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REPUBLIC OF AZERBAIJAN

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AKADEMİK ABDULLA QARAYEV adına FİZİOLOGİYA İNSTİTUTU

Azərbaycan Milli Elmlər Akademiyası
MƏRKƏZİ ELMİ KİTABXANA

# 8th INTERNATIONAL CONGRESS ON MEDICAL & HEALTH SCIENCES

*"Dedicated to the 5th Anniversary of 8 November – Victory Day"*

**August 27-29, 2025 / Baku, Azerbaijan**

Institute of Physiology named after Academician Abdulla Garayev

# PROCEEDINGS BOOK

EDITOR
Prof. Dr. Ulduz Həşimova

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# The local and global dynamics of nonlocal cancer tumor growth model

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We present here, a phase-space analysis of a mathematical model of tumor growth with an immune response. Consider mathematical analysis of the system of nonlocal equations with multipoint conditions regarding to dissipativity, boundedness of solutions, invariance of non-negativity, local and global stability. We derive some features of behavior of the three-dimensional tumor growth models with nonlocal dynamics described in terms of densities of three cells populations: tumor cells, healthy host cells and effector immune cells. We found sufficient conditions, under which trajectories from the positive domain of feasible multipoint conditions tend to one of equilibrium points. Here, cases of the small tumor mass equilibria-the healthy equilibrium point, the death equilibria have been examined. Biological implications of our results are discussed.

Beginning with this talk we intend to investigate the problems of mathematical and biological approaches to model the cancer growth dynamics processes and operations. It is important to take into account the nonlinear property of cancer growth processes in construction of mathematical logistic models. The nonlinearity approach appears very convenient to display unexpected dynamics in cancer growth processes expressed in different reactions of the dynamics to different concentrations of immune cells at different stages of cancer growth developments $[1-41]$. Taking into account all the complex processes, nonlinear mathematical models can be estimated capable of compensation and minimization the inconsistencies between different mathematical models related to cancer growth-anticancer factor affections. The elaboration of mathematical non-spatial models of the cancer tumor growth in the broad framework of tumor immune interactions studies is one of intensively developing areas in the modern mathematical biology, see works $[1-9]$. Of course, the development of powerful cancer immunotherapies requires an understanding of the mechanisms governing the dynamics of tumor growth. One of main reasons for creation of non-spatial dynamical models of this nature is related to the fact that they are described by a system of ordinary differential equations, which can be efficiently investigated by powerful methods of qualitative theory of dynamical systems. Mathematical models for tumour growth have been extensively studied in the literature to understand the mechanism of the disease and predict its future behavior. Interactions of tumour cells with other cells of the body, i.e. healthy host cells and immune system cells are the main components of these models and these interactions may yield different outcomes. Some important phenomena of the tumour progression such as tumour dormancy, creeping through, and escaping from immune surveillance have been investigated by using these models. Kuznetsov et al. $[1]$ proposed a model of second order ordinary differential equations (ODEs), which includes the effector immune cell and the tumour cell populations. They demonstrated that even with two cell populations, these models can provide rich dynamics depending on the system parameters and explained some important aspects of the stages of cancer

