ASSESSMENT FUND

for discipline «Clinical biochemistry»

The level of higher education

SPECIALTY

Direction of preparation

Specialisation 31.05.01. - General medicine

(the code and Direction of preparation)

2024

The assessment fund is designed to control the knowledge of students in the direction of preparation (specialisation) ***General medicine*** in the discipline (practice) "Clinical biochemistry".

The assessment fund is reviewed and approved at the meeting of the department
Chemistry and Biochemistry

Record of 25.10. 2024. № 2

The Head of Department
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1. STUDENTS’ COMPETENCIES RESULTING FROM THE COURCE UNIT (MODULE)

Formation of competence	Planned learning outcomes in the discipline, characterizing the stages of competencies formation	Types of assessment tools/ section code in this document
MPC-9: ability to assess morphofunctional, physiological states and pathological processes in the human body to solve professional problems	**Knowledge:** - goals and main objectives of clinical biochemistry; - characteristics of diagnostic significance of laboratory studies, the main quality indicators of the laboratory diagnostics; - features of changes in the biochemical composition of biological fluids with the most common human diseases, the main biochemical markers of human pathological conditions; - general principles and features of diagnosis of hereditary diseases and congenital anomalies.	**Block A, D -** tasks of the reproductive level - *Oral discussion* - *Control work* - *Control Test* - *Record (oral answer)*
	Skills: - To choose the best analytical method of clinical research; to send for laboratory and instrumental examination, for consultation with specialists. - To interpret the results of laboratory studies of biological material for the diagnosis of basic human pathological conditions.	**Block B, D** - tasks of the reconstructive level: - *Solving situational cases* - *Report*
	Expertise: - skills of effective use of laboratory data to determine the presence of a disease, the effectiveness of its treatment. - the algorithm of the preliminary diagnosis, followed by an additional examination	**Block D** – practice-oriented and/or research level assignments - *Solving situational cases*

2. The planning sheet of discipline

Discipline: Clinical biochemistry
Area/specialization: General Medicine
Course/semester: **5/9**
Credit units (CU): 2

Title of module according to WPD	Type of control	Forms of control	Minimal credit points	Maximal credit points	Week of control
Module 1					
Module 1. Metabolic disorder. Biochemical indicators of metabolic disorders in the human body.	Formative assessment	Activity, attendance, conspectus, performance and presentation of lab works, individual work with tables, discussion of situational tasks	10	20	5
	Midterm examination	Evaluation test	10	15	
Module 2					
Module 2. The mechanisms of the metabolic processes disorders in cells	Formative assessment	Activity, attendance, conspectus, performance and presentation of lab works, individual work with tables, discussion of situational tasks, writing of reports	10	20	8
	Midterm examination	Evaluation test	10	15	
Total for semester			**40**	**70**	
Midpoint assessment			20	30	
Summative assessment			60	100	

3. STANDARD CONTROL TASKS AND OTHER MATERIALS IMPORTANT FOR EVALUATING PLANNED LEARNING RESULTS IN THE DISCIPLINE “BIOCHEMISTRY” (ASSESSMENT TOOLS)

Block A

A.0 Fund of tests for the discipline.

Clinical biochemistry and pathochemistry of nitrogen metabolism

1. The laboratory symptom “creatorrhoea” characterizes:
A) increased content of muscle fibers in feces
B) increased content of neutral fat in feces
C) increased levels of lipase in feces
D) remains of undigested food in the stool

2. The synthesis of uric acid inhibits by:
A) aspirin
B) insulin
C) allopurinol
D) voltaren

3. Localization of arthritis during exacerbation in the first metatarsophalangeal joint is typical for:
A) psoriasis
B) rheumatoid arthritis
C) gout
D) Reiter's disease

4. The mechanism of ammonia neutralization includes:
A) urea synthesis
B) the formation of glutamine
C) formation of ammonium ion
D) all of the above

5. The end products of protein decay are:
A) indole
B) skatole
C) phenol
D) all of the above are true

6. Enzymes and zymogens (proenzymes) are synthesized in the acinar cells of the pancreas. This proenzyme does not have enzymatic activity, but turns into an active enzyme when the peptide bond between arginine-15 and isoleucine-16 is cleaved under the action of trypsin. Identify this zymogen.
A) Chymotrypsinogen;
B) Pepsinogen;
C) Trypsinogen;
D) Procarboxypeptidase;
E) Proelastase.

7. When an ulcer penetrates into the pancreas, the level of which compound increases in the blood:
A) glucose

B) lipases
C) amylase
D) alkaline phosphatase

8. Tophuses are:
A) deposition of urates in tissues
B) deposition of cholesterol in tissues
C) osteophytes
D) compaction of subcutaneous tissue

9. Localization of arthritis during exacerbation in the first metatarsophalangeal joint is typical for:
A) psoriasis
B) rheumatoid arthritis
C) gout
D) Reiter's disease

10. Creatine is found in highest concentration in:
A) liver
B) muscle tissue
C) thyroid gland
D) nervous tissue

11. The end products of the metabolism of purine nitrogenous bases are:
A) urea
B) creatinine
C) uric acid
D) all of the above

12. Uric acid synthesis inhibits by:
A) voltaren
B) allopurinol
C) retabolil
D) insulin

13. The inclusion of protein foods leads to:
A) reducing the acidity of gastric contents
B) increased acidity of gastric contents
C) increasing the level of pepsin in the gastric contents
D) reducing the amount of mucus produced in the stomach

14. The main component of residual nitrogen is:
A) glucose
B) acetone
C) glycerin
D) urea

15. What effect does a low-fat diet have on protein metabolism?
A) Has no effect;
B) The excretion of amino acids and their breakdown products in the urine increases;
C) The excretion of amino acids and their breakdown products in the urine decreases;
D) Protein synthesis increases;
E) There is no correct answer.

Clinical biochemistry of the liver and pigment metabolism

1. The presence of cholestasis is indicated by:
A) increased alkaline phosphatase, bilirubin, cholesterol

B) increase in aspartic and alanine transaminases, glucose
C) decrease in prothrombin, hemoglobin, fibrinogen
D) increase in amylase, total protein, creatinine

2. A biochemical test characteristic for the early diagnosis of viral hepatitis is:
A) protein fractions of blood
B) prothrombin index
C) ALT
D) cholesterol level

3. If there is a threat of hepatic coma, the diet should be limited:
A) proteins
B) carbohydrates
C) salt
D) liquid

4. Free bilirubin in the liver includes in:
A) decarboxylation
B) connection with glucuronic acid
C) connection with proteins
D) hydrolysis

5. In severe liver damage, hyperaminoaciduria is observed due to the release of:
A) Cysteine;
B) Methionine;
C) Tyrosine;
D) All answers are correct;
E) There is no correct answer.

6. When the bile duct is blocked, the following condition occurs:
A) hepatocellular failure
B) cholestasis
C) jaundice of newborns
D) hemolytic jaundice

7. A laboratory sign of a violation of the synthetic function of the liver is:
A) hypoprothrombinemia
B) hypertriglyceridemia
C) haptoglobulinemia
D) hypoenzymemia

8. Cytolysis of hepatocytes in viral hepatitis is reflected by the following biochemical tests:
A) cholesterol level
B) level of total protein and protein fractions of blood
C) level of alanine aminotransferase and asparagine aminotransferase
D) alkaline phosphatase

9. Which markers indicated on cholestasis:
A) increased alkaline phosphatase, bilirubin, cholesterol
B) increase in aspartic and alanine transaminases, glucose
C) decrease in prothrombin, hemoglobin, fibrinogen
D) increase in amylase, total protein, creatinine

10. The activity of γ-glugamyltransferase (GGT) increases significantly in serum when:
A) liver diseases and alcoholism
B) bone damage
C) prostate cancer
D) myocardial infarction

11. Select a protein that takes part in the detoxification of heavy metals in liver cells:
A) Ferritin;
B) Metalloporphyrin;
C) Cytochrome P_{450};
D) Hemosedirin;
E) Metallothionein.
12. If there is a threat of hepatic coma, the diet should be limited:
A) proteins
B) carbohydrates
C) salt
D) liquid
13. An increase in aminotransferase activity is observed with:
A) liver diseases
B) myocardial infarction
C) severe hypoxia and tissue compression
D) all of the above reasons
14. Select a protein that takes part in the detoxification of heavy metals in liver cells:
A) Ferritin;
B) Metalloporphyrin;
C) Cytochrome P_{450};
D) Hemosedirin;
E) Metallothionein.
15. Bilirubin metabolism is carried out according to the scheme. Choose the correct sequence:
A) Biliverdin-urobilin-urobilinogen-conjugate with glucuronic acid - stercobilin;
B) Biliverdin - conjugate with glucuronic acid-urobilinogen-stercobilin-urobilin;
C) Urobilin - biliverdin - conjugate with glucuronic acid - stercobilin - biliverdin;
D) Biliverdin - stercobilin conjugate with glucuronic acid - urobilin - urobilinogen;
E) Urobilinogen - biliverdin - stercobilin - conjugate with glucuronic acid - urobilin

