GGT) functions in the body as a transport molecule, which help to move other molecules around the body. It plays a significant role in helping the liver metabolize drugs and other toxins. GGT is concentrated in the liver, but it's also present in the gallbladder, spleen, pancreas, and kidneys.

Normally, GGT is present in low levels, but when the liver is injured, the GGT level can rise. GGT is usually the first liver enzyme to rise in the blood when any of the bile ducts obstructed. This makes it the most sensitive liver enzyme test for detecting bile duct problems.

However, the GGT test is not very specific and its elevated can be seen with many types of liver diseases. But it can be useful in combination with other tests.

GGT levels are increased with consumption of even small amounts of alcohol. The GGT test may be used in evaluating someone for acute or chronic alcohol abuse.

It may be used to determine the cause of elevated alkaline phosphatase. Both ALP and GGT are elevated in disease of the bile ducts and in some liver diseases, but only ALP will be elevated in bone disease. Therefore, if the GGT level is normal in a person with a high ALP, the cause of the elevated ALP is most likely bone disease.

Some of the conditions that result in increased GGT include:

- overuse of alcohol;
- chronic viral hepatitis;
- lack of blood flow to the liver;
- liver tumor;
- cirrhosis, or scarred liver;
- overuse of certain drugs or other toxins;
- heart failure;
- diabetes;
- pancreatitis;
- fatty liver disease;
- Levels of GGT increase with age in women, but not in men;
- Smoking can also increase GGT level.

Normal results are:

- 0 to 61 IU/L for males
- 0 to 36 IU/L for females

Alkaline phosphatase (ALP) is an enzyme found in the bloodstream. It helps break down proteins in the body and exists in different forms, depending on where it originates. ALP can be found in the liver, bones, placenta of pregnant women, intestine, kidneys, and in other parts of the body.

Another type of ALP testing is an ALP isoenzyme or ALP fractionation test. In this test, specific subtypes of ALP are measured. These subtypes indicate where in the body the ALP was produced.

Reference range can vary from laboratory to laboratory. One common reference range is from 44 to 147 IU/L, but some guidelines recommend a range of 30-120 IU/L. For this reason, it’s important to check the reference range of the lab that analyzed sample.

Elevated ALP can be indicative of liver or bone diseases as well as many other types of conditions. However, high ALP levels are not always a sign of a problem. Interpretation of test results can involve consideration of multiple factors:

The degree of elevation: Very high levels are often seen with blockages of the bile ducts, but high levels alone cannot differentiate between liver problems and other conditions. ALP is usually higher in pregnant women, growing children and teenagers. Elevations of specific sources of ALP can identify the location of tissue damage or other disease in the body.

High levels may also indicate an issue related to the bones such as rickets, Paget's disease, bone cancer, or an overactive parathyroid gland, Metastases of other tumors to the bone, osteomalacia is the softening of bones caused by a lack of calcium. In rare cases, high ALP levels can indicate heart failure, kidney cancer, other cancer, mononucleosis, or bacterial infection. Abnormally low ALP levels are less common but can occur as a result of malnutrition, nutrient deficiencies (celiac disease), hypothyroidism, and some rare inherited disorders.

Causes of decreased activity of ALP:

- Severe anemia;
- Massive blood transfusions;
- Hypothyroidism is a condition in which the function of the thyroid gland is reduced;
- Lack of magnesium and zinc;
- Hypophosphatasia is a rare congenital disorder that causes softening of the bones;
- A pronounced decrease in ALP in pregnant women is a sign of placental insufficiency;
- Bone growth disorders (achondroplasia, cretinism, ascorbic acid deficiency);
- Protein deficiency.

Lactate dehydrogenase (LD) is an enzyme, which plays an important role in making body's energy. It is found in almost all the body's tissues, including those in the blood, heart, kidneys, brain, and lungs.

Elevated levels of LD usually indicate some type of tissue damage. LD levels typically will rise as the cellular destruction begins, peak after some time period, and then begin to fall.

An elevated level of LD may be seen with:

- Hemolytic anemia;
- Pernicious anemia (megaloblastic anemia);
- Infections such as infectious mononucleosis (mono), meningitis, encephalitis, HIV;
- Sepsis;
- Intestinal, myocardial (heart) and lung (pulmonary) infarction;
- Acute kidney disease;

- Acute liver disease;
- Acute muscle injury;
- Pancreatitis;
- Bone fractures;
- Testicular cancer, lymphoma or other cancers;
- Severe shock;
- Lack of oxygen (hypoxia);
- A high LD in the blood may indicate that treatment for cancer (e.g., chemotherapy) has not been successful. A high level is predictive of a poorer outlook for survival for those with cancer.

Low levels are sometimes seen when someone ingests large amounts of ascorbic acid (vitamin C).

Body fluids:

Cerebrospinal fluid—a high LD indicates that meningitis is likely caused by bacteria, while a low or normal level indicates viral meningitis is more likely.

A high LD indicates that pericardial fluid, peritoneal or pleural fluid is an exudate, while a low level indicates it is transudate.

A test for LD isoenzymes is rarely ordered and not widely available nowadays. In the past, the test was used to help diagnose and monitor heart attacks, but it has been replaced by the test for troponin. Though not a routine test, it may be used in differential diagnosis to help determine which organs are likely affected by tissue damage when the cause of an elevated total LD is not clear and cannot be determined using other specific tests.

LD exists in five different forms called isoenzymes. In general, the isoenzyme locations tend to be:

- LD-1: heart, red cells, kidneys, germ cells
- LD-2: kidneys, red blood cells, lungs, heart (lesser amounts than LD-1)
- LD-3: lungs and other tissues
- LD-4: white blood cells, lymph nodes, muscles, liver (lesser amounts than LD-5)
- LD-5: liver, skeletal muscle

Determining which isoenzyme is elevated in the blood may give clues to where tissue damage is occurring in the body and/or which organs may be affected.

Creatine kinase (creatine phosphokinase, CPC) — catalyzes the reaction of the reverse transfer of the phosphoric acid residue from ATP to creatine with the formation of creatine phosphate. From M- and B-type polypeptide chains, 3 isoenzymes of CPC are formed, which have relative specificity: CPC-MM (muscular), CPC-MB (cardiac), CPC-BB (brain). The total activity of CPC is 50-417 ncat/l; 25 IU, the activity of CPC-MB in it is 1-6%.

An increase in the activity of CPC is observed in myocarditis, myocardiodystrophy of various origins, muscular dystrophy, myositis, hypothyroidism, after surgical interventions, intramuscular injections of prednisolone, hypothermia, intensive physical exercises, alcohol intake. The most significant increase in the activity of CPC-MB is noted in myocardial infarction, arrhythmia, after digitalis therapy. A decrease in the activity of CPC is characteristic of thyrotoxicosis, pronounced muscle atrophy.

