

SYLLABUS

The academic year when the cycle of instruction is commenced 2019-2025 [INT]

Module/course name:	Pathophysiology		Module code	LK.3.B.006
Faculty:	Faculty of Medicine MUL			
Major:	Medical			
Specialty:				
Level of study:	I (Bachelor studies) <input type="checkbox"/> II (Master studies) <input type="checkbox"/> Integrated Master studies X Doctoral studies <input type="checkbox"/>			
Mode of study:	full-time X			
Year of study:	I <input type="checkbox"/> II X III <input type="checkbox"/> IV <input type="checkbox"/> V <input type="checkbox"/> VI <input type="checkbox"/>	Semester:	1 <input type="checkbox"/> 2 <input type="checkbox"/> 3 X 4 X 5 <input type="checkbox"/> 6 <input type="checkbox"/> 7 <input type="checkbox"/> 8 <input type="checkbox"/> 9 <input type="checkbox"/> 10 <input type="checkbox"/> 11 <input type="checkbox"/> 12 <input type="checkbox"/>	
Module/course type:	obligatory X elective <input type="checkbox"/>			
Language of instruction:	Polish <input type="checkbox"/> English X			
Form of education				Hours
Lecture				30
Lecture				
Laboratory class				60
E-learning				
Practical class				
Internship				
Other				
TOTAL				90
Student's work input (participation in class, preparation, evaluation, etc.)				Student's hourly workload
1. In class				90
2. Student's own work including: 1 Preparation for class 2 Preparation for partials and finals				85
Summary of the student's workload				175
ECTS points for module/course				7

Educational objectives:

Pathophysiology course will provide an in-depth introduction to basic concepts and fundamental principles of human pathophysiology for medical students. The aim of the course is to provide the student with a systematic approach for understanding disease and rational therapeutic design. Students will examine the phenomena that produce alterations in human physiologic function and the resulting human response. Upon completion of the course, students will understand pathophysiological changes, including how pathological processes are manifested, progress in the body, and the primary and secondary effects of these changes.

Pathophysiology bridges basic science and clinical practice with an emphasis on integrated mechanistic understanding of the molecular, structural, and functional alterations in cells, tissues, and organ systems that underlie human disease. The course will follow the classical division between general pathophysiology (molecular, cellular and tissue reactions) and systemic pathophysiology (integrated tissue and organ system responses). The material will be presented from an epidemiologic perspective that focuses on disease prevalence, incidence, morbidity and

mortality, risk factors, and prevention strategies. It will emphasize mechanisms of development (pathogenesis), progression, pathophysiologic associations with risk factors, structural alterations (morphologic changes) resulting from the disease, and the functional consequences of these structural changes (clinical significance). The course will focus on fundamental concepts of cellular homeostasis; general cellular responses (adaptation, injury, cell death) induced by stress, injurious stimuli, and disease. Following the fundamental, an integrative systemic approach to illustrate the pathophysiology to systemic diseases. Topics presented in this pathophysiology course will include cardiology, pulmonology, hematology, nephrology, gastroenterology, rheumatology, neurology, oncology, and endocrinology.

The matrix of learning outcomes for module/ subject with reference to verification methods of the intended educational outcomes and forms of instruction:

Learning outcome code	A student who has obtained a credit for the module/course has the knowledge/skill to:	Methods of verifying the achievement of the intended learning outcomes:	Form of instruction * provide the symbol
W01 (B.W17.)	knows the methods of intercellular communication, communication between a cell and extracellular matrix and the signal transmission paths in a cell as well as examples of disturbances in these processes, leading to development of cancers and other diseases;	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W02 (B.W22.)	knows the course and control of reproductive functions in males and females	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W03 (B.W23.)	knows the body aging mechanisms	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W04 (B.W25.)	can find relationship between factors disrupting the equilibrium of biological processes and the physiological and pathophysiological changes	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W05 (C.W21.)	knows the development principles and function mechanisms of immune system; including specific and non-specific humoral and cellular defense mechanisms;	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W06 (C.W22.)	can describe major histocompatibility complex;	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W07 (C.W23.)	knows hypersensitive response types, kinds of immune deficiency and basics of immunomodulation	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W08 (C.W27.)	knows basic cell and tissue damage mechanisms;	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W09 (C.W28.)	describes clinical course of specific and non-specific inflammatory processes and tissue and organ regeneration	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W10 (C.W29.)	knows the definition and pathophysiology of shock, with special consideration to differentiation of shock causes, and multiorgan failure	written exam - SSQ - MCQ - MRQ	Lecture / Lab class

W10 (C.W30.)	knows the etiology of hemodynamic disorders, regressive and progressive changes	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W11 (C.W32.)	describes the consequences of developing pathological changes for the neighboring organs	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W12 (C.W33.)	can name internal and external, modifiable and non-modifiable pathogenic factors	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W13 (C.W34.)	knows the clinical manifestations of the most common diseases of systems and organs, metabolic diseases and water-electrolyte and acid-base balance	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W14 (C.W48.)	knows the consequences of vitamins or minerals deficiency and excess	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W15 (C.W49.)	knows enzymes taking part in digestion process, mechanism of hydrochloric acid production in the stomach, the role of bile, the course of digestive products absorption and disorders related to it;	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W16 (C.W50.)	knows the consequences of wrong nutrition, including long starvation, taking too big meals and having unbalanced diet	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W17 (C.W51.)	knows mechanism of hormonal activity	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W18 (E.W1.)	knows the genetic, environmental and epidemiological background of most common diseases	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
W19 (E.W3.)	knows and understands the causes, symptoms, principles of diagnosing and therapeutic procedures of most common children's diseases:		
b)	heart defects, myocarditis, endocarditis, pericarditis, cardiomyopathy, rhythm abnormalities, cardiac failure, arterial hypertension, fainting,	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
c)	acute and chronic diseases of upper and lower respiratory tract, congenital respiratory defects, tuberculosis, mucoviscidosis, asthma, allergic rhinitis, rash, anaphylactic shock, angioedema,		
d)	anemia, bleeding diathesis, bone marrow failure, pediatric malignancies, including solid tumors specific for pediatric cancers		
f)	urinary infections, congestive failure of the urinary system, nephritic syndrome, acute and chronic urolithiasis, renal failure, acute and chronic nephritis, systemic renal disease, abnormal urination, vesicoureteral reflux	written exam - SSQ - MCQ - MRQ	Lecture / Lab class
g)	growth disturbances, disorders of thyroid and parathyroid glands, adrenal disorders, diabetes, obesity, adolescent disorders and gonadal disorders,		

