Federal State Autonomous Educational Institution of Higher Education "Moscow Institute of Physics and Technology (National Research University)"

APPROVED
Head of the Phystech School of Biological and Medical Physics
D.V. Kuzmin

Work program of the course (training module)

course: Cancer and Molecular Cell Biology/Рак и молекулярно-клеточная биология
major: Applied Mathematics and Physics
specialization: Applied Bioinformatics/Прикладная биоинформатика
Phystech School of Biological and Medical Physics
Center for educational programs in bioinformatics
term: 1
qualification: Master

Semester, form of interim assessment: 2 (spring) - Grading test

Academic hours: 30 AH in total, including:
lectures: 0 AH.
seminars: 30 AH.
laboratory practical: 0 AH.

Independent work: 15 AH.

In total: 45 AH, credits in total: 1

Author of the program: A.N. Khamad, candidate of biological sciences

The program was discussed at the Center for educational programs in bioinformatics 04.03.2020

Annotation

The aim of the course is to understand the biological aspects of the onset and development of cancer. After mastering, the student will understand the fundamental concepts and aspects associated with the occurrence of cancer, its development, as well as the physiological and molecular biological characteristics of cancerous tumors; modern problems associated with the treatment of cancerous tumors.

1. Study objective

Purpose of the course

Give a grounding in the molecular and cellular biology that underpins cancer biology and research. Understanding the biological aspects of the emergence and development of cancer and the formation of students' holistic understanding of the process of different cellular processes, especially the regulated cell death and its varieties.

Tasks of the course

- Give a detailed and thorough understanding of the molecular and cellular basis of cancer and its treatments.
- Develop a deep knowledge of cancer biology, pathology, and cell death mechanisms.
- Increasing the understand of cancer cell biology and obtaining basic theoretical knowledge in the field of cell biology, in particular the molecular mechanisms of cell death and cancer development.
- Familiarization with various methods and technologies of detection and induction of cell death for the application of fundamental knowledge in modern biotechnology and medicine.

2. List of the planned results of the course (training module), correlated with the planned results of the mastering the educational program

Mastering the discipline is aimed at the formation of the following competencies:

Code and the name of the competence	Competency indicators
UC-1 Use a systematic approach to critically analyze a problem,and develop an action plan	UC-1.1 Systematically analyze the problem situation, identify its components and the relations between them
	UC-1.2 Search for solutions by using available sources
	UC-1.3 Develop a step-by-step strategy for achieving a goal, foresee the result of each step, evaluate the overall impact on the planned activity and its participants
Gen.Pro.C-4 Successfully perform a task, analyze the results, and present conclusions, apply knowledge and skills in the field of physical and mathematical sciences and ICTs	Gen.Pro.C-4.1 Apply ICT knowledge and skills to find and study scientific literature and use software products
	Gen.Pro.C-4.2 Apply knowledge in the field of physical and mathematical sciences to solve problems, make conclusions, and evaluate the obtained results
	Gen.Pro.C-4.3 Justify the chosen method of scientific research
Pro.C-1 Assign, formalize, and solve tasks, develop and research mathematical models of the studied phenomena and processes, systematically analyze scientific problems and obtain new scientific results	Pro.C-1.2 Make hypotheses, build mathematical models of the studied phenomena and processes, evaluate the quality of the developed model
	Pro.C-1.1 Locate, analyze, and summarize information on current research findings within the subject area
	Pro.C-1.3 Apply theoretical and/or experimental research methods to a specific scientific task and interpret the obtained results
Pro.C-3 Use research and testing equipment (devices and installations, specialized software) in a selected subject field	Pro.C-3.1 Understand the operating principles of the equipment and specialized software
	Pro.C-3.3 Evaluate the accuracy of the experimental (numerical) results
	Pro.C-3.2 Conduct an experiment (simulation) using research equipment (software)

3. List of the planned results of the course (training module)

As a result of studying the course the student should:

know:

- Fundamental concepts and aspects related to the emergence of cancer, its development, as well as physiological and molecular biological characteristics of cancerous tumors;
- Scientific rationale for development of innovative anticancer therapies or combinations in a very competitive and integrated scientific and medical environment;
- Concepts of regulated cell death, apoptosis, necrosis, autophagy, oxidative stress, and cell cycle.

be able to:

- Discuss the mechanisms by which cancers develop, grow, and spread within the body;
- Demonstrate comprehension regarding the ways in which normal cell growth and differentiation is controlled;
- adequately assess the potential prospects for new cancer therapy;
- establish causal relationships between the biological aspects of cancer development and modern methods of therapy and diagnostics, which are increasingly being improved.

master:

- Molecular biological and biomedical terminology related to aspects of cancer biology;
- Skills in mastering a large amount of information and understanding the biological processes associated with cancer;
- Skills of independent work and mastering new knowledge;
- Methods for calculating the detection of various forms of cell death;
- Ability to read high-peered research articles and analyze the data to discuss it in seminars;
- Evaluate critically current issues in cancer research and therapy, and how to translate research findings into therapeutic applications.

4. Content of the course (training module), structured by topics (sections), indicating the number of allocated academic hours and types of training sessions

4.1. The sections of the course (training module) and the complexity of the types of training sessions

№	Topic (section) of the course	Types of training sessions, including independent work			
		Lectures	Seminars	Laboratory practical	Independent work
1	Introduction to Cancer Biology		4		2
2	The role of oncoviruses and epigenetic changes in cancer development		4		2
3	Genetic heterogeneity of tumors. Cancer stem cells		4		2
4	Receptors and signaling pathways in tumors		6		1
5	Cancer targeted therapies.		4		2
6	Types of cell death		2		2
7	Regulators and mechanisms of cell death		4		2
8	Methods for detecting various forms of cell death and application in medicine		2		2
AH in total			30		15
Exam preparation		0 AH.			
Total complexity		45 AH., credits in total 1			

4.2. Content of the course (training module), structured by topics (sections)

Semester: 2 (Spring)

1. Introduction to Cancer Biology

Cancer concept. Features of benign and malignant tumors. Classification of cancerous tumors. Properties of cancerous tumors.

2. The role of oncoviruses and epigenetic changes in cancer development

Types of oncoviruses. Influence of oncoviruses on the stability of the cellular genome. Epigenetic changes, their role in cancer mutagenesis. Epicarcinogens. Influence of epigenetic changes on the expression of microRNA and the activity of mobile elements

3. Genetic heterogeneity of tumors. Cancer stem cells

Cancer stem cells. Cellular (replicative) aging. Telomeric crisis. Overcoming the telomeric crisis by cancer cells

4. Receptors and signaling pathways in tumors

G-protein coupled receptors (GPCRs) and their regulation. Signaling pathways triggered by GPCRs. Tyrosine kinase receptors, their signaling pathways and regulation. Participants in the transmission of intracellular signals: receptors, adapter proteins, G-proteins, secondary messengers, GTPases, kinases, activators and co-activators of transcription. TGFβ receptors. Cytokine receptors (JAK / STAT receptors). Integrin receptors and their ligands and signaling pathways. TNF receptors superfamily, their intracellular domains (TRAF, FADD, TRADD) and signaling. Notch receptors, their role in angiogenesis. Nuclear (intracellular) receptors

5. Cancer targeted therapies.

Chemotherapy, Radiotherapy, Immunotherapy, Oncolytic Viruses, Cancer vaccines. Cancer drug resistance and the role of cancer stem cells. Cancer microenvironment. Studying the different types of each cancer therapy type and the mechanism by which it works. The therapeutic effects of each one and the use of different types in parallel to treat cancer.

6. Types of cell death

Molecular mechanisms of apoptosis. Oxidative stress and apoptosis. Necrosis. Autophagy: mechanisms of survival and death. Other forms of cell death/

7. Regulators and mechanisms of cell death

Regulators of cell death and survival. The role of mitochondria in cell death. Mitochondria in tumor cells. Cell death and cancer. Cell cycle and cell death. Mitotic disaster. Methods for detecting various forms of cell death.