Trypsin catalyzes the cleavage of proteins, peptides, amides and esters, is formed in the lumen of the intestines from trypsinogen and participates in the digestion of food (trypsinogen is an inactive precursor of trypsin that is activated by enterokinase). Normal trypsin activity is 0-50 ncat/l (300-600 IU), α-antitrypsin is 5.1-10.2 μcat/l (0-3 IU).

An increase in trypsin activity is characteristic of acute and chronic pancreatitis, peptic ulcer disease.

A decrease in trypsin activity is observed in emphysema of the lungs, cirrhosis of the liver.

Renin is an enzyme that belongs to the class of hydrolases and the subclass of peptide hydrolases. Renin is characterized by high substrate specificity and catalyzes the conversion of angiotensin I from the M-end of the angiotensinogen molecule. Angiotensin I is converted into an octapeptide - angiotensin II, which has a powerful vasopressor effect. Thus, the participation of renin consists in the formation of substances that regulate blood pressure in the body, determines its role in the pathogenesis of some types of hypertension.

Renin is present in the kidneys, blood plasma, brain, salivary glands, uterus and other tissues.

Increased activity and an increase in the content of renin in the blood plasma are noted in some forms of arterial hypertension, ischemia of the kidneys and other organs. Renin is a sign of disease development. The activity of renin in blood plasma characterizes the weight of hypertensive disease and the risk of developing its complications. Normally, renin activity in blood serum is up to 0.03-0.06 μmol/l/min, or 0.6-1.0 nmol/l/s.

Cholinesterases. There are two cholinesterase enzymes in the body:

- acetylcholinesterase, found in red blood cells, lungs, spleen, nerve endings, and the gray matter of the brain,
- pseudocholinesterase, found in the serum, liver, muscle, pancreas, heart, and white matter of the brain.

Cholinesterase tests measure the activity of these enzymes.A decrease in the activity of the enzyme acetylcholinesterase results in excess acetylcholine at nerve endings. This can lead to overstimulation of nerves within body tissues and organs. Pseudochlinesterase is involved in processing and metabolizing drugs.

PIGMENT METABOLISM

The formation of bile pigments begins with the destruction of erythrocytes and the breakdown of hemoglobin. These processes occur mainly in the liver, spleen and bone marrow. Intermediate products of the breakdown of hemoglobin are verdohemoglobin and biliverdin, which is enzymatically reduced to bilirubin, the main bile pigment of humans. The main organs of bilirubin production are the liver and spleen. The bilirubin formed accumulates in the liver, from where it passes into the gall bladder together with bile and flows into the intestines. About 200-300 mg of bilirubin is excreted in bile per day.

Initially, the bilirubin formed is called free (indirect). It is toxic, poorly soluble in water and easily absorbed by blood plasma proteins, mainly albumins.

Another form of bilirubin — direct or bound, is found in connection with mono- or diglucuronide. Unlike free bilirubin, bound is well soluble in water, non-toxic, easily passes through membranes.

The content of total bilirubin in the blood of an adult is relatively constant — from 8.5 to 20.5 μmol/l. About 75% of this amount is the share of free (indirect) bilirubin (6.5-15.4 μmol/l), and the amount of the bound fraction is normally 2.1-5.1 μmol/l.

The formation of mono- and diglucuronides in the liver plays an extremely important role as a mechanism for neutralizing free bilirubin, which is toxic to the body. This process can be disturbed in severe liver diseases, accompanied by a loss of the ability of liver cells (hepatocytes) to capture free bilirubin from the bloodstream, as well as in the event of damage to the transport system that carries free bilirubin to the hepatocyte microsomal membrane, where bilirubin is conjugated with glucuronic acid. . These disorders contribute to an increase in free bilirubin in the blood, which is accompanied by intoxication of the body.

Thus, an increase in the level of free bilirubin in blood serum is an unfavorable diagnostic sign. Jaundic coloring of the skin appears when the content of bilirubin in the blood exceeds 34 μmol/l. An increase in the level of bilirubin in the blood can be observed when the parenchyma of the liver is damaged and its bilirubin-releasing function is impaired as a result of infectious toxic and alcoholic acute hepatitis and liver cirrhosis (parenchymal jaundice), with increased hemolysis of erythrocytes (hemolytic jaundice), with impaired outflow of bile from the bile ducts into the intestines (obstructive jaundice).

The study of different fractions of bilirubin makes it possible to conduct a differential diagnosis of jaundice and monitor the course of treatment.

Parenchymal jaundice associated with damage to hepatocytes is characterized by an increase in the level of indirect and direct bilirubin in the blood (with a significant preference for direct). At the same time, hyperbilirubinuria is observed. A significant increase in the concentration of indirect bilirubin (more

than 34.2 μmol/l) indicates severe liver damage and is an unfavorable prognostic sign.

In **obstructive jaundice**, the level of bilirubin in the blood serum increases mainly due to direct bilirubin

With **hemolytic jaundice**, the amount of indirect bilirubin that binds to albumin and is not excreted in the urine increases sharply in the blood serum.

Changes in the indicators of the exchange of bile pigments in the blood are combined with the determination of their content in urine and feces. Bile contains bound bilirubin, which enters the intestines. As a result of enzymatic transformations in the lumen of the intestines, urobilinogen is formed from bilirubin, which is partially absorbed and enters the liver through the portal vein, where it undergoes destruction. A small part of stercobilinogen is absorbed through the system of hemorrhoidal veins and enters the blood, from where it is excreted by the kidneys in the form of urobilin (stercobilinogen) in normal urine.

With liver diseases, the content of urobilin in the urine increases sharply, because when the liver function is impaired, its ability to extract urobilinogen from the blood of the portal vein is lost. In parenchymal lesions of the liver, urobilinuria is usually combined with bilirubinuria. Slight urobilinuria is observed with increased hemolysis of erythrocytes, with intestinal diseases and conditions that have a toxic effect on the liver parenchyma.

The complete absence of urobilin in the urine in the presence of bile pigments indicates the cessation of the flow of bile into the intestines due to blockage of the bile duct (obstructive jaundice).

CARBOHYDRATE METABOLISM

Carbohydrates play an important role in the metabolism and energy of the body. They are the main source of energy, providing a large part of the body's energy needs. About 400-500 g of carbohydrates (starch, glycogen, sucrose, lactose, glucose, fructose, etc.) enter the human body with food. Polysaccharides and disaccharides are digested mainly in the digestive tract, turning into monosaccharides, and are absorbed into the blood.

The most important mechanism for maintaining a stable level of glucose in the blood is its neurohormonal regulation, which closely interacts with the glycogenic function of the liver — the ability of this organ to synthesize glycogen and break it down into glucose. The influence of some hormones (adrenaline, thyroxine, glucagon) contribute to the enzymatic breakdown of glycogen in the liver, while insulin activates the processes of glucose use in the synthesis of glycogen and lipids, which leads to a decrease in its level in the blood.