progression. Three equation mathematical models of tumor growth with an immune responses were studied e.g. in [4, 5, 7, 9, 10]. For instance, Kirschner and Panetta [4] examined the tumour cell growth in the presence of the effector immune cells and the cytokine IL-2 which has an essential role in the activation and stimulation of the immune system. de Pillis and Radunskaya [5] included a normal tissue cell population in this model, performed phase space analysis and investigated the effect of chemotherapy treatment by using optimal control theory. In [9], interactions between cancer cells, effector cells, and cytokines (such as IL-2, TGF- $\beta$, IFN- $\gamma$) studied. In [7] interactions between cancer cells, effector cells, and normal tissue cells are investigated. In [6], a four-dimensional model is discussed which can undergo Hopf bifurcations leading to periodic orbits, a possible route to the development of chaotic attractors (for general review see e.g. [1, 3]). In [10] global behavior of the tumour growth population dynamics was investigated. The local stability, the chaotic behavior properties and some features of global behavior tumour growth model of (1.1) were studied in [12] and [11], respectively. The complex oscillations were studied in [16]. Moreover, the model has been also used to define optimal control problems (see e.g. [16 – 18]). Note that nonlinear dynamic systems studied e.g. in [22 – 24]. In contrast to mentioned works, here mathematical analysis of multipoint IVP for (1.1), local and global stability and the multiphase basins of attractions have been investigated. We prove that all orbits are bounded and must converge to one of several possible equilibria. Therefore, the long-term behavior of an orbit is classified according to the basin of attraction in which it starts. Morever, investigation of spatiotemporal pattern formation leads to understanding of the interesting and complex dynamics of prey-predator populations.

In conventional models of population dynamics, consumption of resources by the individuals occurs at the same spatial location as reproduction and death. We assume in this work that the individual located at a point in the spatial domain can consume resources not only at that point but also at some neighboring region surrounding that point. Movement of the individuals to the nearby location occurs in a faster time scale compared to the movement from one location to the other one. This modifies the modeling approach and gives rise to an nonlocal differential equation with convolution terms describing the nonlocal consumption of resources. Such type reaction-diffiusion equations with the nonlocal term is also used to explain the emergence and evolution of biological species and speciation were studied i.e. in [27 – 41]. Reaction-diffusion equation with the nonlocal term is also used to explain the emergence and evolution of biological species and speciation [30-34].

Here, we examine the nonlocal consumption of resources and random motion dynamics to interaction of cancer, immune and normal cells, i.e. we consider the following multi point problem for dynamical system

$$\frac{\partial T}{\partial t} = r_1 T[b_1 - k_1^{-1} J_1(T)] - a_{12} NT - a_{13} IT,$$

$$\frac{\partial N}{\partial t} = r_2 N[b_2 - J_2(N)] - a_{21} NT,$$

$$\frac{\partial I}{\partial t} = r_3 I[b_3 - J_3(I)] + \frac{a_{31} IT}{k_3 + T} - d_3 I,$$

$$T(t_0) = T_0 + \sum_{k=1}^{m} \alpha_{1k} T(t_k), N(t_0) = N_0 + \sum_{k=1}^{m} \alpha_{2k} N(t_k),$$

$$I(t_0) = I_0 + \sum_{k=1}^{m} \alpha_{3k} I(t_k), t_0 \in [0, \eta), t_k \in O_\delta(t_0),$$

where $T = T(t)$ , $N = N(t)$ , $I = I(t)$ denote the densities of tumor cells, healthy host cells and the effector immune cells, respectively at the moment $t$ , $\alpha_{jk}$ are real numbers, $m$ is a natural number and

$$O_\delta(t_0) = \{t \in \mathbb{R} : |t - t_0| < \delta\} \text{ for a } \delta > 0.$$

The assumption $(1.0)$ is given on coefficients $\alpha_{ij}$ and $t_0, t_1, t_2, \ldots t_m$ , where

$$(T_0, N_0, I_0)$$

indicate the given pre-healing vector (or pre-healing vector state) such that $T_0$ is small enough but $N_0$, $I_0$ are big enough. The condition $(1.2)$ links the values of vector function $V(t) = (T(t), N(t), I(t))$ at various points $t_0, t_1, \ldots, t_m$ to each other by healing vector $(T_0, N_0, I_0)$; so, we called $(1.2)$ a multipoint IVP. Moreover,

$$J_i(u) = J_i(u)(x) = \int_\alpha^\beta \Phi_i(x - \tau) u(\tau) d\tau, i = 1, 2, 3,$$

here $\Phi_i$ are given continuous functions on $\mathbb{R}$ .

