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MATHEMATICAL MODEL OF THE IMMUNE SYSTEM OF CONTINUOUS INTERACTION SYSTEM OPERATOR

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Abstract*—The paper presents an integrated mathematical model of the immune system of operator of continuous interaction system, which includes the model of regulation of oxygen modes of the organism, mass transfer and mass exchange of respiratory gases, self-organization of respiratory, blood circulation systems, and immune response. The model can be applied to the study of immunological mechanisms of human organism adaptation to the conditions of professional activity with the purpose of deeper analysis of the state of health, development of methodologies and evaluation of the immune status for the flight and engineering personnel, determination of environmental factors' contribution in the particular immune status of relevant contingent of examined persons, study of influence regularities of a complex of professional factors of activities on human- operator immune system, finding of "cause-effect" connection between ecological-industrial factors and immune system disorders, detection of disorders in the immune system under the influence of a complex of professional factors for the flight crew personnel and development in them future functional abnormalities, development of immunocorrection methods for the treatment and prevention of functional changes and disorders in human- operator.*

Index Terms—Immune system of operator of continuous interaction system; respiratory functional system; model of immune response; organism immune status; dynamic system of respiratory gases mass transfer.

I. INTRODUCTION

Development of new forms and methods for the training of continuous interaction systems' operators is necessary to improve their successful activity; and it can be carried out only on the basis of deep understanding of physiological and psychophysiological processes associated with successful operators' work [1]. It was suggested that human being in conditions of low oxygen partial pressure can cause changes in the immune system [2]. It is known that the sharp immunity decrease occurs often in cases of stress and loading. People at risk are ones whose professions are linked with such factors; flight crews' members and athletes are among them [3]. Chronic psycho-emotional and physical over exertions of organism main systems (including the system of immunity) are inadequate for an organism functional capacity and cause the state of immunodeficiency. This organism state is expressed in organism susceptibility to genetically alien factors, in particular to infections. Analysis of literature data concerning different responses of immune system under the influence of various environmental factors testifies numerical changes in characteristics of immunity. They, from one side, are adaptive in the nature, and from other side they demonstrate a decrease of this system functionality [4]. There are no hesitation that at the stage of adaptation of all organism systems, including the immune system, they are in a state of overstrain. The signs of nonspecific irritation of the immune system could be considered in the aspect of early manifestations of slowly progressing immunological insufficiency.

II. PROBLEM STATEMENT

The foregoing is essential for aviation medicine, where issues related to the early disqualification of flight and engineering personnel are relevant and

unanswered. The solution of this problem from the point of view of adaptation and compensatory responses of immune system, which play significant role in maintaining of the internal organism environment, will allow our understanding of the nature of immunopathological processes. It will give also the possibilities for objective and comprehensive estimation of the state of organism reserves and health as well as new opportunities for pre-nozological diagnosis, correction of immune system in case of its professional deficiency.

If to examine the organism from the standpoint of reliability theory [5], [6], the "weak chains" in organism adaptation to perturbations are the systems of respiration and blood circulation; their functional state determines the nature of processes that occur in the immune system [3]. Therefore, it seems appropriate to analyze the immune system in complex with respiratory and circulatory systems. Also, it should be noted that modern methods of functional diagnostics, however perfect they are, give only a certain slice of its current state and do not answer the question about the nature of these systems future behavior in cases of extreme perturbations, about the course of individual person adaptation to these perturbations. That is why we propose to apply simulation modeling of systems interaction – respiratory, blood circulatory and immune response system. Such modeling will allow identifying of possible compensatory reactions of individual organisms under the extreme loads that happens in professional activity of flight crew members.

The purpose of the work was to develop an integrated mathematical model of the systems – functional respiratory, blood circulatory and immune systems – to study the reserve capabilities of organisms of flight and engineering personnel during adapting to extreme stresses they meet in their professional activity.

To achieve this goal, we use the mathematical models of self-organization of respiratory and blood circulatory systems [7], [8] and the simplest model of immune response [9]. Mathematical model of immune response is a reflection of immunological processes based on theoretical and experimental ideas about the defense system of organism.