The role of glucose and its derivatives in energy metabolism consists in numerous enzymatic reactions, which are accompanied by the conversion of the energy of chemical bonds of carbohydrates into the energy of specific macroergic

phosphate bonds of ATP. Subsequently, ATP energy can be used for muscle work, the biosynthesis of various substances and provide all body functions.

Carbohydrate metabolism plays an important role in the function of the heart, brain, liver and other organs. The role of glucose metabolism processes in brain tissues is extremely important, where it is the main substrate of cellular respiration. In 1 minute, 100 g of human brain tissue consumes about 5 mg of glucose, and the entire brain consumes about 75 mg. At the same time, more than 90% of glucose is oxidized to carbon dioxide and water with the participation of the tricarboxylic acid cycle.

Glucose reserves in brain tissues are small (about 750 mg, that is, for only 10 minutes of its work). This means the high sensitivity of these tissues to a sharp drop in the level of glucose in the blood, which can lead to fainting and hypoglycemic coma with an overdose of insulin, pituitary cachexia, and hypothyroidism.

In addition, carbohydrates are used as components of intracellular structures and play an important role in the construction of immunoglobulins and interferon. Carbohydrate components of glycoproteins — glycosaminoglycans (hyaluronic and chondroitin sulfuric acids) are part of the main substance of connective tissue, are contained in cell membranes of synovial fluid and in other tissues and fluids of the body. Biologically active glycoproteins performing protective functions include α-, β- and γ-interferons, built from protein (160 amino acid residues) and - polysaccharide components.

The most important biological functions are performed by blood plasma glycoproteins, which include globulins, transferrin, ceruloplasmin, and some enzymes. Glycoproteins take part in immunological reactions, ion exchange, intercellular adhesion processes, determining the group characteristics of blood erythrocytes.

Glucose is contained in the blood of healthy people in the amount of 3.3-5.5 mmol/l, 0.8-1.2 g/l.

The main causes of high glucose levels are:

- diabetes mellitus;
- liver disease;
- insulin resistance syndrome;
- acromegaly;
- severe stress (reaction to trauma, heart attack, stroke);
- chronic renal failure;
- Cushing's syndrome;
- taking medications such as corticosteroids, tricyclic antidepressants, diuretics, epinephrines, estrogens, lithium, diphenin (dilantin), salicylates;

- excessive intake of high-carbohydrate foods;
- hyperthyroidism;
- pancreatic cancer;
- pancreatitis.

The causes of low blood glucose levels are:

- insulin overdose (I think this is the most common cause of low glucose in diabetics);
- adrenal insufficiency;
- alcohol abuse, especially in insulin-dependent patients with diabetes mellitus;
- taking medications like acetaminophen and anabolic steroids;
- liver disease;
- hypopituitarism;
- hypothyroidism;
- insulinomas;
- fasting.

Confirmation of the "Viple triad" is required for a confident diagnosis of hypoglycemia:

- glucose level is below of 2.2 mmol / L;
- symptoms of hypoglycemia (disorders of the nervous system (sweating, trembling, hunger, anxiety), and then the brain is also affected (blurred consciousness, hallucinations, blurred vision, sometimes coma and even death);
- disappearance of symptoms when blood glucose returns to normal.

Glycosylated hemoglobin is a specific compound of erythrocyte hemoglobin with glucose, the concentration of which reflects the average blood glucose level over a period of about three months. Glycosylated hemoglobin test helps to estimate the average blood glucose over the past 3 months. The resulting indicator is measured as a percentage. Patients with diabetes should strive to keep their glycosylated hemoglobin levels below 7%.

Reference values are: 4.8 - 5.9%.

With an increase in the level of glycosylated hemoglobin in patients with diabetes, the risk of complications also increases.

The glycosylated hemoglobin analysis results are interpreted as follows:

- 4-6.2 % -the patient has no diabetes;

- 6.5 % and more - the patient has diabetes mellitus;
- 5.7-6.4 % -prediabetes (impaired glucose tolerance associated with an increased risk of diabetes).

In patients with abnormal forms of hemoglobin, for example, in patients with sickle-shaped erythrocytes, the level of glycosylated hemoglobin will be underestimated. In addition, if a person suffers from anemia, severe bleeding, the results of the analysis may also be underestimated. On the contrary, A1c indices are overestimated with a lack of iron and with a recent blood transfusion (since liquid blood preservatives contain a high concentration of glucose).

Connecting peptide, C-peptide. C-peptide is a component of the secretion of the endocrine part of the pancreas, which is an indicator of insulin production and is used to diagnose diabetes mellitus (DM), make its prognosis and control its treatment, as well as for the diagnosis of some pancreatic tumors. This laboratory indicator is used to assess the level of endogenous insulin.

C-peptide is produced in the same amount as insulin, the concentration of C-peptide in the peripheral blood corresponds to the direct production of insulin in the pancreas.

In addition, the concentration of C-peptide does not depend on changes in blood glucose levels and is relatively constant. Therefore, C-peptide analysis is the best method to assess the production of insulin in the pancreas.

Reference values: 1.1 - 4.4 ng / ml.

Reasons for increased serum C-peptide levels:

- obesity (male type);
- pancreatic tumors;
- the use sulfonylurea drugs (glibenclamide);
- long QT interval syndrome.

Reasons for a decrease in serum C-peptide levels:

- diabetes mellitus;
- the use of thiazolidinediones (rosiglitazone).

Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) is the ratio of insulin and blood glucose levels.

Insulin resistance index is calculated according to the formula:

Fasting insulin (U/L) x Fasting glucose (nmol/L)/ 22.5.

The normal Insulin resistance index value of healthy human ranges from 0.5-1.9. Above 1.9 indicates early insulin resistance. Above 2.9 indicates significant insulin resistance.

Pyruvic acid is one of the main metabolites of intermediate metabolism. In the blood of a healthy person, it contains 0.05-0.14 mmol/l; 0.3 — 0.9 mg/100 ml.

An increase in the concentration of pyruvic acid in the blood is observed in diabetes, parenchymal liver diseases, heart failure, increased physical exertion, lack of thiamine, after the administration of certain drugs - strychnine, adrenaline.

Lactic acid is the final product of glycolysis and glycogenolysis, normally its content in venous blood is 0.55-2.22 mmol/l; 5-20 mg/100 ml. An increase in the amount of lactic acid in the blood is determined by increased muscle work, heart failure, and some liver diseases.

