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Main Manifestations of Cellular Damage

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Annotation: This paper describes the fundamental mechanisms of dystrophy and dysplasia. The study demonstrated that disruption of mitochondrial structure leads to significant suppression of mitochondrial respiration and ATP production, as well as to an imbalance of ions within the cell. It also explains that pathobiosis can be exemplified by tissue necrosis, while autolysis is the hydrolysis of cellular components and intercellular substance under the influence of lysosomal enzymes.

Keywords: dystrophy, function, excess transformation, decomposition, infiltration, cell, dysplasia, monster, anemia.

Various pathogenic factors acting on a cell can cause damage. Cell damage refers to changes in its structure, metabolism, physicochemical properties, and functions that lead to disruption of vital functions.

1. Dystrophies. Dystrophies (dys – disorder, disorder, trophe nutrition) are understood as metabolic disorders in cells and tissues, accompanied by disturbances in their functions, plastic manifestations, as well as structural changes leading to disruption of their vital functions.

The main mechanisms of dystrophies are:

- synthesis of abnormal substances in the cell, for example, the protein-polysaccharide complex amyloid;
- excess transformation of some compounds into others, for example, fats and carbohydrates into proteins, carbohydrates into fats;
- decomposition (phanerosis), for example, of protein-lipid complexes of membranes;

- infiltration of cells and intercellular substance by organic and inorganic compounds, for example, cholesterol and its esters of arterial walls in atherosclerosis.

The main cellular dystrophies include protein (dysproteinoses), fatty (lipidoses), carbohydrate and mineral.

2. Dysplasia (dys – disorder, disorder, plaseo – I form) is a disorder in the process of cell development, manifested by a persistent change in their structure and function, which leads to a disruption of their vital activity.

Dysplasia is caused by damage to the cell's genome. This is what causes persistent and, as a rule, inherited changes from cell to cell, unlike dystrophies, which are often temporary, reversible, and can be corrected when the causative factor ceases to act.

The primary mechanism of dysplasia is a disruption of the differentiation process, which involves the formation of structural and functional specialization of cells. Structural features of dysplasia include changes in cell size and shape, their nuclei and other organelles, and the number and structure of chromosomes.

Typically, the cells are enlarged, have an irregular, bizarre shape ("monster cells"), and the ratio of various organelles within them is disproportionate. Such cells often contain various inclusions and signs of degenerative processes.

Examples of cell dysplasia include the formation of megaloblasts in the bone marrow in pernicious anemia, sickle-shaped red blood cells in hemoglobin pathology, large neurons - “monsters” in lesions of the cerebral cortex (tuberous sclerosis), multinucleated giant cells with a bizarre arrangement of chromatin in neurofibromatosis. Recklinghausen. Cellular dysplasia is one of the manifestations of tumor cell atypia.

3. Changes in the structure and function of cellular organelles during cell damage. Cell damage is characterized by a greater or lesser disruption of the structure and function of all its components. However, under the influence of various pathogenic factors, signs of damage to certain organelles may predominate.

When exposed to pathogenic factors, changes are observed in the total number of mitochondria, as well as the structure of individual organelles. A decrease in the number of mitochondria relative to the total cell mass is observed. Stereotypical changes in individual mitochondria caused by most damaging factors include a decrease or increase in their size and shape.

Many pathogenic effects on the cell (hypoxia, endo- and exogenous toxic agents, including drugs in case of their overdose, ionizing radiation, changes in osmotic pressure) are accompanied by swelling and vacuolization of mitochondria, which can lead to rupture of their membrane, fragmentation and homogenization of cristae.

Disruption of the mitochondrial structure leads to a significant suppression of the respiration process in them and the formation of ATP, as well as to an imbalance of ions inside the cell.

Nucleus. Nuclear damage is accompanied by changes in its shape, chromatin condensation at the nuclear periphery (chromatin margination), disruption of the double contour, or ruptures of the nuclear membrane, and its fusion with the chromatin margination band.

Lysosomes. Under pathogenic influences, the release and activation of lysosomal enzymes can lead to " self-digestion " (autolysis) of the cell. The release of lysosomal hydrolases into the cytoplasm can be caused by mechanical ruptures of their membranes or a significant increase in their permeability. This is a consequence of the accumulation of hydrogen ions (intracellular acidosis), lipid peroxidation products, toxins, and other agents in the cells.