W20 (E.W7.)	<p>knows and understands the causes, symptoms, diagnostic principles and therapeutic procedures in respect to most common internal diseases in adults and their complications;</p> <p>a) circulatory disorders, including ischaemic heart disease, heart defects, disorders of the endocardium, myocardium, pericardium, acute and chronic cardiac failure, arterial and venous disorders, primary and secondary arterial hypertension, pulmonary hypertension,</p> <p>b) respiratory disorders, including: diseases of the respiratory tract, chronic bronchial asthma, obstructive pulmonary disease, bronchiectasis, mucoviscidosis, infections of the respiratory tract, interstitial diseases of the lungs, pleura, mediastinum, obstructive and central sleep apnea, respiratory failure (acute and chronic), malignancies of the respiratory system,</p> <p>d) disorders of the endocrine system, including diseases of hypothalamus and pituitary gland, thyroid, parathyroid glands, adrenal cortex and core, ovaries and testes, neuroendocrine tumors, pluriglandular hypofunction, various types of diabetes and metabolic syndrome; hypoglycemia, obesity, dyslipidemia;</p> <p>e) diseases of kidneys and urinary tract, including acute and chronic renal failure, glomerular disorders, interstitial renal disorders, renal cysts, urolithiasis, urinary infections, malignancies of the urinary system, and in particular, cancers of the urinary bladder and kidney</p> <p>f) disorders of the hematopoietic system, including bone marrow aplasia, anemia, granulocytopenia and agranulocytosis, thrombocytopenia, acute leukemia, myeloproliferative and myelodysplastic- myeloproliferative tumors, myelodysplastic syndromes, tumors from mature B and T lymphocytes, bleeding diathesis, thrombophilia, life threatening conditions in hematology, blood disorders in diseases of other organs, blood donation and blood therapy, bone marrow transplantation,</p> <p>g) allergic diseases, including anaphylaxis and anaphylactic shock, angioedema</p> <p>h) water-electrolyte and acid-base disturbances, dehydration, hyperhydration, electrolyte disorders, acidosis and alkalosis.</p> <p>i)</p>	<p>written exam</p> <ul style="list-style-type: none"> - SSQ - MCQ - MRQ 	<p>Lecture / Lab class</p>
U01 (C.U11.)	<p>can associate pictures of tissue and organ damage with clinical symptoms of disease, interview and results of laboratory tests</p>	<ul style="list-style-type: none"> - completion of a given assignment - project - presentation 	<p>Lecture / Lab class</p>
U02 (C.U12.)	<p>analyses the reactive, defense and adaptation response and control disorders caused by an etiological factor</p>	<ul style="list-style-type: none"> - completion of a given assignment - project - presentation 	<p>Lecture / Lab class</p>

U03 (C.U20.)	<p>describes changes in body functioning in states of homeostasis disturbances, and, in particular, specifies its integral response to physical strain, exposure to high and low temperature, loss of blood or water, sudden verticalization, transition from sleep to awake</p>	<ul style="list-style-type: none"> - completion of a given assignment - project - presentation 	Lecture / Lab class
EXAMPLES OF METHODS VERIFYING THE ACHIEVEMENT OF THE INTENDED LEARNING OUTCOMES:			
<p>In terms of knowledge: Oral exam (<i>non-standardized, standardized, traditional, problem-based</i>). Written exam – the student produces/identifies answers (<i>essay, report; structured short-answer questions /SSQ/; multiple choice questions /MCQ/; multiple response questions /MRQ/; matching test; true/false test; open cloze test</i>).</p> <p>In terms of skills: practical exam; Objective Structured Clinical Examination /OSCE/; Mini-CEX (mini – clinical examination); completion of a given assignment; project, presentation.</p> <p>In terms of social competences: A reflective essay; an extended observation by a supervisor/tutor; 360-degree assessment (feedback from teachers, peers, patients, other co-workers); self-assessment (portfolio included).</p>			
Course content: (use keywords referring to the content of each class following the intended learning outcomes):			
<p>Lectures:</p> <p>1. Introduction to endocrinology. Endocrine system and hormones. Disorders of calcium metabolism (Part I). Hypo- and hypercalcemia, hypo- and hyperphosphataemia: causes, symptoms. Tumor hypercalcemia: the importance of PTH-like protein (PTHRP), vitamin D3, cytokines, metastasis. Hyperparathyroidism: 1) primary hyperparathyroidism (PNP): causes. Hypercalcemic syndrome: nephrological symptoms (urolithiasis and calcium, polyuria, PNN), gastroenterological symptoms (peptic ulcer disease, acute pancreatitis), cardiological symptoms (hypertension, arrhythmia), bone symptoms (pain, pathological fractures), psychological symptoms. Hypercalcemic crisis (dehydration, shock). Generalized bone atrophy, periosteal resorption, cysts [osteitis fibrosa cystica], osteopenia, osteoporosis; 2) secondary hyperparathyroidism (induced by hypocalcemia): causes, laboratory symptoms (Ca, P, PTH). The importance of the autonomy of PTH secretion - hypertrophy and hyperplasia of the parathyroid glands (tertiary hyperparathyroidism (TNP) - calcemia, PTH, bone turnover indicators). Hypoparathyroidism: 1) primary hypoparathyroidism - impaired synthesis (deficiency, lack of PTH), impaired PTH secretion, synthesis of inactive PTH molecules.</p> <p>2. Disorders of calcium metabolism (Part II). Symptoms of hypocalcemia: lowering the threshold of neuromuscular excitability (tetany, tetany equivalents [vascular, bronchial, ocular], latent tetany [Chvostek symptom, Troussseau symptom]), trophic changes (skin, nails and hair), psychological symptoms. Symptoms of hyperphosphataemia - calcifications in soft tissues. DiGeorge Syndrome; 2) secondary hypoparathyroidism - causes: PTH-independent hypercalcaemia, PTH-1 or calcium receptor mutation 3) pseudoparathyroidism - causes: PTH resistance of target tissues. Bone metabolic diseases. Primary osteoporosis: postmenopausal, senile - pathomechanism. Risk factors for the development of osteoporosis: genetic and demographic factors, factors related to nutrition and lifestyle (physical activity). Location changes. Symptoms of osteoporosis - low energy fractures, chronic consequences of fractures. Secondary osteoporosis. Osteomalacia: causes, bone and skeletal muscle symptoms. Rickets - clinical manifestations of rickets in infants, young children and older children: "Crooked Rosary", Harrison's furrow, dust and bell-shaped chest, lopsided and deformed knees.</p> <p>. Adrenal gland disease - Adrenal insufficiency. Chronic hypoadrenocorticism: 1) Primary hypoadrenocorticism (Addison's disease) - causes, signs and symptoms. Acute adrenal insufficiency (adrenal crisis): 1) in the course of chronic adrenal insufficiency (stress exposure); 2) as a result of damage to healthy adrenal glands (haemorrhage - Waterhouse and Friderichsen syndrome). Secondary adrenal insufficiency: ACTH deficiency (pituitary or hypothalamic damage), chronic steroid therapy. Diseases with excess of glucocorticosteroids (Cushing's syndrome): Endogenous Cushing's syndrome: 1) ACTH independent of ACTH (primary adrenal hyperfunction); 2) ACTH-dependent Cushing's syndrome (secondary adrenal hyperfunction): pituitary, ectopic secretion of ACTH or CRF. Exogenous Cushing's syndrome - treatment with glucocorticosteroids. Symptoms of Cushing's syndrome: central obesity, symptoms from skeletal muscles, skin, cardiovascular system, skeletal system, mental disorders, disorders of carbohydrate metabolism (impaired glucose tolerance, diabetes), lipid (atherogenic dyslipidemia), water-electrolyte. Diseases with excess of mineralocorticosteroids: primary hyperaldosteronism (Conn's syndrome) - causes, pathomechanism of hypertension. Acid-base balance disorders in the course of Conn's syndrome. Congenital adrenal hyperplasia from 21-hydroxylase deficiency - consequences: cortisol deficiency, no inhibition of ACTH secretion, aldosterone deficiency, excess of adrenal androgens (DHEA, androstenedione, testosterone). Androgenization: hirsutism and virilization, disorders of sex differentiation and puberty. Water-electrolyte disorders (salt loss syndrome); 2) congenital adrenal hyperplasia from 11β-hydroxylase deficiency - consequences: cortisol deficiency, excessive ACTH secretion, excessive androgen formation. Androgenization. Adrenal medulla hormones - adrenaline (epinephrine), noradrenaline (norepinephrine), dopamine - synthesis, metabolism. Metabolic effect of</p>			

