Faculty of Medicine in Rijeka

Curriculum
2026/2027

For course

Physiology and Pathophysiology I

Study program:	**Medical Studies in English (R)** University integrated undergraduate and graduate study
Department:	**Department of Physiology, Immunology and Pathophysiology**
Course coordinator:	**prof. dr. sc. Mahmutefendić Lučin Hana, dipl. ing. biol.**
Year of study:	**2**
ECTS:	**5**
Incentive ECTS:	**0 (0.00%)**
Foreign language:	**Possibility of teaching in a foreign language**

Course information:

The course consists of 66 class hours (38 hours of lectures, 12 hours of seminars, and 16 hours of practicals) and is conducted in the third semester. The course is worth 5 ECTS credits. The main aim of this course is to enable students to apply previously acquired knowledge of physics, chemistry, biology, biochemistry, and normal morphology in order to gain an understanding of normal organism functions and pathophysiological mechanisms that lead to disturbances of normal function and the emergence of disease. Seminars and practicals are designed to prepare students for independent problem-solving and integrative reasoning about health and disease. Individual functions are explained at the molecular level, as well as at the level of the organism as a whole and analyzed in the context of the organism's adaptation to changing environmental conditions. The emphasis is on learning basic "applied" physiology, i.e., on vertically expanding knowledge acquired while explaining basic physiological functions.
Classes are delivered through lectures, seminars, and practicals. Active student participation in the curriculum is fostered through laboratory practicals and the use of computer programs that simulate pathological conditions and generate clinical correlates of specific diseases.
Throughout seminars and practicals, the student actively discusses physiological and pathophysiological mechanisms. The student is required to prepare the material that will be discussed in seminars and practicals. The teacher evaluates student participation throughout seminars (demonstrated knowledge, understanding, ability to formulate a problem, draw conclusions, etc.). The grade points earned are then added to the grade points obtained on the final exam.
Course content:
General concepts of physiology and pathophysiology: Homeostasis and environmental conditions. Physiology and pathophysiology of cell membranes: Transfer of substances through cell membranes. Channelopathies and membrane transport disorders. Membrane and action potential.
Cellular physiology and pathophysiology: Cellular homeostasis. Cellular stress. Energetic homeostasis and energy metabolic disorders. Hypoenergoses. Cellular adaptation, damage, and death. Malignant transformation and tumor growth. Regeneration and stem cells.
Physiology and pathophysiology of muscle cells: Neuromuscular junction. Muscle stimulation and contraction of skeletal muscle. Smooth muscle contraction.
Blood cells and clotting: Erythrocytes. Erythrocyte disorders. Blood types. Platelets, hemostasis, and blood clotting. Hemostasis disorders. White blood cells. White blood cell disorders. Inflammation, repair of damage, and wound healing.

List of assigned reading:

Required textbooks:

1. Lučin P, Mahmutefendić Lučin H, Gulić T. Physiology and Pathophysiology I. Textbook draft – working version, 2026. (available for download on the Merlin e-learning platform).
2. Hall JE, Hall ME. Guyton and Hall Textbook of Medical Physiology. 15th edition. Elsevier, 2026.
3. Gamulin S, Marušić M, Kovač Z. Pathophysiology (Textbook). Medicinska naklada, Zagreb, 2013.
4. Lučin P et al. Practical Exercises in Physiology and Pathophysiology I. Faculty of Medicine, University of Rijeka, 2024. (Available for download on the Merlin e-learning platform).
All materials that are not included in the compulsory reading will be published on the course website.

List of optional reading:

Recommended for additional reading:

1. Alberts B et al. Molecular Biology of the Cell. 7th edition. W. W. Norton & Company, New York, 2022.
2 Abbas AK, Lichtman AH, Pillai S, Henrickson S. Cellular and Molecular Immunology. 11th edition. Elsevier, 2026.
3. Kumar V, Abbas AK, Aster JC, Deyrup AT. Robbins & Kumar Basic Pathology. 11th edition. Elsevier, 2023.

Curriculum:

Lectures list (with titles and explanation):

Lecture 1. Homeostasis and internal environment.

After successfully mastering the material, the student will be able to:

- Define the concept of homeostasis and the importance of maintaining the stability of the internal environment (extracellular fluid).
- Explain the differences between negative feedback, positive feedback, and feed-forward control in the regulation of physiological processes.
- Describe the concept of diurnal (circadian) rhythms and their influence on the variability of physiological values.
- Recognize the concept of hysteresis as a phenomenon where the state of a system depends on its history.
- Demonstrate examples of the physiological interplay of different feedback loops in the systemic integration of the organism.
- Apply the concept of a "tipping point" to predict when physiological compensation ceases to be sufficient.
- Illustrate the difference between an acute response to a stimulus and chronic adaptation, which shifts the boundaries of homeostasis.
- Analyze disease as a pathophysiological disruption of homeostatic mechanisms.
- Compare the efficiency and biological purpose of negative versus positive feedback in maintaining the stability of the organism.
- Critically evaluate the limits of physiological compensation under conditions of chronic stress or pathological changes in the internal environment.

Lecture 2. Membrane transport. Passive and active transport across membranes.

After this lecture, the student will be able to:

- **Define** the concepts of passive transport (diffusion) and active transport across the cell membrane.
- **List** the subtypes of passive transport (simple diffusion through the bilayer, pores/aquaporins, and channels; facilitated diffusion) and active transport (primary and secondary active transport).
- **Identify** the key transport proteins (aquaporins, GLUT transporters, ion pumps, symport, and antiport carriers).
- **Explain** the role of the chemical (concentration) gradient as the driving force for passive transport, contrasted with the energy (ATP) requirement of active transport.
- **Describe** the mechanism of facilitated diffusion using the GLUT transporter model (conformational change of the carrier without energy expenditure).
- **Clarify** the physiological distinction between primary active transport and secondary active transport.
- **Describe** the difference between symport (co-transport) and antiport (counter-transport/exchangers) using concrete physiological examples.
- **Predict** the direction and type of transport for a given substance (e.g., oxygen, glucose, water, ions) based on its physicochemical properties (lipophilicity, charge, size) and concentration distribution across the membrane.
- **Apply** the concept of carrier saturation (saturation kinetics / V_{max}) to distinguish the linear dynamics of simple diffusion from the saturating dynamics of facilitated diffusion.
- **Compare and contrast** simple diffusion, facilitated diffusion, and active transport based on the following criteria: direction relative to the gradient, requirement of a specific protein, energy consumption, and susceptibility to saturation.
- **Analyze** how the pharmacological or pathophysiological inhibition of primary active transport (e.g., blocking the Na+/K+ pump) consequently impairs the function of secondary active transporters within the cell.

Lecture 3. Ion channels. Membrane potential.

After this the student will be able to:

- **List** the main types of ion channels based on selectivity (Na+, K+, Ca2+, Cl-) and gating mechanisms (voltage-gated channels and leak / *semi-leaking* channels).
- **State** the typical values of the resting membrane potential for a general model of a human cell.
- **Explain** the physical meaning of the Nernst potential as a state where the chemical (concentration) gradient and the electrical gradient for a single ion are in a dynamic equilibrium.
- **Describe** how selective membrane permeability (primarily through potassium leak channels) and the action of the electrogenic Ca2+ pump work together to generate the resting membrane potential.
- **Apply** the concept of the Nernst potential to predict the direction of ion movement (influx or efflux) if the actual membrane potential deviates from the equilibrium potential.
- **Compare and contrast** the role of continuously open leak channels, which dominate at rest, with the role of

voltage-gated channels, which are mostly closed at rest.
- **Correlate** the high relative membrane permeability to potassium at rest with the fact that the overall resting membrane potential is very close to the Nernst potential for potassium (Ca2+).
- **Critically assess** the importance of continuous metabolic energy expenditure (ATP) for maintaining the ionic gradients, without which the resting potential would collapse.