The first term of the first equation corresponds to the logistic growth of tumor cells in the absence of any effect from other cells populations with the growth rate of $r_1$ and maximum carrying capacity $k_1$ . The competition between host cells and tumor cells $T(t)$ which results in the loss of the tumor cells population is given by the term $a_{12} NT$

. Next, the parameter $a_{13}$ refers to the tumor cell killing rate by the immune cells $I(t)$ . In the second equation, the healthy tissue cells also grow logistically with the growth rate of $r_2$ and maximum carrying capacity $b_2$ . We assume that the cancer cells proliferate faster than the healthy cells which gives $r_1 > r_2$ . The tumor cells also inactivate the healthy cells at the rate of $a_{21}$ . The third equation of the model describes the change in the immune cells population with time $t$. The first term of the third equation illustrates the stimulation of the immune system by the tumor cells with tumor specific antigens. The rate of recognition of the tumor cells by the immune system depends on the antigenicity of the tumor cells. The model of the recognition process is given by the rational function which depends on the number of tumor cells with growth rate $r_3$ and environmental carrying capacity $b_3$ . The immune cells are inactivated by the tumor cells at the rate of $a_{31}$ as well as they die naturally at the rate $d_3$. We suppose that the constant influx $s$ of the activated effector cells into the tumor microenvironment is zero. One of main aims is derivation of sufficient conditions under which the possible biologically feasible dynamics is local and globally stable, and a converges to one of equilibria. Since these equilibrium points have a biological sense, we notice that understanding limit properties of dynamics of cells populations based on solving problem $(1.1)$ may be of an essential interest for the prediction of health conditions of a patient without a treatment, when the data (e.g. the status of blood cells shown above) that determines the conditions of the patient.

By scaling $y_1 = Tb_1^{-1}$ , $y_2 = Nb_2^{-1}$ , $y_3 = Ib_3^{-1}$ , $\tilde{t} = r_1 t$ in $(1.1)$ and omitting the tilde notation we get the multipoint IVP for the following dynamical system

$$\frac{\partial y_1}{\partial t} = y_1[b_1 - J_1(y_1)] - a_{12}y_1y_2 - a_{13}y_1y_3,$$

$$\frac{\partial y_2}{\partial t} = r_2y_2[b_2 - J_2(y_2)] - a_{21}y_1y_2,$$

$$\frac{\partial y_3}{\partial t} = r_3y_3[b_3 - J_3(y_3)] - a_{31}y_1y_3 - d_3y_3,$$

$$y_1(x,t_0) = y_{10} + \sum_{k=1}^{m} \alpha_{1k}y_1(t_k),$$

$$y_2(x,t_0) = y_{20} + \sum_{k=1}^{m} \alpha_{1k} y_1(t_k),$$

$$y_3(x,t_0) = y_{30} + \sum_{k=1}^{m} \alpha_{1k} y_1(t_k),\ t \in [0,T),,\ t \in [0,T),$$

where $y_k = y_k(x,t)$ and $y_{k0} = y_{k0}(x)$.
We are interested in biologically relevant solutions of the system $(1.3)-(1.4)$ .

## 2. Boundedness and dissipativity

In this section, we shall show that the model are bounded with negative divergence, positively invariant with respect to a region in $\mathbb{R}^3_+$ and dissipative, where

$\mathbb{R}^3_+ = \{x = (x_1,x_2,x_3) \in \mathbb{R}^3 : x_j > 0\}, j = 1,\ 2,\ 3.$

As we are interested in biologically relevant solutions of the system, the next results show that the positive octant is invariant and that the upper limits of trajectories depend on the parameters of multipoint initial conditions.
Let

$$B(l) = \{y = (y_1,y_2,y_3) \in \mathbb{R}^3_+ : 0 \le y_k \le l_k\},$$

where

$$l_1 = \max\{y_{10}, b_1\}, l_k = \max\{y_{k0}, \mu_k\}, k = 2.3,$$

$$\mu_k = \left[ (\beta - \alpha) \max_{x \in [\alpha,\beta]} |\Phi_k(x)| \right]^{-1}.$$