catecholamines and their impact on the cardiovascular system. Pheochromocytoma.

4. Thyroid disease (Part I). Hyperthyroidism (hyperthyreosis) - endo- and exogenous causes. Symptoms of hyperthyroidism (thyrotoxicosis). Graves' disease - the importance of the autoimmune process (anti-TSH receptor [anti-TSHR]), pathogenesis of ophthalmopathy. Toxic nodular goiter (Plummer's disease) - the importance of iodine deficiency. Single autonomic nodule.

5. Thyroid disease (Part II). Hypothyroidism: primary, secondary, tertiary. Congenital and acquired hypothyroidism. Mucous edema. Autoimmune thyroiditis (Hashimoto's disease) - anti-peroxidase (anti-TPO) and anti-thyroglobulin (anti-Tg) antibodies. Neutral goiter: definition, causes, importance of iodine deficiency and excess (Wolff-Chaikoff effect).

6. Partial EXAM - I

7. Hypoxia. Respiratory changes: breath of Kussmaul, Cheyne-Stokes, Pickwick, Biot, agonal. Hemoglobin dissociation curve. Hypoxia, hypoxemia, hypercapnia, hyperoxia, anoxia. Hypoxia - definition, pathogenetic types of hypoxia. Respiratory, circulatory, blood-related, histotoxic and mixed hypoxia. Adaptive mechanisms in hypoxia. Symptoms of hypoxia. Shortness of breath - definition, classification, pathogenesis. Cyanosis - definition, types, differentiation.

8. Diseases of respiratory system (Part I). Respiratory failure: hypoxic and hypo-ventilation. Mechanisms leading to respiratory failure. Obstructive and restrictive ventilation disorders, differentiation of restriction and obstruction in lung function tests. Pulmonary and extrapulmonary causes leading to ventilation disorders. Lung perfusion disorders. Respiratory gas diffusion disorders. Symptoms of respiratory failure. Complications of respiratory failure - pulmonary hypertension, chronic pulmonary heart. Adult respiratory distress syndrome (ARDS; non-cardiogenic pulmonary edema). Pulmonary edema - mechanisms and symptoms. Pulmonary hypertension. Pulmonary embolism. Pneumothorax.

9. Diseases of respiratory system (Part II). Obstructive pulmonary diseases: Bronchial asthma. Acute and chronic obstruction. Bronchial hyperreactivity. Asthma classification. Asthmatic condition. "Aspirin" asthma. Spirometry tests in bronchial asthma. COPD - definition. Etiology of chronic bronchitis and emphysema. Pathomechanism responsible for structural changes in the respiratory tract in the course of COPD. COPD picture - "pink blower", "blue panther". Functional tests in the diagnosis of COPD. Obstructive apnea and central apnea. Neurohormonal reactions. Daytime symptoms, nighttime symptoms. Cardiovascular complications.

10. Lipid disorders and atherosclerosis. Hyperlipidemia: hypercholesterolemia, hypertri-glyceridemia, mixed hyperlipidemia. Hypercholesterolemia. Primary hypercholesterolemia: 1) Family hypercholesterolemia (FH): mutation in the LDL receptor gene, apoB100 gene; 2) Multi-gene hypercholesterolemia. Jaundice, premature symptoms of atherosclerosis and associated diseases. Secondary hypercholesterolemia: hypothyroidism, cholestasis, nephrotic syndrome. Hypertriglyceridaemia: 1) common hypertriglyceridaemia; 2) familial hypertriglyceridaemia; 3) familial complex hyperlipidemia. Atherogenic dyslipidemia. Atherogenic lipid triad: ↑ TG, ↓ HDL-C, ↑ sdLDL. Chylomicronemia syndrome: 1) primary; 2) secondary (diabetes, alcohol, obesity). Hypolipoproteinemia. 1) Tangier disease (↓ apoAI, ↓ ABC, ↓ HDL); Abetalipoproteinemia (↓ apoB, ↓ TG, ↓ TC, acanthocytosis); 3) familial LCAT deficiency. Atherosclerosis as a chronic inflammatory disease. Risk factors for atherosclerosis (modifiable, non-modifiable). Endothelial dysfunction: 1) factors initiating endothelial damage (metabolic, hemodynamic, immunological, humoral); 2) vascular wall regulation disorder (↓ NO, ↓ PGI2, ↑ ET1, ↑ TXA2) increase in leukocyte adhesion and transmigration (↑ MCP-1, ↑ VCAM-1, ↑ M-CSF), increase in release of growth factors (FGF, PDGF), smooth muscle migration and transformation (PDGF, ET1, AngII, thrombin), LDL modification (MM-LDL, oxy-LDL). Foam cell formation (scavenging receptors - classes A, CD36, LOX1). 2. Atherosclerotic phases: 1) fat infiltration, 2) atherosclerotic plaque: structure of the atherosclerotic plaque (cover, lipid core, inflammatory infiltrate). Stable and unstable atherosclerotic plaque. Plaque evolution: neovascularization, calcification, rupture (↑ MMP), detachment.

