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THEORIES OF HUMAN AGING

Abstract: *the article discusses the theories of human aging, the mechanisms of their action in humans and animals.*

Key words: *mutation, DNA, aging, research, thymus gland*

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ТЕОРИИ СТАРЕНИЯ ЧЕЛОВЕКА

Аннотация: *в статье были рассмотрены теории старения человека, механизмы их действия у людей и животных*

Ключевые слова: *мутация, ДНК, старение, исследования, вилочковая железа.*

Introduction: This article will be devoted to biological theories of aging. What we observe as biological aging is a combination of many independent causes, some of which act at the level of individual molecules, others at the level of individual cells, as well as others at the level of tissues, organs and whole organisms. Therefore, when I present various theories of aging, it should be borne in mind that if they are operational, they act in concert, causing biological aging. The number of theories in a particular field usually inversely reflects the level of knowledge in this field. The purpose of the study is to study the theories of aging and identify their mechanisms.

Materials and methods of the study - review of the experience of American scientists, a comparative analysis was carried out in people from 10 to 20 years old (the purpose of this analysis was the work of the immune system in children and older people) and an anonymous survey among middle-aged and elderly people, in order to detect diseases after a decrease in the performance of the immune system. An anonymous survey was conducted in order to identify the statistics of mutational diseases after DNA damage, information about the experiment of American scientists was taken.

The results of the study and their discussion.

Studies of lower organisms have shown that aging genes can be isolated. One of the frequently suggested examples of programmed aging is the limited lifespan of individual human cells that are taken from the body and propagated in artificial laboratory conditions. In semilopast organisms, where only one major reproductive event occurs, reproduction is often followed by surprisingly rapid aging [1]. After conducting an experiment, we found out that a specific salmon has rapid aging caused by excessive secretion of hormones, which occurs in a precisely defined short period of time after spawning. Perhaps the most clearly defined clock in humans is the female menstrual cycle, which turns on somewhere between the ages of 11 and 16 and stops between the ages of 45 and 55. Another very important mechanism that we have discovered is involved in the involution of the human immune system. After conducting an analysis, we realized that the efficiency of the immune system in people after 20 years decreases. These immune clocks are located in the thymus gland, the size of which is greatest in adolescence and decreases to such an extent that it is almost impossible to detect after 50 years. After conducting an anonymous survey among middle-aged and elderly people, we found that decreased immune function leads to many critical age-related conditions, such as (1) increased susceptibility to infectious diseases such as pneumonia and influenza, (2) increased risk of cancer as a result of weakened immune system surveillance, and (3) changes in the walls of blood vessels leading to the most common age-related atherosclerosis.

Among the many proteins in the body are enzymes that catalyze the body's chemical reactions, including the synthesis of RNA and mRNA proteins themselves, hemoglobins that transport oxygen to our cells, antibodies that fight bacteria and viruses, and some of the most important hormones that regulate the activity of many of our organs.

DNA, RNA and proteins are constantly being damaged both from the external environment (for example, radiation) and the internal environment of the body (for example, free radicals, which are highly reactive by-products of normal functions)

[2]. Damage to the DNA of germ cells (sperm or eggs) can lead to the formation of a hereditary mutation for familial Alzheimer's disease or for sickle cell anemia, for example, which can be transmitted from generation to generation and lead to specific human genetic diseases. Based on this, we found out that aging can be the result of the accumulation of mutations in the DNA of somatic cells (liver, brain).
The accumulation of somatic mutations over time can lead to
the formation of an increased amount of altered messenger RNA and, in turn, altered proteins, which can lead to a decrease in the survival of cells, tissues and organs. Survival of cells, tissues and organs.

Studies of specific proteins in young and old animals have not revealed significant age differences in the amino acid sequences that make up cellular proteins [3]. However, studies of cellular proteins with age have revealed an increase in the number of posttranslational modifications (changes in proteins after they have been synthesized).

Calorie restriction. This theory assumes that all organisms have finite metabolic lifespans and that organisms with higher metabolic rates have shorter lifespans. After conducting an experiment, we found that mice, for example, have an extremely high metabolic rate and a very short lifespan. It is possible to experimentally change the metabolic rate in certain species of fish, whose internal body temperature varies depending on the temperature of the surrounding water. Thus, due to a decrease in the temperature of the outer water, the metabolic rate decreases, and fish survive longer than fish living in warmer water.

These experiments show that if you reduce the calorie intake of rodents by about 40 percent, while providing them with the necessary nutrients and vitamins, you extend their life expectancy by about 35 percent. Subsequent studies have shown that calorie restriction is even effective for increasing life expectancy if it is started in middle age. In addition, the restriction of calorie intake by rodents also delayed the onset and development of many age-related diseases and physiological parameters that affect aging rodents, such as cancer. Studies using data from life

insurance companies show that the shortest life expectancy is observed in people with two extreme values of weight: underweight and overweight.

Conclusions:

The theories I have outlined above are not intended to be exclusive.

For example, DNA damage by free radicals can cause somatic mutation. There is also evidence supporting more than one theory of aging. Let's look at recent data regarding the effects of aging on chromosomal telomeres. Most of the genetic material in human cells is located inside a structure called a chromosome [4]. There are 22 pairs of human chromosomes; males have X and Y chromosomes, while females have two X chromosomes. When cells divide, chromosomal DNA replicates, having each DNA for daughter cells. However, the nature of this DNA replication leads to incomplete replication at the very end of the chromosome, called the telomere. Thus, with each division, more and more ends of chromosomes are lost until functional genes are changed. This loss could be considered programmed, since evolution led to a mechanism in which DNA replication was incomplete at the ends of chromosomes. Alternatively, one could consider the loss of telomeric DNA as a time-dependent or random event.

In the future, many of the existing theories of aging may be discarded and new theories developed. The usefulness of current and future theories lies in the fact that they provide a basis for the development of experimental approaches. The many challenges of aging and the relative scarcity of data in the field of aging portend a very exciting and challenging future for aging research.

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