8. Methods for detecting various forms of cell death and application in medicine

Genetic models for studying the process of cell death, phenotypes of knockout mice for key components of cell death mechanisms and “salvation” of the phenotype in double knockouts will be considered

5. Description of the material and technical facilities that are necessary for the implementation of the educational process of the course (training module)

Equipment needed for lectures and seminars: whiteboard with markers, computer and multimedia equipment (projector, sound system)

6. List of the main and additional literature, that is necessary for the course (training module) mastering

Main literature

Provided at the department:

[1] R. Avraham, Y. Yarden, Feedback regulation of EGFR signalling: decision making by early and delayed loops, Nature Reviews Molecular Cell Biology. 12 (2011) 104.
[2] D. Ribatti, The concept of immune surveillance against tumors: The first theories, Oncotarget. 8 (2016) 7175–7180. doi:10.18632/oncotarget.12739.
[3] R. Xu, A. Rai, M. Chen, W. Suwakulsiri, D.W. Greening, R.J. Simpson, Extracellular vesicles in cancer - implications for future improvements in cancer care, Nat Rev Clin Oncol. 15 (2018) 617–638. doi:10.1038 41571-018-0036-9.
[4] Z. Yu, T.G. Pestell, M.P. Lisanti, R.G. Pestell, Cancer stem cells, Int. J. Biochem. Cell Biol. 44 (2012) 2144–2151. doi:10.1016/j.biocel.2012.08.022.
[5] T. Kobets, M.J. Iatropoulos, G.M. Williams, Mechanisms of DNA-reactive and epigenetic chemical carcinogens: applications to carcinogenicity testing and risk assessment, Toxicol Res (Camb). 8 (2019) 123–145. doi:10.1039/c8tx00250a.
[6] R.T. Dorsam, J.S. Gutkind, G-protein-coupled receptors and cancer, Nat. Rev. Cancer. 7 (2007) 79–94. doi:10.1038/nrc2069.
[7] V. Petrova, M. Annicchiarico-Petruzzelli, G. Melino, I. Amelio, The hypoxic tumour microenvironment, Oncogenesis. 7 (2018) 10. doi:10.1038 41389-017-0011-9.
[8] J.B. Swann, M.J. Smyth, Immune surveillance of tumors, J. Clin. Invest. 117 (2007) 1137–1146. doi:10.1172/JCI31405.
[9] N. Kaushik, S. Kim, Y. Suh, S.-J. Lee, Proinvasive extracellular matrix remodeling for tumor progression, Arch. Pharm. Res. 42 (2019) 40–47. doi:10.1007 12272-018-1097-0.
[10] M.A. Caligiuri, Immune surveillance against common cancers: the great escape, Blood. 106 (2005) 773–774. doi:10.1182/blood-2005-05-1887.
[11] J.J. O'Shea, M. Gadina, R.M. Siegel, Cytokines and cytokine receptors, in: Clinical Immunology, Elsevier, 2019: pp. 127–155.
[12] J. Maciejowski, T. de Lange, Telomeres in cancer: tumour suppression and genome instability, Nat. Rev. Mol. Cell Biol. 18 (2017) 175–186. doi:10.1038/nrm.2016.171.
[13] M. Salvadores, D. Mas-Ponte, F. Supek, Passenger mutations accurately classify human tumors, PLOS Computational Biology. 15 (2019) e1006953. doi:10.1371/journal.pcbi.1006953.
[14]. Elmore S. Apoptosis: a review of programmed cell death //Toxicologic pathology. – 2007. – Т. 35. – №. 4. – С. 495-516.
[15]. Berghe T. V. et al. Regulated necrosis: the expanding network of non-apoptotic cell death pathways //Nature reviews Molecular cell biology. – 2014. – Т. 15. – №. 2. – С. 135-147.
[16]. Ouyang L. et al. Programmed cell death pathways in cancer: a review of apoptosis, autophagy and programmed necrosis //Cell proliferation. – 2012. – Т. 45. – №. 6. – С. 487-498.
[17]. Green D. R., Llambi F. Cell death signaling //Cold Spring Harbor perspectives in biology. – 2015. – Т. 7. – №. 12. – С. a006080.

