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## Functional Mechanisms To Ensure The Reactivity Of The Organism.

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### ABSTRACT

The body's reactivity is an important component of ensuring its homeostasis. During the life of the organism and under different conditions, the reactivity of the organism may vary widely. The most optimal, in terms of reactivity, is considered the average period of a person's life, when program response mechanisms are fully deployed and regulatory systems are active. At this time of life, the body's response to various factors becomes complex, and the reactivity reaches its maximum. From about 40-50 years, the reactivity gradually decreases, there is a tendency to increase its imperfections. All this is true for specific and non-specific reactivity. Specific reactivity - the body's ability to respond to the action of an agent specifically. Nonspecific reactivity is the ability of an organism to respond to the action of various agents by the same type of reaction. The same can be said about physiological and pathological reactivity. The isolation of these forms of reactivity is associated with the biological significance of the body's response to external stimuli. It is believed that physiological reactivity is an adequate, by the nature and intensity, the body's responses to the effects of agents that occur within the limits that do not violate its homeostasis. Such a reactivity of a healthy organism ensures its adaptation to the factors of the external and internal environment and often avoids the disease. Pathological reactivity is an inadequate response and response of the body to the effects of agents, accompanied by impaired homeostasis and reducing its adaptation.

**Keywords:** ontogenesis, reactivity, resistance, reaction, organism.

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## INTRODUCTION

The body's reactivity is an important component of ensuring its homeostasis. During the life of the organism and in different states, the reactivity of the organism can vary widely [1, 2]. The most optimal, in terms of reactivity, is considered the average period of a person's life, when program response mechanisms are fully deployed and regulatory systems are active. At this time, the body's response to various factors becomes diverse and complex, and its reactivity is very high [3, 4]. From about 40-50 years, the effectiveness of the response gradually decreases, there is a tendency to increase imperfection of reactivity. In old age, there is a limitation of the lability of the regulatory systems of the response mechanisms, a narrowing of their ability to quickly and timely restructuring [5]. Elderly people become susceptible to infections, they often develop various inflammatory processes, the number of immune disorders increases, they are more susceptible to oncological diseases [6]. The reason for this - the depletion of the immune system, reducing the functions of the barrier systems of the body. There is also a weakening of the regeneration processes, limited freedom of response (the range of response between its upper and lower limits decreases), there is a lack of regulatory and integrative mechanisms (dysfunction of the central nervous system, endocrine and other systems) [7]. Given the importance of this problem, the goal is set in the work: to carefully consider the main functional aspects of reactivity.

There are several groups of theories of aging that address issues of senile involution from different perspectives. Some of them represent aging as "wear" - this is a state of the organism that forms during its life activity and is accompanied by a gradual development of the degradation of cells and their structures [8]. The basics of wear are assumed to be the fluctuation of long-lived colloids of the body, gradual compaction, degradation, coagulation and decrease in the course of aging of the solubility of plasma proteins of the body without their adequate autolysis, as well as the accumulation of somatic mutations [9].

As the molecular mechanisms of "wear of colloids" during aging and the appearance of its other signs, Hermen's theory is currently the most demonstrative. Aging, in his opinion, depends on the imbalance between oxidative and antioxidant cell systems that occurs with age: in the direction of increasing the activity of the first and weakening the effectiveness of the second [10]. Indeed, aging is characterized by increased generation and the prolonged existence of free oxygen radicals and the weakening of antioxidant cell systems. They are capable of causing fluctuations and a decrease in the dispersion of cytoplasmic protein colloids. The result of oxidation is the accumulation of a significant amount of carbonyl groups in the enzymatic proteins of the body, this leads to a gradual slowdown and inactivation of the metabolism of oxidized proteins and, ultimately, impaired function of cells and organs. Aging pigments - lipofuscin, hemosiderin, are formed with the active participation of auto-oxidative, free-radical reactions [11].

The well-known hypothesis II. Mechnikov on the role of auto-intoxication with intestinal poisons in aging processes (products of bacteria in the large intestines). The prolonged action of these compounds (indole, cresol) also causes, in his opinion, senile degradation of cells and their structures. It turned out that neutralization of indole and cresol occurs with the participation of antioxidant systems of the body [12].

Thus, Hermen's theory plays an integrative role in the study of the mechanisms of aging, considered from the point of view of its wear. Another group of hypotheses represents old age - as a genetically programmed process [13].

Back in the 19th century, the Russian physiologist I.R. Tarkhanov suggested that the cause of natural old age is not the wear of the organism, but the gradual restriction of the ability of cells to create and reproduce. He saw the reason for this in a change in nuclear matter. With the development of genetics, the accumulation of factual material, this provision has received objective evidence. The famous experiments of L. Hayflick and P. Moorhead showed that normal diploid fibroblasts of the early human embryo are capable of doubling in cell culture a limited number of times — about 50. This was called the Hayflick cell division limit [14].