Lecture 4. Action potential. Membrane transport disorders.

After this lecture, the student will be able to:

- **Define** the action potential (AP), threshold potential, the "all-or-none" law, and membrane refractoriness.
- **List** and chronologically arrange the phases of the action potential (depolarization, repolarization, hyperpolarization).
- **Identify** the states of hypo- and hyperkalemia, hypo- and hypercalcemia, and hypo- and hypernatremia based on ion concentration alterations in the extracellular fluid.
- **Define** the term channelopathies as primary disorders of ion channels.
- **Explain** the ionic mechanisms underlying the phases of the AP (the gating kinetics of activation, closure, and inactivation of voltage-gated Na+ and K+ channels).
- **Clarify** why hyperkalemia causes initial membrane depolarization but chronically leads to the inactivation of Na+ channels and a loss of excitability.
- **Explain** the role of extracellular calcium as a "membrane stabilizer" through its effect on the activation threshold of voltage-gated Na+ channels.
- **Describe** how alterations in sodium concentration affect the osmotic gradient, cell volume, and the amplitude of the action potential upstroke.
- **Predict** alterations in the shape, amplitude, and slope of the action potential waveform under conditions of severe hyponatremia or hypernatremia.
- **Apply** the concepts of absolute and relative refractory periods to explain the unidirectional propagation of the action potential along the membrane and the maximum firing frequency of impulses.
- **Analyze and contrast** the effects of hypokalemia and hyperkalemia on the resting membrane potential and its distance from the threshold potential (excitability).
- **Correlate** the pathophysiological mechanism of hypocalcemia (lowering the Na+ channel activation threshold closer to the resting potential) with the clinical manifestation of spontaneous muscle contractions (tetany).
- **Differentiate** secondary electrophysiological disorders (caused by electrolyte imbalances) from primary genetic defects of ion channels (channelopathies).

Lecture 5. Physiology and pathophysiology of the cell - maintenance of internal homeostasis

After successfully mastering the material, the student will be able to:

- Explain the main cellular homeostatic mechanisms, including the regulation of pH, ionic balance (Ca^{2+}), and volume maintenance.
- Define the concept of proteostasis and the role of chaperones in ensuring proper protein folding.
- Describe the function of key cellular "sensors" and "controllers" (DDR, UPR) in recognizing protein and DNA damage.
- Recognize the basic metabolic pathways through which the cell maintains energy balance under normal and hypoxic conditions.
- Demonstrate how the cell detects and responds to stress (e.g., the accumulation of misfolded proteins via the UPR pathway).
- Apply knowledge of second messengers and signaling pathways to explain the integration of external signals into the intracellular response.
- Illustrate the phenomenon of metabolic reprogramming (e.g., the switch to anaerobic metabolism) under conditions of limited oxygen availability.
- Explain the importance of contact inhibition and adhesion for tissue organization and the prevention of pathological (tumor) growth.
- Analyze the causes and consequences of cellular homeostasis disruptions, linking them to the development of cellular stress.
- Compare the roles of DDR (DNA Damage Response) and UPR (Unfolded Protein Response) in the cell's decision between survival and programmed cell death.
- Synthesize knowledge of energy homeostasis to assess how systemic hypoxia affects the function of individual organs and tissues.
- Critically evaluate how the collapse of proteostasis contributes to the development of degenerative diseases.

Lecture 6. Cellular stress

After successfully mastering the material, the student will be able to:

Explain the fundamental processes of maintaining cellular homeostasis, including the regulation of pH, calcium concentration, and energy balance.
- Define the concept of proteostasis and explain the role of chaperones in ensuring the integrity of the cellular proteome.
- Recognize various forms of cellular stress (oxidative, ER stress, genotoxic, and metabolic stress) and their characteristic biomarkers.
- Describe the role of sensors and control mechanisms (e.g., DDR, UPR) in recognizing and responding to cellular stress.
- Demonstrate how "metabolic reprogramming" enables cellular survival under conditions of limited resources or hypoxia.
- Apply knowledge of signaling pathways to explain how cells integrate external stimuli with intracellular responses.
- Illustrate the consequences of impaired contact inhibition and adhesion on cellular organization.
- Analyze the link between prolonged (chronic) cellular stress and the loss of cellular function or the development of pathological states.
- Compare acute stress response mechanisms (e.g., temporary adaptation) with permanent changes in cellular phenotype.
- Synthesize information regarding cellular sensor systems to explain how a cell "decides" between repair processes (e.g., DDR response) and programmed cell death.
- Critically evaluate the importance of redox homeostasis and the impact of its disruption on overall cellular pathophysiology.

Lecture 7. Cellular adaptation, damage and death

After successfully mastering the material, the student will be able to:

- Define the basic forms of cellular adaptation to stress (atrophy, hypertrophy, hyperplasia, metaplasia).
- Explain the fundamental differences between apoptosis (programmed death) and necrosis (acute, uncontrolled death) and their respective molecular triggers.
- Describe the phenomenon of autophagy as a process that balances between maintaining cellular homeostasis (survival) and directing the cell toward death.
- Recognize the role of stem cells as the fundamental reservoir for the regenerative capacity of tissues.
- Demonstrate the cascade of events during ischemia-reperfusion injury and explain why reperfusion can further damage tissue.
- Apply the understanding of necroptosis as "programmed necrosis" in explaining certain inflammatory processes.
- Illustrate how the free radical theory links oxidative stress to the process of cellular aging (senescence).
- Analyze the signaling cascades that determine whether a stressed cell will survive, adapt, or be removed by apoptosis.
- Compare different mechanisms of cell death (apoptosis, necrosis, necroptosis) in the context of clinical consequences for the organism (e.g., inflammatory response vs. silent cell elimination).
- Synthesize knowledge regarding the function of stem cells to evaluate the potentials and limitations of regenerative medicine in treating chronic degenerative diseases.
- Critically evaluate the biological role of cellular aging (senescence) as a protective mechanism against the tumor transformation of cells.

Lecture 8. Malignant transformation and growth.

After successfully mastering the material, the student will be able to:

- Define the fundamental characteristics of tumor cells (the "hallmarks of cancer") that enable uncontrolled proliferation and survival.
- Explain the role of genomic instability in initiating tumor transformation.
- Describe the processes of angiogenesis (formation of new blood vessels) and metastasis as key factors in tumor progression.
- Recognize the importance of immune surveillance and the mechanisms by which tumor cells successfully evade it.
- Demonstrate tumor growth kinetics and explain the difference between the rate of cell division and the total volumetric increase of the tumor.
- Apply knowledge of cellular metabolism to explain the concept of "overcoming the energy barrier" in tumor cells.
- Illustrate the clinical significance of tumor cell differentiation in establishing a diagnosis and assessing disease prognosis.
- Analyze biological error as a process that leads from mutation to a clinical entity (tumor).
- Compare invasive growth with normal tissue adhesion and cell movement.

Critically evaluate the translational aspects of tumor biology, with a particular focus on the possibilities of gene therapy and personalized medicine.
- Synthesize theoretical knowledge of tumor molecular biology by participating in a simulated "Molecular Tumor Board," linking the tumor's genetic profile to therapeutic strategies.

Lecture 9. Molecular biomechanics of muscle contraction.

After successfully mastering the material, the student will be able to:

- Define the role of muscle as an electromechanical transducer in the context of converting ionic currents into mechanical force.
- Describe the architecture of the sarcomere, identifying key protein components (actin, myosin, troponin, tropomyosin) as the molecular blueprint of contraction.
- Explain the role of the triad (T-tubules and sarcoplasmic reticulum) as the structural link between the action potential and calcium release.
- Recognize the phases of the cross-bridge cycle and explain the role of ATP and calcium in regulating this process.
- Demonstrate the "length-tension" curve and explain how the overlap of actin and myosin filaments determines the force a muscle develops at different sarcomere lengths.
- Apply the concepts of isotonic and isometric contraction to examples from everyday physical activity (e.g., lifting a load vs. holding a load in a static position).
- Illustrate how changes in intracellular calcium concentration directly correlate with the activation of the contractile apparatus.
- Analyze the "molecular dance" of the cross-bridges as an energy-dependent cycle, assessing critical points where dysfunction may occur (e.g., ATP deficiency or calcium channel dysfunction).
- Critically evaluate the importance of optimal sarcomere length for physiological muscle efficiency, connecting molecular parameters with the macroscopic function of the muscular system.
- Synthesize knowledge of muscle cell structure to explain why the high organization of sarcomeres is a prerequisite for rapid and powerful contraction.