Additional literature

Provided at the department:
[1] R. Avraham, Y. Yarden, Feedback regulation of EGFR signalling: decision making by early and delayed loops, Nature Reviews Molecular Cell Biology. 12 (2011) 104.
[2] D. Ribatti, The concept of immune surveillance against tumors: The first theories, Oncotarget. 8 (2016) 7175–7180. doi:10.18632/oncotarget.12739.
[3] R. Xu, A. Rai, M. Chen, W. Suwakulsiri, D.W. Greening, R.J. Simpson, Extracellular vesicles in cancer - implications for future improvements in cancer care, Nat Rev Clin Oncol. 15 (2018) 617–638. doi:10.1038 41571-018-0036-9.
[4] Z. Yu, T.G. Pestell, M.P. Lisanti, R.G. Pestell, Cancer stem cells, Int. J. Biochem. Cell Biol. 44 (2012) 2144–2151. doi:10.1016/j.biocel.2012.08.022.
[5] T. Kobets, M.J. Iatropoulos, G.M. Williams, Mechanisms of DNA-reactive and epigenetic chemical carcinogens: applications to carcinogenicity testing and risk assessment, Toxicol Res (Camb). 8 (2019) 123–145. doi:10.1039/c8tx00250a.
[6] R.T. Dorsam, J.S. Gutkind, G-protein-coupled receptors and cancer, Nat. Rev. Cancer. 7 (2007) 79–94. doi:10.1038/nrc2069.
[7] V. Petrova, M. Annicchiarico-Petruzzelli, G. Melino, I. Amelio, The hypoxic tumour microenvironment, Oncogenesis. 7 (2018) 10. doi:10.1038 41389-017-0011-9.
[8] J.B. Swann, M.J. Smyth, Immune surveillance of tumors, J. Clin. Invest. 117 (2007) 1137–1146. doi:10.1172/JCI31405.
[9] N. Kaushik, S. Kim, Y. Suh, S.-J. Lee, Proinvasive extracellular matrix remodeling for tumor progression, Arch. Pharm. Res. 42 (2019) 40–47. doi:10.1007 12272-018-1097-0.
[10] M.A. Caligiuri, Immune surveillance against common cancers: the great escape, Blood. 106 (2005) 773–774. doi:10.1182/blood-2005-05-1887.
[11] J.J. O'Shea, M. Gadina, R.M. Siegel, Cytokines and cytokine receptors, in: Clinical Immunology, Elsevier, 2019: pp. 127–155.
[12] J. Maciejowski, T. de Lange, Telomeres in cancer: tumour suppression and genome instability, Nat. Rev. Mol. Cell Biol. 18 (2017) 175–186. doi:10.1038/nrm.2016.171.
[13] M. Salvadores, D. Mas-Ponte, F. Supek, Passenger mutations accurately classify human tumors, PLOS Computational Biology. 15 (2019) e1006953. doi:10.1371/journal.pcbi.1006953.
[14] K.H. Burns, Transposable elements in cancer, Nat. Rev. Cancer. 17 (2017) 415–424. doi:10.1038/nrc.2017.35.
[15] P.S. Moore, Y. Chang, Why do viruses cause cancer? Highlights of the first century of human tumour virology, Nat. Rev. Cancer. 10 (2010) 878–889. doi:10.1038/nrc2961.
[16] A. Albini, M.B. Sporn, The tumour microenvironment as a target for chemoprevention, Nature Reviews Cancer. 7 (2007) 139–147. doi:10.1038/nrc2067.
[17] O. Trédan, C.M. Galmarini, K. Patel, I.F. Tannock, Drug resistance and the solid tumor microenvironment, Journal of the National Cancer Institute. 99 (2007) 1441–1454.
[18] H. Hashizume, P. Baluk, S. Morikawa, J.W. McLean, G. Thurston, S. Roberge, R.K. Jain, D.M. McDonald, Openings between defective endothelial cells explain tumor vessel leakiness, The American Journal of Pathology. 156 (2000) 1363–1380.
[19] I. Gukovsky, N. Li, J. Todoric, A. Gukovskaya, M. Karin, Inflammation, autophagy, and obesity: common features in the pathogenesis of pancreatitis and pancreatic cancer, Gastroenterology. 144 (2013) 1199–1209.
[20] S. Vyas, E. Zaganjor, M.C. Haigis, Mitochondria and Cancer, Cell. 166 (2016) 555–566. doi:10.1016/j.cell.2016.07.002.
[21] V.P. Chauhan, T. Stylianopoulos, Y. Boucher, R.K. Jain, Delivery of molecular and nanoscale medicine to tumors: transport barriers and strategies, Annu Rev Chem Biomol Eng. 2 (2011) 281–298. doi:10.1146/annurev-chembioeng-061010-114300.
[22] H.X. Chao, C.E. Poovey, A.A. Privette, G.D. Grant, H.Y. Chao, J.G. Cook, J.E. Purvis, Orchestration of DNA Damage Checkpoint Dynamics across the Human Cell Cycle, Cell Syst. 5 (2017) 445-459.e5. doi:10.1016/j.cels.2017.09.015.
[23] D. Branzei, M. Foiani, Regulation of DNA repair throughout the cell cycle, Nat. Rev. Mol. Cell Biol. 9 (2008) 297–308. doi:10.1038/nrm2351.
[24] S. Elmore, Apoptosis: A Review of Programmed Cell Death, Toxicol Pathol. 35 (2007) 495–516. doi:10.1080/01926230701320337.
[25] Z. Hongmei, Extrinsic and Intrinsic Apoptosis Signal Pathway Review, Apoptosis and Medicine. (2012). doi:10.5772/50129.
[26] S.I. Grivennikov, Inflammation and colorectal cancer: colitis-associated neoplasia, Semin Immunopathol. 35 (2013) 229–244. doi:10.1007 00281-012-0352-6.
[27] J.W. O'Connor, E.W. Gomez, Biomechanics of TGFβ-induced epithelial-mesenchymal transition: implications for fibrosis and cancer, Clin Transl Med. 3 (2014) 23. doi:10.1186/2001-1326-3-23.
[28] Y. Raz, N. Erez, An inflammatory vicious cycle: Fibroblasts and immune cell recruitment in

