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Original article

# Localization of Solutions to Equations of Tumor Dynamics

*Vardan B. Pogosyan[1], Margarita A. Tokareva[2], Aleksandr A. Papin[3]*

[1]Altai State University, Barnaul, Russia, vardan.pogosyn@yandex.ru
[2]Lavrentyev Institute of Hydrodynamics SB RAS, Novosibirsk, Russia, tma25@mail.ru
[3]Altai State University, Barnaul, Russia, papin@math.asu.ru

Научная статья

# Локализация решений уравнений динамики опухоли

*Вардан Баландурович Погосян[1], Маргарита Андреевна Токарева[2], Александр Алексеевич Папин[3]*

[1]Алтайский государственный университет, Барнаул, Россия, vardan.pogosyn@yandex.ru
[2]Институт гидродинамики им. М.А. Лаврентьева СО РАН, Новосибирск, Россия, tma25@mail.ru
[3]Алтайский государственный университет, Барнаул, Россия, papin@math.asu.ru

This article discusses a mathematical model of tumor dynamics. The tissue is considered as a multiphase three-component medium consisting of extracellular matrix, tumor cells, and extracellular fluid. The extracellular matrix is generally deformable. In the case of the predominant extracellular fluid — tumor cell interaction, the original system of equations is reduced to the one parabolic equation degenerating on the solution with a special right-hand side. The property of a finite perturbation propagation velocity for tumor cell saturation is revealed. The introduction describes the essence of the problem. The second part presents the derivation of a mathematical model of tumor dynamics as a three-phase medium. The third part describes a mathematical model for the case when mechanical interaction with extracellular fluid is neglected. The fourth part considers the case of predominant fluid-cell interaction. The fifth part provides a proof of the theorem on the localization of the solution to the equation for the saturation of tumor cell.

***Keywords:*** differential equations, filtration, tumor, localization, porosity

В данной статье рассматривается математическая модель динамики опухоли. Ткань рассматривается как многофазная среда, состоящая из трех компонентов: внеклеточного матрикса, опухолевых клеток, внеклеточной жидкости. Внеклеточный матрикс, как правило, деформируется. В случае преобладания взаимодействия внеклеточная жидкость — опухолевая клетка исходная система уравнений сводится к одному параболическому вырождающемуся на решении уравнению с правой частью специального вида. Установлено свойство конечной скорости распространения возмущений для насыщенности опухолевых клеток. Во введении описана проблематика задачи. В первом пункте приведен вывод математической модели динамики опухоли как трехфазной среды. Во втором пункте описана математическая модель в случае пренебрежения механическим взаимодействием с внеклеточной жидкостью. В третьей части рассмотрен случай преобладания взаимодействия жидкость — клетка. В четвертой части приведено доказательство теоремы о локализации решения уравнения для насыщенности клеток опухоли.

***Ключевые слова:*** дифференциальные уравнения, фильтрация, опухоль, локализация, пористость

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## Introduction

At the moment, there is no single approach to describing tumor growth, both because tumors have different origins and characteristics, and because there are several parallel causes of tumor development. We can say that the cells that form the tumor, like other cells of the body, live in an aqueous environment saturated with proteins. These include all kinds of nutrients that cells need to survive and reproduce, and chemical factors, in particular growth promoters, growth inhibitors, and chemotactic factors, which set off intracellular chemical cascades of reactions that determine cell behavior. The extracellular space is also filled with a network of cross-linked proteins (elastin, collagen, proteoglycans), collectively known as the extracellular matrix (ECM), which forms the tissue structure. Both in a physiological situation and in a pathological one, the interactions of a cell with its neighbors and with the extracellular matrix are very complex.

