agents. The goal of conservative therapy is to correct valvular insufficiency of the deep, saphenous and peripheral veins; improvement of microcirculation and tissue perfusion; stimulation of lymphatic drainage; inhibition of leukocyte activation and synthesis of inflammatory mediators.

Conclusions. Thus, high prevalence, rapid rejuvenation, as well as a significant number of relapses require modern diagnostics and adequate treatment of varicose veins, which is an important medical and social problem.

THE UBIQUITIN–PROTEASOME PROTEOLYTIC PATHWAY IN NORMAL AND DISEASE STATES

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Introduction. Proteolysis is essential for numerous developmental and physiological processes. However, dysregulation of protease activity underlies many diseases and pathological conditions, including cancer, inflammation and infection. In all tissues, the majority of intracellular proteins are degraded by the ubiquitin (Ub)–proteasome pathway (UPP).

Aim. Carry out an analytical review of the role of protein degradation by the ubiquitin–proteasome pathway in normal and disease states.

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

Results and discussion. Cells contain multiple proteolytic systems to carry out the degradation process and complex regulatory mechanisms to ensure that the continual proteolytic processes are highly selective. The pathological states associated with the ubiquitin system can be classified into two groups: (a) those that result from loss of function-mutation in the ubiquitin system enzyme or in the recognition motif in the target substrate that lead to stabilization of certain proteins, and (b) those that result from gain of function-abnormal or accelerated degradation of the protein target. Studies that employ targeted inactivation of genes coding for specific ubiquitin system enzymes and substrates in animals can provide a more systematic view into the broad spectrum of pathologies that may result from aberrations in ubiquitin-mediated proteolysis. Therefore, excessive breakdown of cell constituents is prevented. Because the UPP is responsible for the turnover of so many different cellular proteins, there are critical mechanisms that regulate its function precisely. The importance of UPP lies in the circulation of transport proteins, in the presentation of antigens to the immune system, and in how uremia activates UPP, causing muscle depletion, because these functions are of particular interest to nephrology. However, the UPP also plays important roles in the regulation of other cellular functions, ranging from the control of the cell cycle to activities that promote cancer. Indeed, inhibitors of proteasome activity, the final component of the pathway, have emerged as novel chemotherapeutic agents.

Conclusions. Better understanding of the processes and identification of the components involved in the degradation of key regulatory proteins will lead to the development of mechanism-based drugs that will target specifically only the involved proteins.

MOLECULAR MECHANISMS OF AGING

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Introduction. Aging is a complex of various mechanisms at the molecular, cellular, organ and system levels, which lead to irreversible changes in organs and tissues with the extinction of body functions. Interest in the subject of aging does not diminish for many centuries. Although modern

medicine has achieved a significant increase in the average life expectancy, aging remains largely mysterious and, unfortunately, an inevitable process.

Aim. The purpose of this work was to study the molecular mechanisms of human aging.

Materials and methods. To achieve this goal, an analysis of literary sources and generalization of the received information was carried out.

Results and discussion. Currently, there are several theories explaining human aging. These are theories that consider aging as a special program, and theories that assume that aging is associated with the accumulation of certain injuries in the body. Theories of programmed aging are based on the fact that the functioning of a living organism is programmed by nature for the period of its active life. After the program is completed, the activity of the hypothalamus and the endocrine system changes, which leads to a decrease in the efficiency of the functioning of the body. The telomeric theory suggests that in somatic cells during each replication, due to the peculiarities of enzyme work, the ends of chromosomes, telomeres, are shortened. At the same time, sections of the genome that are important for cell survival disappear. According to the free radical theory of aging, cell dysfunction is caused by active forms of oxygen, which cause a number of elderly diseases: cardiovascular, tumor, diabetes, etc. The next theory that explains aging is the accumulation of intracellular and extracellular by-products of metabolism that damage cells and tissues: cholesterol, amyloid and glycosylated proteins. Aging is also associated with impaired regulatory mechanisms, for example, the relationship between pro- and anti-inflammatory systems.

Conclusions. Existing theories differ on how much the accumulation of these harmful products is programmed in the genome, and whether such "programming" is a suicide program or simply an inevitable payment for additional evolutionary advantages. In addition to the actual damage, the rate of their accumulation is important, due to the overall intensity of metabolism. The most significant changes in the lifespan of model organisms were associated with mutations that modulate the intensity of metabolism.

MODERN PROBLEMS FOR TREATING MULTIPLE SCLEROSIS

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Introduction. Multiple Sclerosis (MS) is a chronic progressive disease of the central nervous system characterized by demyelination and degeneration of the nerve fiber, has a polymorphic clinical picture, adverse course, early disability, and limitation of living conditions. MS suffer from mainly young people of working age, which testifies to the social significance of this pathology.

Aim. The purpose of this work was to study modern approaches to MS treatment.

Materials and methods. To achieve this goal, an analysis of literary sources and generalization of the received information was carried out.

Results and its discussion. Treatment of MS remains one of the most serious problems of practical neurology. The main tasks of MS therapy are the treatment and prevention of exacerbations and the weakening of the progression of the disease. The current standard for exacerbation of MS is high-dose pulse therapy – 1000 mg of methylprednisolone for 5 days. Methylprednisolone slows the activation and proliferation of T-lymphocytes, reduces the formation of antibodies, reduces the permeability of the blood-brain barrier. For the treatment of MS, immunomodulators (preparations of interferon-beta, glatiramer acetate) and immunosuppressants (mitoxantrone and natalizumab) are used exacerbated. Until now, preparations of the first choice remain interferon-beta and glatiramer acetate. Glatiramer acetate acts on the initial link of the pathogenesis of MS, forming a strong linkage with the main histocompatibility complex of class II, displacing other autoantigens from the tri-molecular complex and becomes a pseudo-target for activated auto-aggressive T-lymphocytes. Currently, along with first-generation drugs for treatment of MS, use of second-generation drugs, in particular, Fingolimod, which is a synthetic modulator of singgosin-1-phosphate receptors on the surface of lymphocytes. It reduces the output of activated T lymphocytes from the lymph nodes and their penetration into the central nervous system, thus reducing the severity of inflammation and degree of damage.