Ribosomes. When exposed to damaging factors, groups of ribosome subunits (polysomes), typically consisting of several ribosomes (monomers), are disrupted, the number of ribosomes decreases, and organelles detach from intracellular membranes. These changes are accompanied

by a decrease in the intensity of protein synthesis in the cell.

Endoplasmic reticulum. When damaged, dilation of the tubules of the reticulum is observed, leading to the formation of large vacuoles and cisterns due to fluid accumulation, focal destruction of the tubular membranes, and their fragmentation.

Golgi apparatus. Damage to the Golgi apparatus is accompanied by structural changes similar to those in the endoplasmic reticulum. This disrupts the removal of waste products from the cell, leading to a disruption of its overall function.

The cytoplasm is a fluid, low-viscosity medium containing the cell's organelles and inclusions. Exposure to damaging factors can cause a decrease or increase in cytoplasmic fluid content, proteolysis or protein coagulation, and the formation of "inclusions" that are not normally found. Changes in the cytoplasm's state, in turn, significantly affect the metabolic processes occurring within it, due to the fact that many enzymes (for example, glycolysis) are located in the cellular matrix. These changes also affect organelle function and the perception of regulatory and other influences on the cell.

4. Necrosis and autolysis. Necrosis (from the Greek " necro" meaning "dead") is the death of cells and tissues, accompanied by the irreversible cessation of their vital functions. Necrosis is often the final stage of dystrophies and dysplasias, as well as a consequence of the direct action of significant damaging factors. Changes preceding necrosis are called necrobiosis or pathobiosis.

Examples of pathobiosis include tissue necrosis in neurotrophic disorders due to tissue denervation, prolonged venous congestion, or ischemia. Necrobiotic processes also occur normally, representing the final stage of the life cycle of many cells. Most dead cells undergo autolysis, i.e., self-destruction of their structures.

The primary mechanism of autolysis is the hydrolysis of cellular components and intercellular substances under the influence of lysosomal enzymes. This is facilitated by the development of acidosis in damaged cells.

phacocytes and microorganisms, can also participate in the process of lysis of damaged cells. In contrast to the autolytic mechanism, the latter is called heterolytic.

Thus, the lysis of necrotic cells (necrolysis) can provide autolytic and heterolytic processes in which enzymes and other factors of both dead cells and living cells in contact with them take part.

5. Specific and nonspecific changes during cell damage. Any damage to a cell causes a range of specific and nonspecific changes.

Specific changes are understood as changes in the properties of cells that are characteristic of a given factor when it acts on various cells, or that are characteristic only of a given type of cell when it is exposed to damaging agents of various kinds.

Thus, exposure of any cell to mechanical factors is accompanied by disruption of its membrane integrity. Under the influence of uncouplers of oxidation and phosphorylation, coupling between these processes is reduced or blocked. High blood concentrations of aldosterone, one of the adrenal cortex hormones, cause the accumulation of excess sodium ions in various cells.

On the other hand, the action of damaging agents on certain types of cells causes changes specific to them.

For example, the influence of various pathogenic factors on muscle cells is accompanied by the development of myofibril contracture, on neurons – by the formation of the so-called damage potential, on erythrocytes – by hemolysis and the release of hemoglobin from them.

Damage is always accompanied by a complex of nonspecific, stereotypical changes in cells.

These are observed in various types of cells when exposed to a variety of agents. Common nonspecific manifestations of cellular alterations include acidosis, excessive activation of free-radical and peroxide reactions, denaturation of protein molecules, increased membrane permeability, and increased cellular absorption properties.

Identification of a complex of specific and non-specific changes in the cells of organs and tissues makes it possible to judge the nature and strength of the action of the pathogenic factor, the degree of damage, as well as the effectiveness of medicinal and non-medicinal treatments used for treatment.