There is information about the localization of "old age" genes in chromosome 1 and 4 — turning off the c-foc gene located there prevents cell proliferation [15]. A gene in yeast (LAG) responsible for the extension of life has been identified, it is similar to some human genes. The involvement of apoptosis mechanisms in the senile process, which depends on the expression or inhibition of specific genes (p 53, bcl-2, etc.), is also

assumed. An indirect confirmation of this is the "immortality" of tumor cells, "leaving" from the mechanisms of apoptosis and not having the limit of cell division of Hayflick. All these data indicate the recent role of genetic control in the mechanisms of aging. In the mechanisms of development of age-related changes there are several options [16]:

- option one - the causes of aging equally and simultaneously affect the various elements of the body, leading them to a uniform violation [17];
- version of the second - allocate one link in the body, which due to its weakness or increased load on it first fails. It further becomes a peculiar driver of age-related changes and causes secondary changes in other, more stable links. Integrating reactivity systems - the central nervous system, the endocrine system and the immune system [18] primarily claim the role of the pacemaker of aging.
- option three - aging occurs as a result of the activity of certain mechanisms for which the generation of age-related changes in other organs and tissues is a normal function. Moreover, it is not necessary that this generation belong to the same entities, it can consistently move from one to another [19].

To date, there is no convincing evidence in favor of any of the three options, i.e. the "main place" of aging has not yet been allocated [20].

In age-related and pathological changes, a large role is played by specific and non-specific reactivity. Specific reactivity is the ability of an organism to respond to the action of an agent specifically and differentially. A classic example is the development of immunity to antigenic effects. In this case, the body reacts to the action of the antigen by producing antibodies or a complex of cellular reactions that are specific to the antigen and [21, 22].

Manifestations of specific reactivity are: allergy, specific immunity, autoimmune diseases, immunodeficiency and immunodepressive states, immunoproliferative diseases [23].

Nonspecific reactivity is the ability of an organism to respond to the action of various agents by the same type of reaction. It is implemented using such mechanisms as stress, the acute phase response, activation of phagocytosis to foreign cells, bacteria, viruses, protozoa, inorganic substances [24]. In response to excessive stimuli (massive injuries, blood loss, pain), the body responds by developing a typical process — shock [25]. In so many diseases, inflammation, fever, and hypoxia can be given as an example of non-specific reactivity. Mechanisms of specific and non-specific reactivity are often observed simultaneously [26]. So with infections, autoimmune diseases, malignant tumors, the mechanisms include both specific (antibody production, activation of cellular immunity), and nonspecific response (leukocytosis, fever, hypoxia).

The severity of the response of the organism is determined by hereditary and acquired properties [27]. The hereditary can be attributed - genetically determined threshold of receptor sensitivity and / or their number on the reacting substrate (cell), the isozyme composition of the individual, which determines the nature of biochemical reactions and features of their development in each person [28, 29].

There is a physiological and pathological reactivity. The isolation of these forms of reactivity is explained by the biological significance (expediency) of the body's response to an agent [30]. Physiological reactivity is an adequate, by nature and intensity, responses of the body to agents, within the limits that do not violate its homeostasis [31]. This is the reactivity of a healthy organism, ensuring its adaptation to the factors of the external and internal environment and often avoiding the disease [32]. For example, the inclusion of mechanisms of contractile and non-contractile thermogenesis with moderate hypothermia, tachycardia and tachypnea during exercise, digestive leukocytosis [33].

Pathological reactivity is inadequate in terms of its severity and nature of the body's response to the effects of agents, accompanied by a violation of its homeostasis and reducing adaptive capacity [34]. In fact, the development of the disease and its manifestations are the pathological reactivity [35]. The manifestation of this reactivity, for example in shock, will be a decrease in resistance to infectious pathogens, inhibition of phagocytosis, and a change in drug sensitivity [36]. One of the response to inflammation is the formation of biologically active substances that cause damage to cells that are not affected by a meeting with an agent that caused inflammation - secondary alteration develops. Allergy, immunodeficiency and immunosuppressive

states are also examples of pathological reactivity [37]. First of all, reactivity depends on the state of the body. Compensatory-adaptive mechanisms of a healthy and sick person are not the same. Reactivity of the patient is characterized by a decrease in optimal vital activity, limiting the breadth of response [38]. In patients with anemia there will be less opportunity to adapt to hypoxia than in healthy people, hence the decline in their vital activity.

### CONCLUSION

The most optimal, in terms of reactivity, is considered the average period of a person's life, when program response mechanisms are fully deployed and regulatory systems are active. At this time, the body's response to various factors becomes diverse and complex, and the reactivity is very high. With aging, the effectiveness of the response gradually decreases, there is a tendency to increase imperfection of reactivity. The specific and nonspecific reactivity suffers. Specific reactivity is the ability of an organism to respond to the action of an agent specifically, differentially. Nonspecific reactivity is the ability of an organism to respond to the action of various agents by the same type of reaction. The severity of the response of the organism is determined by hereditary and acquired properties. There is a physiological and pathological reactivity. The isolation of these forms of reactivity is explained by the biological significance of the body's response to a particular agent. Physiological reactivity - these are adequate, in nature and intensity, the body's responses to agents, within the limits that do not violate its homeostasis. Reactivity of a healthy organism, ensuring its adaptation to the factors of the external and internal environment, and often avoids the disease. Pathological reactivity is an inadequate response to the effects of agents in terms of severity and nature of the organism, accompanied by a violation of its homeostasis and reducing adaptive capacity, aggravating the pathology.

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