Clinical biochemistry and pathochemistry of lipid metabolism, biochemical diagnosis of myocardial infarction
1. The most characteristic sign of lipid metabolism disorders in diabetes mellitus is an increase in the level of:
A) triglycerides
B) total cholesterol
C) low density lipoproteins
D) high density lipoproteins
2. Reducing the energy value of the diet for obese patients is carried out by reducing:
A) carbohydrates
B) proteins
C) fats
D) proteins, fats and carbohydrates
3. Low-density lipoproteins include:
A) apolipoprotein B-100
B) apolipoprotein A
C) apolipoprotein B-48
D) apolipoprotein C
4. Serum turbidity is caused by excess:

A) cholesterol
B) TAG
C) bilirubin
D) phospholipids
5. A group of natural compounds under the general name “tocopherols” (vitamin E group) are bioantioxidants and protect lipid fragments of cells from oxidation. What is the chemical nature of this function?
A) Oxidation to retinals, and then to retinoic acids;
B) Possibility of complex formation with a variety of cationic particles;
C) The presence of a polyisoprenoid chain that ensures liposolubility;
D) Lipophilicity of the carbon skeleton and the tendency to oxidize the hydroquinone fragment;
E) Donation of methyl groups for various biochemical reactions.
6. Reactive oxygen species are especially dangerous for:
A) Neutrophils;
B) Red blood cells;
C) Eosinophils;
D) Monocytes;
E) Mast cells.
7. The TAG level, which is a risk factor for the development of cardiovascular diseases, is ______mmol/l:
A) 1.0
B) 1.2
C) 1.7
D) 0.7
8. Changes in the blood characteristic of the acute stage of myocardial infarction are:
A) increase in amylase activity
B) increase in troponin levels
C) decrease in de Ritis coefficient
D) increase in gamma-glutamyltransferase activity
9. Sources of polyunsaturated fatty acids include:
A) meat
B) olive oil
C) dairy products
D) green vegetables
10. The pathology of lipid metabolism includes:
A) hyperbilirubinemia
B) glucosuria
C) hyperproteinemia
D) hyperlipemia
11. Neutral fats are synthesized along the main reaction pathways. In which reaction the glycerol formed during the breakdown of triacylglycerols is included?
A) Phosphorylation;
B) Oxidation;
C) Acylation;
D) Alkylation;
E) Methylation.
12. Hormones that regulate lipid metabolism are:
A) adrenaline
B) insulin

C) glucocorticoids
D) all of the above
13. Type IIa of hyperlipidemia according to the WHO classification is characterized by an increase in blood plasma:
A) LDL and VLDL
B) VLDL
C) LDL
D) LP (a)
14. Optimal cholesterol concentration for healthy individuals:
A) up to 8 mmol/l
B) up to 6 mmol/l
C) up to 7 mmol/l
D) up to 5 mmol/l
15. Atherogenic lipoproteins do not include:
A) VLDL
B) HDL
C) LDL
D) chylomicrons

Clinical biochemistry and pathochemistry of carbohydrate metabolism. Diabetes
1. The diagnosis of diabetes mellitus is established when the level of glycated hemoglobin is ______%
A) ≥6.0
B) ≥6.5
C) ≥6.8
D) ≥7.0
2. Serum insulin level is often increased:
A) with type 1 diabetes mellitus
B) with type 2 diabetes mellitus
C) after stimulation with glucose in type 1 diabetes mellitus
D) after stimulation with glucose in type 2 diabetes mellitus
3. An early metabolic marker of type 2 diabetes mellitus is:
A) disappearance of the early phase of insulin secretion
B) high fasting glucose levels
C) the presence of frequent episodes of hypoglycemia at night
D) ketonuria
4. The normal blood sugar level during glucose tolerance test after 2 hours of its administration is no more than _____mmol/l:
A) 7.8
B) 6.8
C) 7.0
D) 11.0
5. To diagnose impaired glucose tolerance, it is necessary to:
A) determination of insulin in the blood
B) glucose tolerance test
C) determination of glucosylated hemoglobin
D) all of the above are incorrect
6. Glycosylated hemoglobin:
A) is constantly present in the blood in small quantities

B) increases in the blood in insulin-dependent diabetes
C) increases in the blood in non-insulin-dependent diabetes
D) all of the above are true

7. In June 2017 a level of HbA1c of 46-year-old patient was 4%, in October of the same year, after examination, it turned out that HbA1C was 11%, based on which you suspect:
A) type I diabetes;
B) type II diabetes;
C) Impaired glucose tolerance;
D) This is a variant of the norm;
E) Correct answers B and C.

8. The maximum blood sugar level during glucose tolerance test after 2 hours of its administration is no more than ______mmol/l:
A) 5.5
B) 6.1
C) 7.8
D) 6.4

9. Type 2 diabetes mellitus develops due to:
A) autoimmune destruction of pancreatic beta cells
B) genetically determined defects in pancreatic beta cell function
C) insulin resistance and relative insulin deficiency
D) tumors or injuries of the pancreas

10. Glucagon is produced by:
A) δ-cells of the pancreas
B) β-cells of the pancreas
C) L-cells of the gastrointestinal tract
D) α-cells of the pancreas

11. When glucosuria is detected, first of all it is necessary to:
A) determine fasting blood glucose levels
B) prescribe sulfonylurea drugs
C) limit your carbohydrate intake
D) determine the level of basal insulin

12. Ketone bodies in the blood cause:
A) alkalosis
B) acidosis
C) glucosuria
D) polyuria

13. When a glucose solution is administered to patients with impaired renal function, “water poisoning” is observed, sometimes leading to death. Explain the reason:
A) There is an isotonic increase in fluid volume due to a decrease in the concentration of proteins in plasma;
B) Hypotonic accumulation of water occurs in all cell compartments without an equivalent amount of salts;
C) There is a hypertonic increase in fluid volume due to the accumulation of sodium ions inside the cells;
D) There is a hypotonic decrease in fluid volume due to insufficiency of the adrenal cortex;
E) An isotonic decrease in fluid volume occurs due to large losses of sodium ions.

14. After abnormally large sweat production, not compensated by adequate water intake, and in persons with diabetes mellitus, fluid loss occurs in the cell and extracellular space. Describe the type of water metabolism disorder:

A) Isotonic volume reduction;
B) Hypotonic volume increase;
C) Hypertensive volume reduction;
D) Hypotonic volume reduction;
E) Isotonic volume increase.

15. Insulin is produced by the __________pancreas:
A) α-cells
B) β-cells
C) δ-cells
D) PP cells

Clinical biochemistry of acid-base status

1. The constancy of the acid-base state is maintained by:
A) lymph
B) kidneys
C) bone tissue
D) myocardium

2. What is reacts most quickly to changes in blood pH
A) renal system
B) pulmonary ventilation
C) bone tissue
D) Liver
E) carbonate buffer

3. Increased production of lactic acid in tissues occurs
A) with iron deficiency anemia
B) with alcohol poisoning
C) due to stress
D) with renal failure
E) in case of circulatory disorders in tissues

4. The cause of metabolic acidosis in renal failure is
A) decrease in tubular secretion of H+ ions
B) reduction in the number of working nephrons
C) deterioration in the reabsorption of Na+ ions
D) decrease in H_2CO_3 excretion
E) violation of ammoniogenesis

5. The source of acids in cell metabolism are reactions
A) oxidation of sulfur-containing amino acids
B) anaerobic glycolysis
C) oxidative phosphorylation
D) glycogen synthesis
E) Transamination

6. The concentration of hydrogen ions in the blood and intercellular space is about 40 nM, which corresponds to a pH value of 7.4. Indicate the exogenous source of protons:
A) Ascorbic acid, methionine;
B) Cholesterol, α-tocopherol;
C) Thiamine pyrophosphate, pyridoxal phosphate;
D) Maltose, lactose;
E) Lysine, arginine.