7. List of web resources that are necessary for the course (training module) mastering

www.molbiol.ru
http://www.biosyn.com/Gizmo/Tools/Oligo/Oligonucleotide%20Properties%20Calculator.htm
http://www.ncbi.nlm.nih.gov/

8. List of information technologies used for implementation of the educational process, including a list of software and information reference systems (if necessary)

Internet access. For some of the lessons, you need Zoom. Google Drive to access course materials. The presence of smartphones / laptops during classes is encouraged to participate in interactive exercises.

9. Guidelines for students to master the course

A student who studies discipline must, on the one hand, master a general conceptual apparatus, and on the other hand, must learn to apply theoretical knowledge in practice.
As a result of studying the discipline, the student should know the basic definitions of the discipline, be able to apply this knowledge to solve various problems.

Successful learning requires:
- visits to all classes provided by the curriculum for the discipline;
- conducting the abstract of occupations;
- intense independent work of the student.

Independent work includes:
- reading recommended literature;
- study of educational material, preparation of answers to questions intended for self-study;
- solving problems offered to students in the classroom;
- preparation for performance of tasks of the current and intermediate certification.

An indicator of possession of the material is the ability to answer questions on discipline topics without an outline.

It is important to achieve an understanding of the material being studied, and not its mechanical memorization. If it is difficult to study individual topics, questions, you should seek advice from the teacher.

Intermediate control of students' knowledge in the form of problem solving in accordance with the subject of classes is possible

SUPPLEMENT

Assessment funds for course (training module)

major:	Applied Mathematics and Physics
specialization:	Applied Bioinformatics/Прикладная биоинформатика Phystech School of Biological and Medical Physics Center for educational programs in bioinformatics
term:	1
qualification:	Master

Semester, form of interim assessment: 2 (spring) - Grading test

Author: A.N. Khamad, candidate of biological sciences

1. Competencies formed during the process of studying the course

Code and the name of the competence	Competency indicators
UC-1 Use a systematic approach to critically analyze a problem,and develop an action plan	UC-1.1 Systematically analyze the problem situation, identify its components and the relations between them
	UC-1.2 Search for solutions by using available sources
	UC-1.3 Develop a step-by-step strategy for achieving a goal, foresee the result of each step, evaluate the overall impact on the planned activity and its participants
Gen.Pro.C-4 Successfully perform a task, analyze the results, and present conclusions, apply knowledge and skills in the field of physical and mathematical sciences and ICTs	Gen.Pro.C-4.1 Apply ICT knowledge and skills to find and study scientific literature and use software products
	Gen.Pro.C-4.2 Apply knowledge in the field of physical and mathematical sciences to solve problems, make conclusions, and evaluate the obtained results
	Gen.Pro.C-4.3 Justify the chosen method of scientific research
Pro.C-1 Assign, formalize, and solve tasks, develop and research mathematical models of the studied phenomena and processes, systematically analyze scientific problems and obtain new scientific results	Pro.C-1.2 Make hypotheses, build mathematical models of the studied phenomena and processes, evaluate the quality of the developed model
	Pro.C-1.1 Locate, analyze, and summarize information on current research findings within the subject area
	Pro.C-1.3 Apply theoretical and/or experimental research methods to a specific scientific task and interpret the obtained results
Pro.C-3 Use research and testing equipment (devices and installations, specialized software) in a selected subject field	Pro.C-3.1 Understand the operating principles of the equipment and specialized software
	Pro.C-3.3 Evaluate the accuracy of the experimental (numerical) results
	Pro.C-3.2 Conduct an experiment (simulation) using research equipment (software)

