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