7. Ammoniogenesis is

A) synthesis of urea in the liver
B) NH_3 binding in tissues
C) deamination of glutamate in the liver
D) neutralization of ammonia in nervous tissue
E) formation of NH_4^+ ions in the kidney tubules

8. The source of phosphoric acid in body fluids is metabolism of:

A) nucleic acids
B) Phospholipids
C) Homopolysaccharides
D) Proteins
E) hyaluronic acid

9. Amino acid groups are involved in the regulation by the protein buffer system

A) $–CH_3$
B) $–NH_2$
C) $–C_6H_6$
D) –SH
E) –OH

10. The source of acids in cell metabolism are reactions of:

A) aerobic metabolism
B) Glycogenolysis
C) oxidative phosphorylation
D) cholesterol synthesis
E) Ketogenesis

11. The pH value is an indicator that depends on the concentration of H^+ ions:

A) as an arithmetic progression
B) as a geometric progression
C) as a parabolic function
D) Linear
E) Logarithmically

12. The HbOsat indicator (SO2, hemoglobin saturation with oxygen) shows the ratio of the fraction of oxygenated hemoglobin:

A) to carboxyhemoglobin
B) to carbohemoglobin
C) to "workable" blood hemoglobin
D) to glycosylated hemoglobin in the blood
E) to non-oxy- and non-carbo-hemoglobin

13. What is not refer to acids which are formed in the cell:

A) uric acid
B) Urea
C) lactic acid
D) Glutamine
E) carbonic acid

14. The pH limits compatible with life is the range:

A) 6.8-7.8
B) 7.6-8.2
C) 6.1-8.8
D) 7.3-7.7
E) 5.5-6.5

15. An increase in lactic acid production in tissues occurs when

A) as a result of anemia
B) with thrombosis
C) due to renal failure
D) against the background of hyperventilation
E) with increased blood viscosity

Clinical biochemistry of water-salt metabolism
1. Violation of the concentration function of the kidneys is characterized by:
A) proteinuria
B) leukocyturia
C) hyposthenuria
D) hematuria
2. Minerals in the body are contained in the form of:
A) insoluble salts
B) ions
C) bound to proteins
D) in all of the above forms
3. The ultrafiltrate of primary urine does not contain proteins whose molecular weight is higher
A) 50000 D
B) 20000 D
C) 15000 D
D) 100000 D
4. Glucosuria is observed when the blood glucose level increases above
A) 5.55 - 6.0 mmol/l
B) 8.3 - 8.8 mmol/l
C) 9.6 - 10.3 mmol/l
D) Above 10 mmol/l
5.Oliguria is observed at:
A) chronic nephritis
B) acute nephritis
C) diabetes insipidus
D) diabetes mellitus
6. Which chemical compound reflects the kidneys condition:
A) uric acid
B) urea
C) residual nitrogen
D) creatinine
7. The main potential-forming ion is:
A) calcium
B) potassium
C) sodium
D) chlorine
8. All substances in primary urine are divided into
A) threshold
B) non-threshold
C) penetrating
D) Non-penetrating
9. What stones form in acidic urine?

A) oxalate
B) phosphate
C) urates
D) Arbonate
10. A decrease in urine pH is observed when:
A) fasting
B) vegetarian diet
C) diabetes mellitus
D) diabetes insipidus
11. In case of chronic renal failure in the pathogenetic treatment of anemia, the following is used:
A) iron supplements
B) erythropoietin
C) red blood cell transfusion
D) vitamin B12
12. The main ion that determines the osmotic transport of water through cell membranes:
A) calcium
B) potassium
C) sodium
D) chlorine
13. Which types of transmembrane transport are included in reabsorption?
A) simple diffusion
B) facilitated diffusion
C) active transport
D) vesicular transport
14. False proteinuria is observed in pathology of:
A) kidney
B) adrenal glands
C) urinary tract
D) Glomerular apparatus
15. An increase in urine pH is observed when:
A) fasting
B) vegetarian diet
C) diabetes mellitus
D) diabetes insipidus

Clinical biochemistry of vitamin metabolism disorders
1. Vitamin K-dependent coagulation factors include:
A) first, fifth, sixth, eighth
B) second, seventh, ninth, tenth
C) first, third, fourth, sixth
D) third, fourth, eleventh, twelfth
2. A single dose of vitamins A and D is sufficient to cover the need for several weeks. Why do you need to take B vitamins more often?
A) High solubility leads to their rapid elimination from the body;
B) Low accumulation in tissues and cells requires a higher concentration from the outside;
C) Low concentration in cells and tissues leads to loss of biological activity of vitamins;
D) Low concentrations of vitamins do not cover the needs of enzymes in coenzyme structures;

E) There are no special mechanisms that allow the retention of B vitamins in the body as well as vitamins A and D.

3.Lack of which vitamin leads to increased permeability of cell membranes:

A) N

B) D3

C) E

D) K

E) B1

4.Which disorder is not typical for C-deficiency:

A) Disruption of mineralization processes in supporting tissues

B) Degeneration of odontoblasts with the development of caries and tooth loss

C) Impaired collagen synthesis in bones and dental tissues

D) Impaired synthesis of purine and pyrimidine nucleotides

E) Impaired synthesis of connective tissue proteoglycans

5.Which of the following vitamins is not synthesized by the human intestinal microflora:

A) C

B) H

C) PP

D) B_{12}

E) B_9

6.The main place of absorption of vitamin B_{12} is:

A) distal jejunum

B) proximal ileum

C) proximal jejunum

D) distal ileum

7. The following vitamin is involved in the decarboxylation reactions of pyruvic acid, 2-oxoglutaric acid and other branched keto acids:

A) Riboflavin;

B) Thiamine;

C) Cobalamin;

D) Pantothenic acid;

E) Folic acid.

8.Which vitamin is responsible for the absorption of Ca^{2+} and phosphates from the intestines:

A) D

B) E

C) B_1

D) B_2

E) B_{12}

9.Which vitamins mutually enhance the effect of reducing capillary fragility and permeability:

A) P and C

B) A and E

C) K and PP

D) B_6 and B_2

E) H and B_3

10.Which disorder is not typical for C-vitaminosis:

A) Disruption of mineralization processes in supporting tissues

B) Degeneration of odontoblasts with the development of caries and tooth loss

C) Impaired collagen synthesis in bones and dental tissues
D) Impaired synthesis of purine and pyrimidine nucleotides
E) Impaired synthesis of connective tissue proteoglycans

11. During pregnancy, the use of which synthetic vitamins may lead to the development of defects in the child:

A) A
B) B_1
C) B_2
D) C

12.Which vitamins belong to the fat-soluble group:

A) PP, riboflavin, folate, B_{12}
B) B_6, P, cobalamin, ascorbate
C) Biotin, B_9, niacin, B_2
D) B_1, C, pyridoxine, thiamine
E) K, tocopherol, A, calciferol

13.Which vitamin is a biological antioxidant:

A) B1
B) F
C) E
D) B_5
E) D

14. Which vitamin deficiency leads to the development of polyneuritis:

A) Thiamine
B) Ascorbic acid
C) Cobalamin
D) Folic acid
E) Riboflavin

15.Which vitamins mutually enhance the effect of reducing capillary fragility and permeability:

A) P and C
B) A and E
C) K and PP
D) B_6 and B_2
E) H and B_3

Clinical biochemistry of the development of oncological processes

1. Multiple myeloma is characterized by an increase in total protein levels due to:

A) albumin
B) gamma globulin
C) paraprotein
D) alpha globulin

2. Select the incorrect mechanism of avoiding of tumor tissue from immunological surveillance:

A) The appearance of heteroorganic antigens in tumor tissue does not cause an immunological reaction due to the fact that there is a immunological tolerance;
B) Masking of tumor antigens. Thus, chorionepithelioma cells have a neutral polysaccharide capsule;
C) Antigenic determinants are found in some tumors that proactively stimulate T-suppressor cells, which leads to inhibition of the immune response against the tumor;

D) Carcinogenic factors can cause immunosuppression;
E) Creating an overload of the immunological system and suppressing the immune response by tumor tissue after reaching a certain mass

3. Is it true that the majority of spontaneously occurring tumors have low antigenic activity?
A) Yes
B) No

4. Indicate possible causes of tumor recurrence:
suppression of local immune factors
A) penetration of a DNA fragment of a tumor cell into the genome of a normal cell
B) preservation of viable tumor cells after its removal
C) penetration of a fragment of “tumor” RNA into a normal cell

5. Is it true that the Pasteur effect is inhibited in tumor tissue?
A) Yes
B) No

6. Confirmation of the greatest importance for an irradiated cell is:
A) nucleic acids
B) proteins
C) lipopolysaccharides
D) polysaccharides

7. A tumor is characterized by: 1-Increased water content; 2-Decreased potassium; 3-Decreased calcium; 4-Increasing magnesium; 5-Increasing the content of hydrogen ions.
A) 1, 3, 5;
B) 3, 5;
Cc) 1, 3, 4, 5;
D) 1, 2, 5;
E) 2, 3, 5.