## 1. Basic three-phase model. Formulation of the problem

A mathematical model of tumor dynamics is considered [1] – [5]. A tumor is composed of at least three major components occupying an appropriate percentage of space: tumor cells, extracellular matrix (ECM), and extracellular fluid. In addition, the components and chemical factors that diffuse into the fluid and are taken up or produced by the cells should be taken into account. However, they will not be considered in the work. When describing the process, mass balance equations are used for each of the phases

$$\frac{\partial(1-\phi)}{\partial t} + \nabla\cdot((1-\phi)\upsilon_0) = \Gamma_0, \quad (1)$$

$$\frac{\partial\phi s}{\partial t} + \nabla\cdot(\phi s\upsilon_T) = \Gamma_T, \quad (2)$$

$$\frac{\partial\phi(1-s)}{\partial t} + \nabla\cdot(\phi(1-s)\upsilon_l) = \Gamma_l, \quad (3)$$

where $(1-\phi)$ is the volume ratios occupied by ECM, $\phi$ is the porosity, $s$ is the saturation of the cellular phase, $(1-s)$ is the saturation of the extracellular fluid, $\upsilon_0, \upsilon_T, \upsilon_l$ are true velocities of the extracellular matrix, cells and extracellular fluid, respectively, $\Gamma_0, \Gamma_T, \Gamma_l$ are intensity of mass transfer from one component to others ($\Gamma_0+\Gamma_T+\Gamma_l = 0$). It is assumed that the phase densities are the same: $\rho_0 = \rho_T = \rho_l = \rho$. The momentum balance equations for each component are written as follows

$$\rho(1-\phi)(\frac{\partial\upsilon_0}{\partial t} + \upsilon_0\cdot\nabla\upsilon_0) = \nabla\cdot T_0 + b_0 + m_0^\sigma, \quad (4)$$

$$\rho\phi s(\frac{\partial\upsilon_T}{\partial t} + \upsilon_T\cdot\nabla\upsilon_T) = \nabla\cdot T_T + b_T + m_T^\sigma, \quad (5)$$

$$\rho\phi(1-s)(\frac{\partial\upsilon_l}{\partial t} + \upsilon_l\cdot\nabla\upsilon_l) = \nabla\cdot T_l + b_l + m_l^\sigma, \quad (6)$$

where $T_i$ is the stress tensor, $b_i$ are external forces, and $m_i^\sigma$ is the interaction force acting on the $i$ component due to its interaction with other components. It is assumed that the main contribution to the interaction forces is proportional to the difference in velocities between the components. In accordance with thermodynamics, the saturation assumption implies the existence of the Lagrange multiplier $P$, which is then identified with the pressure of the extracellular fluid in the constitutive equations, so that one can write

$$\begin{aligned}
m_0^\sigma &= P\nabla(1-\phi) - M_{T0}(\upsilon_0-\upsilon_T) - M_{l0}(\upsilon_0-\upsilon_l)\\
&\quad - \frac{\Gamma_0}{2}\upsilon_0 + \frac{\Gamma_0-\Gamma_T}{6}\upsilon_T + \frac{\Gamma_0-\Gamma_l}{6}\upsilon_l,\\
m_T^\sigma &= P\nabla\phi s - M_{lT}(\upsilon_T-\upsilon_l) - M_{T0}(\upsilon_T-\upsilon_0)\\
&\quad - \frac{\Gamma_T}{2}\upsilon_T + \frac{\Gamma_T-\Gamma_l}{6}\upsilon_l + \frac{\Gamma_T-\Gamma_0}{6}\upsilon_0,\\
m_l^\sigma &= P\nabla\phi(1-s) - M_{lT}(\upsilon_l-\upsilon_T) - M_{l0}(\upsilon_l-\upsilon_0)\\
&\quad - \frac{\Gamma_l}{2}\upsilon_l + \frac{\Gamma_l-\Gamma_T}{6}\upsilon_T + \frac{\Gamma_l-\Gamma_0}{6}\upsilon_0,
\end{aligned} \quad (7)$$