Sialic acids are acylated derivatives of neuraminic acid. They are part of glycoproteins, glycolipids (gangliosides), mucins and other glycoconjugates. Sialic acids are involved in the formation of a number of biologically active compounds containing carbohydrates. In healthy people, the level of sialic acids in blood serum varies between 2.0-2.33 mmol/l. These indicators increase significantly with various inflammatory and necrotic processes accompanying arthritis, in particular, rheumatoid arthritis and polyarthritis. The level of sialic acids reflects the degree of activity of the rheumatic process, some acute and chronic inflammatory diseases.

LIPID METABOLISM

Lipids are a large group of compounds that differ significantly in their chemical structure and the functions they perform. There are several classifications of lipids. They are characterized by the following general features: insoluble in water, soluble in non-polar solvents, such as ether, chloroform; contain higher alkyl radicals in their composition; widely distributed in living organisms. We will give the characteristics of some of them.

Lipid metabolism disorders play an important role in the development of vascular atherosclerosis and diseases of the cardiovascular system. It has been scientifically proven that elevated blood cholesterol (hypercholesterolemia) and local inflammatory changes in the vascular wall increase the risk of thickening and hardening of the arterial wall with subsequent disorders of local circulation. Atherosclerotic vascular disease, according to statistics, increases the likelihood of myocardial infarction, stroke, kidney pathology. A lipidogram allows to assess atherogenicity (tendency to develop atherosclerosis) of blood plasma even at normal levels of total cholesterol. In the study of lipid profile, indicators such as triglycerides, total cholesterol (cholesterol), high, low and very low density lipids are determined. The coefficient of atherogenicity is calculated.

Cholesterol is an essential organic substance. It is synthesized mainly by the liver (endogenous cholesterol), and also partially enters the body with food (exogenous cholesterol). Cholesterol forms the cell membranes of all organs and

tissues of the body, is a precursor of steroid hormones necessary for full development, growth and puberty, takes part in the synthesis of bile acids, which ensure the absorption of nutrients from the intestines. In the blood, cholesterol circulates in a complex with lipoprotein proteins.

Cholesterol (CS) is a fat-like substance that is vital for the body. It participates in the formation of cell membranes in all organs and tissues of the body. On the basis of cholesterol, hormones are created, without which growth, development of the body and the implementation of the reproduction function are impossible. Bile acids are formed from it, due to which fats are absorbed in the intestines.Cholesterol is insoluble in water, therefore, to move around the body, it is "packed" in a shell consisting of special proteins - apoproteins. The resulting complex ("cholesterol + apoprotein") is called lipoprotein. High-density lipoproteins (HDL) remove excess free cholesterol accumulated in peripheral cells. They transport cholesterol to the liver, where it is catabolized to form fatty acids, or they transfer it to very low density lipoproteins (VLDL), as a result of which the latter are converted to low density lipoproteins (LDL). HDL are antiatherogenic factors that prevent the formation of atherosclerotic plaque in the vessel. A lowered HDL indicates the possibility of developing the disease.The total blood cholesterol is 60-70% represented by LDL, which are able to stay in the vascular wall and promote the accumulation of cholesterol in tissues. It is the levels of LDL and, to a lesser extent, total cholesterol in blood plasma that determine the risk of developing atherosclerosis and cardiovascular diseases. Even if the norm of cholesterol remains, an increase in LDL is indicative of atherogenic properties of blood lipids.Elevated blood triglyceride levels are also associated with the risk of atherosclerosis, coronary heart disease, and cerebrovascular disease.

Reference values (cholesterol norm): 2.9 - 5.2 mmol / l.

The concept of "norm" is not quite applicable in relation to the level of total cholesterol. For different people with different numbers of risk factors, the cholesterol level will differ. The total cholesterol (cholesterol) test is used to determine the risk of cardiovascular disease, however, in order to determine this risk for a particular patient most accurately, it is necessary to evaluate all the predisposing factors.

Causes of increased total cholesterol levels (hypercholesterolemia)

Hypercholesterolemia can be the result of a hereditary predisposition (familial hypercholesterolemia) or excessive intake of animal fats with food. In most people with high cholesterol, both are involved to some extent.

In adults, during a preventive examination, the results are divided into three groups according to the degree of risk of cardiovascular diseases:

- acceptable cholesterol level - below 5.2 mmol / l - low risk;
- borderline level - 5.2-6.2 mmol / l - average risk (in this case, the determination of the so-called lipid profile (lipid profile) may additionally be

prescribed to determine due to which fraction of cholesterol its total level is increased - LDL (" bad ") or HDL (" good ");

- high cholesterol level - more than 6.2 mmol / l - high risk (again, a lipid profile can be assigned - to clarify the reason for the increase in total cholesterol).

Before prescribing treatment, it is necessary to exclude other causes of an increase in total cholesterol:

- cholestasis - stagnation of bile, which can be caused by liver disease (hepatitis, cirrhosis) or stones in the gallbladder;
- chronic inflammation of the kidneys leading to nephrotic syndrome;
- chronic renal failure;
- decreased thyroid function (hypothyroidism);
- poorly treated diabetes mellitus;
- alcoholism;
- obesity;
- cancer of the prostate or pancreas.

Reasons for lowering total cholesterol levels (hypocholesterolemia)

- Heredity;
- Severe liver disease;
- Oncological diseases of the bone marrow;
- Increased thyroid function (hyperthyroidism);
- Disorders of absorption in the intestines;
- Folate or B12 deficiency anemia;
- Commonburns;
- Tuberculosis;
- Acutediseases, acuteinfections;
- Chronicobstructivepulmonarydisease.

Cholesterol concentration may change from time to time, this is normal. A single measurement does not always reflect the usual level, therefore, it may sometimes be necessary to retake the analysis after 1-3 months.

Increase total cholesterol levels:

- Pregnancy (a cholesterol test must be taken at least 6 weeks after delivery);
- Prolonged fasting;

- Donating blood while standing;
- Anabolic steroids, androgens, corticosteroids;
- Smoking;
- Eating food containing animal fats;

Reduce total cholesterol levels:

- Donating blood while lying down;
- Taking some medicines (allopurinol, clofibrate, colchicine, antifungal drugs, statins, cholestyramine, erythromycin, estrogens);
- Intense physical activity;
- A diet high in polyunsaturated fatty acids.

High density lipoprotein cholesterol (HDL-C).

The concept of "norm" is not quite applicable in relation to the level of HDL cholesterol. For different people with different numbers of risk factors, HDL cholesterol levels will differ. To determine the risk of developing cardiovascular disease more accurately for a particular person, it is necessary to assess all the predisposing factors.

In general, we can say that a low level of HDL predisposes to the development of atherosclerosis, and sufficient or high - prevents this process. Reference values: 1.03 - 1.55 mmol / L.