III. PROBLEM SOLUTION

Developed mathematical model is described in present sub-chapter. The simplest model of immune response [9] is based on the fundamental principles of immune defense, formulated in the clonal-selecting theory of Barnet and the basic principles of pathophysiology. The model considers the interaction of four components of the system: antigen (virus, bacterium), antibody, plasmatic cell and quantitative characteristics of damaged organ. The model is based on the following provisions of immunology:

- the antibody binds the antigen, forming antibody-antigen complex;
- through time interval τ, plasmatic cells appeared in organism in proportion to the number of antibody-antigen complexes producing antibodies;
- the number of plasmocytes that were formed as a result of antigenic stimulation depends on the viability of damaged organ: with increasing organ damage, the formation of plasmocytes decreases, that consequently affects the activity of immune system.

Experimental data and theoretical studies make it possible to argue that the nature of the dynamics of blood minute volume in healthy organism depends on the intensity of metabolic processes in tissues, gas composition and other characteristics of inhaled mixture, the temperature state of the environment, other external and internal perturbations. It is also evident that the state of immunodeficiency is significantly dependent on the state of blood circulatory system.

Lets suppose that according to the model of immunity [9] $V(t)$ is the concentration of pathogenic antigens that reproduce progressively, $C(t)$ is the concentration of plasma cells of population of carriers and producers of antibodies (immunocomponent cells and immunoglobulin products), $F(t)$ is the concentration of antibodies (substrates of immune system that neutralize antigens (immunoglobulines, cells' receptors)), $m(t)$ is the relative characteristic of the affected organ.

Then the changes in antigens number in organism can be written by the ratio:

$$dV = \beta V dt - \gamma F V dt, \qquad (1)$$

where dV is the increase of antigens' number over the time interval dt due to the reproduction. Obviously, it is proportional to V and some number β, which we will call as reproduction factor of antigens. The member $\gamma FVdt$ describes the number of antigens that will be neutralized by antibodies F over the time interval dt. The number of such viruses will be proportional to the number of antibodies in organism and number of antigens, γ is the coefficient linked with probability of antigen neutralization by antibodies. We have an equation:

$$\frac{dV}{dt} = (\beta - \gamma F)V. \qquad (2)$$

Basing on the hypothesis of the formation of cascade populations of plasmatic cells, let's write a ratio that describes the multiplication of plasmocytes number in comparison with C^* is the constant level of plasmocytes in healthy organism:

$$d\left(C - C^*\right) = dC = Q\left(t - \tau\right)dt, \qquad (3)$$

$$Q\left(t\right) = \alpha FV. \qquad (4)$$

This equation describes the following process: immunocompetent B-lymphocyte is stimulated by the complex of antigen with Ig-receptor (receptor of immunoglobulin) in the presence of signal from a specific T-helper, which is activated by antigen on macrophages; and it initiates the cascading process of formation of cells synthesizing antibodies that neutralize the antigens of defined type. Since in suggested model the antibodies mean substrates capable to bind with antigens so, the number of lymphocytes stimulated in this way will be proportional to FV.

A more complete equation will be as follows:

$$dC = \alpha F(t-\tau)V(t-z)dt - \mu_C(C - C^*)dt, \qquad (5)$$

where $\alpha F(t-\tau)V(t-z)dt$ is the generation of plasmocytes in time interval τ, during which the formation of plasma cells cascade is going; α is the coefficient that takes into account the probability of antigen-antibody contact, excitation of cascade reaction and the number of newly formed cells; $\mu_C(C - C^*)dt$ are describes the decrease of plasmatic cells' numbers due to aging; μ_C is the coefficient, inverse proportional to plasmatic cells' living time.

Then

$$\frac{dC}{dt} = \alpha F(t-\tau)V(t-\tau) - \mu_C(C - C^*). \qquad (6)$$

The balance in the number of bodies that react with antigen is determined by the ratio:

$$dF = \rho Cdt - \eta\gamma FVdt - \mu_f Fdt, \qquad (7)$$

where ρCdt describes the generation of antibodies by plasma cells over a time interval dt; ρ is the velocity of antibodies production by one plasmatic cell; $\eta\gamma FVdt$ describes the decrease in antibodies number in time interval dt due to the binding with antigens; $\mu_f Fdt$ describes a decrease of antibodies population due to aging; μ_f is the coefficient, inverse proportional to plasmatic cells' living time.

Indeed, as it was noted during the derivation of equation (2), the number of antigens eliminated during time interval dt by neutralizing them by antibodies is equal to $\gamma FVdt$, and η-number of antibodies required to neutralize one antigen.