8. Indicate the factors inhibiting cell division:
A) cAMP
B) growth factors
C) decrease in cell surface tension
D) keylons
E) cGMP

9.What are oncoproteins?
A) proteins that stimulate tumor progression
B) proteins that block cellular respiration
C) proteins that inhibit glycolysis
D) proteins that cause tumor transformation of normal cells

10.What factors are aimed at destroying tumor cells in the body?
A) macrophage phagocytosis
B) allogeneic inhibition
C) Killer T lymphocytes
D) “natural killers” NK

11. The main cytochemical marker of acute myeloblastic leukemia is a positive reaction to:
A) myeloperoxidase
B) ß-glucuronidase
C) ATPase
D) acid phosphatase

12. Tumor markers are

A) proto-oncogenes
B) proteins synthesized in tumor cells
C) Antioncogenes

13. Is it true that skin cancer is less common in people of the Negroid race than in people of the white race?

A) yes
B) no

14. An increase in plasma concentrations of alpha-fetoprotein may indicate the presence

A) colon tumors
B) stomach tumors
C) liver tumors
D) trophoblast tumors

15.What metabolic changes are characteristic of malignant tumor cells?

A) increased glucose uptake, weakened anaerobic glycolysis
B) activation and qualitative changes in protein synthesis
C) increased uptake of cholesterol and higher fatty acids,
D) hypohydration of tumor tissue
E) absence of qualitative changes in protein metabolism
F) activation of nucleic acid metabolism

A.1 Questions for discussion:

Module 1. Metabolic disorders. Biochemical indicators of metabolic disorders in the human body.

Topic 1. Determination of proteins and indicators of protein metabolism. Protein homeostasis. Blood plasma proteins, diagnostic value of their determination.

1.1 Requirement and nutritional value of proteins. Nitrogen balance. The main stages of protein digestion.

1.2 Hydrochloric acid of gastric juice: role, mechanism of biosynthesis, regulation. Causes and consequences of hypo- and hyperchlorhydria. Rotting of proteins in the intestines. Neutralization of rotting products.

1.3 Biological role of proteins. Total protein and protein fractions of blood serum: quantitative and qualitative characteristics, role, clinical and diagnostic significance. Method for determining total serum protein.

1.4 Amino acid pool of blood. Pathways for the formation and utilization of amino acids. The role of the liver in amino acid metabolism.

1.5 Decarboxylation of amino acids. Formation, role and neutralization of biogenic amines.

1.6 Formation and neutralization of ammonia in tissues. Ammonia toxicity. Biosynthesis of urea. Diagnostic value of determining urea content in blood and urine.

1.7. Residual blood nitrogen and total urine nitrogen. Definition, components. Diagnostic value of determining residual nitrogen and its components.

1.8 Exchange of phenylalanine and tyrosine. Hereditary metabolic disorders (causes, biochemical mechanisms of development of clinical manifestations, diagnosis, principles of correction).
1.9 Decay of purine nucleotides. Violation of purine nucleotide metabolism - gout, Lesch-Nyhan syndrome; biochemical defect, clinical manifestations, diagnosis. Ways to correct hyperuricemia.
1.10 Synthesis of pyrimidine nucleotides, regulation. Use of thymidylate synthase reaction inhibitors in clinical practice. Decomposition of pyrimidine nucleotides. Use of final products.

Topic 2. Clinical diagnostic assessment of carbohydrate metabolism.
2.1. The main representatives of carbohydrates in food. Biological role of carbohydrates. Digestion and absorption of carbohydrates. Characteristics of enzymes.
2.2. Congenital disorders of digestion and absorption of carbohydrates.
2.3. Glycogen mobilization. Sequence of reactions. Regulation. Glycogen store diseases. Causes, biochemical disorders and clinical manifestations.
2.4. Biosynthesis of glycogen from glucose: sequence of reactions, regulation.
2.5. Glucose pool. Neurohumoral regulation of blood glucose. Hypo- and hyperglycemia, the causes of their occurrence.
2.6. Pathochemistry of diabetes mellitus. The main disorders of carbohydrate, protein, lipid and water-salt metabolism, their clinical manifestations.
2.7. Insulin, proinsulin. Physiological effects, mechanism of action.
2.8. Diabetes mellitus: insulin-dependent and non-insulin-dependent.
2.9. Modern diagnosis of diabetes mellitus. Glucose testing, determination of C-peptide, glycosylated hemoglobin, albumin, ketone bodies, triglycerides.
2.10. Complications of diabetes.

Topic 3. *Clinical methods for assessing lipid metabolism. Peroxide lipid oxidation. Biochemical aspects of atherosclerosis. Laboratory tests for typing dyslipoproteinemia. Hereditary pathology of lipid metabolism.*
3.1. Dietary fats. Biological role. Digestion and absorption. Bile acids: biosynthesis and participation in digestion. Characteristics of enzymes. Resynthesis of fats in intestinal cells. The role of chylomicrons in fat metabolism.
3.2. Biosynthesis of fatty acids. Organ and intracellular localization. Conditions required for synthesis. Sequence of reactions, regulatory mechanisms.
3.3. Biosynthesis and use of ketone bodies. The role of ketone bodies. Energy efficiency. Detection of ketone bodies in urine (Legal test).
3.4. Cholesterol. Structure. Biorole. Biosynthesis of cholesterol and its regulation, pathways of utilization, transport in the body.

3.5. Hypercholesterolemia: causes and consequences. The principle of quantitative determination of cholesterol content in blood serum using the cholesterol oxidase method.
3.6. Blood lipoproteins. Classification. Features of the composition, place of synthesis and utilization, functions of various lipoproteins. Atherogenic lipoproteins.
3.7. Modified lipoproteins. The mechanism of their utilization and role in the pathogenesis of atherosclerosis.
3.8. Lipid peroxidation: initiators, substrates, intermediate and final products. Concept of the mechanism, biological role, causes and consequences of activation, diagnostics.
3.9. Antioxidant system: representatives and mechanism of their antioxidant action. Causes and consequences of inhibition of the antioxidant system.
3.10. Sphingolipids. Structure, main function. Glycolipids. Classification. Distribution in the body, biological role. Disorders of glycolipid metabolism.

Topic 4. *Study of pigment metabolism disorders.*
4.1. Represent heme synthesis as a diagram. Name the regulatory enzyme for heme synthesis and its effector.
4.2. Diagram the catabolism of heme. Indicate the localization of each stage of formation of bile pigments.
4.3. Give the characteristics of direct bilirubin (place of formation, solubility in water and lipids, features of transport in the blood, place of further transformations).
4.4. Indicate the normal content of indirect bilirubin in the blood. Name the types of jaundice in which the amount of indirect bilirubin in the blood increases.
4.5. Indicate its normal content in the blood. Name the types of jaundice in which the amount of direct bilirubin in the blood increases.
4.6. Specify the main reason for the development of conditionally physiological jaundice in newborns. Describe the changes in the content of bile pigments in the blood, urine and feces in this type of jaundice.
4.7. Specify the main reason for the development of posthepatic jaundice. Describe the changes in the content of bile pigments in the blood, urine and feces in this type of jaundice.
4.8. Specify the main reason for the development of hepatic jaundice. Describe the changes in the content of bile pigments in the blood, urine and feces in this type of jaundice.
4.9. Specify the main reason for the development of prehepatic jaundice. Describe changes in the content of bile pigments in the blood, urine and feces in this type of jaundice.

4.10. Give the characteristics of indirect bilirubin (place of formation, solubility in water and lipids, features of transport in the blood, place of further transformations).

Topic 5. *Biochemical methods for diagnosing liver diseases and digestive diseases.*
5.1. List the main metabolic functions of the liver.
5.2. List the pathways for the formation of glucose in the liver, name their physiological role.
5.3. Present in the form of a diagram the ways in which glucose is used in the liver, indicate the significance of each process.
5.4. Give examples of blood plasma proteins synthesized in hepatocytes. Indicate their biological role.
5.5. Write the reaction for the formation of mevalonic acid from β-hydroxy-β-methyl-glutaryl-CoA, indicate the enzyme. Explain how and why the rate of this reaction will change with excess cholesterol in food.
5.6. Present as a diagram the synthesis of acetoacetate in liver cells. Name the conditions accompanied by increased ketogenesis.
5.7. List the bile acids synthesized in the liver and their possible conjugates. Describe the biological role of bile acids.
5.8. Name the main stages (phases) of neutralization of toxic products in the liver, indicate the essence of the reactions.
5.9. Write the reaction catalyzed by alcohol dehydrogenase. Diagram the breakdown of ethanol into carbon dioxide and water.
5.10. Explain how and why the rate of gluconeogenesis in the liver will change when taking large amounts of ethanol.

Topic 6. *Acid-base state in normal and pathological conditions. Pathobiochemistry of the kidney. Water-electrolyte metabolism, its regulation and disorders.*
6.1. List the physicochemical properties of urine. Give the values of normal indicators. Explain the dependence of these indicators on the nature of nutrition and water consumption in a healthy person.
6.2. List the nitrogen-containing compounds in normal adult urine. Name the possible reasons for changes in the daily excretion of each of these compounds.
6.3. List the inorganic components of normal adult urine (at least 6). Indicate the possible reasons for changes in the daily excretion of these components.
6.4. List the changes in daily diuresis and urine composition that can be observed a) with an increase in aldosterone secretion, b) with a decrease in aldosterone secretion.
6.5. Name the nitrogen-containing compounds that are absent in the normal urine of an adult, but excreted in the urine under pathological conditions. Indicate the reasons for such violations.