Lecture 10. Skeletal Muscle Excitation, Neuromuscular Transmission, Excitation-Contraction Coupling, Smooth Muscle Contraction, Energetics, and Pathophysiology.

After successfully mastering the material, the student will be able to:

- Explain the process of neuromuscular transmission, with a specific focus on the role of presynaptic mechanisms, neurotransmitter release, and the function of the postsynaptic membrane.
- Define the phenomenon of "quantal release" of acetylcholine and the role of acetylcholinesterase in regulating signal duration.
- Describe the differences between muscle fiber types (fast vs. slow-twitch) and their specific metabolic adaptations.
- Recognize the role of myoglobin in supplying oxygen to muscle during contraction.
- Demonstrate the role of calcium as the key mediator linking presynaptic depolarization with neurotransmitter release.
- Apply knowledge of energy systems (phosphocreatine, glycolysis, oxidative phosphorylation) to explain metabolic support during different types of muscle work (e.g., explosive power vs. endurance).
- Illustrate the development of tetanic contraction through stimulus summation and link this phenomenon to the physiological control of muscle force.
- Explain the molecular mechanism of smooth muscle activation (e.g., in blood vessels) and highlight the fundamental differences compared to skeletal muscle.
- Analyze the causes of muscle fatigue, distinguishing metabolic causes (ATP depletion, lactate accumulation) from signaling disturbances.
- Compare pathophysiological mechanisms in clinical cases of neuromuscular transmission disorders and muscle function impairment.
- Critically evaluate the links between the pathophysiology of excitable membranes and the onset of epileptic seizures (epilepsy as a disorder of systemic excitability).
- Synthesize translational aspects, connecting molecular defects in muscle proteins or channels with the clinical presentation of the disease.

Lecture 11. Hematopoiesis. Physiology of erythrocytes.

After this lecture, the student will be able to:

- **Define** the terms hematopoiesis and erythropoiesis and list the primary sites of blood cell production in the body across different life stages.
- **List** the developmental stages of erythrocytes (from stem cell to mature erythrocyte).

Identify the key structural components of the hemoglobin molecule.
- **Explain** the role of erythropoietin (EPO) and the feedback mechanism in the regulation of erythropoiesis.
- **Describe** the cycle of iron metabolism in the body, including its absorption, transport (transferrin), storage (ferritin, hemosiderin), and recycling.
- **Clarify** the role of hepcidin as the master regulator of iron homeostasis.
- **Apply** knowledge of hemoglobin structure and affinity to explain oxygen and carbon dioxide transport in different tissues (the Bohr effect).
- **Analyze** the impact of hypoxia at the cellular level (the role of HIF - hypoxia-inducible factor) and its effect on stimulating erythropoiesis.

Lecture 12. Blood types.

After this lecture, the student will be able to:

- **Define** the terms antigen (agglutinogen) and antibody (agglutinin) in the context of blood groups.
- **List** the four main blood groups of the ABO system and identify the presence or absence of the Rh factor (D-antigen).
- **Explain** the rule regarding the presence of naturally occurring antibodies in plasma (e.g., why a person with blood type A has anti-B antibodies).
- **Describe** the basic genetic inheritance pattern of the ABO system (codominance and recessiveness) and the Rh system.
- **Clarify** the process of agglutination as a specific antigen-antibody reaction.
- **Determine** blood compatibility between donors and recipients within the ABO and Rh systems.
- **Predict** the possible blood types of offspring based on the known blood types of the parents (simple genetic crosses).
- **Analyze** the basic pathophysiological sequence of events leading to hemolytic disease of the newborn (Rh incompatibility between an Rh-negative mother and an Rh-positive fetus).
- **Differentiate** between the concepts of a "universal donor" and a "universal recipient," noting the limitations of this concept in real clinical practice.
- **Evaluate** the potential dangers and the urgency of intervention in the event of administering ABO-incompatible blood (acute transfusion reaction).
- **Propose** a safe and rapid protocol for blood type selection in emergency transfusions when the patient's exact blood type has not yet been laboratory-confirmed.

Lecture 13. Red blood cells disorders.

After this lecture, the student will be able to:

- **Define** anemia and polycythemia and state their classification into four major etiopathogenetic groups.
- **List** the key laboratory parameters required for the evaluation of the red blood cell line (erythrocyte indices, reticulocytes, iron parameters, bilirubin, LDH).
- **Identify** the main representatives within the categories of hemoglobinopathies (e.g., thalassemias, sickle cell anemia) and hemolytic anemias.
- **Explain** the molecular and cellular mechanisms leading to impaired erythrocyte production (as seen in iron, vitamin B12, or folic acid deficiency).
- **Describe** the pathophysiological difference between quantitative disorders of hemoglobin synthesis (thalassemias) and qualitative disorders (structural variants such as hemoglobin S).
- **Clarify** the distinction between intravascular and extravascular hemolysis and explain why accelerated erythrocyte destruction causes a rise in unconjugated bilirubin and a drop in haptoglobin.
- **Outline** the basic concept of anemia caused by acute blood loss (outside the context of circulatory shock).
- **Differentiate** between the pathophysiological mechanisms leading to iron deficiency anemia versus anemia of chronic disease.
- **Analyze** the cause-and-effect relationship between a specific pathophysiological defect (e.g., iron deficiency vs. a defect in globin chain synthesis) and the patient's final laboratory profile.
- **Evaluate** the diagnostic value and specificity of individual laboratory markers (e.g., ferritin vs. serum iron, or bilirubin vs. LDH) when confirming suspected hemolysis versus a production defect.
- **Differentiate** the pathophysiological basis of primary polycythemia (Polycythemia vera) as a clonal disorder from secondary polycythemias (acting as a compensatory response to hypoxia).

Lecture 14. Hemostasis and blood clotting.

After this lecture, the student will be able to:

- **Define** the term hemostasis and **list** its four consecutive phases.
- **List** the key coagulation factors (physiological cofactors and zymogens) and the primary cells involved in the process (platelets, endothelial cells).

Identify the most important natural anticoagulants (e.g., antithrombin, protein C and S, TFPI) and clinical anticoagulants (heparin, warfarin, DOACs).
- **Explain** the role of local vascular spasm (vasoconstriction) and the mechanisms of platelet adhesion, activation, and aggregation during platelet plug formation.
- **Describe** the coagulation cascade model through the activation of the extrinsic, intrinsic, and common pathways leading to the conversion of prothrombin to thrombin and fibrinogen to fibrin.
- **Clarify** the process of clot retraction and its importance in stabilizing the clot and bringing the edges of the damaged vessel closer together.
- **Explain** the mechanisms by which natural anticoagulant systems prevent clot extension onto undamaged endothelium and how clinical anticoagulants interfere with these processes.
- **Predict** how the administration of specific drugs (such as aspirin as an antiplatelet agent or heparin as an anticoagulant) affects individual phases of hemostasis.
- **Analyze** the cause-and-effect relationships between a deficiency of a specific factor (e.g., Von Willebrand factor or factor VIII) and a defect in a specific phase of hemostasis (primary vs. secondary hemostasis).
- **Differentiate** between the mechanisms of action of natural anticoagulants and clinical pharmacological agents (e.g., how warfarin inhibits the hepatic synthesis of vitamin K-dependent factors, whereas heparin accelerates antithrombin activity).
- **Synthesize** a comprehensive conceptual map (schematic diagram) that integrates all four phases of hemostasis, illustrates their temporal overlap, and marks the exact sites of action of natural inhibitors and clinical anticoagulants.