11. Ischemic heart disease (Part I). Hypoxia-induced myocardial metabolism and function disorders: reduced ATP generation, metabolic acidosis, ion pump dysfunction, oxidative stress. Hypoxia (ischemic preconditioning), "stunned" and frozen myocardium - pathomechanisms. Ischemic heart disease - definition, causes. Coronary artery disease. Stage of coronary artery changes: significant (subcritical) and critical stenosis. Coronary reserve. Coronary artery disease: 1) stable coronary syndromes (stable angina pectoris, Prinzmetal angina pectoris, cardiac syndrome X, coronary sternal angina); 2) acute coronary syndromes (unstable angina [unstable coronary artery disease], myocardial infarction, sudden cardiac death). Pathomechanism of ischemia - coronary vasoconstriction, adrenergic stimulation. Features of angina pain (character, location, duration, provoking factors, resolution). Equivalent to angina pectoris. Mute myocardial ischemia. Acute coronary syndromes. Factors leading to the formation of a blood clot on the atherosclerotic plaque. Atherosclerotic plaque damage: 1) erosion, rupture 2) damaging factors.

12. Ischemic heart disease (Part I). Factors leading to ischemia progression: occlusion time, impaired endogenous fibrinolysis, increased coagulation, increased ATP demand, reduced O₂ supply. Unstable angina pectoris (unstable coronary artery disease). Features of pain in unstable angina. Categories of pain - angina at rest, newly formed, increasing. Myocardial infarction - definition, types: without ST elevation and with ST elevation (NSTEMI, STEMI),

with and without Q wave (NQ-MI, Q-MI). Cellular mechanisms of myocardial infarction: the role of neutrophils, preservation of K⁺, Na⁺, and Ca⁺⁺ ions. Markers of cardiomyocyte necrosis (troponin, CK-MB, myoglobin). Pathophysiological basis of ECG changes in the course of myocardial infarction. Acute and chronic complications of myocardial infarction. Postinfarction heart remodeling.

13. Renin-angiotensin-aldosterone system and cardiac arrhythmias. Tachyarrhythmias - pathomechanism: increased automatism of the sinoatrial node, increased automatism of ectopic centers, automatism triggered by early and late follow-up depolarization, re-entry phenomenon. Bradyarrhythmias - pathomechanism: reduced automatism of the sinoatrial node, nodal and ventricular replacement rhythms, wandering pacemaker. Sinus node dysfunction: sick sinus syndrome, bradycardia-tachycardia syndrome. Atrioventricular (AV) block: AV I° block, Wenckebach type II II block (Mobitz I), Mobitz II type II AV block, III block AV. Intraventricular blocks: right bundle branch block, left bundle branch block (duration and morphology of QRS complexes). Supraventricular arrhythmias. Additional supraventricular beats (premature, surrogate) - changes in P wave morphology. Supraventricular tachycardia: atrial tachycardia [AT], AV relapsed tachycardia [AVNRT], atrioventricular recurrent tachycardia [AVRT] (changes in P wave and RP interval). Wolf-Parkinson-White syndrome (WPW). Symptoms of ventricular pre-excitation (shortening of PQ, widening of the QRS complex, delta wave). Atrial flutter (flutter wave). Atrial fibrillation (wave 'f', irregular rhythm of the ventricles). Types of atrial fibrillation: paroxysmal, persistent. Ventricular arrhythmias. Additional (premature) ventricular beats (PVC) - single, steam, salvo. Ventricular tachycardia (VT). Classification of ventricular rhythms: ventricular tachycardia, accelerated idioventricular rhythm, mono-multiform tachycardia. Torsade de pointes tachycardia. QT segment elongation - causes. Morgagni-Adams-Stokes Syndrome (MAS) - sudden cardiac arrest: ventricular fibrillation, asystole. PEA - pulseless electrical activity.

14. Shock: Pathogenesis of shock: oligovolemic, cardiogenic and distributive shock (anaphylactic, septic, toxic, neurogenic). Shock Phases. Neuroendocrine mechanisms responsible for hemodynamic changes in shock. The concept of centralization of circulation and "autotransfusion". Organ complications in shock: ARDS syndrome, DIC, acute kidney damage, acute liver failure, gastrointestinal bleeding. Metabolic and electrolyte changes in shock. Criteria for the diagnosis of developed shock. Septic shock - etiology, inflammatory mediators responsible for vascular and thrombotic reactions in septic shock. Hyper- and hypodynamic phase of septic shock.

15. Partial EXAM - II

Laboratory classes:

1. Orientation to course. Syllabus review. Introduction to pathophysiology.

Introduction to endocrinology. Diseases of the hypothalamic-pituitary system and gonads. Primary, secondary and tertiary hypothyroidism and overactive endocrine glands. Ectopic hormone secretion. ACTH deficiency and excessive ACTH secretion. GH deficiency: causes, symptoms in childhood and adulthood. Tissue resistance to GH (Laron's dwarfism). Excessive GH secretion: causes of symptoms in children and adults. Hyperprolactinemia: causes, symptoms (hypogonadism). Panhypopituitarism. Sheehan syndrome. Gliński-Simmonds syndrome. Maturation Disorders. Premature puberty: 1) central origin (GnRH-dependent); 2) peripheral (GnRH-independent). Delayed puberty: 1) hypogonadotropic hypogonadism; 2) hypergonadotropic hypogonadism. Menstrual disorders. Primary amenorrhea - causes. Feminizing testicles syndrome. Secondary amenorrhea. Polycyclic ovary syndrome (PCOS). Dysmenorrhea. Menopause.

2. Eating disorders. Malnutrition - definition, diagnosis, pathogenesis. Clinical signs of malnutrition. Anorexia nervosa - characteristics, hypothalamic-pituitary axis disorders. Bulimia - characteristics. Gluttonous food. Obesity - exo- and endogenous obesity. Localization of fat tissue. Central obesity (male type). Hip-femoral obesity (female type). Metabolic syndrome (cardiovascular risk syndrome). Components of the metabolic syndrome. The role of visceral fat in the development of insulin resistance: free fatty acids (FFA), TNF-alpha, IL-6, leptin, adiponectin, resistin. Effects of insulin resistance in the liver, skeletal muscles and adipose tissue. Diagnosis of insulin resistance. Glucose homeostasis.