where $M_{ij}$ is the interaction between the $i$-th and $j$-th components and $T_l = -(P\phi(1-s))I + \hat{T}_l$, $T_T = -(P\phi s)I + \hat{T}_T, T_0 = -(P(1-\phi))I + \hat{T}_0$, where $\hat{T}_i$ is named excess stresses. It is assumed that the main contribution to the interaction forces is made by terms proportional to the difference in velocities between the components. The terms in (7) that are proportional to the mass production rates of $\Gamma_i$ are, however, negligible [6]. When describing biological phenomena, inertia can be neglected, and the force of interaction between the extracellular matrix and fluid is negligible in relation to the force of interaction between the cell and the fluid, and, above all, the

cell and the ECM. However, this last assumption is not essential and can be discarded. Also, to a first approximation, we consider the ECM to be rigid. In accordance with these assumptions, the equations (1)–(6) can be rewritten as

$$\begin{cases} \frac{\partial(1-\phi)}{\partial t} = \Gamma_0, \\ \frac{\partial \phi s}{\partial t} + \nabla(\phi s v_T) = \Gamma_T, \\ \frac{\partial \phi(1-s)}{\partial t} + \nabla(\phi(1-s)v_l) = \Gamma_l, \\ 0 = -\phi s \nabla P + \nabla \cdot \hat{T}_T + b_T + M_{lT}(v_T - v_l) - M_{T_0} v_T, \\ 0 = -\phi(1-s)\nabla P + \nabla \cdot \hat{T}_l - M_{lT}(v_T - v_l), \end{cases} \tag{8}$$

since the ECM rigidity assumption implies that the stress tensor $T_0$ simply responds to forces applied to the ECM. The bulk force $b_T$ reflects, for example, the influence of chemotactic or haptotactic factors on tumor cells, and it is also assumed that $b_l$ disappears. If we assume that $\hat{T}_l = 0$, then the last equation can be written as Darcy's law $v_l - v_T = -\mathrm{K}\nabla P$, where K is the permeability and is a function of the volume fraction of the liquid phase. By adding the two equations of momentum, it is possible to exclude from the system the forces of interaction between the liquid and the cells

$$-\phi \nabla P + \nabla \cdot \hat{T}_T - \mathrm{K}_0^{-1} v_T + b_T = 0,$$

where $\widetilde{\mathrm{K}}_0 = M_{T0}^{-1}$ is the permeability of the sticky granular flow in the porous structure formed by the extracellular matrix network. In the general case, it can depend on the porosity and saturation of tissue cells. The model equations will take the form

$$\begin{cases} \frac{\partial(1-\phi)}{\partial t} = \Gamma_0, \\ \frac{\partial \phi s}{\partial t} + \nabla(\phi s v_T) = \Gamma_T, \\ \nabla(\phi s v_T + \phi(1-s)v_l) = 0, \\ v_l - v_T = -\mathrm{K}\nabla P, \\ v_T = \mathrm{K}_0\Big[-\phi \nabla P + \nabla \cdot \hat{T}_T + b_T\Big], \end{cases} \tag{9}$$

where the third equation in (9) was obtained by summing the mass balance equations.

## 2. Neglecting of mechanical interaction with extracellular fluid

Consider the case when the permeability tensor is isotropic. Since $M_{T0} \geq M_{lT}$, $\widetilde{K_0} \ll K$. Then, substituting the pressure gradient from Darcy's law into (8), we get that

$$v_T = \widetilde{\mathrm{K}}_0(\nabla \cdot \hat{T}_T + b_T). \tag{10}$$

Assuming that $b_T$ is proportional to the chemical concentration gradient $b_T = \chi \nabla c$, and neglecting the stress tensor, the (10) equation implies the usual chemotactic closure $v_T = w\nabla c$, where $w = \widetilde{\mathrm{K}_0}\chi$. In particular, there are classical chemotactic models of the form

$$\frac{\partial \phi s}{\partial t} + \nabla \cdot (w \phi s \nabla c) = \Gamma_T. \tag{11}$$