In adults, HDL cholesterol, depending on the level, can be assessed as follows:

- less than 1.0 mmol / l in men and 1.3 mmol / l in women - a high risk of developing atherosclerosis and cardiovascular diseases, regardless of other risk factors;
- 1.0-1.3 mmol / L in men and 1.3-1.5 mmol / L in women - the average risk of developing atherosclerosis and cardiovascular diseases;
- 1.55 mmol / L and above - low risk of developing atherosclerosis and cardiovascular diseases; while the vessels are protected from the negative effects of excess cholesterol.

Reasons for low HDL levels:

- heredity (Tangier disease);
- cholestasis - stagnation of bile, which can be caused by liver disease (hepatitis, cirrhosis) or stones in the gallbladder;
- severe liver disease;
- untreated diabetes mellitus;
- chronic inflammation of the kidneys leading to nephrotic syndrome;

- chronic renal failure.

Causes of elevated HDL levels:

- hereditary predisposition;
- chronic liver disease;
- alcoholism;
- frequentintenseaerobicexercise.

HDL cholesterol levels may change from time to time. A single measurement does not always reflect the "normal" amount of cholesterol, so sometimes it may be necessary to retake the test after 1-3 months.

Sometimes, HDL cholesterol levels can be higher or lower for a short period of time. This phenomenon is called biological variation and reflects the normal fluctuations in cholesterol metabolism in the body.

Reduce HDL levels:

- stress, a recent illness (after them you need to wait at least 6 weeks);
- anabolic steroids, androgens, corticosteroids.

Increase HDL levels:

- pregnancy (a lipid profile should be taken at least 6 weeks after the baby is born);
- statins, cholestyramine, phenobarbital, fibrates, estrogens, insulin.

Low-density lipoprotein cholesterol (LDL-C). An LDL cholesterol test is used to determine the risk of cardiovascular disease, however, in order to accurately determine it for a person, all factors must be taken to attention.

An increase in LDL cholesterol may be the result of a hereditary predisposition (familial hypercholesterolemia) or excessive intake of animal fats. In most people with high cholesterol, both are involved to some extent. Reference values: 0 - 3.3 mmol / L.

The level of LDL cholesterol can be estimated as follows:

- less than 2.6 mmol / l – optimal;
- 2.6-3.3 mmol / l – close to optimal;
- 3.4-4.1 mmol / l – borderline high;
- 4.1-4.9 mmol / l – high;
- above 4.9 mmol / l – very high.

Possible causes of high LDL cholesterol levels:

- cholestasis – stagnation of bile, which can be caused by liver disease (hepatitis, cirrhosis) or stones in the gallbladder;

- chronic inflammation of the kidneys leading to nephrotic syndrome;
- chronic renal failure;
- decreased thyroid function (hypothyroidism);
- poorly treated diabetes mellitus;
- alcoholism;
- obesity;
- cancer of the prostate or pancreas.

The lowered level of LDL cholesterol is not used in diagnostics due to low specificity. Nevertheless, its reasons may be:

- hereditary hypocholesterolemia;
- severe liver disease;
- oncological diseases of the bone marrow;
- increased thyroid function (hyperthyroidism);
- inflammatory diseases of the joints;
- B12 or folate deficiency anemia;
- common burns;
- acute diseases, acute infections;
- chronicobstructivepulmonarydisease.

The cholesterol concentration may change from time to time, this is normal. A single measurement does not always reflect the usual LDL level, therefore, it may sometimes be necessary to retake the analysis after 1-3 months.

Increase the level of **very low density lipoprotein cholesterol** (VLDL cholesterol):

- pregnancy (lipid profile should be done at least 6 weeks after the baby is born);
- prolonged fasting;
- standing blood donation;
- anabolic steroids, androgens, corticosteroids;
- smoking;
- eating food containing animal fats.

Reduce the level of VLDL cholesterol:

- lying down;

- allopurinol, clofibrate, colchicine, antifungal drugs, statins, cholestyramine, erythromycin, estrogens;
- intense physical activity;
- a diet low in cholesterol and saturated fatty acids and, on the contrary, high in polyunsaturated fatty acids.

Triglycerides are a compound of esters of fatty acids and glycerol and are the main source of energy for the body. The predominant amount of triglycerides is found in adipose tissue, and only a small amount is found in the blood. They come from food or are resynthesized in the liver. Most triglycerides are transported in the blood as very low density lipoprotein (VLDL). Elevated triglyceride levels are often combined with diabetes mellitus, obesity, arterial hypertension and changes in other lipid profile indicators.

Triglycerides are fats that are the body's main source of energy. Most triglycerides are found in adipose tissue, but some are found in the blood, providing energy to muscles. After eating, triglyceride levels rise as the body converts the energy it doesn't need into fat. Triglycerides are absorbed in the intestine and, transported through the blood, are stored in adipose tissue in reserve. They are burned between meals, releasing energy for the body. Since triglycerides are insoluble in water, they are carried in the blood with a protein in a complex called lipoprotein. Most triglycerides in the body are carried by very low density lipoproteins (VLDL).

An increase in triglycerides increases the risk of developing cardiovascular disease, although their causes are not fully understood. There are several factors that contribute to this: decreased physical activity, overweight, smoking, alcohol abuse, and diabetes.In addition, triglycerides significantly increase the risk of developing acute inflammation of the pancreas - acute pancreatitis.

Reference values (norm of triglycerides): 0 - 2.25 mmol / l.

The interpretation of the results should be made taking into account other analyzes included in the lipid profile.

Causes of increased triglyceride levels (hypertriglyceridemia). Hypertriglyceridemia can be the result of a hereditary predisposition or excessive intake of animal fats from food. In most people with high cholesterol, both are involved to some extent.

In adults, during a preventive examination, the results are divided into several groups according to the degree of risk:

- an acceptable TG level - below 1.7 mmol / L - reflects a low risk of cardiovascular diseases;
- borderline level - 1.7-2.2 mmol / l;
- a high level of triglycerides - 2.3-5.6 mmol / l - indicates a high probability of developing cardiovascular diseases;

- very high - above 5.6 mmol / l - in addition to the high risk of heart problems, in this case there is a very high risk of developing pancreatitis, therefore, early prescription of drugs that reduce the concentration of TG is required (optimally - to 1.3 mmol / l or below).

Before prescribing treatment, it is necessary to exclude other causes of an increased triglyceride content:

- alcoholism;
- chronic inflammation of the kidneys leading to nephrotic syndrome;
- chronic renal failure;
- decreased thyroid function (hypothyroidism);
- poorly treated diabetes mellitus;
- pancreatitis;
- myocardial infarction - in this case, elevated levels can persist for up to several months;
- gout.

Reduced triglyceride levels (hypotriglyceridemia) are of no particular clinical significance. It can occur under the following conditions:

- hereditary hypoliproproteinemia;
- increased thyroid function (hyperthyroidism);
- malabsorption in the intestine;
- chronic obstructive pulmonary disease;
- cerebral infarction.