Lecture 15. Hemostasis disorders.

After this lecture, the student will be able to:

- **Define** the terms thrombocytopenia, thrombocytopathy, coagulopathy, and vascular purpura.
- **List** the primary causes of quantitative platelet disorders (decreased production versus increased consumption or destruction).
- **Identify** hereditary coagulopathies (hemophilia A and B, von Willebrand disease) and key vascular disorders leading to bleeding (e.g., scurvy, senile purpura, hereditary hemorrhagic telangiectasia).
- **Explain** the pathophysiological mechanisms causing qualitative platelet disorders (thrombocytopathies, whether hereditary or acquired, such as the effect of aspirin).
- **Describe** the role of von Willebrand factor (vWF) as a crucial bridge between primary and secondary hemostasis, explaining why its deficiency impairs both processes.
- **Clarify** how alterations in blood vessel walls (vascular disorders) or perivascular connective tissue lead to increased bleeding despite normal platelet counts and coagulation factors.
- **Apply** knowledge of pathophysiology to differentiate clinical bleeding patterns: superficial bleeding (petechiae, purpura, ecchymoses, epistaxis) in platelet/vascular disorders versus deep tissue bleeding (hematomas, hemarthroses) in coagulopathies.

Lecture 16. White blood cells.

After this lecture, the student will be able to:

- **Define** the term leukopoiesis and state the primary sites of production for different types of white blood cells.
- **List** the five main types of leukocytes and classify them into granulocytes (neutrophils, eosinophils, basophils) and agranulocytes (lymphocytes, monocytes).
- **State** the normal reference values for the total leukocyte count and the standard percentage proportions in a healthy individual's differential blood count.
- **Describe** the lifespan and localization dynamics of leukocytes (the difference between the circulating pool, the marginal pool along vessel walls, and tissue localization).
- **Clarify** the core physiological roles of each specific leukocyte type (e.g., neutrophils and monocytes as professional phagocytes, eosinophils in anti-parasitic defense, basophils in inflammatory responses).
- **Interpret** a differential blood count report to identify abnormalities such as leukocytosis, leukopenia, neutrophilia, lymphocytosis, or eosinophilophilia.
- **Formulate** a temporal and spatial schematic diagram (the lifecycle) of a selected leukocyte type, integrating its production phase in the bone marrow, its transport and redistribution within the circulation, its emigration into tissues, and its functional execution up to its eventual destruction.

Lecture 17: White blood cells disorders.

After this lecture, the student will be able to:

- **Define** the terms leukocytosis, leukopenia, agranulocytosis, leukemia, and lymphoma.
- **List** the most common causes of reactive leukocytosis (neutrophilia, lymphocytosis, eosinophilia).
- **Classify** leukemias based on the rate of progression (acute vs. chronic) and the lineage of differentiation

(myeloid vs. lymphoid).
- **Explain** the pathophysiological difference between reactive leukocytosis (leukemoid reaction) and neoplastic proliferation in leukemias.
- **Describe** the mechanisms behind leukopenia/neutropenia development (decreased bone marrow production due to toxins/drugs vs. increased peripheral consumption or sequestration).
- **Relate** a finding of extreme leukopenia (agranulocytosis) to the immediate risk of developing opportunistic infections and sepsis in a patient.
- **Analyze** how the uncontrolled proliferation and accumulation of blast cells in the bone marrow leads to the suppression of normal hematopoiesis and subsequent bone marrow failure (pancytopenia: anemia, thrombocytopenia, infections).
- **Compare and contrast** the pathophysiological characteristics and clinical course of acute leukemias (non-functional blasts, abrupt onset) versus chronic leukemias (partially differentiated cells, indolent course).
- **Analyze** the cause-and-effect relationship between acute tissue damage and the subsequent mobilization of cells from the bone marrow (explaining the concept of a "left shift" through the appearance of immature/band neutrophils).
- **Relate** an isolated increase in a specific leukocyte type to a general type of stimulus (e.g., neutrophilia in acute bacterial inflammation, eosinophilia in an allergic reaction).

Lecture 18: Inflammation, tissue repair after damage, and wound healing.

After this lecture, the student will be able to:

- Define inflammation as a biological response of the organism to tissue damage and list its main purposes.
- Explain the difference between PAMPs (Pathogen-Associated Molecular Patterns) and DAMPs (Damage-Associated Molecular Patterns) in the initiation of the inflammatory response.
- Describe the role of PAMP/DAMP sensors and the inflammasome as "molecular switches" of inflammation.
- Recognize the key mediators of acute inflammation and the role of cytokines and chemokines as communication molecules of white blood cells.
- Outline the vascular response and the leukocyte recruitment cascade (margination, adhesion, diapedesis) as a mechanism for directing cells to the site of injury. *(Napomena: hrvatski pojam "rublje" odnosi se na margination/rolling)*
- Apply the understanding of chemotaxis to explain how leukocytes locate a specific target within the tissue.
- Illustrate the timeline of acute inflammation, from initiation to the tissue repair phase, emphasizing the transition from pro-inflammatory to anti-inflammatory (pro-resolving) mediators.
- Correlate the systemic response of the organism (e.g., fever, acute-phase proteins) with the local inflammatory process.
- Analyze the functional plasticity of macrophages (the distinction between pro-inflammatory [M1] and M2/reparative macrophages) in the process of chronic or resolving acute inflammation.
- Compare the specific mechanisms of inflammation based on etiology (infectious, physical, chemical, ischemic, or immunological causes), identifying common and specific activation pathways.
- Critically evaluate the clinical value of inflammatory biomarkers in patient diagnosis and monitoring (translational aspect).
- Synthesize knowledge of the physiology of inflammation to analyze selected clinical cases, predicting potential complications if homeostatic mechanisms (e.g., tissue repair) are impaired.

Lecture 19: Regeneration and stem cells.

After this lecture, the student will be able to:

- **Define** the terms regeneration, repair (healing), and stem cells.
- **Classify** stem cells based on their differentiation potential (totipotent, pluripotent, multipotent, and unipotent).
- **Explain** the difference between physiological regeneration (routine tissue renewal, e.g., hematopoiesis, epithelium) and reparative regeneration (response to tissue injury).
- **Describe** the concept of the stem cell "niche" and its role in regulating stem cell self-renewal and differentiation.
- **Relate** general concepts of stem cell plasticity and asymmetric cell division to previously acquired knowledge regarding hematopoietic stem cells (HSCs) from the hematology block.
- **Analyze** why different tissues in the human body possess varying regenerative capacities (classification into labile, stable, and permanent tissues) in the context of their cell cycle characteristics and stem cell availability.
- **Evaluate** the advantages, limitations, and potential biological risks (such as tumorigenicity) of using embryonic stem cells versus adult stem cells and induced pluripotent stem cells (iPSCs) in modern medicine.
- **Synthesize** concepts from general cell pathophysiology (injury, division) and hematology (bone marrow transplantation) into a comprehensive final outlook on how stem cell biology is transforming contemporary therapeutic approaches (regenerative medicine).

Practicals list (with titles and explanation):

Practical 1. Laboratory excercise on cell membrane transport.