We have an equation

$$\frac{dF}{dt} = \rho C - \left(\eta\gamma V + \mu_f\right)F. \qquad (8)$$

These ratios do not take into account organism weakening in course of disease associated with the decrease of organs' activities that supply the immunological material (leukocytes, lymphocytes, antibodies, etc.); they are necessary to "struggle" with reproduced antigens. It is logical to assume that productivity of such organs is in relation with sizes of target organ lesions.

Let's suppose that M is the characteristics of healthy organ (mass or area), M' is corresponding characteristics of the healthy part of affected organ. Then the relative characteristics of target organ lesion can be written as:

$$m = 1 - \frac{M'}{M}. \qquad (9)$$

Obviously, $m = 0$ for the unaffected organ, and $m = 1$ for the fully affected organ.

We have an equation:

$$\frac{dm}{dt} = \sigma V(1-m) - \mu_m m, \qquad (10)$$

where $\sigma V(1-m)$ characterizes the degree of target organ damage; dt is the time interval over which the relative size of affected organ is increased; σV is proportional to the number of antigens; σ is the some constant (unique for each disease); $(1-m)$ is the factor that characterizes the effect of antigens on the still unaffected part of target organ; μ_m is the coefficient of proportionality, characterizing the velocity of mass (area) changes of affected organ.

The reduction of $(1-m)$ happens due to the restorative activity of organism.

Obviously, with significant damage of vitally important organs, the production of antibodies decreases that cause the fatal case. In order to take into account the factor of damaging of vitally important organs in (6), we replace α by function $\alpha\xi(m)$. The graph $\xi(m)$ looks like [9].

The function $\xi(m)$ in the interval $0 \le m \le m^*$ is equal to one (Fig. 1). This means that performance of immunological organs in this interval is independent of the severity of immunodeficiency

state. But further, at $m^* \le m \le 1$, their productivity sharply decreases, according to the linear part of the curve at this interval. It should be noted that in real conditions the graph of the function $\xi(m)$ may be of more complex form, but qualitatively it consists on the constant $\xi = 1$ at the beginning of the argument change and going down function. Obviously, for different diseases and different immunodeficiency states, the steepness of this part of the curve and value m^* will be different. Function $\xi(m), 0 \le \xi(m) \le 1$ characterizes the degree of damage of immune system normal functioning due to the significant damage of organ – the targets were stated as [9]:

$$\xi(m) = \begin{cases} 1, m \le m^*, m^* = 0.1, \\ \dfrac{1-m}{1-m^*}, m > m^*, m^* = 0.1. \end{cases} \quad (11)$$

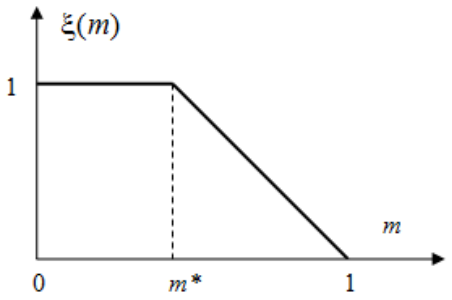

Fig. 1. Graph of the function $\xi(m)$

Thus, the dynamics of immunodeficiency can be represented by the system of nonlinear ordinary differential equations:

$$\begin{aligned} \frac{dV}{dt} &= (\beta - \gamma F)V, \\ \frac{dC}{dt} &= \xi(m)\alpha F(1-\tau)V(t-\tau) - \mu_C\left(C - C^*\right), \\ \frac{dF}{dt} &= \rho C - \left(\mu_f + \eta\gamma V\right)F, \\ \frac{dm}{dt} &= \sigma V(1-m) - \mu_m m. \end{aligned} \quad (12)$$

Let's note that in the above described model there is a combined population of immunocompetent and antibody-forming cells $C(t)$. In the absence of viruses in organism $C(t) = C^* > 0$, or C^* is a normal level of immunocompetent cells in healthy organism.

If such cells are absent, or $C^* = 0$, that means that organism is tolerant (unfavorable) against the given antigen. However, it may happen so, that organism does not have information about this antigen, and respectively, does not have immunocompetent cells. It is possible that in such cases, immunocompetent cells with similar specific receptors capable to cause an immune response against this antigen will enter into reaction. In the following work we will assume that organism has a non-zero level of cells C^* with their receptors F^*, which can cause an immune response.