6.6. Describe the mechanisms by which the kidneys maintain a constant pH of the internal environment of the body.
6.7. Give examples of threshold substances in urine. Indicate the immediate cause of the appearance of these substances in the urine and the diseases in which these changes are observed.
6.8. List the compounds whose determination in urine is used to diagnose jaundice. Indicate what changes in the excretion of these compounds are observed in different types of jaundice. How does this change the color of urine?
6.9. Describe the functioning of the renin-angiotensin system and the effect of this system on urine composition and daily diuresis.
6.10. List the pathological conditions in which ketonuria is observed. Specify the immediate cause of ketonuria. Indicate how urine pH changes during ketonuria. Give the names of ketone bodies.

Topic 7. *Vitamins and their clinical use. Hypo- and hypervitaminosis. Vitamin-like compounds.*
7.1. Vitamins. Definition, classification. Functions of vitamins. Sources of intake and stages of metabolism in the body. Causes of a-, hypo- and hypervitaminosis. The concept of antivitamins.
7.2. Vitamin A. Structure. Requirement, sources of intake and metabolism in the body. Metabolic role. Biochemical mechanisms of development of disorders and manifestations of deficiency. Causes and consequences of acute and chronic hypervitaminosis A.
7.3. Vitamin D. Structure. Sources of income. Metabolism, role in the regulation of calcium and phosphorus metabolism. Manifestations of hypervitaminosis D. Pathogenesis of rickets.
7.4. Vitamin E. Structure, properties, sources of supply, daily requirement, biological role. Biochemical mechanisms of development and clinical manifestations of deficiency in humans.
7.5. Vitamin K. Structure. Requirement, sources of income, biological role. Manifestations of vitamin K deficiency.
7.6. Vitamin B1. Structure, sources of income, daily requirement, biorole: coenzyme and non-coenzyme functions. Biochemical mechanisms of development and clinical manifestations of deficiency.
7.7. Vitamin B2. Structure, sources of intake, daily requirement, biorole. Biochemical mechanisms of development and clinical manifestations of deficiency.
7.8. Folic acid. Structural components, sources of income and daily requirements. Formation of coenzyme form, biological role. Folic acid deficiency: causes and manifestations. Folic acid antivitamins and their use in medicine.
7.9. Vitamin B12. Concept of structure, sources of income, daily requirement, coenzyme forms and biorole. Causes of occurrence, biochemical mechanisms of development and clinical manifestations of deficiency.

7.10. Vitamins C and P. Structure, properties, needs, sources of intake, metabolic functions. Biochemical mechanisms of development and clinical manifestations of deficiency and hypervitaminosis C.

Topic 8. *Pathochemistry of microelements metabolism. Iron metabolism. Disorders of the metabolism of other trace elements - metals and the most important trace elements - non-metals. Deficiency of microelements.*

8.1. Causes, biochemical characteristics and consequences of hypercalcemia and hypocalcemia.

8.2. Deficiency of microelements (endemic goiter, dysthyroidism, fluorosis, Kashin-Beck disease, Keshan disease).

8.3. Causes, biochemical characteristics and consequences of hypo- and hyperphosphatemia.

8.4. The role of macroelements (sodium, potassium, calcium, phosphorus, sulfur, chlorine and others).

8.4. Metabolism of iron in the body. The role of transferrin and ferritin. Iron deficiency and its etiology. Hemosiderosis. Primary and secondary hemochromatosis.

8.5. Zinc, content in food, its bioavailability, role in the body. Hypozincosis.

8.6. Copper. The role of ceruloplasmin. Biological effects in the body. Hypercupremia, hypocupremia. Wilson-Konovalov disease and Menkes disease.

8.7. Cobalt and its importance in metabolism. Cobalt deficiency.

8.7. Manganese. The role of transmanganin. Hypermanganemia. Manganese deficiency.

8.8. Molybdenum, tungsten, chromium. Content in food products. Role in the body.

8.9. Vanadium, nickel, cadmium, strontium. Participation in metabolism.

8.10. Iodine, fluorine, selenium, silicon. Content in food products. Influence on metabolic processes. Excess and deficiency in the body.

Topic 9. *Biochemistry of signaling systems Diagnosis of endocrine disorders Systems.*

9.1. Define the concept of hormones. Chemical classification of hormones. Give the names of hormones belonging to different groups.

9.2. Classification of hormones according to their mechanism of action? Give examples. Mechanism of hormones action according to their chemical nature?

9.3. Present in the form of a diagram the hierarchy of regulatory systems of the human body.

9.4. What is the mechanism of action of catecholamines? What metabolic changes occur in target tissues under the influence of catecholamines?

9.5. Explain what metabolic changes can develop: a) with an increase in the production of hormones with a mineralcorticoid effect, b) with a decrease in the

production of hormones with a mineralcorticoid effect. Explain the mechanism of development of metabolic disorders in these pathological conditions.
9.6. Describe the functioning of the renin-angiotensin mechanism and its role in maintaining homeostasis.
9.7. Explain what metabolic changes can develop: a) with an increase in thyroxine production, b) with a decrease in thyroxine production. Explain the mechanism of development of metabolic disorders in these pathological conditions.
9.8. Name the peptide hormones that regulate calcium metabolism. Where is each of these hormones synthesized? Name the target tissues for each of these hormones. What metabolic changes occur in target tissues and in the body as a whole under the influence of each of these hormones?
9.9. Where is vasopressin synthesized? What is its chemical nature? What disease develops when the production of vasopressin decreases? What disorders develop with this disease? Explain the mechanism of development of these disorders.
9.10. What is the mechanism of action of glucocorticoids? What metabolic changes occur in target tissues under the influence of glucocorticoids? What changes in biochemical blood parameters occur under the influence of glucocorticoids?

Section 2. Mechanisms of metabolic disorders in cells

Topic 1. *Intracellular regulation of metabolism. Enzymes in the diagnosis of diseases of internal organs.*
1.1. Enzyme map of the cell. Compartmentalization. The structure of enzymes and their distribution in cells and tissues. Vitamins and their coenzyme functions.
1.2. Enzymes having a quaternary apoenzyme structure.
1.3. Organ specificity in the distribution of enzymes.
1.4. Isoenzymes (lactate dehydrogenase, creatine phosphokinase, alkaline and acid phosphatases), their characteristics. Multienzymes. Allosteric (regulatory) enzymes.
1.5. Clinical enzymology. Classification of enzymopathies (hereditary, congenital, acquired).
1.6. Enzymodiagnostics. The principle of determining enzyme activity in biological fluids. Changes in enzyme activity in diseases.
1.7. Enzyme therapy. The use of enzymes for medicinal purposes
1.8. Use of enzyme activity regulators as medicines.

Topic 2. *Hypoxia as a cause of metabolic disorders in cells. Oxidative stress.*
2.1. What is common to compounds called AFO (collective concept)?
2.2. Name three types of cellular targets to which the action of AFO is directed in the body.
2.3. Define the concept of “oxidative stress”, what is the mechanism of its development?
2.4. How does a “respiratory explosion” develop during inflammatory processes?

2.5. Why does antioxidant protection decrease during pathological processes against the background of increased AFO generation?

Topic 3. *Laboratory assessment of free radical processes, lipid peroxidation, antioxidant system - diagnostic and prognostic significance.*
3.1. Free radical oxidation: normal metabolic mechanisms and a nonspecific pathological process.
3.2. The contribution of free radical oxidation to the development of diseases.
3.3. Methods for assessing the intensity of free radical oxidation processes in the patient's body.
3.4. Methods for assessing the functional activity of the antioxidant defense system.
3.5. The imbalance between the intensity of free radical oxidation and the functional activity of the antioxidant system is an important criterion for assessing the disease.
3.6 Why do hemolysis of erythrocytes and the development of hemolytic anemia occur with hereditary deficiency of glucose-6-phosphate dehydrogenase?
3.7. What is the damaging effect of AFO on connective tissue?
3.8. What is the mechanism of the damaging effect of AFO in cataracts?
3.9. Why is the damaging effect of AFO considered as a source of hereditary pathology and tumor growth?
3.10. What laboratory indicators better characterize the balance of the "LPO-antioxidant defense of the body" system?