Chemotaxis can then be viewed as a force balanced by the resistance force created by the nutrient medium, rather than as a convenient closure of the mass balance equation. The (11) equation with a given or varying concentration in accordance with the classical reaction–diffusion equation can be characterized by a solution that experiences a discontinuity. On the other hand, [7] shows that if the mechanics are properly taken into account, that is, if (10) is used with a suitable stress constitutive equation, the discontinuity of the solution is prevented. In case of more chemotactic/haptotactic effects, the model can be used

$$\begin{cases} \frac{\partial(1-\phi)}{\partial t} = \Gamma_0, \\ \frac{\partial \phi s}{\partial t} + \nabla \cdot \Big[\widetilde{K_0}\phi s\Big(\nabla \cdot T_T + \sum_i \chi_i c_i\Big)\Big] = \Gamma_T, \end{cases}$$

which must be supplemented with appropriate reaction—diffusion equations for the chemical factors $c_i$. The first equation describes the possible deposition or degradation of the extracellular matrix. Suppose that the stress tensor is isotropic $T_T = -\Sigma(s)\mathrm{I}, \Sigma(s) = s - s^0, b_T = 0$ (no chemotactic effect), where $s^0 = s|_{t=0}$ is a given function [2]. Substituting $v_T$ into the second equation from (9), we get a system of equations for finding $\phi$ and $s$

$$\begin{cases} \frac{\partial(1-\phi)}{\partial t} = \Gamma_0, \\ \frac{\partial \phi s}{\partial t} - \nabla \cdot \Big(K_0(\phi)a(s)\nabla s\Big) = \Gamma_T, \end{cases}$$

where $\widetilde{K_0} = K_0(\phi)a(s)$,

$$a(s) = \begin{cases} 1, & s \geq 1, \\ s^\alpha, & 0 < s < 1, \alpha \geq 1. \\ 0, & s \leq 0. \end{cases}$$

Systems of equations similar in structure have been studied in the works [8]– [10].

## 3. Predominance of liquid-cell interaction

If there are no interactions cell - ECM , ECM - liquid, then $\Gamma_0 = 0$. Let's put $\Gamma_T = \gamma\delta(s)R(\phi)$ [2], where $\gamma$ – const $> 0$,

$$\delta(s) = \begin{cases} 0, & s \geq 1, \\ s(1-s), & 0 < s < 1, \\ 0, & s \leq 0, \end{cases}$$

$$R(\phi) = \begin{cases} 0, & \phi \geq 1, \\ \phi(1-\phi), & 0 < \phi < 1, \\ 0, & \phi \leq 0. \end{cases}$$

Then the system takes the form:

$$\begin{cases} \frac{\partial \phi}{\partial t} = 0, \\ \frac{\partial \phi s}{\partial t} - \nabla \cdot \Big(K_0(\phi)a(s)\nabla s\Big) = \Gamma_T. \end{cases}$$

From the first equation we have that $\phi = \phi^0(x)$, and the second takes the form

$$\phi^0(x)\frac{\partial s}{\partial t} - \nabla \cdot \Big(K_0(\phi^0(x))a(s)\nabla s\Big) = \Gamma_T.$$

## 4. Finite perturbation propagation velocity

In the one-dimensional case, the equation for $s$ can be written as

$$\phi^0(x)\frac{\partial s}{\partial t} = \frac{\partial}{\partial x}\left(a(s)K_0(\phi^0(x))\frac{\partial s}{\partial x}\right) + \Gamma_T, \quad (12)$$