As it was noted above, infectious disease is an internal disorder that leads to a pathological state, the course of which is controlled by the immune system.

It is obvious that the disease course is significantly influenced by the parameters used in model (12). It is natural to assume that values σ and μ_m are the functions of the values of volumetric velocities of regional blood circulations through the capillaries of the target organ. However, basing on the main function of respiratory system [10], [11], the dynamics of systemic and regional circulation is directly related with the change in the gas portrait of organism and intensity of metabolic processes in individual tissue regions. Therefore, to study the state of immunodeficiency and the impact of the reaction of blood circulatory system in this process, let's use a mathematical model of respiratory gases mass transfer in organism in the dynamics of respiratory cycle, which describes the gases mass exchange as a mechanism controlled by organism self-regulation [12]. The action of self-regulation mechanisms is aimed on the maintaining of equilibrium states in organism, stable oxygen and carbon dioxide organism modes in perturbations of internal and external environment. Obviously, immunodeficiency can be considered as internal perturbation.

In the case of infectious disease, the criterion of self-organization quality is estimated by the minimum of functional [12]

$$\begin{aligned} \int_{t^*}^{t^*+\Delta t} \Bigg[& \rho_1 \sum_{i=1}^{n} \lambda_{t_i} \left(G_{t_i} O_2(\tau) - q_{t_i} O_2(\tau) \right)^2 \\ & + \rho_2 \sum_{i=1}^{n} \lambda_{t_i} \left(G_{t_i} CO_2(\tau) + q_{t_i} CO_2(\tau) \right)^2 \\ & + \rho_3 \sum_{i=1}^{n} \lambda_{t_i} \left(G_{t_i} N_2(\tau) \right)^2 + \rho_4 f_k^2 \left(m(\tau), V(\tau) \right) \Bigg] d\tau, \end{aligned} \quad (13)$$

where $G_{t_i} O_2(\tau), G_{t_i} CO_2(\tau), G_{t_i} N_2(\tau)$ are flows of oxygen, carbon dioxide and nitrogen through the capillary-tissue membranes of ith tissue in moment of time τ; $q_{t_i} O_2(\tau)$, $q_{t_i} CO_2(\tau)$ are velocities of oxygen utilization and carbon dioxide removal from ith tissue; $f_k\left(m(\tau), V(\tau)\right)$ is the function that

characterizes the degree of damage by viruses of target organ of *k*th tissue reservoir; ρ_4 is the coefficient characterizing the degree of influence of modeled type of disease being on the level of gas homeostasis; t^* is the moment of time from which the criterion of quality of control is evaluated; Δt is the time during which were estimated the dynamics of gases and parameters of immune system to determine the quality of control.

The function $f_k(m,V)$ used in (13) determines the degree of damage of the target organ at given moment of time and, in a simplified version, can be written as $f_k(m,V) = a_k m + b_k V$, where a_k, b_k are some coefficients. According to our assumption $a_k = b_k = 1$. The function $f_k(m,V)$ haven't to be negative, and $f_k(m,V) = 0$ only if the organism is free of viruses ($V = 0$) and tissue of the target organ is not damaged ($m = 0$).

Obviously, a mutual influence of respiratory system and immune system exists. We can assume that energy processes' courses in the tissues of target organ are provided by its unaffected part only. In this case, tissue mass of metabolizing target organ will be determined by the expression:

$$V_{t_k}(\tau) = V_{t_k}^0\left(1 - m(\tau)\right), \qquad (14)$$

where $V_{t_k}^0$ is the total mass of tissues of healthy target organ.

The value V_{t_k} appears in the equations describing the dynamics of oxygen, carbon, and nitrogen stresses in *k*th tissue reservoir and influences on the level of gas homeostasis of tissue region under constant characteristics of other conditions. Velocity of oxygen utilization $q_{t_{ki}}O_2$ in *k*th tissue reservoir plays more significant role in interaction determining [10], [11]. The change of value $q_{t_{ki}}O_2$ leads to the almost instantaneous change in blood circulatory through the capillaries of target organ tissues, which consequently leads to the adjustments of immune system parameters. During the simulation of target-organ damage, it is necessary to take into account the degree in which the healthy target organ cells are able to reflect the metabolic functions of affected cells, and what is the energy value of these actions. Taking into account the mutual influence of the immune system, respiratory and blood circulatory ones in process of mathematical modeling allows us to predict the course of immunodeficiency state. Possible reactions of respiratory and blood circulatory systems in the form of these systems state changes and its necessary regulatory correction have to be taken into account.