Topic 4. *Metabolic syndrome. Molecular mechanisms of insulin resistance formation.*
4.1. Relationship between carbohydrate metabolism and other types of metabolism. Possible pathways for the synthesis of carbohydrates in the body.
4.2. Insulin and contra-insular hormones - as regulators of the deposition and mobilization of glycogen and lipids.
4.3. What differences are there in the structure and functioning of the extracellular and intracellular parts of the insulin receptor?
4.4. How does insulin activate phosphatidylinositol 3-kinase?
4.5. How does insulin-stimulated protein phosphatase via Ras pathway participate in the regulation of glycogen metabolism?
4.6. How does transmembrane glucose transfer from intestinal cells into the blood differ from glucose transfer into muscle cells?
4.7. How does transmembrane glucose transfer from blood to adipocytes differ from glucose transfer from blood to hepatocytes?
4.8. How does transmembrane glucose transport in renal tubules differ from the transfer of glucose from the blood to adipocytes?
4.9. How does the metabolism of basic energy carriers change with insulin deficiency?
4.10. Why is the formation of ketone bodies increased in diabetes?

Topic 5. *Interrelation of metabolic pathways. Pathochemistry of diabetes mellitus. Laboratory tests*

5.1. Laboratory indicators of carbohydrate metabolism disorders and their clinical and diagnostic significance: glucose in the blood and urine; fructosamine in blood serum; glycosylated hemoglobin.
5.2. Diagnostics of diabetes mellitus – biochemical constellations. Monitoring of patients.
5.3. How does the level of fructosamine in the blood change during pregnancy, uremia, myeloma, and acute inflammatory diseases?
5.4. Technology for screening assessment of glucose levels in blood and urine.
5.5. Determination of blood glucose using test strips.
5.6. Glucose tolerance tests. Load tests (single glucose load, double load, test with adrenaline, insulin).
5.7. Analysis of glycemic curves. Glycemic curves during a single glucose load and in the case of certain types of pathology. Hyperglycemic coefficient (Baudoin coefficient). Postglycemic coefficient (Rafalsky coefficient).
5.8. Name promising laboratory tests for diagnosing diabetes mellitus and the nature of emerging metabolic disorders.
5.9. What is the basis for predicting diabetes mellitus?
5.10. List the indications for determining C-peptide in serum and urine.

Topic 6. *Laboratory assessment of biochemical markers in the blood during myocardial infarction, their diagnostic and prognostic significance.*

6.1. Features of myocardial metabolism and its disorders in the initial stage of myocardial infarction.
6.2. Principles of enzyme diagnostics of myocardial infarction.
6.3. Non-enzymatic marker proteins in the blood during myocardial infarction.
6.4. Markers of unstable angina.
6.5. Clinical and biochemical periodization of myocardial infarction.
6.6. Name the substrates of oxidation in the myocardium and specify how the utilization of a particular substrate depends on the physiological state of the body.
6.7. Name the conditions in which myocardial infarction has developed, and the activity of CK and CK-MB is practically not determined.
6.8. Name a protein marker that allows you to diagnose myocardial infarction both in early and late stages.

Topic 7. *Molecular mechanisms of bone tissue formation and resorption. Osteoporosis.*

7.1. What physiological and biochemical processes are regulated by calcium ions?
7.2. How does hormonal regulation of calcium-phosphorus metabolism occur?
7.3. What are the physiological effects of hormones: PTH, calcitonin, vitamin D3?
7.4. How is the synthesis of PTH and calcitonin regulated?

7.5. How is vitamin D3 synthesized and activated?
7.6. What foods contain calcium, phosphorus, vitamin D?
7.7. What are the main causes and clinical manifestations of hypocalcemia?
7.8. What are the main causes and clinical manifestations of hypercalcemia?
7.9. What laboratory criteria are used to diagnose hypo- and hypercalcemia?
7.10. Osteoporosis, causes and mechanisms of development. Factors contributing to the development of osteoporosis.

Topic 8. *Biochemical basis of hereditary pathology (disorders of carbohydrate metabolism, metabolism of individual amino acids, metabolism of purine and pyrimidine nitrogenous bases, vitamins, porphyrins, non-enzymatic proteins)*
8.1. Disorders of monosaccharide metabolism. Essential fructosuria, hereditary fructose intolerance, galactosemia. Causes, clinical picture, laboratory diagnostics, aspects of treatment.
8.2. Disaccharidase deficiency. Intolerance to maltose, isomaltose, lactose. Symptoms, principles of treatment.
8.3. Glycogen metabolism and its defects. Glycogenosis: diseases of Gierke, Pompe, Corey-Forbes, Andersen, McArdle, Hers, Terje, Hoidzhin.
8.4. Disorders of glycosaminoglycan metabolism. Hurler, Scheie, Gunter, Sanfilippo, Morquio, Maroto-Lami syndromes.
8.5. Molecular pathology of the metabolism of individual amino acids. Phenylketonuria, tyrosinemia, alkaptonuria, homocystinuria, cystinuria, histidinemia, Hartnup disease, blue diaper syndromes, Thad, Price, Knapp-Komrover.
8.6. Disturbances in the metabolism of purine and pyrimidine nitrogenous bases. Lesch-Nyhan syndrome, gout, xanthinuria, orotaciduria.
8.7. Primary endogenous hypovitaminosis. Darier's syndrome, vitamin D-resistant rickets (phosphate diabetes), Leigh's disease, Wernicke-Korsakoff syndrome, methylmalonic aciduria, Imerslund-Gresbeck disease, pyridoxine-dependent seizures, pyridoxine-dependent anemia, cystathioninuria, Knapp-Komrover syndrome, Möller-Barlow disease, propionic acidemia .
8.8. Disorders of porphyrin metabolism. Porphyrias: acute intermittent, cutaneous, erythropoietic, erythropoietic. Hepatic coproporphyria.
8.9. Defects of proteins of non-enzymatic nature. Combined immunodeficiency (T- and B-lymphocytes), deoxyadenosinuria, hypouricemia.

Topic 9. *Disturbances of intercellular interactions. Biochemical basis of carcinogenesis.*
9.1. What is a tumor marker?
9.2. Features of the modern classification of tumor markers.
9.3. Name the indications for determining tumor markers.
9.4. What markers can be used in screening for malignant neoplasms?

9.5. Name the main tumor markers in the diagnosis of breast, liver, ovarian, and pancreatic cancer.
9.6. Algorithm for blood testing for tumor markers
9.7. Laboratory examination plan for a patient with a malignant neoplasm
9.8. Biochemical screening. Tumor markers.
9.9. Range of application of tumor markers
9.10. Laboratory tests for examining patients at risk of cancer.

A.2 Questions for midterm control (colloquium)

***Section 1.* Metabolic disorders. Biochemical indicators of metabolic disorders in the human body.**
1. Name the main types of proteins determined in clinical laboratories.
2. What diseases are classified as human prion diseases?
3. Compare the “heterodimeric” and “polymerization” models of prion protein transformation.
4. Why are BEFs of inflammation called adaptive and how is their heterogeneity manifested?
5. What methods are used to determine BF and their constellations in a number of diseases (exacerbation of rheumatoid arthritis, lupus erythematosus, stomach cancer)
6. Formulate the principles of biochemical diagnostics for liver diseases.
7. What is the role of the liver in the metabolism of proteins, carbohydrates, lipids, hormones, pigments, vitamins, xenobiotics?
8. Indicate the compartmentalization of enzymes determined in CD-laboratory in liver diseases.
9. Name laboratory syndromes for diffuse liver damage.
10. Name serum markers of chronic viral hepatitis B, C, D.
11. Name the stages of metabolism of exogenous and endogenous lipids.
12. What classifications of lipoproteins exist and what separation methods are they based on?
13. Name the reasons that cause an endogenous block of active transport of polyene fatty acids into cells and its consequences for the body.
14. What is the modern concept of the pathogenesis of atherosclerosis?
15. What is the logical structure of the stages of diagnosing dyslipoproteinemia and assessing their atherogenicity?

Section 2. *Mechanisms of metabolic disorders in cells*
1. Formulate the difference between the concepts of “enzymes”, “isoenzymes”, “enzyme isoforms”.
2. What factors influence the content of enzymes in the blood and what is the mechanism of development of hyperenzymemia in pathological conditions?

3. Name the factors that determine the rate of release of enzymes from damaged tissues.
4. What specific reasons do you know for increased levels of enzymes in the blood?
5. What is the clinical decision threshold for determining enzyme activity relative to the upper reference limit?
6. Hereditary enzymopathies of amino acid metabolism. Biochemical diagnostics.
7. Pathology of carbohydrate metabolism. Diabetes. Glycogenoses. Biochemical diagnostics.
8. Pathology of lipid metabolism. Obesity, atherosclerosis. Biochemical diagnostics.
9. Hereditary enzymopathies of porphyrins, purines, pyrimidines. Biochemical diagnostics.
10. Ketosis. Biochemical diagnostics.
11. Clinical use of prostaglandins, prostacyclins, thromboxanes.
12. The mechanism of development of tolerance to drugs and physiologically active substances.
13. Lipid peroxidation and oxidative modification of proteins and lipoproteins.
14. Changes in biochemical parameters of blood and urine during viral and alcohol intoxication.

