ARTICLE IN PRESS

Computers and Mathematics with Applications ▮ (▮▮▮▮) ▮▮▮–▮▮▮

Contents lists available at ScienceDirect

Computers and Mathematics with Applications

journal homepage: www.elsevier.com/locate/camwa

On the existence of weak solutions of nonlinear degenerate parabolic system with variable exponents

L. Shangerganesh [a], N. Nyamoradi [b,*], V.N. Deiva Mani [c], S. Karthikeyan [c]

[a] Department of Humanities and Sciences, National Institute of Technology, Goa, India
[b] Department of Mathematics, Razi University, 67149 Kermanshah, Iran
[c] Department of Mathematics, Periyar University, Salem, India

ARTICLE INFO

Article history:
Received 3 April 2017
Received in revised form 3 August 2017
Accepted 9 September 2017
Available online xxxx

Keywords:
Cancer invasion parabolic system
Weak solution
Faedo–Galerkin method

ABSTRACT

The main goal of the present paper is establishing the existence and uniqueness of weak solutions for the nonlinear degenerate reaction–diffusion system with variable exponents. A model also is proposed to characterize the invasion of cancer cells towards healthy cells with acidification environment. Moreover, the main results of this paper are obtained using regularization problem, the Faedo–Galerkin approximation method, some apriori estimates, compactness results and the Gronwall Lemma.

1. Introduction

Invasion and spread of cancer cells surrounding the tissue plays an important role in solid tumor progression and it plays key role for the metastasis spread of the disease. In recent years there have been many reaction–diffusion models on tumor invasion which has grown immensely as one of the challenging problems for mathematical analysis. Over the last two decades, various cancer invasion models have developed to understand the stages of cancer invasion towards healthy cells, for example, see, Anderson [1], Anderson et al. [2], Gatenby [3], Gatenby and Gawlinski [4], Jiang et al. [5], Kerangueven et al. [6], Perumpanani et al. [7], Preziosi [8] and also the references therein.

In this paper, we have considered the tumor dynamics mathematical model with two types of cancer cells. It means that we have assumed that during the development of the cancer disease, the second sub-population emerging in the tumor mass arises from mutations in the cells of the first sub-population. We also hypothesize that production and the diffusion of the acid into surrounding healthy tissue, creates an environment in which cancer cells survive and proliferate. We should emphasize that the diffusion of cancer cells and degrading enzymes are highly nonlinear and they have growth conditions in terms of variable exponents. The third equation of the model incorporates the drug toxicity effect. In particular, it describes the dynamic growth of the normal cells of the host organ which are affected by the cancerous tumor growth. The fourth equation of the model is the acidification caused by the presence of a tumor. Cancer cells affect the surrounding medium by the excess of ions H^+ that are produced in a proportional rate to the density of cancer cells. Here, we have considered that its natural reabsorption is proportional to the excess of ions H^+. Moreover, the diffusivity property of fourth equation also follows Fick's law and it is represented as nonlinear diffusion.

We have considered a mathematical model with four unknown variables namely two cancer cells density, normal cells density and acidification medium concentration as proposed in [9]. To such model we have assumed the nonlinear density

* Corresponding author.
E-mail addresses: shangerganesh@nitgoa.ac.in (L. Shangerganesh), nyamoradi@razi.ac.ir (N. Nyamoradi), deivamani.vn@gmail.com (V.N. Deiva Mani), skkmathpu@gmail.com (S. Karthikeyan).

http://dx.doi.org/10.1016/j.camwa.2017.09.019
0898-1221/

dependent diffusion for unknowns instead of standard Laplacian diffusion operator. Thus, the dynamics of the cancer invasion system with nonlinear diffusion is governed by the following reaction–diffusion system :

$$\left.\begin{aligned} &\partial_t u_1 - \operatorname{div}(A_1(x,t,u_1,\nabla u_1)) = u_1(1-u_1) - \beta_1 u_1 u_2 - \rho u_1 - \gamma_1 u_1 u_3 \text{ in } Q_T,\\ &\partial_t u_2 - \operatorname{div}(A_2(x,t,u_2,\nabla u_2)) = r_2 u_2(1-u_2) - \beta_2 u_1 u_2 + \rho u_1 - \delta_1 u_2 u_3 \text{ in } Q_T,\\ &\partial_t u_3 = r_3 u_3(1-u_3) - \gamma_2 u_1 u_3 - \delta_2 u_2 u_3 - \sigma u_3 u_4 \text{ in } Q_T,\\ &\partial_t u_4 - \operatorname{div}(A_4(x,t,u_4,\nabla u_4)) = \xi(u_1+u_2-u_4) \text{ in } Q_T \end{aligned}\right\} \tag{1.1}$$

with initial and boundary conditions

$$\begin{aligned} &u_i(x,0) = u_{i,0}(x),\ i = 1,2,3,4 \text{ in } \Omega,\\ &u_i(x,t) = 0, \qquad i = 1,2,4 \quad \text{in } \Sigma_T, \end{aligned}$$