Consider the problem $(1.2)$ with $t_0 = 0$.
**Theorem 3.1.** Assume

$$b_1 \le d_3, \frac{\partial}{\partial y_1} J_1(y_1) \le 2,$$

$$b_k \le J_k(y_k) + y_k \frac{\partial}{\partial y_k} J_k(y_k) \text{ for all } t \in [0,T], k = 2,3.$$

Then:(1) $B(l)$ is positively invariant with respect to $(1.3)-(1.4)$; (2) all solutions of $(1.3)$ are uniformly bounded and are attracted into the region $B(l)$ ; (3) the system $(1.3)$ is with the negative divergence; (4) the system $(1.3)$ is dissipative.

### 3. The Lyapunov stability of equilibria points

In this section, we will derive the stability properties of equilibria points of the system $(1.3)$. The equilibria points of the system $(1.3)$ are obtained by solving the system of isocline equations

$$y_1\{[b_1 - J_1(y_1)] - a_{12}y_2 - a_{13}y_3\} = 0,$$

$$y_2\{r_2[b_2 - J_2(y_2)] - a_{21}y_1\} = 0,$$

$$y_3\{r_3[b_3 - J_3(y_3)] - a_{31}y_1 - d_3\} = 0,$$

where $J_i(y_i)$ is convolution operators defined by $(1.2)$ and $\Phi_i$ are given continuous functions on $\mathbb{R}$.

**Theorem 3.1.** The points $P_1(0,0,0)$, $P_2(0,c_2,0)$, $P_3(0,0,c_3)$ $P_4(d_1,d_2,0)$ are equilibria points for the system $(1.3)$, where $c_2, c_3$, $d_1$ and $d_2$ are solutions of the following equations

$$J_2(y_2) = b_2,$$

$$J_3(y_3) = b_3 - \frac{d_3}{r_3},$$

$$[b_1 - J_1(y_1)] - a_{12}y_2 = 0,$$

$$r_2[b_2 - J_2(y_2)] - a_{21}y_1 = 0.$$

Let

$$\mathbb{R}^3_+ = \{x \in \mathbb{R}^3 : x_i \geq 0, i = 1,2,3\}, B_r(\bar{x}) = \{x \in \mathbb{R}^3, \|x - \bar{x}\|_{\mathbb{R}^3} < r\}.$$

In this section we show the following results:

**Theorem 3.2.** Assume (1) $r_2 - a_{21} < 0$, $\frac{r_3}{k_3} - a_{31} < 0$; (2) $c_{13} = \frac{r_3}{1+k_3} - a_{31} - d_3 < 0$, $a_{12} > a_{13}$. Then the system $(1.3)$ is asymptotically stable at the equilibria point $E_1(1,0,0)$ in the Lyapunov sense. If $a_{21} > r_2$ or $c_{13} > 0$, then the system $(1.3)$ is unstable at $E_1(1,0,0)$.

Now, we consider the equilibria point $E_2(0,1,0)$ and prove the following result:

**Theorem 4.2.** Assume: (1) $c_{11} = 1 - a_{12} < 0$, (2) $a_{21}^2 > r_2(r_2 - c_{11})$ . Then the system $(1.3)$ is asymptotically stable at the equilibria point $E_1(0,1,0)$ in the sense of Lyapunov .

Let $a_{\pm}$, $b_{\mp}$ and $D$ are the numbers defined by $(4.0)$. Now, we prove the following result:

**Theorem 4.3.** Assume

$$a_{21}a_{\pm} > r_2,\ a_{\pm} + a_{31}a_{\pm} + d_3 > \frac{r_3 a_{\pm}}{a_{\pm} + k_3},\ (2 + k_3)a_{31} + d_3 > r_3 + \sqrt{D}.$$

Moreover, suppose

$$a_{13} > a_{12},\ a_{31} > \frac{k_1 r_3}{(a_{\pm} + k_1)^2},\ d_{31}d_{12} > d\rho.$$

Then system $(1.3)$ is asymptotically stabile at equilibria points $E_3(a_{\pm},0,b_{\mp})$ in the sense of Lyapunov .