Triglyceride levels can remain significantly (up to 5-10 times) elevated even several hours after a meal.

Fasting blood samples taken at different times may also vary. In some people, triglyceride levels change by 40% within one month. This phenomenon is called biological variation and reflects the normal fluctuations in cholesterol metabolism in the body. As a result, a single measurement does not always reflect the "normal" triglyceride level, so sometimes a retest is required.

Increase triglyceride levels:

- food rich in fat;
- drinking alcohol;
- pregnancy (the test should be taken at least 6 weeks after delivery);
- oral contraceptives, cholestyramine, furosemide, veroshpiron, cordarone, corticosteroids.

Reduce triglyceride levels:

- intense physical activity;
- statins, metformin.

Cholesterol / HDL ratio. The coefficient of atherogenicity is calculated based on lipid metabolism indicators: CA = (total cholesterol - HDL) / HDL or CA = (LDL + VLDL) / HDL. A coefficient of atherogenicity that exceeds the normal range indicates an increased risk of cardiovascular disease. Reference values: 2.2 - 3.5.

A result above 3 indicates a predominance of "bad" cholesterol, which may be a sign of atherosclerosis.

CA increase:

- pregnancy (cholesterol should be taken at least 6 weeks after the baby is born);
- prolonged fasting;
- donating blood while standing;
- anabolic steroids, androgens, corticosteroids;
- smoking;
- eating food containing animal fats.

CA decrease:

- donating blood while lying down;
- allopurinol, clofibrate, colchicine, antifungal drugs, statins, cholestyramine, erythromycin, estrogens;
- intensephysicalactivity;
- a diet low in cholesterol and high in polyunsaturated fatty acids.

Medicines that increase total cholesterol: beta-blockers, corticosteroids, lansoprazole, lithium salts, oral contraceptives, phenobarbital, thiazides.

Medicines that lower total cholesterol: estrogens, allopurinol, androgens, statins, fibrates, fatty acid sequestrants, levothyroxine, filgrastim, tamoxifen.

Drugs that increase HDL levels: steroid drugs, progestins, androgens, alpha-blockers, carbamazepine, lipid-lowering drugs, estrogens, hydroxychloroquine, indapamide, insulin, hypoglycemic drugs, phenobarbital, phenytoin.

Medicines that lower HDL levels: oral contraceptives, beta-blockers, methimazole, methyldopa, tamoxifen, thiazides.

Drugs that increase LDL levels: anabolic steroids, aspirin, carbamazepine, corticosteroids, oral contraceptives, phenothiazides, progestins, sulfonamides.

Medicines that lower LDL levels: cholestyramine, clofibrate, estrogens, neomycin sulfate, nicotinic acid, statins, thyroxine.

Medicines that increase triglyceride levels: beta-blockers, cholestyramine, corticosteroids, estrogens, oral contraceptives, thiazide diuretics.

Medicines that lower triglycerides: ascorbic acid, asparaginase, colestipol, clofibrate, metformin, niacin.

In the presence of the disease, the type of hyperlipoproteinemia (HLP) helps to establish the type of lipid metabolism disorder. Each type of HLP is characterized by a specific clinical picture.

- HLP type I (increased level of chylomicrons, cholesterol, triglycerides, atherogenicity has not been proven) is detected since childhood, has a familial nature, the presence of this disorder is not characteristic of atherosclerosis, it is extremely rare.
- HLP IIa type (increased LDL, cholesterol, triglycerides - normal.) is often detected in coronary heart disease, in cases of sudden death of young people due to myocardial infarction.
- HLP IIb type (increased LDL and VLDL, cholesterol, triglycerides) often develops with coronary heart disease.
- HLP III type (increased LDL, cholesterol, triglycerides) — in atherosclerosis with damage to the arteries of the lower extremities, mainly in the elderly.
- HLP type IV (increased VLDL), cholesterol is normal or slightly elevated, triglycerides are elevated) is observed in the elderly, with atherosclerosis of the coronary arteries, obesity and diabetes.
- Type V HLP (increased VLDL and chylomicrons, cholesterol, triglycerides) is often determined in coronary heart disease and diabetes, mainly in the elderly.

MINERAL METABOLISM

Mineral substances are vital components of nutrition that ensure normal life and development of the body.

Sodium is the main cation of the extracellular fluid of blood plasma. It is contained in a concentration of 135-150 mmol/l. Sodium is part of all body tissues and fluids. Of the total amount of sodium in the body, 50% is in extracellular fluid, 6% in cells, and 44% in cysts. Bones are the main depot of sodium. In the body, sodium is mainly found in the form of salts NaCl, $NaHCSO_3$, Na_2HPO_4.

Sodium is the main cation of the extracellular fluid, maintains osmotic pressure, determines the movement of water, the state of neuromuscular excitability, regulates the constancy of the bioelectric potential of cell membranes.

An increase in sodium content in the blood plasma (hypernatremia) is observed with oliguria or anuria of any origin, hyperfunction of the adrenal cortex

(Cushing's syndrome, primary aldosteronism), prolonged use of corticosteroids, parenteral administration of hypertonic sodium solution, and severe fluid restriction.

A decrease in the concentration of sodium in the blood plasma (hyponatremia) is caused by a salt-free diet, profuse sweating, severe and prolonged labor, acute and chronic adrenal insufficiency, and diabetic acidosis.

Potassium is the main cellular cation. Blood serum contains 3.6-6.3 mmol/l. In the intercellular fluid and blood plasma, potassium is in an ionized form, in the cells - in the form of weak compounds with proteins, carbohydrates, creatinine, phosphorus.

Potassium supports osmotic and acid-base homeostasis in the cell, participates in ensuring transmembrane potential difference, synthesis of protein, glycogen, ATP, creatine phosphate, acetylcholine, glucose phosphorylation. In the transmission of disturbances along the neuromuscular fiber, the potassium ion is a synergist of the sodium ion and an antagonist of the calcium ion.

An increase in the content of potassium in the blood plasma (hyperkalemia) is noted: in hemolytic anemias, disintegrating tumors, necrosis, long-term crushing syndrome and positional muscle necrosis; with impaired excretion of potassium from the urine (oliguria, anuria, chronic nephritis), dehydration, anaphylactic shock, parenteral administration of potassium-rich solutions against the background of kidney failure; when treated with triamterene.

A decrease in the concentration of potassium (hypokalemia) occurs with insufficient intake of potassium with food, increased excretion of it with urine, which can be observed with hyperfunction of the cortical layer of the adrenal glands and the anterior lobe of the pituitary gland, primary and secondary aldosteronism, increased secretion of antidiuretic hormone, diabetic acidosis, respiratory alkalosis, parenteral administration of large amounts of fluid that does not contain potassium, treatment with gentamicin, overdose of ACTH, preparations of the adrenal cortex, saluretics.