where it is assumed that there exists a constant $M > 0$ such that the following estimates hold $0 \le s \le M < \infty$. The main result of this paper can be formulated as follows: let $s(x,t)$ be a weak solution of (7) in $K_{\rho_0}(x_0) \times (0,\infty)$, $K_{\rho_0}(x_0) = \{(x,x_0) : |x - x_0| < \rho_0\}$ such that $s_0(x) \equiv s(x,0) = 0$ in $K_{\rho_0}(x_0)$. Then there exist $T > 0$ and $\rho(t) \in (0,\rho_0)$ such that $s(x,t) = 0$ for all $t \le T$ and $x \in K_\rho(x_0)$. Under additional assumptions on the nature of the vanishing of $s_0(x)$ we prove that $s(x,t) = 0$ in $K_{\rho_0}(x_0)$. The question of the existence of a corresponding solution is not touched upon here. The proof uses the local energy method developed in [11], [12]. Consider a number of function spaces on $\Omega$ and $Q_T$, following the notation adopted in [13]. Let $||\cdot||_{q,\Omega}$ be the norm in the Lebesgue space $L_q(\Omega)$, $q \in [1,\infty]$. For brevity, we put $||\cdot||_q = ||\cdot||_{q,\Omega}$, $||\cdot|| = ||\cdot||_{2,\Omega}$. We also use the space $\overset{o}{C}{}^\infty$, the set of infinitely differentiable functions with compact support in $\Omega$, and the Sobolev space $W_p^l(\Omega)$, where $l$ – natural, $p \in [1,\infty]$ with the norm $||f||_{W_p^l(\Omega)} = \sum_{m=0}^{l} ||D_x^m f||_{p,\Omega}$.

**Definition.** A non-negative bounded measurable function $s(x,t)$ $(0 \le s(x,t) \le M)$ defined in $\Omega \times (0,\infty)$ is a weak solution of the equation (12) with initial condition $s_0(x)$ if $\forall T > 0$ and any open subset $\Omega_1 \subset R^1$ the following assumptions hold

$$s \in L_\infty(0,T,W_2^1(\Omega)), \frac{\partial}{\partial x}\left(s^{\alpha+1}\right) \in L_2[(0,T)\times\Omega_1], \quad (13)$$

$$\lim_{t\to 0}\int_Q s\,dx = \int_Q s_0\,dx \quad (14)$$

and $\forall \Phi(x,t) \in \overset{o}{C}{}^\infty((0,T)\times\Omega_1)$

$$\int_0^\infty\int_\Omega \left[a(s)K_0(\phi^0)\frac{\partial s}{\partial x}\frac{\partial \Phi}{\partial x} - \Gamma_T\,\Phi\right]dx\,dt =$$

$$= \int_0^\infty\int_\Omega s\phi^0(x)\frac{\partial \Phi}{\partial t}\,dx\,dt + \int_\Omega \phi^0(x)s(x,0)\,\Phi(x,0)\,dx. \quad (15)$$

We introduce the notation

$$A(\rho,t) \equiv \int_{K_\rho(x_0)} s^2(x,t)dx,$$

$$B(\rho,t) \equiv \int_{K_\rho(x_0)} s^\alpha\left(\frac{\partial s}{\partial x}\right)^2 dx,$$

and without loss of generality we assume $x_0 = 0$.

**Lemma.** Suppose that (13), (14) are fulfilled. Then $s(\rho,t)$ satisfies the estimates

$$s^\sigma(\rho,t) \le C_i A^{\frac{1-\theta}{r}}(\rho,t)[B^{\frac{1}{2}}(\rho,t) + \rho^{-\delta}A^{\frac{1}{r}}(\rho,t)]^\theta, \quad i = 1,2, \quad (16)$$

where

$$\sigma = \frac{\alpha}{2} + 1 > 0, \quad \theta = \frac{2}{2+r}, \quad \delta = \frac{1}{\theta r}.$$

If $i = 1$, then $\frac{4}{\alpha+2} < r < 2, 0 < \alpha < 2$,

$$C_1 = CM^{\frac{(r\sigma-2)(1-\theta)}{2}}\max(\sigma, M^{\frac{r\sigma-2}{r}}),$$

and if $i = 2$, then $\alpha = 2$,

$$r = \frac{4}{\alpha+2} = 1, \quad C_2 = 2C,$$

$C$ is a positive constant independent of the radius $\rho$.