After successfully mastering the material, the student will be able to:

- **Analyze** the energy requirements of different modes of transport: distinguish between passive transport (diffusion, facilitated diffusion) and primary and secondary active transport.
- **Explain** the molecular basis of "energy on credit" (secondary active transport, i.e., co-transport and counter-transport) and the role of the sodium gradient in driving these processes.
- **Define** the physical basis of osmosis and distinguish between the concepts of osmolality and osmotic pressure in the context of maintaining cell volume.
- **Calculate and interpret** the importance of the differences in concentrations of key ions (Na+, K+, Ca2+, Cl-) between the extracellular and intracellular fluid for the establishment of membrane potential.
- **Link** the Nernst equation to the ionic equilibrium potential and evaluate how changes in extracellular ion concentrations affect the polarization state of the membrane.
- **Connect** disorders in ion pump function (e.g., Na+/K+ -ATPase) to subsequent changes in osmotic balance and cellular edema.
- **Differentiate** between clinical scenarios of electrolyte disturbances (hyper-/hyponatremia, hyper-/hypokalemia) based on an understanding of membrane transport and ion distribution.
- **Discuss** examples from clinical practice where pharmacological blockade of membrane transporters is a therapeutic target (e.g., proton pump inhibitors or digoxin as an Na+/K+ -ATPase inhibitor).
- **Evaluate** the importance of maintaining ionic gradients as a "biological battery" that allows the cell to perform work (signal transmission, muscle contraction, secretion).
- **Synthesize** knowledge to predict the impact of acute changes in pH levels on the stability of ion channels and overall cellular homeostasis.

Practical 2. Laboratory excercise on membrane and action potential.

After successfully mastering the material, the student will be able to:

- **Explain** the resting membrane potential (RMP) and the underlying electrochemical basis.
- **Explain** the effects of changes in sodium (Na+) and potassium (K+) concentrations on the resting membrane potential.
- **Describe** the ion channels essential for the generation and maintenance of the resting membrane potential.
- **Explain** the action potential and the concept of the stimulus threshold.
- **Describe** the effects of changes in extracellular ion concentrations on the action potential's characteristics.
- **Explain** the periods of absolute and relative refractory periods.
- **Analyze** the relationship between stimulus frequency and the generation of action potentials.
- **Analyze** the relationship between stimulus intensity and the frequency of action potentials.
- **Explain** the mechanisms responsible for action potential conduction velocity and predict how myelination and neuronal diameter influence this process.

Practical 3. Laboratory excercise on muscle physiology.

After successfully mastering the material, the student will be able to:

- **Describe** the motor unit, twitch, latent period, contraction phase, relaxation phase, threshold stimulus, summation of muscle contraction, tetanus, muscle fatigue, isometric contraction, and isotonic contraction.
- **Explain** how nerve impulses trigger muscle movement (excitation-contraction coupling).
- **Describe** the phases of a muscle twitch.
- **Explain** the concepts of threshold stimulus and maximal stimulus.
- **Analyze** the effect of increasing stimulus intensity on muscle force.
- **Analyze** the effect of increasing stimulus frequency on muscle force (summation).
- **Explain** the physiological mechanisms of muscle fatigue.
- **Describe** the differences between isometric and isotonic muscle contractions.

Practical 4. Erythrocytes and blood group.

After this practical class, the student will be able to:

- **Explain** the principle of slide blood typing based on the agglutination reaction.
- **Interpret** basic laboratory parameters of the red blood cell count (e.g., Hb, Hct, MCV, MCH, MCHC, reticulocytes) and iron status panels.
- **Demonstrate** the correct and safe technique for capillary blood sampling from a patient's (peer's) fingertip, adhering to aseptic and antiseptic guidelines.

Perform the slide method for ABO and Rh blood typing according to the established protocol.
- **Execute** manual erythrocyte counting under a microscope using a hemocytometer.
- **Calculate** MCV, MCH, and MCHC values using experimentally obtained data (RBC count, hemoglobin, and hematocrit).
- **Analyze** the observed pattern of cell clumping on the slide to accurately determine the patient's blood type and Rh factor.
- **Analyze** potential sources of error in their own laboratory technique (e.g., poor blood flow expression, pipetting errors, miscounting chamber squares) by comparing obtained results with standard reference values.
- **Formulate** a complete and well-structured laboratory report (panel) that includes patient data, determined blood group, measured parameters, calculated indices, and a brief conclusion on whether the findings fall within physiological limits.

Practical 5. Platelets and clotting. Bleeding time and clotting time.

After this practical class, the student will be able to:

- **State** the normal reference values for platelet count, bleeding time, and clotting time.
- **Apply** knowledge of hemostasis phases to interpret basic laboratory tests (e.g., prothrombin time/PT for the extrinsic pathway, activated partial thromboplastin time/aPTT for the intrinsic pathway).
- **Explain** the physiological principle of bleeding time as an indicator of primary hemostasis (platelets and vessels) and clotting time as an indicator of secondary hemostasis (fibrin formation).
- **Clarify** the principle of manual platelet counting in a hemocytometer (the significance of specific dilution and cell recognition).
- **Describe** (theoretically) the role of PT in assessing the extrinsic coagulation pathway and aPTT in assessing the intrinsic coagulation pathway, explaining their importance for patient panel interpretation.
- **Perform** manual platelet counting under a microscope using a hemocytometer and the appropriate protocol.
- **Demonstrate** the correct execution of the bleeding time test (e.g., Duke's method) and the capillary clotting time test, ensuring precise time measurement.
- **Analyze** the interrelationship between the obtained platelet count and bleeding time to distinguish a quantitative deficiency (thrombocytopenia) from a potential qualitative defect (thrombocytopathy).
- **Relate** a pathologically prolonged clotting time to a potential PT and aPTT profile (e.g., predict which of these two tests would be prolonged in an isolated intrinsic pathway disorder).
- **Evaluate** the accuracy and precision of their own manually measured parameters, considering technical limitations and potential sources of error in the lab (e.g., temperature effects, puncture depth, tissue fluid contamination).
- **Formulate** a comprehensive laboratory report that integrates manually obtained results (platelet count, bleeding/clotting time) with a theoretical presentation (simulation) of expected PT and aPTT findings for a healthy subject versus a patient with a coagulation disorder.

Practical 6. Leukocytes. Differential leukocyte count (DLS).

After this practical class, the student will be able to:

- **Recognize** and recall the characteristic morphological features of the five main leukocyte types under a microscope (nuclear shape, cell size, presence and color of granules).
- **State** the reference intervals for the total leukocyte count and the relative percentages of individual subpopulations in a differential blood count (CBC with diff).
- **Describe** the pathophysiological significance of reactive changes observed on the slides (e.g., why a left shift occurs during bacterial inflammation and what it represents morphologically).
- **Perform** the manual procedure for counting the total leukocyte number in a hemocytometer according to the established protocol.
- **Demonstrate** the correct technique for scanning a peripheral blood smear under a microscope (utilizing the oil immersion objective and a systematic zigzag movement across the slide).
- **Calculate** the relative percentages of leukocyte subpopulations based on the cells independently counted on the assigned slide.
- **Differentiate** among neutrophils (segmented and band forms), lymphocytes, monocytes, eosinophils, and basophils while evaluating the peripheral blood smear.
- **Analyze** the calculated percentages of counted cells to identify a specific laboratory pattern (e.g., isolated lymphocytosis, eosinophilia, or neutrophilia with a left shift).
- **Evaluate** the accuracy of their own manual counting and identify potential technical errors (e.g., misidentification of cells or counting in the wrong zone of the smear).
- **Judge** which type of pathological stimulus or infection the analyzed slide suggests (e.g., matching an eosinophilia finding with allergies/parasites, or lymphocytosis with a viral infection).
- **Formulate** and propose an official differential blood count report based on the data independently gathered through microscopy, complete with a written pathophysiological explanation and clinical interpretation of the panel.