Block B *(example)*

B.1 Clinical cases:

Module 1.

Case 1. In poteinogram of a patient with an unknown pathology of the gastrointestinal tract, the following data were found: total protein - 58 g/l; albumins – 48%; alpha-1-globulins – 6%; alpha-2-globulins – 10%; beta globulins – 13%; Gamma globulins – 23%. What possible reasons could provoke the detected changes in serum proteins?

Case 2. The parents of a 5-year-old girl consulted an endocrinologist. During the examination, the child showed manifestations of secondary sexual characteristics: muscle hypertrophy, excessive hair growth, and decreased timbre of the voice. The level of ACTH in the blood is increased. The doctor diagnosed andrenogenital syndrome. Justify the doctor's diagnosis. To do this: a) present a scheme for the synthesis of steroid hormones, name them and indicate their functions; b) name the enzymes whose deficiency causes these symptoms; c) indicate which corticosteroid synthesis increases in this pathology; d) why the ACTH concentration is increased.

***Module* 2.**

Case 1. An elderly woman was transported to hospital after she fell at home and broke her hip. She lived as a recluse and left the house only on those occasions when there was no one to make the necessary purchases for her. In addition to the hip fracture, radiographic examination revealed typical signs of osteomalacia.
Laboratory data:

Serum: calcium 1.75 mmol/l
phosphate 0.7 mmol/l
alkaline phosphatase 440IU/l
albumin 30g/l
What is the cause of osteomalacia in an elderly woman? What confirms the diagnosis?

Case 2. A patient with suspected acute liver damage (acute hepatitis) was admitted to the infectious diseases department. The patient has a bright icteric coloration of the skin and mucous membranes, the content of direct and indirect bilirubin is increased in the blood, the urine is intensely colored, direct bilirubin and urobilinogen are determined in it; the amount of bile pigments in feces is reduced. What type of jaundice does the patient have? To answer the question
- write a diagram of heme catabolism
-indicate the diagnostic criteria for this pathology.

Topics for reports
1. Medical and diagnostic value of determining some blood proteins (haptoglobin, C-reactive protein, ceruloplasmin, etc.)
2. Pathological cascade of metabolic syndrome.
3. Biochemical basis of hemochromatosis.
4. Biochemistry of thalassemia.
5. Hemophilia: types, causes, treatment prospects.
6. Hormonal and local factors regulating bone tissue remodeling.
7. Causes, biochemical characteristics and consequences of hypercalcemia.
8. Causes, biochemical characteristics and consequences of hypocalcemia.
9. Causes, biochemical characteristics and consequences of hypo- and hyperphosphatemia.
10. Characteristics of the most important disorders of acid-base balance (acidosis and alkalosis).
11. Osteoporosis, causes and mechanisms of development. Factors contributing to the development of osteoporosis.
12. Gout: characteristics, pathological cascade, causes.
13. Biochemical causes and characteristics of proteinuria and nephrotic syndrome.
14. Isoenzymes are genetically determined multiple forms of enzymes. Clinical and diagnostic significance.
15. Hereditary enzymopathies of amino acid metabolism. Biochemical diagnostics.
16. Pathology of carbohydrate metabolism. Diabetes. Glycogen store diseases. Biochemical diagnostics.
17. Pathology of lipid metabolism. Obesity, atherosclerosis. Biochemical diagnostics.

18. Hereditary enzymopathies of porphyrins, purines, pyrimidines. Biochemical diagnostics.
19. Ketosis. Biochemical diagnostics.
20. Clinical usage of prostaglandins, prostacyclins, thromboxanes.
21. The mechanism of development of tolerance to drugs and physiologically active substances.
22. Lipid peroxidation and oxidative modification of proteins and lipoproteins.
23. Changes in biochemical parameters of blood and urine during viral and alcohol intoxication.
24. Immunoglobulins, interleukins, their clinical and diagnostic significance.
25. Biochemistry of nutrition. Principles of rational nutrition in various age groups.

Block D

Yhe list of questions and tasks for intermediate certification (test/exam):

Questions to check the level "KNOWLEDJE":

1. Enzyme map of the cell. Compartmentalization. The structure of enzymes and their distribution in cells and tissues. Vitamins and their coenzyme functions.
2. Enzymes that have a quaternary apoenzyme structure. Organ specificity in the distribution of enzymes. Isoenzymes (lactate dehydrogenase, creatine phosphokinase, alkaline and acid phosphatases), their characteristics. Multienzymes. Allosteric (regulatory) enzymes.
3. Clinical enzymology. Classification of enzymopathies (hereditary, congenital, acquired).
4. General methods for determining enzyme activity. Factors influencing the level of enzymes in extracellular fluid.
5. The main directions of using enzymes in medical practice: a) enzyme diagnostics. The principle of determining enzyme activity in biological fluids; b) enzyme therapy; c) the use of enzyme activity regulators as drugs.
6. Pathobiochemistry of the myocardium. Regulation of cardiac muscle metabolism at rest and during exercise. The effect of hypoxia on metabolism in the heart muscle. Biochemical aspects of laboratory diagnosis of myocardial infarction (enzyme studies).
7. Changes in enzymatic activity of blood serum in pathology of the pancreas (acute and chronic pancreatitis).
8. The relationship between the processes of anabolism and catabolism of lipids with other types of metabolism. Pyruvate and acetyl-SCoA and the ways of their formation from other metabolites.
9. Anabolic and catabolic stages of nitrogen-containing compounds. Connection with other metabolic pathways.
10. Insulin and contrainsular hormones - as regulators of the deposition and mobilization of glycogen and lipids.

11. Disturbances in the synthesis of glycogen and lipids due to insulin deficiency.
12. Regulation of acid-base balance. Blood buffer systems (bicarbonate, hemoglobin, phosphate, protein).
13. Indicators of the acid-base balance (Siggor-Andersen nomogram): - the value of the active reaction of the solution (pH); - partial pressure of carbon dioxide (pCO2); - true blood bicarbonate (AB); - standard bicarbonate (SB); - the sum of the bases of all blood buffer systems (BB); - normal buffer bases
(NBB); - excess (deficiency) of buffer bases (BE = BB – NBB).
14. The role of potassium and sodium cations in the metabolism of substances.
15. Phosphorus-calcium metabolism
16. Methods for studying the functional state of the kidneys.
17. Proteinuria: glomerular, tubular, mixed.
18. Metabolism of lipoproteins, their role.
19. System "lipid peroxidation – anti-free-radical protection".
20. Hereditary pathology of lipid metabolism (sphingolipidosis, gangliolipidosis).
21. Disaccharidase deficiency. Intolerance to maltose, isomaltose, lactose. Symptoms, principles of treatment.
22. Molecular pathology of the metabolism of individual amino acids. Phenylketonuria, tyrosinemia, alkaptonuria, homocystinuria, cystinuria, histidinemia, Hartnup disease, blue diaper syndrome, Thad, Price, Knapp-Komrover.
23. Porphyrias: acute intermittent, cutaneous, erythropoietic, erythropoietic. Hepatic coproporphyria

Questions to check the level "SKILLS"

1. Describe the distribution of enzymes in cells and tissues, taking into account their functions. Explain the coenzyme functions of vitamins.
2. Explain the organ specificity in the distribution of enzymes: isoenzymes (lactate dehydrogenase, creatine phosphokinase, alkaline and acid phosphatases), their characteristics.
3. Determine enzyme activity. Explain the effect of factors influencing the level of enzymes in the extracellular fluid.
4. Use enzymes in medical practice: a) enzyme diagnostics. Principle of activity determination enzymes in biological fluids; b) enzyme therapy;
5. Describe the effect of hypoxia on metabolism in the heart muscle. Conducting laboratory diagnostics of myocardial infarction (enzyme studies).
6. Relationship between carbohydrate metabolism and other types of metabolism.
7. Describe the relationship between the processes of anabolism and catabolism of lipids with other types of metabolism.
8. Describe the relationship of the anabolic and catabolic stages of nitrogen-containing compounds with other metabolic pathways.
9. Regulation of the deposition and mobilization of glycogen and lipids.