3. Diabetes mellitus. Etiopathogenetic classification of diabetes: type 1 diabetes (DMT1), type 2 diabetes (DMT2), other types of diabetes, gestational diabetes. Autoimmune late-onset adult diabetes (LADA). Adult diabetes in young people (MODY). Type 1 diabetes - pathogenesis, the role of genetic and environmental factors (infectious, nutritional). Pathophysiological foundations of therapeutic management in DMT1 (insulin therapy). Type 2 diabetes - pathogenesis, the importance of genetic and environmental factors (obesity, aging, physical activity). DMT2 pathomechanism: insulin resistance, beta cell dysfunction (changes in insulin secretion). The natural course of DMT2 (progression of changes): compensatory hyperinsulinemia, insufficient insulin secretion, impaired glucose homeostasis. Pathophysiological basis of therapeutic management in DMT2 (oral antidiabetic drugs). Incorrect glucose tolerance (IGT). Retrospective indicators of glycemic control (HbA1c, fructosamine). Metabolic disorders due to insulin secretion / defect: hyperglycaemia, atherogenic dyslipidemia, protein synthesis disorder, electrolyte disorder. Hyperglycemia - metabolic consequences: toxic effects of glucose, nonenzymatic glycation of proteins, activation of the sorbitol pathway, oxidative stress. Pathophysiological signs of hyperglycemia: polyuria (osmotic diuresis, sodium-glucose transporters - SGLT-2, SGLT-1), polyphagia, polydipsia. Chronic complications of

diabetes: 1) microangiopathic complications: retinopathy, nephropathy, 2) macroangiopathic complications (dynamic atherosclerosis). Diabetic neuropathy. Diabetic foot syndrome. Acute complications of diabetes. Ketoacidosis, hyperglycemic-hypermolar condition, lactic acidosis. Conditions that favor acute diabetic complications. Hypoglycemia: mild, moderate, severe. Symptoms of hypoglycemia: neurovegetative, neuroglycopenia. The importance of hormonal counterregulatory mechanisms. Impact of hypoglycemia on the cardiovascular system. Recognition criteria. Pathophysiological foundations of therapeutic management.

4. Water and electrolyte disturbances. Water-electrolyte disorders associated with excessive and insufficient vasopressin release (SIADH, diabetes insipidus). Iso-, hypo- and hypertonic dehydration (clinical causes and symptoms). Symptoms of dehydration. Orthostatic hypotension. Iso-, hypo- and hypertonic over-hydration. Over-hydration symptoms. Local and generalized edema. Edema pathomechanisms: hydrostatic, oncotic, increase of vascular wall permeability, disturbance of lymph outflow. Pathogenesis of edema in cardiovascular disease (venous stasis, decrease in effective arterial blood volume, "cardiac cirrhosis"). Pathogenesis of edema in liver diseases (portal hypertension, visceral vasodilation, relative hyperaldosteronism). Hepato-renal syndrome. Pathogenesis of edema in kidney diseases. Nephritic syndrome. Nephrotic syndrome: protein and lipid disorders. Edema associated with endocrine and metabolic disorders. Sodium disorders: Causes and symptoms of hyper- and hyponatraemia. Potassium metabolism disorders: causes and symptoms of hyper- and hypo-kalemia. Impact of electrolyte disorders on cardiovascular function.

5. Acid-base disturbance: Electrolyte disturbances (H^+ , Na^+ , K^+) associated with acid-base imbalance. Respiratory acidosis: acute, chronic. Metabolic acidosis: additive, retention, subtractive - causes and symptoms. Lactic and ketoacidosis. Adjusted and unadjusted changes in acid-base disorders. Respiratory and metabolic alkalosis - causes and symptoms. Adjusted and unadjusted changes in acid-base disturbances.

6. Acute kidney impairment and chronic renal failure. Acute kidney impairment (AKI) - definition, prerenal, renal and non-renal causes. Diagnosis of AKI: prerenal AKI: causes of relative and absolute hypovolaemia leading to acute prerenal damage. Clinical and laboratory symptoms of acute renal uremia. Renal AKI: exo- and endogenous nephrotoxins. Renal AKI pathogenesis: tubular cell damage, intraurethral roller formation, "kidney soaking into the kidney", tubulo-glomerular reflex. AKI in rhabdomyolysis - pathomechanism. AKI in the course of glomerulonephritis and interstitial nephritis. Clinical manifestation of acute renal uremia. Differentiation of prerenal and renal AKI. Non-renal AKI - causes of urinary tract obstruction, pathomechanism. Chronic kidney disease (CKD) - pathogenesis, criteria for diagnosis. The "intact nephron" theory. Factors accelerating the progression of kidney diseases. Impact of CKD on the hematopoietic, circulatory, skeletal, nervous, digestive, endocrine, skin and mucous membranes systems. Clinical picture of uremia. "Uremic toxins". Chronic renal failure end stage renal disease - diagnosis (GFR). Proteinuria - diagnostic criteria (albuminuria, proteinuria), classification: causes of pre-glomerular, glomerular and extra-glomerular proteinuria. Urolithiasis.

7. Inflammation: Inflammatory reaction in the human body - definition of inflammation. Exogenous and endogenous inflammatory factors. Stages of inflammatory response. External and internal factors leading to cell and tissue damage. Local symptoms and systemic inflammation. Vascular reactions in inflammation. Mechanisms of expansion and increase of vascular permeability in an inflammatory focus. Inflammatory exudate and its composition. Hemodynamics of exudate formation. Inflammatory infiltrate. Mechanisms initiating inflammatory infiltrate. Stages of leukocyte migration: adhesion, diapedesis, chemotaxis. Adhesive proteins: selectins, integrins, immunoglobulin superfamily. Endo- and exogenous chemotactic substances. The role of bacterial lipopolysaccharides (LPS) in inflammation. Angiogenesis. Phagocytosis. Opsonization. Specific and non-specific opsonins. Bactericidal mechanisms of phagocytes. Oxygen explosion of phagocytes. Oxygen free radicals. NADPH oxidase, myeloperoxidase, inducible nitric oxide synthase (iNOS). Peroxynitrite. Antioxidative defense of the system. Antioxidant enzymes: superoxide dismutase (SOD), catalase (CAT) glutathione peroxidase (GPx). Non-enzymatic antioxidants. Oxygen-independent bactericidal mechanisms: cathepsins, defensins, lysozyme, the main cationic protein. Cellular mediators of inflammation. Mediators preformed and generated after the activation of the inflammatory stimulus. Serotonin, histamine, prostaglandins, prostacyclin, thromboxanes, leukotrienes, lipoxins, platelet activating factor (PAF). Cyclooxygenases (COX1 and COX2), phospholipase A2. The importance of omega-3 acids in silencing the inflammatory process. Protectins, resolvins, maresins. Cytokines - sources, action. The role of interleukin 6 (IL-6), tumor necrosis factor (TNF) and interferon γ (INF γ) in inflammation. Growth factors and chemokines. Extracellular inflammatory mediators. Kinin system: kinins, kininogens, kininogenases, kininases, convertases. Complement system. Classic and alternative ways to activate the complement system. The role of active components (C3a, C4a, C5a, C3b, C4b) in the inflammatory reaction. Membrane attack complex (MAC). Local signs of inflammation: redness, warming, swelling, pain and functional impairment. Pathomechanisms of local signs of inflammation. The role of inflammatory mediators. Acute phase reaction. Acute phase proteins: C-reactive protein (CRP), serum amyloid A (SAA). Criteria for the distribution of inflammation. Acute, subacute and chronic inflammation. Normo-, hypo- and hyper-ergic inflammation. Positive and negative hypoergia. Acute and chronic complications of inflammation. Septic shock. Spreading inflammation. Specific and non-specific inflammations. Tissue regeneration and repair. Wound healing. Thermoregulation and basic thermoregulation disorders (fever,