**Condition 4.4.** Let $a_{12} \geq 1$, $a_{12} \geq r_2$ , $a_{12}a_{21} > r_2$, $d_{33} < 0$ and

$$Re\left\{(d_{11} + d_{22}) + \sqrt{(d_{11} + d_{22})^2 - 4(d_{12}a_{21}\bar{x}_2 + d_{11}d_{22})}\right\} < 0.$$

**Theorem 4.4.** Assume the Condton 4.4 is satisfied. Then system $(1.3)$ is asymptotically stable at the equilibria point $E_4(\bar{x}_1,\bar{x}_2,0)$ in the Lyapunov sense .

**Theorem 4.5.** Assume the Condition 4.5 is satisfied. Moreover, suppose

$$b_{22} + b_{33} > 0,\ -(b_{11} + b_{33})(b_{22} + b_{33}) < b_{12}b_{21},$$

$$4p_{12}^2 \leq p_{11}p_{22},\ 4p_{13}^2 \leq p_{11}p_{33},\ 4p_{23}^2 \leq p_{22}p_{33}\ .$$

Then the system $(1.3)$ is asymptotically stable at equilibria points $E_{ij}$ in the Lyapunov sense.

**Conclusion.** Taking into account different and effective features of mathematical modelling and its possibilities to figure out a problem in dynamics on the basis of its logic properties, it was surely pointed out the characteristics of a mathematical model to use in description of needed processes of a given dynamic system with identified problems. In this paper, a three dimensional model was devoted to mathematical description and regulation possibilities of uncontrolled tumor processes by organism as a complex system. The dynamics of interactions of the dimensions corresponded to tumor

cells, immune cells and healthy -- host -- cells were given as forces of vectors, negatively or positively converging to basins of attractions, depending on their importance for the complex system. In order to make the model subjected to control, there was included IVP, describing the system's important parameters to operate with it in the farther processes of stages of development. The model was undergone different changes to determine its limits of survival: it was determined the conditions of boundedness the system can be restricted, invariance in non- negativity, which means the model keeps its properties of reactions to changing in proper way, being subjected to different analysis, and the circumstances the system can be forced to be dissipated in. The system was exposed to changing pressures to estimate its convenience to biologically important properties as points of equilibria and Lyapunov stability conditions. The next step in exploring of the model were very complex and logistic approaches to its properties for verification of the conditions, providing the global equilibria points and multimodal attraction sets, having biologically strong value in regulation of the processes towards the positive effects of feasible medical external implementation at the convenient stages, determined by multimodal attraction basins.

**Biological implications.** Here we study a multiphase host-tumor model that enhances the type of effector immune cells that can fight a tumor, and stimulates effector immune cells to proliferate. Interactions between cancer tumor cells, healthy host cells and the effector immune cells can explain long-term tumor relapse. Here, the sufficient conditions is derived that under which the possible biologically feasible dynamics is stable in the Lyapunov sense, and a converges to one of equilibrium points. Since these equilibrium points have a biological sense, we notice that understanding limit properties of dynamics of cells populations based on solving the problem $(1.3)-(1.4)$ may be of an essential interest for the prediction of health conditions of a patient without a treatment, when the data (e.g. the status of blood cells shown above) that determines the condition of the patient are compared at various times $t_0, t_1, \ldots, t_m$ and correlated. In the section 4, lyapunov stability of the system $(1.3)$ at the corresponding equilibria points are studied. We show that the system $(1.3)$ is global stable at the "free tumor " equilibria point $E_2(0,1,0)$ . In the section 5, the basins of multiphase attractors of the system $(1.3)-(1.4)$ are constructed dependent on multipoint parameters of IVP.

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