Chlorine is contained in the serum in the amount of 95-110 mmol/l. The concentration of chlorine anion of the extracellular fluid is 70% of the total amount of chlorine, and the rest of it (30%) is unevenly distributed between tissues.

The biological value of chlorine consists in maintaining osmotic pressure, the acid-base state of the extracellular fluid, and the gas exchange function of erythrocytes, since chlorine ions are easily exchanged with bicarbonate ions. Chlorine plays an important role in neutralizing the products of pathological decay of tissues. Chlorine ions are a component of hydrochloric acid in gastric juice and are an activator of amylase. The daily need for chlorine varies from 41 to 140 mmol (1.5-5 g). Chlorine ions enter the body with food in the form of sodium chloride.

Normally, the concentration ratio of chlorine ions in erythrocytes and blood plasma is 0.45-0.5. With acidosis, the content of chlorine ions in erythrocytes increases and this ratio exceeds 0.5, with alkalosis, it decreases to 0.4 and below. Therefore, the ratio of concentrations of chlorine ions in erythrocytes and blood plasma is an important indicator of the acid-base state.

An increase in the content of chlorine in the blood (hyperchlorplasmia) is observed with decompensation of the mechanisms of regulation of water-salt exchange - pituitary insufficiency, heart failure, arterial hypertension, alkalosis, resorption of edema, exudates, transudates, ingestion of large doses of chlorides.

A decrease in the level of chlorine in the blood (hypochlorplasma) is noted with muscle strain, sweating, vomiting, diarrhea, poisoning with sulema, nephritis, nephrosis, renal diabetes, respiratory acidosis, cholera, and a decrease in the intake of chlorine with food.

Calcium is concentrated almost exclusively in blood serum, its content is 2.25 — 3.0 mmol/l. In bones, calcium is represented by phosphates — $Ca_3(PO_4)_2$ (85%), carbonates — $CaCO_3$ (10%) and salts of organic acids — citric and lactic (about 5%). The plasma contains calcium in the form of forms that perform different functions: non-diffusing or protein-bound (0.9 mmol/l), and diffusing — ionized (1.25 mmol/l) and non-ionized (0.35 mmol/l l). Only ionized calcium is biologically active.

Calcium is involved in the construction of bone tissue, the process of blood clotting. Calcium ions affect the activity of a number of enzymes, the permeability of membranes, energy processes in mitochondria, are able to activate various metabolic processes in cells, gluconeogenesis, glycogenesis, participate in the generation of action potentials in nerve and muscle cells, in the transmission of disorders along the neuromuscular ulcer fiber, act as a connecting factor between contraction processes in muscles, including in the myocardium, regulate the excitability of nerve and muscle cells, support the contractile ability of the myocardium, contribute to the release of mediators, the release and physiological action of hormones, enhance the effect of vasopressin.

The daily calcium requirement of an adult is normally 20 — 37.5 mmol (0.8 — 1.5 g), pregnant and lactating women - 2 times higher. Infants should receive about 15 mmol (0.6 g), children aged 1 year and older - 25 mmol (up to 1 g) of calcium.

Calcium combines with fatty and bile acids and enters the liver through the portal vein. Calciferol contributes to the transport of calcium ions through the enterocyte membrane into the blood. The calcium/phosphorus ratio is important in the absorption process.

Homeostasis of calcium ions is regulated by parathyroid hormone and thyroid hormone (calcitonin). When the level of calcium ions in the blood plasma decreases, the secretion of parathyroid hormone increases, which increases the resorption of these ions in the intestines, reabsorption in the renal tubules, and also

increases the dissolution of mineral substances by osteoclasts in the bone tissue and the influx of calcium ions into the blood.

When the concentration of calcium ions in the blood increases, calcitonin is secreted, which causes a decrease in the content of these ions due to the fixation of calcium in bone tissue and calciuria.

Calciferol participates in the regulation of the amount of calcium ions in the blood plasma, which ensures the synthesis of specific calcium-binding proteins necessary for the absorption of calcium in the intestines and its reabsorption in the kidneys. In addition to the listed hormones, thyroxine, androgens (increase) and glucocorticoids (decrease) can change the level of calcium ions in the blood.

An increase in the content of calcium in the blood (hypercalcemia) is observed in the last period of pregnancy, with parathyroidism, leukemia, destructive processes in bone tissue, hypervitaminosis D, alimentary hypercalcemia, Addison's disease, Cushing's syndrome, acromegaly, gangrene, peritonitis, jaundice, heart failure, physiological hypercalcemia babies (after 4 days of life), in premature babies.

A decrease in the amount of calcium in the blood (hypocalcemia) is noted during pregnancy, vitamin D deficiency (rickets), insufficient function of the parathyroid glands, spasmophilia, kidney diseases (nephritis, nephrosis), fluoride poisoning, diarrhea, acute pancreatitis. Hyperreflexia and tetany are observed when the calcium content in blood plasma drops below 1.8-1.5 mmol/l.

The concentration of inorganic phosphates in whole blood and serum is 1-1.5 mmol/l.

Phosphorus is concentrated mainly in the skeleton - bones and teeth (90%) and intracellularly. As the main intracellular anion, phosphorus is included in the composition of phosphoric esters, coenzymes, proteins, nucleic acids, lipids, etc. In bones, phosphorus is represented by poorly dissolved calcium phosphate (2/3) and soluble compounds (1/3).

An increase in the amount of phosphates in the blood (hyperphosphatemia) is most pronounced in nephritis and nephrosis (an unfavorable prognostic sign), is observed in hypoparathyroidism, acromegaly, hypervitaminosis D, bone diseases (fractures in the healing stage), myeloma, diabetes, spasmophilia, Cushing's disease, Addison's disease illness, toxicosis of pregnancy, heavy muscle work.

A decrease in the concentration of phosphorus in the blood (hypophosphatemia) is noted in hyperparathyroidism, hyperinsulinism, myxedema, poor nutrition, impaired absorption of phosphates in the intestines, vitamin D rickets in the early stage (in children). A decrease in phosphorus concentration in the blood is considered to be below 1 mmol/l.

Magnesium participates in most enzymatic processes, is a cofactor and activator of enzymes. Magnesium ions are necessary for the process of

neuromuscular disorders, while magnesium is in synergy with calcium and antagonism with potassium, and potassium plays a leading role.

The content of magnesium in blood plasma is 0.74-1.23 mmol/l. 5-75% of it is presented in an ionic form, the rest is bound to proteins.

An increase in the content of magnesium in the blood (hypermagnesemia) is observed in anuria, chronic renal failure, hypothyroidism, diabetic acidosis, hypertension, atherosclerosis.