Seminars list (with titles and explanation):

Seminar 1. Cell membrane transport

After successfully mastering the material, the student will be able to:

- **Analyze** the energy requirements of different modes of transport: distinguish between passive transport (diffusion, facilitated diffusion) and primary and secondary active transport.
- **Explain** the molecular basis of "energy on credit" (secondary active transport, i.e., co-transport and counter-transport) and the role of the sodium gradient in driving these processes.
- **Define** the physical basis of osmosis and distinguish between the concepts of osmolality and osmotic pressure in the context of maintaining cell volume.
- **Calculate and interpret** the importance of the differences in concentrations of key ions (Na+, K+, Ca2+, Cl-) between the extracellular and intracellular fluid for the establishment of membrane potential.
- **Link** the Nernst equation to the ionic equilibrium potential and evaluate how changes in extracellular ion concentrations affect the polarization state of the membrane.
- **Connect** disorders in ion pump function (e.g., Na+/K+ -ATPase) to subsequent changes in osmotic balance and cellular edema.
- **Differentiate** between clinical scenarios of electrolyte disturbances (hyper-/hyponatremia, hyper-/hypokalemia) based on an understanding of membrane transport and ion distribution.
- **Discuss** examples from clinical practice where pharmacological blockade of membrane transporters is a therapeutic target (e.g., proton pump inhibitors or digoxin as an Na+/K+ -ATPase inhibitor).
- **Evaluate** the importance of maintaining ionic gradients as a "biological battery" that allows the cell to perform work (signal transmission, muscle contraction, secretion).
- **Synthesize** knowledge to predict the impact of acute changes in pH levels on the stability of ion channels and overall cellular homeostasis.

Seminar 2. Membrane and action potential.

After successfully mastering the material, the student will be able to:

- **Explain** the resting membrane potential (RMP) and the underlying electrochemical basis.
- **Explain** the effects of changes in sodium (Na+) and potassium (K+) concentrations on the resting membrane potential.
- **Describe** the ion channels essential for the generation and maintenance of the resting membrane potential.
- **Explain** the action potential and the concept of the stimulus threshold.
- **Describe** the effects of changes in extracellular ion concentrations on the action potential's characteristics.
- **Explain** the periods of absolute and relative refractory periods.
- **Analyze** the relationship between stimulus frequency and the generation of action potentials.
- **Analyze** the relationship between stimulus intensity and the frequency of action potentials.
- **Explain** the mechanisms responsible for action potential conduction velocity and predict how myelination and neuronal diameter influence this process.

Seminar 3 Muscle physiology

After successfully mastering the material, the student will be able to:

- **Describe** the motor unit, twitch, latent period, contraction phase, relaxation phase, threshold stimulus, summation of muscle contraction, tetanus, muscle fatigue, isometric contraction, and isotonic contraction.
- **Explain** how nerve impulses trigger muscle movement (excitation-contraction coupling).
- **Describe** the phases of a muscle twitch.
- **Explain** the concepts of threshold stimulus and maximal stimulus.
- **Analyze** the effect of increasing stimulus intensity on muscle force.
- **Analyze** the effect of increasing stimulus frequency on muscle force (summation).
- **Explain** the physiological mechanisms of muscle fatigue.
- **Describe** the differences between isometric and isotonic muscle contractions.

Seminar 4. Red blood cells disorders.

After this seminar, the student will be able to:

- **Recognize** and **explain** the abbreviations and measurement units of key hematological and biochemical parameters (Hb, Hct, MCV, MCH, MCHC, RDW, ferritin, iron, TIBC, bilirubin, reticulocytes) in a laboratory report.
- **Identify** deviations (elevated or decreased values) in a given laboratory report relative to the provided reference intervals for age and sex.
- **Explain** the pathophysiological cause of isolated or combined abnormalities in a laboratory report (e.g., why

ferritin is elevated despite low serum iron in anemia of chronic disease).
- **Apply** the provided diagnostic algorithm step-by-step to a real patient laboratory report to systematically narrow down differential diagnoses.
- **Demonstrate** the correct sequence of analysis: from confirming the presence of anemia (Hb/Hct), through morphological classification (MCV), to biochemical confirmation of the specific etiology.
- **Correlate** hematological findings with accompanying biochemical markers into a coherent pathophysiological picture of a patient with anemia or polycythemia.
- **Judge** and select the most likely definitive diagnosis based on the case analysis and **defend the conclusion with arguments** before peers and the seminar instructor.
- **Evaluate** the body's compensatory capacity based on the reticulocyte count (differentiating an adequate regenerative response from an aregenerative state).

Seminar 5. Disorders of hemostasis.

After this seminar, the student will be able to:

- **Interpret** basic laboratory tests (platelet count, bleeding time, PT, aPTT) in patients with a suspected hemostasis disorder.
- **Identify** pathological deviations in a coagulation panel relative to adult-specific reference intervals.
- **Explain** the pathophysiological basis of an isolated prolongation of a specific test (e.g., why a deficiency of factor VIII prolongs aPTT while leaving PT normal).
- **Critically assess** coagulation panel results to determine whether a patient is under adequate anticoagulant therapy or at an increased risk of bleeding.
- **Demonstrate** the correct interpretation of PT/INR and aPTT for evaluating and monitoring the safety and efficacy of a patient's anticoagulant therapy.
- **Formulate** a coherent pathophysiological case summary linking the patient's bleeding phenotype (e.g., petechiae versus hemarthrosis) to the coagulation panel
- **Analyze** the specific laboratory profile of von Willebrand disease and explain why both bleeding time (platelet defect) and aPTT (due to vWF's role in stabilizing factor VIII) can be prolonged in this condition.
- **Compare and contrast** the pathophysiology and laboratory findings of immune thrombocytopenia (ITP) versus coagulation disorders like hemophilia.

Seminar 6. White blood cell disorders. Inflammation.

After this seminar, the student will be able to:

- **Recognize** and correctly read the relative (%) and absolute values of specific leukocyte types (neutrophils, lymphocytes, eosinophils, basophils, monocytes) and the presence of pathological forms (blasts).
- **Identify** quantitative abnormalities (leukocytosis, leukopenia, neutrophilia, lymphocytosis) in a given laboratory report.
- **Explain** the pathophysiological basis of the observed laboratory changes (e.g., why an acute bacterial infection causes neutrophilia)
- **Demonstrate** the recognition of classic laboratory patterns in a differential blood count (e.g., a "left shift" in response to inflammation).
- **Interpret** a differential blood count report to identify abnormalities such as leukocytosis, leukopenia, neutrophilia, lymphocytosis, or eosinophilophilia.
- **Correlate** concurrent alterations across the red, white, and platelet lineages (pancytopenia) with the pathophysiological concept of bone marrow failure.
- **Relate** an isolated increase in a specific leukocyte type to a general type of stimulus (e.g., neutrophilia in acute bacterial inflammation, eosinophilia in an allergic reaction).
- **Utilize** a differential blood count report to initially distinguish a severe bacterial infection from chronic myeloid leukemia (CML).

Student obligations:

ECTS Grading System

Student assessment is conducted in accordance with the current Regulations on Studies of the University of Rijeka and the Regulations on Student Assessment of the Faculty of Medicine, University of Rijeka.

Students' performance and achievements are expressed as points earned, which form the basis for determining the final grade. Students' performance and acquired competencies are assessed through continuous assessment during the course, with a maximum of 70 points (70%), and the final examination, with a maximum of 30 points (30%).

Student achievement is graded using both the ECTS grading scale (A–F) and the numerical grading scale (1–5). ECTS grades are assigned according to an absolute grading scale and the assessment criteria established for the integrated undergraduate and graduate university study programme in Medicine.

I. Continuous Assessment (maximum of 70 points)

The following activities are assessed during the course (maximum of 70 points):

1) Attendance

Attendance at lectures, seminars, and practical exercises is mandatory. Students may be absent from a maximum of 30% of lectures and 30% of seminars and 30% of practicals, provided that all absences are justified.

Each absence from a seminar or practical exercise must be compensated for by an oral assessment conducted by the respective seminar or practical exercise instructor in order for the student to be eligible to take the final examination.

2) Assessment of Acquired Knowledge

During the course, students' acquired knowledge will be assessed through two partial examinations, each consisting of 50 questions, held in November (Partial Examination 1: L1-L10, S1-S3, P1-P3) and January (Partial Examination 2: L11-L19; S4-S6; P4-P6).