References

1. Ф.И. Висмонт. Лекции по патофизиологии // Минск 1997, С.18
2. Turakulov I.Sh. Patologik fiziologiya asoslari. – Toshkent: TTA nashriyoti, 2019.
3. Л.Н. Осколок, Г.В. Порядиню Основные механизмы повреждения клеток // учебное пособия, Россия, Москва. 2016 г., 55 с.
4. Elmurotova D.B., Ro‘zimatova Sh. Sh., Umarova F.S. Insonning estetik tafakkuri // Лучшие интеллектуальные исследования, ISSN:3030-3680, Ч.45, Т.1, С.130-135, май-2025, Россия. https://scientific-jl.com/luch/article/view/13889
5. Elmurotova D.B., Farmonova Sh.Sh., Umarova F.S. Borliq va bo‘shliq: mavjudlik chegaralari haqida tafakkur // Лучшие интеллектуальные исследования, ISSN:3030-3680, Ч.44, Т.5, С.411-416, май-2025, Россия. https://scientific-jl.com/luch/article/view/13407
6. Elmurotova D.B., Jo’rayeva R.A, Umarova F.S. Bilimning chegarasi va rad etilishi”: eskeptitsizm va bilimga bo‘lgan ishonchsizlik muammosi // Лучшие интеллектуальные исследования, ISSN:3030-3680, Ч.44, Т.5, С.417-423, май-2025, Россия. https://scientific-jl.com/luch/article/view/13408
7. Elmurotova D.B., Kattaxodjayeva D.U., Komiljonov J.O. Blood and its functional structure // Texas Journal of Medical Science, Peer Reviewed International Journal, V.45, ISSN NO: 2770-2936, June 2025, P.40-44. https://zienjournals.com/index.php/tjms/article/view/6318
8. Elmurotova D.B., Odilova N.J., Odilova E.U. Invasive Radiation Diagnostic Methods // American Journal of Bioscience and Clinical Integrity, ISSN: 2997-7347, V.2., N.10, 2025. P.21-27.Amerika, https://biojournals.us/index.php/AJBCI/article/view/1555/1308
9. Elmurotova D.B., Kattaxodjayeva D.U., Samatov A.X., G’ulomov F.O. Classification of bone joints // Miasto Prysztosci, IF-9.9, ISSN-2544-980X, V.67, 2025, P.126-130. Poland. https://miastoprzyszlosci.com.pl/index.php/mp/article/view/7255/6825
10. Elmurotova D.B., Yusupov N.N., Sattarov Y.K., G'oyibnazarov R.B. Remature hearing loss// Miasto Prysztosci, IF-9.9, ISSN-2544-980X, V.66, 2025, P.206-210. Poland. https://miastoprzyszlosci.com.pl/index.php/mp/article/view/7156
11. Elmurotova D.B., Kattaxodjayeva D.U., Komiljonov J.O. Structure of the brain section of the head // Miasto Prysztosci, IF-9.9, ISSN-2544-980X, V.65, 2025, P.313-317. Poland. https://miastoprzyszlosci.com.pl/index.php/mp/article/view/7033
12. Elmurotova D.B., Kattaxodjayeva D.U., Tanirbergenova N.O. The Main Tasks of Biophysics and the Physical Foundations of Biological Structures// American Journal of Bioscience and Clinical Integrity, ISSN: 2997-7347, V.2, N.10, 2025, October P.71-77. https:///biojournals.us/index.php/AJBCI/article/view/1596/1345
13. Elmurotova D.B., Imamova L.N., Orifqulova M.F. Indications for holter monitoring // Miasto Prysztosci, IF-9.9, ISSN-L:2544-980X, V.62, 2025, P.155-160. Poland. https://miastoprzyszlosci.com.pl/index.php/mp/article/view/6828

14. Elmurotova D.B., Qo'ziyev S.R. Retinitis pigmentosa: clinical picture and diagnostic insights // Western European Journal of Linguistics and Education, ISSN (E): 2942-190X, V.3, Issue 10, October 2025, P.113-120. Germany, https://westerneuropeanstudies.com/index.php/2/article/view/2901/20091
15. Elmurotova D.B., Qo'ziyev S.R. Current Perspectives on the Etiology and Epidemiology of Retinitis Pigmentosa // American Journal of Bioscience and Clinical Integrity, ISSN: 2997-7347, V.2, N.10, 2025, October P.119-124. America. https://biojournals.us/index.php/AJBCI/article/view/1641/1380
16. Elmurotova D.B., Urmanbekova D.S., Bozorov M.B. Main risk factors of myocardial infarction // American Journal of Bioscience and Clinical Integrity, ISSN: 2997-7347, V.2, N.12, 2025, Dekabr P.6-9. https://biojournals.us/index.php/AJBCI/article/view/1763/1476