A decrease in the concentration of magnesium in the blood (hypomagnesemia) is noted in cancer, toxemia in pregnant women, chronic heart failure, starvation, diabetes, metabolic (hypokalemic) alkalosis, acute and chronic pancreatitis, hyperthyroidism, chronic alcoholism, rickets.

Iron is part of hemoglobin, its content is 60-70% (48.2 mmol/l, 86-120 μg%). Blood serum contains 6-29 μmol/l of iron. Myoglobin, a protein that supplies muscles with oxygen, contains 3-5% of all iron in the body. The part of iron-containing enzymes accounts for 0.1-0.2%, or 0.14 mmol (6-8 mg), of all body iron. Iron reserves make up 30-40% (18.0-26.8 mmol, or 1.0-1.5 g) of the body's total iron and is in the form of ferritin and hemosiderin.

An increase in the amount of iron in the blood is characteristic of pernicious anemia, increased hemolysis of erythrocytes, sideroachrestic anemia, erythremia, hemochromatosis, ingestion of iron preparations.

A decrease in the level of iron in the blood is observed in hypochromic anemia, acute and chronic blood loss, purulent and septic diseases, some conditions with an increased need for iron (pregnancy, acute infectious diseases).

Iodine is a vital element. It is part of the structure of the thyroid hormone thyroxine, and affects the synthesis of proteins and fats.

The total content of iodine in the body is approximately 0.4 mmol (50 mg), and the thyroid gland contains 30% of this amount in the form of organic compounds and 1% in the form of inorganic compounds. Iodine is also found in the adrenal glands, liver, kidneys, ovaries, and skin. 550-1200 nmol/l of iodine in the form of iodides is determined in the blood. The content of protein-bound iodine in the blood plasma is 315.2-630.4 nmol/l.

An increase in the concentration of iodine in the blood is found in hyperfunction of the thyroid gland, leukemia.

A decrease in the level of iodine in the blood is found in hypoproteinemia, nephrotic syndrome, dystrophy, hypofunction of the thyroid gland - endemic goiter, cretinism, myxedema.

TESTS FOR SELF-CONTROL

1 . The number of globulins in blood serum is (in g/l):

A. 20-23
B. 23-35
C. 35-50
D. 65-85
E. 11-16

2. The amount of gamma globulins in the blood serum is (in g/l):

A. 2-5
B. 1,2-2,2
C. 5-16
D. 4-12
E. 20-25

3. Total bilirubin is (in mmol/l):

A. 20-40
B. 1,7-20,5
C. 1,7-17,1
D. 0,86-5,1
E. 20,5-28,1

4. Indirect bilirubin is normal (in μmol/l):

A. 20-40
B. 1,7-20,5
C. 1,7-17,1
D. 0,86-5,1
E. 5. 20,5-28,1

5. Normal blood cholesterol is:

A. 3,35-6,26
B. 2-2,5
C. 2,97-8,79
D. 8,79-12
E. 2-4

6. Blood glucose level (in mmol/l):

A. 3,1-5,5
B. 4,4-6,6
C. 6,6-12,2
D. 5,1-8,4
E. 6,6-7,1

7. Normal blood iron is (in μmol/l):

A. 6-26
B. 13-30
C. 3,8-5,2
D. 2,0-2,5
E. 10-20

8. Normal blood urea is (in mmol/l):

A. 4,2-8,3
B. 14,28-28,56
C. 3,33-0,132
D. 0,165-0,389
E. 0,062-0,132

9. Normal blood creatinine is (in mmol/l):

A. 0,0008-0,0028
B. 14,28-28,56
C. 3,3-8,32
D. 0,050-0,115
E. 0,165-0,389

10. Potassium in the blood plasma is (in mmol/l):

A. 13-30
B. 3,6-6,3
C. 134-169
D. 97-108
E. 2-2,5

Norms of laboratory biochemical indicators of a healthy person

Indicators of protein metabolism	
Blood serum proteins	65-85 g/l
albumins	35-45 g/l
α1-globulins	1-4 g/l (3-6%)
α2-globulins	4-12 g/l (7-13%)
β-globulins	5-11 g/l (8-14%)
γ-globulins	5-16 g/l (15-22%)
Hemoglobin	men 132-164 g/l; women 115-145 g/l
C-reactive protein	does not appear
Non-protein nitrogenous components of blood	
blood nitrogen	14-28 mmol/l (0.2-0.4 g/l)
creatinine	blood 50-115 μmol/l; urine 4.42-17.6 mmol/day
urea	blood 4.2-8.3 mmol/l; urine 330-580 mmol/l
uric acid	men's blood 214-458 μmol/l; a woman's blood 149-404 μmol/l; urine 2.4-6.0 mmol/l
Enzyme composition of blood	
α-amylase	blood 12-32 mg/h*ml; urine - up to 64 g/h*l
aspartate aminotransferase (AST)	0.1-0.45 μmol/h*ml
alanine aminotransferase (ALT)	0.1-0.68 μmol
creatine phosphokinase (CPK)	50-417 ncat/l; 25 ME
lactate dehydrogenase (LDH)	0.8-4.0 μmol/h*ml (LDH1-17-27%; LDH2- 27-37%; LDH3- 18-25%; LDH4-3-8%; LDH5-0-5%)
trypsin	0-50 ncat/l (300-600 IU)
α-antitrypsin	5.1-10.2 μcat/l (0-3 IU)
cholinesterase	77000-240000 nmol/(s*l)
alkaline phosphatase	men - 0.9-2.3 micrometers/l; women – 0.7-2.1 micrograms/l; children under 14 years of age 1.2-6.3 micrograms/l
renin	0.03-0.06 μmol/l/min 0.6-1.0 nmol/l/s
Pigment metabolism	
total bilirubin	8.5-20.5 μmol/l
indirect	6.5-15.4 μmol/l
direct	2.1-5.1 μmol/l
Carbohydrate metabolism	
glucose	3.1-5.5 mmol/l
pyruvic acid	0.05-0.14 mmol/l (90.3-0.9 mg/100ml)
lactic acid	0.55-2.22 mmol/l (5-20 mg/100 ml)
Lipid metabolism	
cholesterol	3.35-6.26 mmol/l
triglycerides	0.7-1.7 mmol/l
LP lipoproteins	
α-LP (HDL)	>0.9 mmol/l
β-LDL (LDL)	1.68-4.53 mmol/l
pre-β-LP (VLDL)	0.26-1.04 g/l
Mineral metabolism	
sodium	135-150 mmol/l
potassium	3.6-6.3 mmol/l
chlorine	95-110 mmol/l
calcium	2.25-3.0 mmol/l
phosphorus	1-1.5 mmol/l
iron	6-29 μmmol/l
iodine	in the blood 550-1200 nmol/l

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