Both partial examinations will be conducted in person at the Faculty of Medicine, University of Rijeka.

A maximum of 30 points can be earned on each partial examination, according to the following scoring system:

Number of Correct Answers	Points Awarded		Number of Correct Answers	Points Awarded
49-50	35		35	25
47-48	34		34	24
45-46	33		33	23
43-44	32		32	22
42-41	31		31	21
40	30		30	20
39	29		29	19
38	28		27-28	18
37	27		25-26	17,5
36	26			

Examination Procedure: Partial examinations will consist of 50 multiple-choice questions, with a time limit of 50 minutes for each examination.

Retaking Partial Examinations. Students who fail to earn the minimum required number of points on one or both partial examinations may retake the corresponding partial examination(s). Points earned on the retake will be awarded according to the scoring table above.

Improvement of Continuous Assessment Results. Students who have earned the minimum required number of points on a regular partial examination but wish to improve their continuous assessment results may also retake the corresponding examination.

Exam (exam taking, description of the written/oral/practical part of the exam, point distribution, grading criteria):

II. Final Examination

Students who have earned 35–70 points during the course are required to take the final examination, where they can earn additional points. The final examination consists of a written multiple-choice question (MCQ) examination and an oral examination.

Students who have earned fewer than 35 points during the course or have been absent from more than 30% of classes are not eligible to take the final examination.

A maximum of **30 points** can be earned on the final examination. The final examination consists of written and oral components, in which students must demonstrate at least 50% of the required knowledge, skills, and competencies.

Students who demonstrate more than 50% of the required knowledge, skills, and competencies in both the written and oral components will be awarded points according to their performance. These points will be added to those earned during the course.

Written Component of the Final Examination

The written component consists of **two tests, each containing 30 multiple-choice questions**, administered together as a single examination (**60 questions in total**).

Test A covers general and cellular physiology and pathophysiology, as well as the physiology and pathophysiology of the muscular system (Lectures 1–10, Seminars 4-6, and Practicals 4-6).

Test B covers the physiology and pathophysiology of the hematopoietic system and inflammation (Lectures 11–19, Seminars 4-6, and Practicals 4-6).

Students must answer **at least 50% of the questions correctly on each test** and can earn **14–28 points** based on their overall performance across all 60 questions, according to the following scoring system:

Number of Correct Answers	Points Awarded		Number of Correct Answers	Points Awarded
96-100%	28		69-71,9%	20
91-95,9%	27		66-68,9%	19
87-90,9%	26		63-65,9%	18
84-86,9%	25		60-62,9%	17
81-84,9%	24		57-59,9%	16
78-80,9%	23		54-56,9%	15
75-77,9%	22		50-53,9%	14
72-74,9%	21			

Oral Component of the Final Examination

Students are eligible to take the oral component of the final examination **if they have answered at least 50% of the questions correctly on each of the two written tests.**

Students can earn 1–5 points on the oral examination, divided into five performance categories (1, 2, 3, 4, and 5).

Points earned on the written and oral components are added together.

The final examination is considered passed if the student earns a minimum of 14 points on the written component and at least 1 point on the oral component. The final examination is assessed as a single, integrated examination. Therefore, if a student fails the oral component, the results of the written component will not be carried forward to subsequent examination periods.

Other notes (related to the course) important for students:

Course content and all information regarding the course, including exam dates, can be found on the SharePoint platform of Merlin - accessed via an AAI address.

COURSE HOURS 2026/2027

Physiology and Pathophysiology I

Lectures (Place and time or group)	**Practicals** (Place and time or group)	**Seminars** (Place and time or group)
02.10.2026		
Lecture 1. Homeostasis and internal environment.: • P01 (11:15 - 13:00) [210] ○ PAPI		
prof. dr. sc. Mahmutefendić Lučin Hana, dipl. ing. biol. [210]		
07.10.2026		
Lecture 2. Membrane transport. Passive and active transport across membranes.: • P08 (13:15 - 15:00) [210] ○ PAPI		
prof. dr. sc. Mahmutefendić Lučin Hana, dipl. ing. biol. [210]		
09.10.2026		
Lecture 3. Ion channels. Membrane potential.: • P08 (13:15 - 15:00) [210] ○ PAPI		
prof. dr. sc. Mahmutefendić Lučin Hana, dipl. ing. biol. [210]		
14.10.2026		
Lecture 4. Action potential. Membrane transport disorders.: • P08 (13:15 - 15:00) [210] ○ PAPI		
prof. dr. sc. Mahmutefendić Lučin Hana, dipl. ing. biol. [210]		
16.10.2026		
Lecture 5. Physiology and pathophysiology of the cell - maintenance of internal homeostasis: • P08 (12:15 - 14:00) [143] ○ PAPI		
prof. dr. sc. Lučin Pero, dr. med. [143]		
20.10.2026		
Lecture 6. Cellular stress: • ONLINE (18:00 - 19:45) [143] ○ PAPI		
prof. dr. sc. Lučin Pero, dr. med. [143]		
21.10.2026		
Lecture 7. Cellular adaptation, damage and death: • P08 (14:15 - 16:00) [143] ○ PAPI		
prof. dr. sc. Lučin Pero, dr. med. [143]		
28.10.2026		

Lecture 8. Malignant transformation and growth.: • P08 (13:15 - 15:00) [143] ○ PAPI		
prof. dr. sc. Lučin Pero, dr. med. [143]		
29.10.2026		

	Practical 1. Laboratory excercise on cell membrane transport.:
	• Department of Physiology - Seminarska (15:30 - 17:00) [213]
	  ○ Group II (B)
		Seminar 1. Cell membrane transport:
		• Department of Physiology - Seminarska (14:00 - 15:30) [213]
		  ○ Group II (B)

doc.dr. sc. Karleuša Mujkić Ljerka, dipl. ing. bioteh. [213]		
30.10.2026		

	Practical 1. Laboratory excercise on cell membrane transport.:
	• P04 (13:30 - 15:00) [211]
	  ○ Group I (A)
		Seminar 1. Cell membrane transport:
		• P04 (12:00 - 13:30) [211]
		  ○ Group I (A)

izv. prof. dr. sc. Gulić Tamara, mag. biol. [211]		
04.11.2026		
Lecture 9. Molecular biomechanics of muscle contraction.: • P08 (14:15 - 16:00) [143] ○ PAPI		
prof. dr. sc. Lučin Pero, dr. med. [143]		
05.11.2026		

	Practical 2. Laboratory excercise on membrane and action potential.:
	• Department of Physiology - Exercise room (15:30 - 17:00) [213]
	  ○ Group I (A)
		Seminar 2. Membrane and action potential.:
		• Department of Physiology - Exercise room (14:00 - 15:30) [213]
		  ○ Group I (A)

doc.dr. sc. Karleuša Mujkić Ljerka, dipl. ing. bioteh. [213]		
06.11.2026		

	Practical 2. Laboratory excercise on membrane and action potential.:
	• v (13:30 - 15:00) [211]
	  ○ Group II (B)
		Seminar 2. Membrane and action potential.:
		• v (12:00 - 13:30) [211]
		  ○ Group II (B)

izv. prof. dr. sc. Gulić Tamara, mag. biol. [211]		
11.11.2026		
Lecture 10. Skeletal Muscle Excitation, Neuromuscular Transmission, Excitation-Contraction Coupling, Smooth Muscle Contraction, Energetics, and Pathophysiology.: • P08 (14:15 - 16:00) [143] ○ PAPI		