hyperthermia, hypothermia).

8. Hypersensitivity: Immune tolerance. Reasons for breaking the immune tolerance. Autoantigens. Effector mechanisms of autoaggression: cellular, humoral. Autoantibodies. Examples of autoimmune diseases. Hypersensitivity. Definition of hypersensitivity. Types of hypersensitivity reactions (according to Gell and Coombs). Type I hypersensitivity reaction - anaphylaxis, atopy, reagins, anaphylactoxins, anaphylactogens. Genetic and environmental factors conditioning excessive IgE production. Stages of anaphylactic reaction. The role of cells in anaphylaxis. Anaphylaxis mediators. Atopic diseases. Diagnosis of atopy. Anaphylactic shock. Quincke's edema - prevalence, pathogenesis. Hypersensitivity reaction type II. Location of surface antigens on blood cells and organs. Factors responsible for the formation of neoantigens. Pathomechanisms of type II allergic reactions. The role of complement system and antibody dependent cytotoxicity (ADCC). Serological conflict, post-transfusion reaction, muscle fatigue, drug-induced cytopenia. Type III hypersensitivity reactions. Types of immune complexes - with excess of antigens, with excess of antibodies. Factors conditioning survival of immune complexes. Extra- and intrinsic antigens in pathological immune complexes. Effector mechanisms in immune complex reactions. Experimental models - serum sickness, Arthus phenomenon. Examples of diseases developing in the mechanism of type III allergic reaction. Type IV allergic reactions. The role of cellular response in late-type reaction. Mantoux reaction. Allergic contact dermatitis.

9. Hemostatic disorders: Hypercoagulability. Pathogenesis of vascular thrombi: 1) factors causing vascular endothelial damage; 2) factors causing blood coagulability (congenital, acquired thrombophilia); 3) factors causing blood flow disorder. Venous and arterial blood clots - complications. Embolism. Embolic materials. Congestion wandering. Congestion complications. Deep vein thrombosis. Risk factors for deep vein thrombosis. Complications of deep vein thrombosis: pulmonary embolism, post-thrombotic syndrome. Disseminated intravascular coagulation (DIC) syndrome. DIC initiating factors, phases, forms. The role of the coagulation system, fibrinolysis. Multi-organ complications in the DIC syndrome. Plasma haemorrhagic diathesis: 1) congenital - hemophilia A (\downarrow part VIII), hemophilia B (\downarrow part IX), von Willebrand's disease (\downarrow vWF, \downarrow VIII); 2) acquired disorders of plasma coagulation impaired synthesis / II, VII, IX, X ~ vitamin K). Platelet haemorrhagic diathesis: Thrombocytopenia: 1) central - congenital, acquired; 2) peripheral - immunological, non-immunological. Platelet dysfunction: 1) congenital (Glanzmann's thrombasthenia); 2) acquired (drug / aspirin). Vascular haemorrhagic diathesis: 1) congenital; 2) acquired (Henoch and Schenlein purpura, purpura caused by acquired vascular wall disorders (GKS excess, C avitaminosis).

10. Anemia: Etiopathogenesis of anemia: hemorrhagic, aplastic, sideroblastic, (deficiency of iron deficiency, vitamin B12, folic acid), hemolytic, secondary in the course of various diseases. Division based on the morphological features of erythrocytes: microcytes, normocytes, macrocytes. General and specific symptoms of anemia. Iron deficiency. Absorption, transport and storage of iron in the body - the role of DMT1 and HFE protein, ferroportin, hepcidin, hemojuvillin, transferrin, ferritin, lactoferrin, transferrin receptors (TfR). Symptoms of iron deficiency. Assessment of iron management: serum iron, iron transferrin saturation (Tfs), total iron binding capacity (TIBC), soluble transferrin receptors (sTfR), ferritin concentration (F). Megaloblastic anemia. Causes of vitamin B12 and folic acid deficiency. Addison's and Birmer's disease. Symptoms of vitamin B12 deficiency not associated with anemia. Hemolytic anemia: ovalocytosis, nocturnal paroxysmal hemoglobinuria. Hemolytic enzymes. Hemoglobinopathies: sickle cell disease, thalassemia. Symptomatic anemia in the course of chronic diseases, kidney diseases, alcoholism, endocrine disorders. Hypersensitivity (polycythemia): 1) polycythemia vera; 2) secondary polycythemia - associated with hypoxia, not associated with hypoxia. The role of erythropoietin (EPO).

11. Liver diseases. Markers of hepatocyte damage (ALT, AST, GGTP), indicators of hepatocyte dysfunction (albumin, coagulation factors, cholinesterase), cholestasis indicators (AP, GGTP). Cirrhosis - causes, classification. Symptoms of cirrhosis: 1) associated with loss of hepatocyte function, 2) associated with portal hypertension and portal-systemic collateral circulation (esophageal varices, hypersplenism, ascites, hepatorenal syndrome, hepatopulmonary syndrome, portal encephalopathy). Congenital liver disease: hemochromatosis, Wilson's disease. Autoimmune liver disease. Steatosis - definition, pathogenesis, etiological factors. Non-alcoholic fatty liver disease (NAFLD). Non-alcoholic fatty hepatitis (NASH). Alcoholic liver disease - steatosis, inflammation, cirrhosis. The role of acetaldehyde, free fatty acids, oxidative stress, proinflammatory cytokines. Functional and anatomical cholestasis. Clinical and laboratory symptoms of cholestasis. Jaundice: hemolytic, hepatic, cholestatic - etiopathogenesis, differentiation. Functional jaundice: Gilbert, Rotor, Dubin-Johnson, Crigler-Najjar syndrome types I and II. Neonatal jaundice. Kernicterus. Hepatitis types A, B and C. HBV serological markers, "Serological window". Seroconversion in the HBs and HBe systems. HCV infection.

