forms. This can be avoided if manufacturers provide reliable information about the drug, and a person who buys a vitamin drug will carefully study the composition and instructions.

Absorption of vitamins from multivitamin complexes is also important. There are two opinions. At first, the components of the vitamin drug are not absorbed (or absorbed only partially). The fact is that the absorption of many vitamins and minerals depends on the substances that come with them. For example, fat-soluble vitamins are metabolized only when consumed together with fats. Or the opposite example: iron and calcium «mutually destroy». Therefore, different vitamins and minerals in one tablet (capsule, dragee) can disrupt each other's absorption. The opposite view – vitamins and minerals from multivitamin drugs are absorbed even better than natural ones. And with the problem of «mutually destroying» vitamins and minerals, the pharmacological industry copes with microencapsulation.

If you suspect hypovitaminosis, you should definitely go to a polyclinic. This can be a standard biochemical blood test, a comprehensive analysis that checks the level of essential vitamins, or urine analysis.

The intake of vitamins is necessary for those people who use diets, regardless of the season. Experts recommend to regularly take vitamins to those involved in sports, children during their active growth, pregnant, lactating women and vegetarians. Preventive course of vitamin therapy in the winter-spring period is recommended to take place all people.

Conclusions. Thus, the question of whether to recommend the use of multivitamins or refuse them, there is no precise answer so far. In modern conditions, the habit of using multivitamins is a useful part of a healthy lifestyle. Before buying a vitamin complex, consult your doctor. With proven hypovitaminosis, take a specific vitamin or group of necessary vitamins. Minimize and optimize the heat treatment of foods, eat a variety of foods, regularly eat seasonal fruits and vegetables, replace white bread and pastries with more useful cereals.

THEORIES OF AGING

Nai Abraham
Scientific Supervisor: associate professor Myronchenko S.I.
National University of Pharmacy, Kharkiv, Ukraine
Abraham.nai123@gmail.com

Introduction. Aging is a combination of age-related changes in the body, leading to the formation of age-related pathology and an increase in the likelihood of death. This deterioration is the primary risk factor for major human pathologies including cancer, diabetes, cardiovascular disorders, and neurodegenerative diseases. The study of the fundamental mechanisms of aging is needed to develop a more effective treatment of diseases of the elderly and senile age.

Aim. Carry out an analytical review of the mechanisms for the development of aging and the most effective methods of its prevention.

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

Results and discussion. Many theories have been proposed to explain the process of aging, but neither of them appears to be fully satisfactory. Modern biological theories of aging in humans fall into two main categories: programmed and damage or error theories. The programmed theories imply that aging follows a biological timetable, perhaps a continuation of the one that regulates childhood growth and development. This regulation would depend on changes in gene expression that affect the systems responsible for maintenance, repair and defense responses. The damage or error theories emphasize environmental assaults to living organisms that induce cumulative damage at various levels as the cause of aging. The programmed theory has three sub-categories: programmed longevity (aging is the result of a sequential switching on and off of certain genes, with senescence being defined as the time when age-associated deficits are manifested; endocrine theory (biological clocks act through hormones to control the pace of aging; aging is hormonally regulated and the evolutionarily conserved insulin/IGF-1 signaling (IIS) pathway plays a key role in the hormonal regulation of aging); immunological theory (the immune system is programmed to decline over time, which leads to an increased vulnerability to infectious disease

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

Nasteka M.V.
Scientific supervisor: ass. prof. Hnatiuk V.V.
National University of Pharmacy, Kharkiv, Ukraine
nastekamaria@gmail.com

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.