prof. dr. sc. Lučin Pero, dr. med. [143]		
12.11.2026		
	Practical 3. Laboratory excercise on muscle physiology.: • Department of Physiology - Exercise room (13:45 - 16:00) [143] ○ Group I (A)	Seminar 3 Muscle physiology: • Department of Physiology - Exercise room (13:00 - 13:45) [143] ○ Group I (A)
prof. dr. sc. Lučin Pero, dr. med. [143]		
13.11.2026		
Lecture 11. Hematopoiesis. Physiology of erythrocytes.: • P08 (12:15 - 14:00) [210] ○ PAPI	Practical 3. Laboratory excercise on muscle physiology.: • Department of Physiology - Seminarska (15:00 - 17:15) [213] ○ Group II (B)	Seminar 3 Muscle physiology: • Department of Physiology - Seminarska (14:15 - 15:00) [213] ○ Group II (B)
doc.dr. sc. Karleuša Mujkić Ljerka, dipl. ing. bioteh. [213] · prof. dr. sc. Mahmutefendić Lučin Hana, dipl. ing. biol. [210]		
19.11.2026		
	Practical 4. Erythrocytes and blood group.: • Department of Physiology - Exercise room (14:00 - 17:00) [1102] ○ Group I (A)	
Kostelac Elizabeta, dr.med. [1102]		
20.11.2026		
Lecture 12. Blood types.: • P08 (12:15 - 13:00) [210] ○ PAPI	Practical 4. Erythrocytes and blood group.: • Department of Physiology - Exercise room (14:00 - 17:00) [3613] ○ Group II (B)	
asistentica Bilić Sara, mag. biol. exp. [3613] · prof. dr. sc. Mahmutefendić Lučin Hana, dipl. ing. biol. [210]		
02.12.2026		
Lecture 13. Red blood cells disorders.: • P08 (13:15 - 16:00) [210] ○ PAPI		
prof. dr. sc. Mahmutefendić Lučin Hana, dipl. ing. biol. [210]		
09.12.2026		
Lecture 14. Hemostasis and blood clotting.: • P08 (13:15 - 15:00) [211] ○ PAPI		
izv. prof. dr. sc. Gulić Tamara, mag. biol. [211]		
10.12.2026		

		Seminar 4. Red blood cells disorders.: • v (13:15 - 14:45) [210] ○ Group II (B)
prof. dr. sc. Mahmutefendić Lučin Hana, dipl. ing. biol. [210]		
11.12.2026		

Lecture 15. Hemostasis disorders.:
• P08 (12:15 - 14:00) [210]
  ○ PAPI
		Seminar 4. Red blood cells disorders.:
		• Department of Physiology - Seminarska (14:15 - 15:45) [212]
		  ○ Group I (A)

prof. dr. sc. Mahmutefendić Lučin Hana, dipl. ing. biol. [210] · doc.dr. sc. Marcelić Marina, mag. pharm. inv. [212]		
16.12.2026		
Lecture 16. White blood cells.: • P08 (13:15 - 15:00) [143] ○ PAPI		
prof. dr. sc. Lučin Pero, dr. med. [143]		
17.12.2026		
	Practical 5. Platelets and clotting. Bleeding time and clotting time.: • Department of Physiology - Exercise room (13:30 - 15:45) [3613] ○ Group II (B)	
asistentica Bilić Sara, mag. biol. exp. [3613]		
18.12.2026		

Lecture 17: White blood cells disorders.:
• P08 (12:15 - 14:00) [143]
  ○ PAPI
	Practical 5. Platelets and clotting. Bleeding time and clotting time.:
	• Department of Physiology - Exercise room (14:15 - 16:30) [1102]
	  ○ Group I (A)	

Kostelac Elizabeta, dr.med. [1102] · prof. dr. sc. Lučin Pero, dr. med. [143]		
07.01.2027		
		Seminar 5. Disorders of hemostasis.: • v (13:15 - 14:45) [211] ○ Group II (B)
izv. prof. dr. sc. Gulić Tamara, mag. biol. [211]		
08.01.2027		

Lecture 18: Inflammation, tissue repair after damage, and wound healing.:
• P08 (12:15 - 15:00) [143]
  ○ PAPI
		Seminar 5. Disorders of hemostasis.:
		• v (15:15 - 16:45) [1132]
		  ○ Group I (A)

prof. dr. sc. Lučin Pero, dr. med. [143] · Omerović Alen, dr. med. [1132]		
11.01.2027		
	Practical 6. Leukocytes. Differential leukocyte count (DLS)..: • Department of Physiology - Exercise room (13:45 - 15:15) [3613] ○ Group II (B)	Seminar 6. White blood cell disorders. Inflammation.: • Department of Physiology - Seminarska (11:15 - 13:30) [210] ○ Group II (B)
asistentica Bilić Sara, mag. biol. exp. [3613] · prof. dr. sc. Mahmutefendić Lučin Hana, dipl. ing. biol. [210]		
13.01.2027		
Lecture 19: Regeneration and stem cells.: • P08 (11:15 - 12:00) [210] ○ PAPI	Practical 6. Leukocytes. Differential leukocyte count (DLS)..: • Department of Physiology - Exercise room (16:00 - 17:30) [3613] ○ Group I (A)	Seminar 6. White blood cell disorders. Inflammation.: • Department of Physiology - Seminarska (13:30 - 15:45) [212] ○ Group I (A)
asistentica Bilić Sara, mag. biol. exp. [3613] · prof. dr. sc. Mahmutefendić Lučin Hana, dipl. ing. biol. [210] · doc.dr. sc. Marcelić Marina, mag. pharm. inv. [212]		

List of lectures, seminars and practicals:

LECTURES (TOPIC)	Number of hours	Location
Lecture 1. Homeostasis and internal environment.	2	P01
Lecture 2. Membrane transport. Passive and active transport across membranes.	2	P08
Lecture 3. Ion channels. Membrane potential.	2	P08
Lecture 4. Action potential. Membrane transport disorders.	2	P08
Lecture 5. Physiology and pathophysiology of the cell - maintenance of internal homeostasis	2	P08
Lecture 6. Cellular stress	2	ONLINE
Lecture 7. Cellular adaptation, damage and death	2	P08
Lecture 8. Malignant transformation and growth.	2	P08
Lecture 9. Molecular biomechanics of muscle contraction.	2	P08
Lecture 10. Skeletal Muscle Excitation, Neuromuscular Transmission, Excitation-Contraction Coupling, Smooth Muscle Contraction, Energetics, and Pathophysiology.	2	P08
Lecture 11. Hematopoiesis. Physiology of erythrocytes.	2	P08
Lecture 12. Blood types.	1	P08
Lecture 13. Red blood cells disorders.	3	P08
Lecture 14. Hemostasis and blood clotting.	2	P08
Lecture 15. Hemostasis disorders.	2	P08
Lecture 16. White blood cells.	2	P08
Lecture 17: White blood cells disorders.	2	P08
Lecture 18: Inflammation, tissue repair after damage, and wound healing.	3	P08

Lecture 19: Regeneration and stem cells.	1	P08

PRACTICALS (TOPIC)	Number of hours	Location
Practical 1. Laboratory excercise on cell membrane transport.	2	Department of Physiology - Seminarska P04
Practical 2. Laboratory excercise on membrane and action potential.	2	Department of Physiology - Exercise room v
Practical 3. Laboratory excercise on muscle physiology.	3	Department of Physiology - Exercise room Department of Physiology - Seminarska
Practical 4. Erythrocytes and blood group.	4	Department of Physiology - Exercise room
Practical 5. Platelets and clotting. Bleeding time and clotting time.	3	Department of Physiology - Exercise room
Practical 6. Leukocytes. Differential leukocyte count (DLS).	2	Department of Physiology - Exercise room

SEMINARS (TOPIC)	Number of hours	Location
Seminar 1. Cell membrane transport	2	Department of Physiology - Seminarska P04
Seminar 2. Membrane and action potential.	2	Department of Physiology - Exercise room v
Seminar 3 Muscle physiology	1	Department of Physiology - Exercise room Department of Physiology - Seminarska
Seminar 4. Red blood cells disorders.	2	Department of Physiology - Seminarska v
Seminar 5. Disorders of hemostasis.	2	v
Seminar 6. White blood cell disorders. Inflammation.	3	Department of Physiology - Seminarska

EXAM DATES (final exam):