2. Competency assessment indicators

As a result of studying the course the student should:

know:

- Fundamental concepts and aspects related to the emergence of cancer, its development, as well as physiological and molecular biological characteristics of cancerous tumors;
- Scientific rationale for development of innovative anticancer therapies or combinations in a very competitive and integrated scientific and medical environment;
- Concepts of regulated cell death, apoptosis, necrosis, autophagy, oxidative stress, and cell cycle.

be able to:

- Discuss the mechanisms by which cancers develop, grow, and spread within the body;
- Demonstrate comprehension regarding the ways in which normal cell growth and differentiation is controlled;
- adequately assess the potential prospects for new cancer therapy;
- establish causal relationships between the biological aspects of cancer development and modern methods of therapy and diagnostics, which are increasingly being improved.

master:

• Molecular biological and biomedical terminology related to aspects of cancer biology;
• Skills in mastering a large amount of information and understanding the biological processes associated with cancer;
• Skills of independent work and mastering new knowledge;
• Methods for calculating the detection of various forms of cell death;
• Ability to read high-peered research articles and analyze the data to discuss it in seminars;
• Evaluate critically current issues in cancer research and therapy, and how to translate research findings into therapeutic applications.

3. List of typical control tasks used to evaluate knowledge and skills

• What types of tumors do you know by origin? What are the differences between benign and malignant tumors?
• The cell is at the G1 stage of the cell cycle. What mechanisms of DNA double-strand break repair can it use at this stage of the cycle?
• What mechanisms are implemented by cells in the repair of single-strand DNA breaks?
• What mechanism of DNA repair is realized without the participation of nucleases?
• What mechanisms are responsible for maintaining the stability of the cell genome during the passage of the cell cycle?
• Which category are genes responsible for the functioning of DNA repair systems: oncogenes or tumor suppressor genes? Why?
• Can a cell be reborn into a cancer cell if it “lost” one of the alleles of the tumor suppressor gene and continues to divide further? Why?
• Through what mechanisms can epigenetic changes affect the proliferative potential of cells and their transformation into cancerous ones?
• Under what conditions are oncoviruses capable of transforming a cell from normal to cancerous? What mechanisms can be involved in this?
• Activation of which receptors and signaling pathways are mainly responsible for proliferation? For resistance to apoptosis? For migration and metastasis?
• Why is genetic heterogeneity a major barrier to cancer therapy?

4. Evaluation criteria

• Why does hypoxia occur in tumors? What are the consequences of hypoxia in the tumor tissue?
• What signaling pathways are responsible for "switching" to glycolytic metabolism?
• Why is the architecture of tumor vessels disordered, while the vessels themselves have numerous morphological defects?
• Why are oxygen starvation and the appearance of a necrotic focus in the tumor negative prognostic factors?
• Why, despite the increased expression of collagenases, tumors can show increased collagen in the stroma? How does this affect the metastatic potential of cancer cells?
• How are cancer cells able to “defend” against the cytolytic activity of natural killer cells?
• By what mechanisms can a tumor avoid "immune surveillance" by adaptive immunity?
• What cells besides cancer cells are present in the tumor? How do cancer cells interact with their microenvironment?
• What physiological features of tumors prevent drug delivery?
• What are the reasons for the resistance of tumors to chemotherapy drugs?
• What types of cancer is hormone therapy used for? What are its principles?
• Based on what criteria can a patient be prescribed adoptive T-cell therapy?
• What are the advantages of gene therapy for cancer compared to classical therapies?
• What is the main advantage of nanopreparations compared to classical chemotherapy drugs? Why are nanodrugs still not widely used for cancer therapy despite positive preclinical results?
• What properties of nanomaterials are especially important if they are used to deliver hydrophilic low molecular weight drugs to the cytosol?
• What is the advantage of “targeting” nanoparticles to improve their therapeutic properties? What opposite effects can this lead to?
• What are the advantages of 3D cell models over cells in a monolayer

