Conclusions. A review of the results of the studies showed high efficacy of FPD in the treatment of diabetes, blood and liver diseases, oncological, neurodegenerative, as well as the accompanying effects of cell transplantation associated with improving psycho-emotional state, disappearance of chronic insomnia and fatigue, increased sexual function in men and women, slow dawn aging etc.

MODERN METHODS OF CYSTIC FIBROSIS TREATMENT

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Introduction. Cystic fibrosis (CF) is the most common hereditary disease with an autosomal recessive mode of inheritance, universal multisystem exocrinopathy. Cystic fibrosis gene is Cystic Fibrosis Transmembrane conductance Regulator (CFTR) that was mapped and cloned at 1989 year. The protein encoded by this gene is an epithelial cell membrane protein. Its main function is to regulate the transport of chloride channel ions through the cell membrane. The presence of a defective gene leads to increase of the mucus viscosity of the excretory glands. Primarily it is manifested by pathology from the respiratory and digestive systems.

Aim is to study modern methods of cystic fibrosis treatment from the position of etiopathogenesis.

Materials and methods. The regulatory and scientific sources of Ukraine, Russia, the EU, Israel, and the USA about the questions of the cystic fibrosis treatment have been analyzed and compared.

Results and discussion. Treatment of patients with cystic fibrosis in most countries is provided according to protocols that are approved at the state level. In Ukraine the treatment depends on the Unified Clinical Protocol (approved by the Order of the Ministry of Health of Ukraine on July 15, 2016 No. 723). It has a symptomatic and pathogenetic character and is provided throughout life with the use of Pancreatin replacement enzyme therapy, daily activities by the dilution viscous sputum and clean the patient's bronchial ways from it, with antibacterial therapy of respiratory tract infections. Modern methods of CF treatment include drugs of gene therapy and modulators (correctors and potentiators).

Gene therapy is a group of methods basic on transferring nucleic acids (DNA and RNA) into cells in order to replace a defect caused by a gene mutation. Recombinant adenoviruses, adeno-associated vectors, liposome-mediated CFTR gene transfer use as modern vectors for transferring the normal CFTR gene into the cell. They can be carried to the target organs (lungs) through inhalation

Also in recent years, drugs from the group of modulators (correctors and potentiators) have widely distributed. They compensate for the effects of mutations and are essentially the drugs of pathogenetic therapy. Correctors improve the maturation of CFTR protein, potentiators increase the probability that the defective channel will be open and allow chloride ions pass through the channel pore.

The first drug of this group is Ivacaftor (VX-770, Kalydeco). It is the potentiator, that increase the opening of the CFTR ion channel on the cell membrane through activation of adenylate cyclase way.

The main mechanism of corrector’s action is to improve the maturation of the CFTR protein through direct binding to it, or through adaptation of protein homeostasis and a decrease in protein degradation. The most famous corrector is Lumacaftor (VX-809). It was found that this medicine stabilizes the CFTR protein and increases its movement to the surface of the cell membrane. Also it is able to partially restore the function of the protein through stabilizing the N-terminal domain of the CFTR protein. However, monotherapy of Lumacaftor leads to a slight decrease in chlorides level in the sweat test. Therefore, an important event in the treatment of CF was the opening of the combination drug Ivacaftor/Lumacaftor (Orkambi) in 2015. The combination of a potentiator and a corrector made it possible to get a clinical effect in patients with the most common mutation by cystic fibrosis – a deletion of phenylalanine in position 508.

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