**Proof** follows [14], [15].

**Theorem.** Assume that the conditions (13) – (15) are fulfilled and additionally $t \in [0,T], T \le T^*$, where

$$T^* \le min\left(\frac{1}{2}min(\phi^0(x)), \left((\rho_0^{1+2\delta} - \right.\right.$$

$$\left.\left. -\rho^{1+2\delta})\frac{2\theta-1}{(2\delta+1)4K_i^2}w^{1-2\theta}(\rho_0,t)\right)^{\frac{1}{1-\theta}}\right), i = 1,2.$$

If $s(x,t)$ is weak solution to (7) and $s_0(x) = 0$ in $K_{\rho_0}(x_0)$, $0 < \rho_0 < dist(x_0,\partial G)$, then $s(x,t) = 0$ almost everywhere in $K_{\rho_1(t)}(x_0)$ for $0 \le t \le T \le T^*$. Moreover

$$\rho_1(t) = \left(\rho_0{}^{1+2\delta} - L\,t^{1-\theta}(w(\rho_0,t))^{2\theta-1}\right)^{\frac{1}{1+2\delta}},$$

where if $0 < \alpha < 2$, then $L = 4C_1^2 \cdot Q(r), \quad r \in (1,2)$, and if $\alpha = 2$, then $L = 4C_2^2 \cdot Q(r), \quad r = \frac{4}{\alpha+2} = 1$. In both cases

$$w(\rho_0,t) = \sup_{0\le\tau\le t}\int_0^\tau B(\rho_0,s)ds,$$

$$Q(r) = \frac{2\delta+1}{2\theta-1}\left(\frac{1}{2}\rho_0^\delta + T^{\frac{1}{2}}M^{2(\frac{1}{2r}-1)}\rho_0^{\frac{1}{2r}-1}\right)^{2\theta},$$

$$K_i = C_i\,[\frac{1}{2}\rho_0{}^\delta + T^{\frac{1}{2}}\,\rho_0{}^{\frac{1}{2r}-1}\,M^{2(\frac{1}{2r}-1)}]^\theta, \quad i = 1,2,$$

and constants $C_1$ and $C_2$ are determined in Lemma.

**Proof of the theorem.** Equation (12) after standard transformations, similar to work [14], is

transformed to the form:

$$\int_0^\rho \phi^0(x)s^2dx+$$

$$+\int_0^t\int_0^\rho\left[a(s)K_0(\phi^0(x))\,(\frac{\partial s}{\partial x})^2-s\Gamma_T\right]dx\,d\tau=$$

$$=\int_0^t sa(s)K_0(\phi^0(x))\,\frac{\partial s}{\partial x}(\rho,\tau)\,ds]\,d\tau. \tag{17}$$

Let's put

$$a(\rho,t)=\sup_{0\le\tau\le t}A(\rho,\tau).$$

It follows from (17) that

$$\min_x(\phi^0(x))a(\rho,t)+\min_x(K_0\quad(\phi^0(x)))w(\rho,t)\le$$
$$\le I_1+\nu I_2, \tag{18}$$

where $\nu=\gamma\max_x(\phi^0(x)(1-\phi^0(x)))$, and follows [14], we obtain

$$I_1=\int_0^t s(\rho,\tau)^{\alpha+1}\left|\frac{\partial s}{\partial x}(\rho,\tau)\right|d\tau\le$$

$$\le K_i t^{\frac{1-\theta}{2}}\,\rho^{-\delta\theta}\,[a(\rho,t)+w(\rho,t)]^\theta\left(\frac{\partial w}{\partial\rho}\right)^{\frac12},$$

$$I_2=\int_0^t\int_0^\rho s^2(1-s)dxd\tau\le t(a+w).$$

Therefore, (18) takes the form

$$min(\phi^0(x))(a(\rho,t)+w(\rho,t))\le I_1+\nu I_2\le$$
$$\le t^{\frac{1-\theta}{2}}\,K_i\rho^{-\delta\theta}\,(a(\rho,t)+w(\rho,t))^\theta\left(\frac{\partial w}{\partial\rho}\right)^{\frac12}+$$
$$+\nu t(a+w),\quad i=1,2.$$