10. Describe the mechanism: hyperglycemia; hyperketonemia and hypercholesterolemia; hyperazotemia, hyperaminoacidemia, hypercarbamidemia;
11. Mechanism of development of tissue hypoxia.
12. Physiological mechanisms of regulation of ABB: a) buffering; b) removal of volatile acids (CO_2); c) generation of bicarbonate (HCO_3^-) by the kidneys; removal of non-volatile acids (organic acids, H_2PO^{4-}, HSO^{3-}) and ammonium salts (NH_4^+); d) participation of the liver in the regulation of ABB; e) the role of the pancreas and stomach.
13. Renal control of acid-base balance: a) conversion of dibasic phosphates into monosubstituted ones; b) conversion of bicarbonates to carbonic acid; c) synthesis of ammonia in the kidneys and excretion of ammonium salts (acid equivalents).
14. Interpretation of acid-base values and diagnostic principles.
15. Regulation of phosphorus-calcium metabolism.
16. Features of ketogenesis; characteristics of the anabolic and catabolic stages of cholesterol metabolism.
17. Dislipoproteinemia. Classification. Laboratory tests for typing.
18. Factors that stimulate LPO: reactive oxygen species, hypoxia, ischemia, infectious agents.
19. Disorders of monosaccharide metabolism. Essential fructosuria, hereditary fructose intolerance, galactosemia.
20. Explain the mechanism of development of glycogenoses: diseases of Gierke, Pompe, Corey-Forbes, Andersen, McArdle, Hers, Terje, Hoidzhin.
21. Explain the mechanism of development of metabolic disorders of purine and pyrimidine nitrogenous bases. Lesch-Nyhan syndrome, gout, xanthinuria, orotaciduria.
22. Explain the mechanism of development of porphyrin metabolism disorders. Porphyrias: acute intermittent, cutaneous, erythropoietic, erythropoietic. Hepatic coproporphyria.

Questions to check the level "EXPERTISE"

1. Multienzymes. Allosteric (regulatory) enzymes.
2. Use of enzyme activity regulators as medicines.
3. Regulation of cardiac muscle metabolism at rest and during exercise.
4. Interpretation of enzymatic activity of blood serum in pathology of the pancreas (acute and chronic pancreatitis).
5. Possible pathways for the synthesis of carbohydrates in the body.
6. Diabetes mellitus. Forms. Epidemiology. Etiology. Risk factors.
7. Explain why protein glycosylation is one of the most important causes of late complications of diabetes mellitus.
8. Diagnostic tests for suspected diabetes.
9. Typical forms of acid-base balance disorders: a) compensated and decompensated acidosis; b) stage of compensation and decompensation in respiratory acidosis; c)

metabolic alkalosis during compensation and decompensation; d) phases of compensation and decompensation of respiratory alkalosis.
10. Changes in the parameters of water-salt metabolism in case of its violations.
11. Violation of phosphorus-calcium metabolism.
12. Biochemical blood test for kidney diseases.
13. Biochemical aspects of atherosclerosis (etiology, risk factors, pathogenesis, laboratory diagnostics).
14. Antioxidant protection of the cell: antioxidant enzymes, antioxidants of non-enzymatic origin (redox vitamins - cell membrane stabilizers).
15. Causes, clinical picture, laboratory diagnostics, aspects of treatment.
16. Disorders of glycosaminoglycan metabolism. Hurler, Scheie, Gunter, Sanfilippo, Morquio, Maroto-Lami syndromes.
17. Diagnosis of primary endogenous hypovitaminosis. Darier's syndrome, vitamin D-resistant rickets (phosphate diabetes), Leigh's disease, Wernicke-Korsakoff syndrome, methylmalonic aciduria, Imerslund-Gresbeck disease, pyridoxine-dependent seizures, pyridoxine-dependent anemia, cystathioninuria, Knapp-Komrover syndrome, disease Meller-Barlow, propionic acidemia.
18. Diagnostics of defects in proteins of non-enzymatic nature. Combined immunodeficiency, deoxyadenosinuria, hypouricemia.

INTERMEDIATE CONTROL EXAMINATION TICKET № 1 (record)

TICKET № 1

№ 1. In the laboratory, the blood was examined and the following content of protein fractions (g/l) was obtained: albumins - 40, α1-globulins - 2, α2-globulins - 7, β-globulins - 8, γ-globulins - 12. What method could the analysis be performed? What is its essence? Are there any deviations from the norm in the results? Give a name of this analysis result.

№ 2. When examining a patient admitted to the hospital with suspected viral hepatitis, it was found that the activity of the ALT enzyme in the blood serum exceeds the normal level by 10 times (the norm is 0.1-0.45 mmol/l×hour). What does the result indicate?

№ 3. One student has had dinner and is lying on the couch, another is doing a 40-minute run.

1. Describe the differences in lipid metabolism.
2. Name blood indicators that can confirm your assumptions.
3. How will these indicators change in the blood?

№ 4. To determine the cause of hypoglycemia, the newborn was given a glucagon test, which did not cause an increase in blood glucose levels.

a) How does glucagon increase blood glucose levels?

b) Name the possible reasons for the absence of the hyperglycemic effect of glucagon.

c) What biochemical studies will make it possible to diagnose the child?

4. METHODOLOGICAL MATERIALS DETERMINING PROCEDURES FOR ASSESSING KNOWLEDGE, ABILITIES, SKILLS

The assessment scales

Report evaluation criteria

Points	5	4	3	2
Content	The work is fully completed.	Practically the most important components of the work are completed but not fully.	Not all important components of the work are completed.	The work is done partially with the teacher's help.
	The work demonstrates deep understanding of the described processes.	The work demonstrates understanding of the main points but some details are not specified.	The work demonstrates partial understanding of the described processes.	The work demonstrates little understanding of the described processes.
	The work contains interesting discussion material. Scientific vocabulary is used correctly.	There is some discussion material. Scientific vocabulary is used, but sometimes not correctly.	Discussion material is present but does not help to understand the problem. Scientific terminology is used but not correctly.	Minimal discussion material is present. Few scientific terms are used.
	The student provides his own interpretation or development of the topic (generaliza-tions, applications, analogies).	The student offers his own interpretation or development of the topic in most cases.	The student sometimes offers his interpretation.	Interpretation is limited or is not related to the subject.
Literacy	No error - grammatical or syntactic.	Minimal errors are present.	There are errors making the meaning difficult to understand.	A lot of errors, making the material difficult to read.
Report	The student clearly explains the abstract content and makes visual contact with the audience.	The student clearly explains the content of the essay.	The student clearly explains the content of the essay.	The student reports incorrect information.

51-- 60 points – 5
41 – 50 points – 4

31 – 40 points – 3
Less than 30 points – 2.

Assessment criteria for answers to situational tasks:
5 points - student gives precise answers to all questions on situational problems using terms and definitions from basic, main and additional literature.
4 points - student correctly, but not in details, answers to all the questions with minimal errors using references from basic and main literature.
3 points - student correctly solves the problem, but doesn't answer all the questions (70 - 89%), omitting details, answers with errors using references from basic literature.
2 points – student correctly solves the fragments of the task, doesn't answer all the questions making mistakes and using references from basic literature.
1 point – student demonstrates isolated fragments of knowledge could not solve the whole problem.
0 points - student does not solve the task, gives wrong answers, the answers do not relate to the task questions.

The criteria for test assessment
5 points – 85 - 100% of correct answers
4 points – 76 - 85% of correct answers
3 points – 61 - 75% of correct answers
2 points – 0 - 60% of correct answers

Assessment criteria for exam questions:
1. Knowledge of the basic processes, functions and laws of the subject, depth and completeness of answers to question.
2. Knowledge of terms and concepts, their use in the response.
3. Ability to explain the meaning of processes, laws, mechanisms, to make conclusions
and summarization, ability to explain cause-and-effect relations.
4. Ability to answer questions.
5. Use of literary language, scientific terms, logic and consistency of answers, ability to express his/ her opinion.

The exam is assessed **excellently** (85-100% of the information presented; according to the RPS - 28-30 points) if the student:
❒ shows a comprehensive, systematic and deep knowledge of the program material;
❒ demonstrates knowledge of modern educational and scientific literature;
❒ able to creatively apply knowledge of theory to solve professional problems;
❒ knows terminology;
❒ demonstrates the ability to analyze and compare different approaches to solving the problems stated in the ticket;
❒ confirms theoretical postulates with examples from biochemical processes.

The exam is assessed **well** (76-85% of the information presented; according to the RPS - 24-27 points) if the student:

❒ reveals a knowledge of program material;
❒ mastered the basic and most significant additional literature;
❒ is able to apply knowledge of theory to solving problems of a professional nature;
❒ allows for some errors and inaccuracies when answering.

The exam is assessed **satisfactorily** (60-75% of the information presented; according to the RPS - 20-23 points) if the student:

❒ basically knows the program material to the extent necessary for the upcoming work in the profession;
❒ generally mastered the basic literature;
❒ allows significant errors in answering questions on the exam paper.

The exam is graded **unsatisfactorily** (less than 60% of the information presented; according to the RPS - 0 points) if the student:

❒ reveals significant gaps in knowledge of the main program material;
❒ makes fundamental mistakes in answering questions on the exam paper;
❒ demonstrates ignorance of theory and biochemical processes.