and thus aging and death. Indeed, dysregulated immune response has been linked to cardiovascular disease, inflammation, Alzheimer's disease, and cancer). Many of the proposed theories interact with each other in a complex way.

Conclusion. Thus, the study of the mechanisms of aging and the search for methods of extending healthy life is not only the most promising scientific direction, but also could lead to interventions that slow or alter aging.

THE MITOCHONDRIAL DYSFUNCTION IMPORTANCE IN DISEASES OF THE NERVOUS SYSTEM

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Introduction. Neuron injury is a cardinal problem of neurology and psychiatry. It is know that programmed neuron death is a main factor in the pathogenesis of neurodegenerative diseases and damage of the central nervous system. Despite numerous research works, the mechanisms involved in the loss of neurons are still not fully understood. This greatly complicates the development of the method of etiotropic and pathogenetically substantiated conservative treatment of Parkinson's disease, Alzheimer's disease, and neurodegenerative processes in the retina. The recent research findings indicate that the disturbances of mitochondrial function have an important place in the development of these diseases. Disturbance of cellular energy is one of the universal pathophysiological mechanisms of central nervous system damage in neurodegenerative, cerebrovascular, demyelinated, dysmetabolic and other diseases. The reason for this is that neurons are the most "energy-dependent" type of cells in the body.

Aim. To study the role of mitochondrial dysfunction in the pathogenesis of the central nervous system diseases.

Materials and methods. The analysis of scientific works for the years 2010-2019 on the problems of mitochondrial diseases, neurodegenerative diseases, molecular mechanisms of necrosis and apoptosis was carried out with using the search engine Google, PubMed, eLIBRARY and etc.

Results and discussion. Energy mitochondrial insufficiency is divided into primary and secondary. Primary mitochondrial insufficiency is characteristic of mitochondrial encephalopathy – a group of diseases caused by structural, biochemical, and genetic defects of mitochondria and mitochondrial DNA (mtDNA). The nature and severity of clinical manifestations of mitochondrial diseases is determined by the severity of mtDNA mutation, the percentage of mutant mtDNA in specific organs and tissues, the threshold of sensitivity of organs and tissues containing mtDNA to oxidative phosphorylation defects. The main mutations of mtDNA include: mutations associated with deletions of more or less significant fragments of the molecule (progressive external ophthalmoplegia, Pearson syndrome); point mutations – in which cells are formed that accumulate normal and mutant molecules in different ratios (Leber optic neuropathy, melas-syndrome).

Secondary mitochondrial insufficiency and energy dysfunction are one of the main mechanisms for the development of neurodegenerative diseases, acute and chronic brain ischemia. It's characterized by disorders of mitochondrial movement inside the neuron, their conjugation and separation, the formation of excessive fragmentation. Thus, in Alzheimer's disease excessive mitochondrial fragmentation with damage to the internal membrane develops. β-Amyloid Peptide and Tau-protein, which accumulate in cells during Alzheimer's disease, are able to suppress axonal transport of mitochondria, resulting in impaired neurotransmitter release and synaptic plasticity in the neuron.

Suppression of mitochondrial separation in Parkinson's disease leads to the accumulation of oxidized dopamine. This causes the accumulation of α-synuclein and dysfunction of lysosomes. The latter factor makes a negative effect on mitochondrial function, and thus a metabolic vicious circle is formed.

Mutations in the HTT (huntingtin) protein disrupt the dynamics and then the function of mitochondria in Huntington's disease, indirectly affecting the Drp1 protein.

Thus, the lack of energy production in neurons with mitochondrial dysfunctions leads to depolarization of their membranes, the opening of the channels of glutamate receptors, the Ca^{2+} ions in an excessive amount enter the cell and activate caspases and other enzymes that initiate autolysis and apoptosis. Currently, caspase activation is considered as a possible mechanism for neuronal death in neurodegenerative diseases and AIDS-dementia. It has been founded that huntingtin, a product of the Huntington's chorea gene, and presenilins (PS-1 and PS-2) in Alzheimer's disease are a target for caspase-3.

Conclusions. Disturbance of the mtDNA structure and the processes of mitochondrial movement inside the neuron, as well as the processes of their conjugation and separation, lead to disorder of the energy processes inside the nervous cells and the development of a some neurodegenerative diseases and mitochondrial encephalopathies. The study of the mitochondrial metabolic activity provides information that allows not only to understand the pathogenesis of the disease, but also to find out which drugs will be effective for their treatment.

RISK FACTORS OF THE DEVELOPMENT OF NEWBORN'S CONJUGMENT JAUNDICE

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Introduction. Jaundice – the appearance of jaundice on the skin and mucous membranes. According to literary data in the early neonatal period, it appears in 50-60% of full-term and 70-90% of premature babies. The main cause of this condition is hyperbilirubinemia, the clinical significance of which lies in the development of deep metabolic disorders, changes in the functional state of the liver and neurological damage.

Despite numerous studies conducted in different years to study the causes of this state of the neonatal period, the relevance of this problem does not decrease. During this period, metabolic disorders are most often associated with an increase in serum bilirubin levels.

A certain part of these states is transient for the child and does not require special correction. However, in recent years, most neonatal jaundice occurs with a high level of bilirubin in the blood serum and takes a protracted course, resulting in a high risk of complications due to the neurotoxicity of indirect bilirubin, which in turn necessitates early diagnosis and adequate treatment of this condition. In the literature there is no sufficient data on the risk factors for the formation of conjugation hyperbilirubinemia in children. Along with this, there is a shortage of drugs that contribute to the rapid and effective relief of hyperbilirubinemia. The underestimation of the dynamics of the development of the pathological process with severe hyperbilirubinemia, delayed therapeutic intervention can lead to death or severe disability. On the other hand, jaundice in the neonatal period is the most common cause of unreasonable and prolonged treatment with the use of invasive methods and a large number of drugs that are harmful to the body of the newborn.

Aim. The aim of the study is to identify risk factors associated with the formation of conjugation hyperbilirubinemia in newborns, the development of prevention methods and the consideration of the possibility of creating new dosage forms for the treatment of this pathological condition.

Materials and methods. This retrospective study was conducted to study the clinical and biochemical parameters of newborns with manifestations of hyperbilirubinemia using neonatal development cards "KCH RT №2, filia «HC»", stock partnership «Ukrzaliznytsya». Biomedical risk factors for conjugated jaundice have been studied for many years.

The main group (MG) consisted (18) of children with signs of conjugative hyperbilirubinemia. The control group (CG) (15) of children was selected by the method of “random control” 1 of practically healthy newborns without manifestations of physiological jaundice. The main and control groups were compared in absolute terms (maternal age, gestational age, baby weight at birth, etc.) were comparable. Risk factors were divided into 2 groups: 1. Premorbid factors (state of health and age of parents, bad habits (smoking, alcohol, drugs); 2. Perinatal factors (viral infections, transferred during pregnancy; the