Now we choose $t$ in such a way that $t\le\frac{1}{2\nu}min(\phi^0(x))$. Therefore,

$$\rho^{2\delta\theta}w^{2(1-\theta)}\le K_i^*t^{1-\theta}\frac{\partial w}{\partial\rho}, \tag{19}$$

where $K_i^*=4(K_i)^2, i=1,2$. Integrating (19) by $\rho$ from $\rho_1$ to $\rho_0$, we find that $(1\le r<2)$

$$\rho_1^{1+2\delta\theta}-\rho_0^{1+2\delta\theta}+\frac{2\delta\theta+1}{2\theta-1}K_i^*t^{1-\theta}w^{2\theta-1}(\rho_0,t)\ge$$
$$\ge\frac{2\delta\theta+1}{2\theta-1}K_i^*t^{1-\theta}w^{2\theta-1}(\rho_1,t).$$

Choosing $t$ in such a way that the equality

$$\rho_1^{1+2\delta\theta}=\rho_0^{1+2\delta\theta}-\frac{2\delta\theta+1}{2\theta-1}\,K_i^*\,t^{1-\theta}\,w^{2\theta-1}(\rho_0,t),$$

holds, we obtain that $w(\rho,t)=0$ for all $\rho\le\rho_1$, i.e. $s(x,t)=0$ almost everywhere in $K_\rho(0)$ for $\rho\le\rho_1$ and

$$0\le t\le min\left(\frac{1}{2\nu}min(\phi^0(x)),\left((\rho_0^{1+2\delta\theta}-\right.\right.$$
$$\left.\left.-\rho^{1+2\delta\theta})\frac{2\theta-1}{(2\delta\theta+1)K_i^*}w^{1-2\theta}(\rho_0,t)\right)^{\frac{1}{1-\theta}}\right).$$

The theorem is proved.

**Conclusion**

This article describes a mathematical model of tumor dynamics takes into account phase transitions and based on the theory of fluid filtration in porous media. After some natural assumptions characteristic of the process under consideration, the system of equations under consideration is reduced to a first-order equation for the porosity function and a parabolic equation for the saturation of the cell phase with a special right-hand side. The property of a finite speed of propagation of disturbances for cell saturation has been established.

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***Information about the authors***

**V.B. Pogosyan,** Master Student of the Department of Differential Equations, Altai State University, Barnaul, Russia;

**M.A. Tokareva,** Candidate of Sciences in Physics and Mathematics, Researcher at the Laboratory of Boundary Problems in Continuum Mechanics, M.A. Lavrentiev Institute of Hydrodynamics SB RAS, Novosibirsk, Russia;.

**A.A. Papin,** Doctor of Sciences in Physics and Mathematics, Head of the Department of Differential Equations, Altai State University, Barnaul, Russia.

***Информация об авторах***

**В.Б. Погосян,** магистрант кафедры дифференциальных уравнений, Алтайский государственный университет, Барнаул, Россия;

**М.А. Токарева,** кандидат физико-математических наук, научный сотрудник лаборатории краевых задач механики сплошных сред, Институт гидродинамики им. М.А. Лаврентьева СО РАН, Новосибирск, Россия;

**А.А. Папин,** доктор физико-математических наук, заведующий кафедрой дифференциальных уравнений, Алтайский государственный университет, Барнаул, Россия.