• What morphological features distinguish programmed cell death and necrotic cell death, and why is PCD less harmful for the organism?
• What is the role of programmed cell death in an organism?
• What are caspases, why are they produced as proproteins?
• What are initiator caspases and executioner caspases, what are their targets and how are both types related to adaptor complexes?
• What are death receptors and describe how they function in apoptosis?
• Which classes of proteins are essential in programmed cell death? Briefly describe their functions.
• What is/are the roles of Bcl2 family of proteins in apoptosis?
• What is the role of phagocytes in Cell death?
• What is Granzyme/Perforin pathway?
• What is Necroptosis and its key effectors?
• What is Netosis and its key effectors?
• What is Pyroptosis and its key effectors?
• What is Autophagy-associated cell death and its key effectors?
• What is Entosis and its key effectors?
• What is Ferroptosis and its key effectors?
• What is Programmed Cell Death and regulated cell Death?
• What are the key regulators of Panoptosis and their role?
• What is the mechanism of autophagy?
• What are the main molecular messengers released by PCD and their role?
• What are the Membrane permeability/damage detection methods?
• What are the Mitochondrial damage/alteration detection methods
• What are the Gel electrophoresis-based methods?
• What is the role of inhibitors of apoptosis?
• What is the role of survival factors in apoptosis, and briefly describe one example of how a survival factor could perform its function?
• Explain how some apoptotic genes can function as proto-oncogenes while others function as tumor suppressor genes
• What factors cause cell death?

The mark is excellent (10 points) - it is given to a student who has shown comprehensive, systematic, deep knowledge of the curriculum of the discipline, who has an interest in this subject area, has demonstrated the ability to confidently and creatively put them into practice in solving specific problems, and a free and proper substantiation of decisions.

The mark is excellent (9 points) - it is given to a student who has shown comprehensive, systematic, in-depth knowledge of the curriculum of the discipline and the ability to confidently put them into practice in solving specific problems, free and proper substantiation of the decisions made.

The mark is excellent (8 points) - given to a student who has shown comprehensive, systematic, in-depth knowledge of the curriculum of the discipline and the ability to confidently apply them in practice in solving specific problems, correct justification of decisions made, with some shortcomings.

A mark is good (7 points) - it is put up for a student, if he knows the material firmly, sets it up competently and in essence, knows how to apply the knowledge gained in practice, but does not competently substantiate the results obtained.

Evaluation is good (6 points) - it is put up to a student, if he knows the material firmly, sets it up correctly and in essence, knows how to apply this knowledge in practice, but admits some inaccuracies in the answer or in solving problems.

A mark is good (5 points) - it is given to a student, if he basically knows the material, correctly and essentially sets it out, knows how to apply this knowledge in practice, but allows a sufficiently large number of inaccuracies to answer or solve problems.

Grade satisfactorily (4 points) is given to a student who has shown the fragmented, fragmented nature of knowledge, insufficiently correct formulations of basic concepts, violations of the logical sequence in the presentation of program material, but at the same time he has mastered the main sections of the curriculum necessary for further education and can apply knowledge is modeled in a standard situation.

Grade satisfactorily (3 points) - given to a student who showed the fragmented, scattered nature of knowledge, making mistakes in formulating basic concepts, disrupting the logical sequence in presenting program material, poorly masters the main sections of the curriculum required for further education and even applies the knowledge gained in a standard situation.

The rating is unsatisfactory (2 points) - is given to a student who does not know most of the main content of the curriculum of the discipline, makes gross mistakes in the wording of the basic principles and does not know how to use this knowledge when solving typical tasks.

Unsatisfactory mark (1 point) - is given to a student who does not know the main content of the discipline's curriculum, makes gross errors in the wording of the basic concepts of the discipline and does not have any skills to solve typical practical problems.

5. Methodological materials defining the procedures for the assessment of knowledge, skills, abilities and/or experience

During the oral differentiated credit, the student is given 30 minutes to prepare. The interview for a student in an oral differentiated credit must not exceed one astronomical hour.