It is necessary to mention that in the case of infectious disease, it is advisable to integrate the mathematical model (13) with the mathematical model of heat transfer and heat exchange [14].

IV. CONCLUSIONS

Today proposed mathematical model has only theoretical importance. However, in the case of model individualization [10], [11] and existence of appropriate data array it is hoped that the proposed approach will allow to investigate the immunological mechanisms of adaptation of human organism to the conditions of professional activity. This is necessary for such purpose as deeper health state analysis for the development of methodology for the estimation of immune status of flying and engineering staff representatives. Also, it is valuable for:

- determining of the contribution of environmental factors in the peculiar immune state of corresponding contingent of persons surveyed;
- studying of influence regularities of complex of professional factors on the activity of immune system of human-operator;
- finding of the relationship "cause-effect" between the environmental-industrial factors and immune system disorders;
- detecting of disorders in the immune system under the influence of complex of professional factors in flight crew members and to predict on the base of this the development of functional deviations in the future;
- development of methods of immunocorrection in the treatment and prevention of functional changes and disorders of immunity in human operators.

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Н. І. Аралова, О. М. Ключко, В. Й. Машкін, І. В. Машкіна. Математична модель імунної системи оператора системи неперервної взаємодії

В роботі представлено інтегровану математичну модель імунної системи оператора системи неперервної взаємодії, до складу якої входить модель регулювання кисневими режимами організму, масопереносу та масообміну респіраторних газів, самоорганізації системи дихання та кровообігу та імунного відгуку. Модель може бути застосована для дослідження імунологічних механізмів адаптації організму людини до умов професійної діяльності з метою більш глибокого аналізу стану здоров'я, розробки методології оцінювання імунного статусу у осіб льотного та інженерно-технічного складу, визначення внеску факторів середовища в особливості імунного статусу відповідного контингенту обстежуваних осіб, дослідження закономірності впливу комплексу професійних факторів діяльності на імунну систему людини-оператора, встановлення причинно-наслідкового зв'язку між екологовиробничими факторами та порушеннями імунної системи, виявлення порушень в імунній системі під впливом комплексу професійних факторів у осіб льотного складу та розвитком у них в майбутньому функціональних відхилень, розробки методів імунокорекції при лікуванні та профілактиці функціональних змін та порушень імунітету у людини-оператора.

Ключові слова: імунна система оператора системи неперервної взаємодії; функціональна система дихання; модель імунного відклику; імунний статус організму; керована динамічна система масопереносу респіраторних газів.

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Напрям наукової діяльності: біофізика, екологія, біоінформатика, науки про мозок.
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Н. И. Аралова, Е. М. Ключко, В. И. Машкин, И. В. Машкина. Математическая модель иммунной системы оператора системы непрерывного взаимодействия

В работе представлена интегрированная математическая модель иммунной системы оператора системы непрерывного взаимодействия, в состав которой входит модель регулирования кислородными режимами организма, массопереноса и массообмена респираторных газов, самоорганизации системы дыхания и кровообращения и иммунной отзыва. Модель может быть использована для исследования иммунологических механизмов адаптации организма человека к условиям

профессиональной деятельности с целью более глубокого анализа состояния здоровья, разработки методологии оценки иммунного статуса у лиц летного и инженерно-технического состава, определения вклада факторов среды в особенности иммунного статуса соответствующего контингента обследуемых лиц, исследования закономерности влияния комплекса профессиональных факторов деятельности на иммунную систему человека-оператора, установления причинно-следственной связи между эколого-производственными факторами и нарушениями иммунной системы, выявления нарушений в иммунной системе под влиянием комплекса профессиональных факторов у лиц летного состава и развитием у них в будущем функциональных отклонений, разработки методов иммунокоррекции при лечении и профилактике функциональных изменений и нарушений иммунитета у человека-оператора.
Ключевые слова: иммунная система оператора систем беспрерывного взаимодействия; иммунная система; модель иммунного отклика; иммунный статус организма; управляемая динамическая система массопереноса респираторных газов.

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