12. Diseases of the exocrine pancreas and GI tract. Etiopathogenesis of acute pancreatitis (ACS) - factors leading to intra-pancreatic activation of digestive enzymes. Chronic pancreatitis - definition, etiological factors, pathomechanism. Pancreatic neoplasms: 1) neuroendocrine (carcinoid, insulinoma, gastrinoma [Zollinger-Ellison syndrome], VIP-oma, glucagonoma); 2) hormonally inactive. Diseases of the esophagus, stomach, intestines. Gastroesophageal Reflux Disease (GERD). Anti-reflux barrier. Reasons for reflux - functional and anatomical disorders, the importance of diet, medications taken and associated diseases. Barrett's esophagus. Gastric and

duodenal ulcer. Primary and secondary factors damaging the gastric and duodenal mucosa. Helicobacter pylori infection. Clinical consequences of infection - asymptomatic infection, mucositis, ulcer, cancer, lymphoma; relationship between bacterial biology and settlement location; the meaning of VacA, CagA. The effect of nonsteroidal anti-inflammatory drugs (NSAIDs) on gastric mucosal homeostasis - direct and indirect effects. Symptoms and complications of peptic ulcer disease. Functional dyspepsia - symptoms, causes, differentiation. Disorders of digestion and absorption of nutrients: carbohydrates, proteins, fats. Celiac disease: pathogenesis - the importance of genetic (HLA) and environmental factors (gluten - gliadin, secalin, hordein, avenin), intestinal and parenteral symptoms, endomysial antibodies (EMA), anti-tissue transglutaminase (tTG). Bacterial overgrowth in the small intestine - causes, symptoms. Non-inflammatory bowel diseases: 1) ulcerative colitis (UC), 2) Crohn's disease (CD). Location of lesions, UC and CD complications. Diarrhea - acute and chronic; osmotic, motor, secretory. Upper and lower gastrointestinal bleeding - symptoms, differentiation.

13. Hypertension. Hypertension - definition and classification. Optimal pressure, normal, high normal, hypertension grade 1, 2 and 3. Isolated systolic hypertension. Primary hypertension. Pathogenesis of primary hypertension. Renal, vascular and neural mechanisms in hypertension. The role of genetic and environmental factors (salt intake, physical activity, obesity). Secondary hypertension: 1) kidney disease: parenchymal disease, vascular renal hypertension; 2) endocrinopathies: primary hyperaldosteronism (Conn's syndrome), Cushing's syndrome, chromo-lymphoma, hyperthyroidism and hypothyroidism, hyperparathyroidism; 3) obstructive sleep apnea syndrome (OSA); 4) obesity / metabolic syndrome; 5) coarctation of the aorta. Complications of hypertension: cardiovascular, neurological, ocular, renal. Malignant hypertension. Critical hypertension. Hypertensive crisis.

14. Heart failure. Cardiac failure (heart failure) - types of heart failure: chronic heart failure, acute heart failure, left ventricular failure, right ventricular failure, double ventricular failure, systolic failure, diastolic failure. Cardiogenic shock. Pathophysiological mechanisms leading to heart failure: 1) primary contractile impairment 2) increased preload (volume overload), 3) increased afterload (pressure overload), 4) diastolic impairment, 5) cardiac arrhythmias. Compensatory mechanisms in circulatory failure: 1) hemodynamic / myocardial hypertrophy: eccentric (volume overload), concentric (pressure overload); 2) neurohormonal mechanisms: sympathetic system, RAAS system, natriuretic peptides (ANP, BNP), vasopressin. Progression of chronic heart failure. Acute heart failure: 1) acute left ventricular failure: factors causing a sudden decrease in left ventricular ejection volume, pathomechanism of changes. Pulmonary edema of cardiac origin. 2) acute right ventricular failure: factors leading to an increase in pulmonary artery pressure, impaired diastolic and right ventricular systolic function. Acute right ventricular overload, impaired left ventricular filling, decreased discharge capacity.

15. Neurological and psychiatric disorders. Schizophrenia. Psychopathological symptoms of schizophrenia - positive symptoms, negative symptoms, cognitive deficits, affective symptoms, disorganization. Pathophysiological causes of schizophrenia: (a) disturbed balance of neurotransmitters in the CNS - dopamine, serotonin, glutamate theories; (b) genetic factors; (c) environmental factors; (d) psychosocial factors; (e) neurodevelopmental theory; (f) neurodegenerative theory; (g) inflammatory theory (leaky bowel syndrome, IgG-dependent hypersensitivity, the role of gluten-free diet). Pathophysiological basis of anti-psychotic treatment.

Depression: etiopathogenesis of depression: the role of hypothalamic-pituitary-adrenal axis dysfunction. Monoamine deficiency hypothesis (dysfunction of the central noradrenergic, serotonergic and dopaminergic systems). Neurotrophic hypothesis of depression (neurotoxicity, neurotrophic factors, neurogenesis). The role of brain-derived neurotrophic factor (BDNF). Changes in glutaminergic and GABAergic neurotransmission. Disorders of circadian rhythms.

Epilepsy: etiological classification of epilepsy: symptomatic, idiopathic, cryptogenic, genetic, metabolic, of unknown etiology. Theories of epileptic seizures: 1) the importance of impaired neurotransmission mechanisms: the role of γ-aminobutyric acid (GABA), GABA_A and GABA_B receptors. The importance of glutamic and aspartic acid. NMDA receptors, AMPA, KA, mGluR. Channelopathies: the role of sodium, calcium and potassium channels in the formation of epileptic seizures.

Parkinson's disease (PD) and Parkinson's syndrome. Pre- and postsynaptic parkinsonism. Pathogenesis of PD - the role of the ubiquitin-proteasome system, mitochondrial dysfunction, oxidative stress and lipid peroxidation. Lewy body, α-synuclein. The importance of genetic and environmental factors in the development of PD. Gene mutation for kinase 2, mitochondrial kinase, DJ-1 protein, ubiquitin hydroxylase.

Alzheimer's disease (AD) and etiological classification of dementia syndromes: degenerative, vascular, metabolic, inflammatory dementia. Pathogenesis of AD: the amyloid cascade hypothesis. Neurofiber degeneration. Tau protein. Cholinergic transmission disorder. Risk factors for AD.

