Conclusions. The emergence of new drugs, the mechanism of action of which is aimed at correcting the causes and mechanisms of the development of cystic fibrosis, gives hope for a significant improvement of the prognosis of patients' lives in the coming years.

MOLECULAR MECHANISMS OF CONSTANT PROLIFERATION OF TUMOR CELL

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Introduction. In normal tissues, growth processes and the cell cycle are carefully monitored. Stimulation to division in a cell is carried out by growth factors that bind to a receptor on the cell surface, that has an intracellular domain with tyrosine kinase activity. The activation of the tyrosine kinase domain leads to the activation of the intracellular pathways that regulate the cell cycle.

Aim. The aim of this work was to study the molecular mechanisms of chronic tumor cell proliferation.

Materials and methods. To achieve this goal, an analysis of literary sources and summary of the received information was carried out.

Results and its discussion. In a tumor, this signaling system is impaired, and cell growth and division are stimulated in the absence of external stimuli. In this case, the stimulation of cell division in tumor cells can be carried out in different ways. Tumor cells can themselves produce growth factors as a result of amplification or mutation in genes, encoding growth factors. An increase in the concentration of growth factors leads to stimulation of proliferation. An increase in the content of receptor proteins located on their surface can lead to a change in the signal system in tumor cells. This, is turn, leads such cells to a hypersensitive state in relation to growth factor. Similar effects can be caused by mutations or rearrangements in genes, encoding receptors of growth factor, which will lead to changes in the receptor molecule. In addition, the activation of the components of the tumor cell signaling system can occur independently from growth factors and their receptors at lower levels of regulation. This excludes the need for their stimulation by forming of a ligand-receptor complex. In recent years, studies of the tumor cell genome have shown that somatic mutations contribute to the activation of signaling systems involving growth factor receptors. It is known that mutations and rearrangements of various genes lead to activation of signaling systems both at the level of growth factors and their receptors, and at a lower level of signal transmission through a cascade of proteins into the cell nucleus.

Conclusion. Changing and/or disrupting the stages, weakening the signaling systems, can contribute to the development of adaptive resistance towards to drugs whose targets are mitogenic signals. It is proved that the more signaling proteins are in cell, the more intensive is the cell proliferation and, respectively, tumor growth.

THE ROLE OF LIPOFUSCIN IN AGING AND PATHOLOGY

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Introduction. The age-pigment, lipofuscin that accumulates in cells intrinsically and progressively with age is considered as the hallmark of aging. Lipofuscin is a pigment consisting of

oxidized and cross-linked proteins and lipids, which makes it difficult to degrade and remove it from cells. To date, there is still no clear understanding of the mechanisms of its formation, its role in the body in general and in aging processes in particular.

Aim. The aim of this review is to provide an overview of the current knowledge on the mechanisms of formation and accumulation of lipofuscin based on the recent publications.

Materials and methods. Data analysis of literature and Internet sources.

Results and discussion. The presence of lysosomal enzymes (acidic phosphotases) in lipofuscin suggested that lipofuscin may appear due to oxidative stress, which results in the accumulation of "residual bodies" in the cell that are products of oxidation and peroxidation of lipids that are not susceptible to degradation by lysosomal enzymes. Mitochondrial enzymes, fragments of mitochondria and the endoplasmic reticulum are also found in lipofuscin granules. Therefore, the nature of the accumulation of lipofuscin in cells can also be associated with the destruction of cellular organelles that have not been utilized by lysosomes. So, mitochondria are most susceptible to degradation into lipofuscin granules (mitolipofuscin). It is known that with age, there is an increase in the accumulation of lipofuscin in different cells: in the cells of the brain, heart, retina, skeletal muscle, and skin. Besides, it accumulates with an increase in the functional activity of the organ, with atrophy, and decreases with dystrophy and necrosis. At the moment, drugs and substances that reduce the amount of lipofuscin can be divided into the following groups: stabilizing membranes (piracetam); activating and improving the metabolism of fats and proteins (metformin, monacolin, curcumin); protecting against reactive oxygen species (flavonoids, meclonoxate, monacolin); able to bind to lipofuscin and activate its oxidation (flavonoids, meclonoxate); autophagy (direct removal of lipofuscin by autophagasome).

Conclusions. Thus, the variability of data on the importance of lipofuscin and its role in intracellular exchange processes, in particular its place in involutive processes and in pathology, make it necessary to carry out further studies in this direction.

PROSPECTIVE USE OF GENOME EDITING TECHNOLOGIES: THE COMPLEX OF CRISPR-CAS9

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Introduction. CRISPR stands for "clustered regularly interspaced short palindromic repeats", which are repeated short DNA sequences that are palindromes (reading the same in both directions) interspersed with short, nonrepeat "spacers". CRISPRs originally were discovered in archaea and bacteria, where the spacers were bits of DNA from infecting viruses. The spacers are transcribed into short CRISPR RNAs that attract a cutting enzyme, such as Cas9 (CRISPR-associated protein 9). The complex of CRISPR-Cas9 then searches the DNA for matching spacer sequences and, using natural DNA repair, cuts them out by breaking the double helix across both strands. In this way, a bacterial cell can recognize bits of viral DNA in future encounters and promptly remove them, akin to an animal's immune system. Cas9 was the first DNA-cutting enzyme used with CRISPRs. Others, with differing targets, include Cas13 and CPF1.

Aim. To analyze and study of genomic editing technologies using the complex of CRISPR-Cas9.

Materials and methods. Google Scholar, EMBASE, MEDLINE and Medscape resources have been applied for search and analysis up to March 2020 using terms "genome editing technologies" and "CRISPR-Cas9".

Results and discussion. CRISPR systems are attractive for their high efficiency, programmability and inheritance not only for bacteria and archaea, but also for humans. The complex of