IMMUNITY OF AGING

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Introduction. The human immune system is one of the most interesting and poorly understood in modern medicine. It can be affected by many processes and factors, but the impact of aging on immunity is considered the most powerful and irreversible.

Aim. Analysis of literature, studying the basic patterns and properties of aging

Materials and methods. Acquaintance with the literature, revealing important aspects.

Results and discussion. In the age imbalance of immunity, the most important is the involution of thymus and the decrease in the level of thymic hormones. This leads to a change in the function of T cells and the violation of their immunoregulatory properties. Reduced T-lymphocyte reactivity is manifested:

In reducing the ability of proliferation and blast transformation under the influence of PGA; Increased survival of allogeneic skin transplates and induction of induced tumors.

In the 50-65 years, the number of T-lymphocytes decreases, while B-lymphocytes increase, T-killers are not formed, the response to T-dependent antigens sharply decreases and is stored on T-independent. The quantitative and functional defect of T cells leads to a decrease in the response to exogenous antigens and amplifies them with respect to endogenous ones. The mortality of such persons is high and is caused by diseases of the vessels of the brain and heart.

Many signs affect aging by their own, independent from other mechanisms.

According to the hypothesis of hypothalamic hours old age is seen as a violation of the internal environment of the organism, which contains an increase in the activity of the hypothalamus. As a result of the elderly, the secretion of hypothalamic hormones (liberine) and a number of pituitary hormones (gonadotropins, somatotropins) and insulin increases sharply. But with the stimulation of some structures of the hypothalamus, others at the start destroy their activity, which leads to the "solution" of many aspects of the metabolism and functions of the organism.

Our DNA is damaged by internal and external factors every day. Although cells are able to fight mutations in the genes, this ability persists only to a certain level of damage. Spontaneous hydrolytic reactions, active forms of oxygen, ultraviolet radiation – all of these factors lead to the accumulation of damaged DNA in cells over time. If it is not "repaired" in time, the risk of developing cancerous diseases increases. It is the violation of DNA repair mechanisms that is the cause of some of the diseases associated with accelerated aging – the Bloom and Werner syndromes. If mutations in DNA exclude genes-suppressors of tumors or include oncogenes – there is cancer. However, for the transformation of the cell into cancer, not one but several mutations is required. The cessation of the ability of cells to restore the genome leads to the fact that the incidence of oncology increases with age.

Epigenetic changes are functional changes in the inherited DNA, but are not direct modifications to the genetic code. Epigenetic mechanisms include DNA methylation and modification of structural proteins – histones. The study of these mechanisms began only in the past decade, so it is difficult to specify the exact mechanisms of connection of epigenetic changes and aging. However, some facts are already known. So, according to the DNA methylation of individual cells, one can judge their age, and the restraining of histone modifications extends the life span of worms. Further epigenetic studies promise to give a better understanding of general aging processes and possibly create effective therapies.

Proteostase is a term that indicates the constancy of all the proteins in the body. For the normal functioning of the cell, it is vital that the proteins have the correct conformation. Conformational stability is provided by a number of enzymes, the most significant of which are chaperones and proteases. Like DNA, proteins are eventually damaged, and these damage accumulate. It is known that structurally inappropriate proteins result in aging-related diseases such as Alzheimer's and Parkinson's disease, as well as cataracts. There are works in which it is argued that gene therapy is the best way to maintain proteostase.

Excessive nutrition leads to obesity and accelerated aging. Restriction of food consumption, on the other hand, is one of the most well-known methods used in practical bioheronthology and leads to prolonged life expectancy in many mammals.

Radioactive radiation as a special effect. The higher the total dose of radiation, the more the life expectancy decreases. In some experiments with irradiation in small doses showed an increase in life expectancy. To explain the causes of this phenomenon, the following assumptions were made: irradiation may have a therapeutic effect for already developed diseases, or radiation acts as a prophylactic, preventing new infectious diseases.

Conclusion.For functional nutrition a balanced composition of food is required: micro- and macroelement (zinc, selenium, calcium, magnesium, etc.), vitamin (vitamins A, C, E, D, B) and protein composition of products, level of antioxidant content. It is these components that determine the quality of the food. Thus, on the basis of modern ideas about the important physiological role of natural immunity, it can be argued that the normal functioning of the immune system is the most important condition for increasing the life expectancy.

HUMAN PAPILLOMA VIRUS AND CERVICAL CANCER: FEATURES OF THE COURSE AND PREVENTION

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Introduction. The papillomavirus infection is highly contagious, it can initiate malignant diseases, which is why it is an actual problem of modern society. This disease is transmitted sexually and therefore, according to the WHO, HPV occurs in every sixth inhabitant of the Earth. Diseases associated with HPV: cervical intraepithelial neoplasia and cervical cancer. In the world of cervical cancer, the fourth most common cancer among women is cancer (it accounts for 7.5% of all deaths from cancer)**.**

Aim. Study of the role of human papillomavirus in the pathogenesis of cancer, in particular, cervical cancer. Analysis of methods for the prevention and treatment of human papillomavirus infections in the world and in Ukraine

Materials and methods. Analysis of the scientific literature and the results of the advanced research in the field of medicine and pharmacology.

Results and discussion. Human papillomavirus belongs to the family Rapillomaviridae, DNA-containing, epithelotropic with pronounced cytopathic effect (destruction of the nucleus and cellular organelles). The genome of double-stranded DNA is divided into three functionally active regions, one of which (LCR) is responsible for transcription of genes, the other (L) encodes the structural proteins of the capsid. The third (E) has a leading role in carcinogenesis. Under the action of proteins E 1,2,6,7, the control of suppressor genes of tumor growth P 53 and P Rb is violated, which leads to the development of neoplasia.

In infected cells, the viral genome can exist in two forms: episomal (more often found in the tissue of flat candida and epithelial dysplasia of low degree) and integrated (manifested in epithelial dysplasia of high degree and cervical cancer), where, in the presence of favorable factors, the implementation of the coding in The genome of the virus program aimed at its reproduction. This, in turn, leads to proliferation and enhanced synthesis of DNA and RNA by the host cell. Due to what it increases the ability to divide. Since the synthesis of own proteins in infected cells is suppressed, their differentiation (maturation) does not occur. Such cells, reaching the 2-3rd row of the intermediate layer of the epithelium of the cervix, are prone to destruction, which violate the dynamics of cellular renewal of the epithelial layer. Dysplasia occurs, which is based on proliferation and structural rearrangement of epithelial cells. The virus is highly contagious. The incubation period lasts from 1 to 20 months. IDPs can stay (persist and multiply) for a long time in the surface layer of the epithelium. In the literature there is evidence that the "entry" of PVI occurs at the level of immature cells of the epithelium of the skin and