Stroke. General characteristics of CNS vascular disorders. Ischemic stroke. Embolic material. Cellular changes in the focus of ischemia: necrosis, apoptosis. The importance of astrocytes and microglia cells in an ischemic focus. Penumbra. Transient Ischemic Attack (TIA). Hemorrhagic stroke (intracerebral hemorrhage). Risk factors for intracranial hemorrhage. Hydrocephalus. Causes of hydrocephalus (fluid absorption disorder, excessive fluid production). Non-communicating hydrocephalus, communicating hydrocephalus. Signs of hydrocephalus before closing the cranial sutures and after closing the sutures. Increase in intracranial pressure.

Others (please specify): (self-study)

1. Excess and deficiency of vitamins and their impact on human health.
2. Minerals, trace elements and their impact on human health.
3. Pathophysiology of pregnancy and childbirth.
4. Pathophysiology of the aging process.

(unnecessary delete)

Obligatory literature for Lecture:

1. Robbins and Cotran Pathologic Basis of Disease – Kumar, Abbas, Aster, W.B. Saunders, 9th, 2015

Complementary literature for Lecture:

1. Harrison's Principles of Internal Medicine, McGraw-Hill, 17th Edition

Obligatory literature for Lab classes:

1. Robbins and Cotran Pathologic Basis of Disease – Kumar, Abbas, Aster, W.B. Saunders, 9th, 2015

Complementary literature for Lab classes:

1. Harrison's Principles of Internal Medicine, McGraw-Hill, 17th Edition

Requirements for didactic aids (e.g. laboratory, multimedia projector, others...)

1. Laptop
2. Multimedia projector
3. TV

Conditions for obtaining a credit for the subject:

Methods of evaluation: The overall course grade will be determined by the results of 2 partial exams, 1-2 quizzes, 1 in-class activity and 1 final comprehensive written exam, which verify if the student acquired the knowledge, of the information as stated in the syllabus. A passing score confirms the satisfactory fulfilment of course requirements and is based on student's class attendance and mid-semester grades.

Exams

- Questions for the exams will be drawn from reading, Lecture and lab activities.
- Regular classroom attendance, participation in class discussion and studying according to class objectives will contribute to student's success on the exams.
- In order to pass this course, each student must pass the final written examination with a grade of 60% or higher. In addition, each student must earn an overall grade of 60% or higher for each section of the course. There will be no exceptions to this rule.
- Exams must be taken on scheduled dates and times. If illness or emergencies prevent a student from meeting deadlines, the coordinator must be notified before the exam date. A different exam (format and/or questions) may be substituted for exams missed for any reason, potentially including a completely essay-based exam.
- Make-up work will be at the instructor's discretion.
- The use of electronic devices with electronic data bases is not permitted during written or oral exams.

Comprehensive Final Exam

- A comprehensive final exam for all Lecture and lab topics.
- Exam takes place during the end-of-term examination sessions.
- The student is informed about the criteria of evaluation before approaching the exam, additionally, the student has the right to have an insight into his/her paper within 7 days from the release of examination results. If the exam is conducted in the form of a test , there is one set of questions for all students taking it at the same time.
- The date of the comprehensive exam ought to be announced at least 4 weeks before the examination session and at least 10 days before the make-up session. If a student does not appear at the exam, it is recorded in the protocol as "absent". A justification ought to be handed in or sent to the Dean's office within 3 days from the exam date. In case of unexcused absence, the Dean writes unsatisfactory mark (fail). After receiving Dean's approval, the examiner sets a new date. The same rules apply to pass, make up and commission exams. Exam results are available within up to 7 days but no later than 3 days before the make-up exam.

Grading scale: Course grades are dependent upon meeting the learning objectives and completing course requirements. Each student will receive the actual grade earned based on graded activities according to the designated evaluation criteria and percentages. Scores and final course grades will not be rounded up.

The grading scale is listed below:

95-100%	5.0 (very good)
90-94%	4.5 (better than good)
80-89%	4.0 (good)
70-79%	3.5 (quite good)
60-69%	3.0 (satisfactory)
<60%	2.0 (unsatisfactory)

In case of retake, the grading scale is listed below:

95-100%	5.0 (very good)
90-94%	4.5 (better than good)
85-89%	4.0 (good)
80-84%	3.5 (quite good)
70-79%	3.0 (satisfactory)
<70%	2.0 (unsatisfactory)

Attendance: Attendance is required. Student must participate in Lecture and lab sessions according to the schedule. During the semester only 1 absence is possible. All excused absences from class must be reported. Participation performance will not be penalized for excused absences. In the case of absences with excuse in the form of Lectures, labs or practical classes, the content of classes the student missed shall be made up according to the schedule given by the instructor.

Tardiness: Students are expected to arrive at class on time. Students that arrive after class begins will not be permitted in the classroom until the break. 3 tardiness will be considered 1 complete absence.

Uniforms: Students will be in uniform the first day of class. Students are expected to attend class in white lab coat and lab shoes or shoe covers (otherwise student won't be able to participate in the lab). Students are not permitted to wear heavy outside coats or jackets to any lab.

- Using mobile phones during the Lecture and labs is forbidden.

Missed exams/Assignments/Make-up policy: Student not present to take an assigned examination may receive a grade of zero (0) for that examination. The student may be allowed to make-up an examination under the following circumstances:

- Absence is due to serious illness/hospitalization of the student or an immediate family member. Documentation by a health care provider will be required at the time the student requests a make-up exam for the day of illness.
- Absence is due to family emergency, verified by a note from the professional person in attendance.
- Absence is due to a death in the immediate family. Documentation will be required.
- An absence that the faculty and/or Department Head deems as unavoidable.

To be eligible for a make-up exam in the above circumstances, the student must notify their instructor PRIOR to the absence, and must make arrangements within 48 hours after the absence for the retake. Faculty has the right to offer an alternative form of the exam.

The name and address of the department/clinic where the course is taught (module/course); contact details (phone number/ email address):

Department of Pathophysiology
Medical University of Lublin
8b Jacewskiego Street
20-090 Lublin
Office #: (+48) 81 448 6500

Names of the author/authors of this syllabus:

1. Prof. dr hab. Jarogniew J. Łuszczki

Names of the teacher/teachers conducting classes:

1. Prof. dr hab. Jerzy Belłowski
2. Dr n. med. Aleksandra Walczak
3. Dr n. med. Katarzyna Załuska
4. Dr n. med. Monika Dudra- Jastrzębska
5. Dr n. med. Marta Rusek

Signature of the head of the department/clinic

Dean's signature

KIEROWNIK
Katedry i Zakładu Patofizjologii
Uniwersytetu Medycznego w Lublinie

Prof. dr hab. Stanisław Czuczyński

Date of submission:

Katedra i Zakład Patofizjologii
Uniwersytetu Medycznego w Lublinie
20-090 Lublin, ul. Jacewskiego 8 b
tel. 81 448-65-00; 81 448 65 02
fax. 81 448 65 01