where $Q_T = \Omega \times (0,T)$, $\Sigma_T = \partial\Omega \times (0,T)$, Ω is an open bounded domain in $\mathbb{R}^N$ with boundary $\partial\Omega$. Further, $u_1(x,t)$ and $u_2(x,t)$ denote the density of two cancer cells sub-populations respectively, $u_3(x,t)$ represents the density of normal cells and $u_4(x,t)$ represents the medium acidification at position x and time t. Here, β_1 and β_2 are the coefficients of the interaction terms between both types of cancer cells. The positive constant ρ denotes the intrinsic mutation rate of cancer cells from type I to type II. Moreover, the competition between healthy cells and cancer cells are assumed as Lotka–Volterra functional competition form, therefore, γ_1 and δ_1 represent the rate of consumption of corresponding cancer cells. The constants r_2 and r_3 measure the proliferation rates and ξ represents production rate of the ions H^+. Finally, γ_2 and δ_2 respectively denote the interaction rate of two cancer cells with normal cells and σ denotes the degradation rate of normal cells due to acidification. Further, we have assumed the Dirichlet boundary conditions for the system (1.1), it means that the model is self contained and has no population density on the boundary $\partial\Omega$.

Here, $A_i(x,t,u_i,\nabla u_i)$, $i = 1,2,4$ are nonlinear density dependent diffusion functions. Further, these divergence form diffusion operators are Carathéodory function defined on $Q_T \times \mathbb{R} \times \mathbb{R}^N$ to $\mathbb{R}^N$. Moreover, it is worth mentioning that to prove the existence of weak solutions of the given nonlinear parabolic system (1.1), we are in need of the following hypotheses throughout this article. For $i = 1,2,4$,

(H1) $A_i(x,t,\eta,\zeta) \geq d_i|\zeta|^{p(x)}$, $d_i > 0$,
(H2) For any $\varrho_i > 0$ and the functions $\Lambda_i(x,t)$ in $L^{p'(x)}(Q_T)$ such that $|A_i(x,t,\eta,\zeta)| \leq \varrho_i\left[\Lambda_i(x,t) + |\eta|^{p(x)-1} + |\zeta|^{p(x)-1}\right]$,
(H3) $(A_i(x,t,\eta,\zeta) - A_i(x,t,\eta,\zeta'))(\zeta - \zeta') \geq 0$,

for almost every $(x,t) \in Q_T$, for every $\eta \in \mathbb{R}$, for every $\zeta, \zeta' \in \mathbb{R}^N$, with $\zeta \neq \zeta'$ and $p(x)$ is a continuous function. For more details regarding the hypotheses for the constant case, see [10–12], and for the variable exponent case, see [13,14]. A good amount of work is available on the literature for elliptic and parabolic type partial differential equations with variable exponents and no growth conditions during the last two decades, for example, see [13–18] and the references therein.

We now briefly discuss the literature related to this work. Studies on the existence of solutions of various biological models using mathematical techniques are very interesting and have studied by many researchers in the literature, see [19–28] and the references therein. However, existence of solutions of tumor dynamics mathematical models are very few. Global existence of unique classical solution to the chemotaxis–haptotaxis model of cancer invasion of tissue is proved in [29] using contraction mapping principle and apriori estimates. Further, Tao and Wang used a similar idea in [30] to prove the global existence of solutions in two and three spatial dimension domains for cancer invasion model which incorporates interaction of cancer cells with extra cellular matrix. A free boundary problem modeling the cell cycle and cell movement in multi cellular tumor spheroids is considered in [31] and existence of solutions is established with the help of fixed point argument and L^p theory of parabolic problems. Further, Walker and Webb used fixed point arguments and proved the existence of unique global classical solutions for a system of nonlinear cancer invasion model in [32]. Global existence of solution is proved for mathematical model of cancer cell invasion using semigroup theory method and apriori estimates in [33].

In most of the previous studies, the diffusion coefficients of unknown functions are considered as a constant or a function of space variables. However, diffusion functions of cancer invasion models need not be constant always. It has been well established theoretically and numerically. The density dependent nonlinear diffusion of unknown is more realistic than the constant diffusion coefficient, for example, see [34–37]. Moreover, Ito et al. established local existence and uniqueness of solutions to approximate systems of 1D tumor invasion model with nonlinear diffusion in [38]. A mathematical model focusing on the effect of HSPs on the tumor cell migration is proposed and the local existence of a unique positive weak solution is obtained using iterative procedure in [39]. Tao and Winkler studied global existence of solutions for chemotaxis–haptotaxis nonlinear diffusion model using the Schauder fixed point theorem in [40]. Tao and Cui considered a nonlinear diffusion model consists of three reaction–diffusion–taxis equations describing interactions between cancer cells, matrix degrading enzymes, and the host tissue and established existence of solutions using fixed point argument in [41].

The main aim of present work is establishing the existence and uniqueness of weak solution of highly nonlinear density dependent diffusion cancer invasion system (1.1). A major difficulty in the analysis of (1.1) is the strong degeneracy of the density dependent diffusion terms and the presence of highly nonlinear coupled source terms. Therefore, in order to handle this difficulty, we introduce the regularized non-degenerate problem. Then, we establish the existence of a weak solution of regularized system using the Faedo–Galerkin approximation method. Finally, using some apriori estimates, compactness results and Gronwall's Lemma, we prove the main result of the work.

Please cite this article in press as: L. Shangerganesh, et al., On the existence of weak solutions of nonlinear degenerate parabolic system with variable exponents, Computers and Mathematics with Applications (2017), http://dx.doi.org/10.1016/j.camwa.2017.09.019.