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Boundedness of solutions to a quasilinear chemotaxis–haptotaxis model

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ABSTRACT

We study global solutions of a class of chemotaxis–haptotaxis systems generalizing the prototype

$$\begin{cases} u_t = \nabla\cdot((u+1)^{m-1}\nabla u) - \nabla\cdot(u(u+1)^{q-1}\nabla v) \\ \qquad - \nabla\cdot(u(u+1)^{p-1}\nabla w) + H(u,w), \\ 0 = \Delta v - v + u, \\ w_t = -vw, \end{cases}$$

in a bounded domain $\Omega \subset \mathbb{R}^N (N \geq 1)$ with smooth boundary, $H(u,w) := u(1-u^{r-1}-w)$, with parameters $m \geq 1, r > 1$ and positive constants p, q. It is shown that either $\max\{q+1, p, 2p-m\} < \max\{m+\frac{2}{N}, r\}$ or $\max\{q+1, p, 2p-m\} = r$ and $b > 0$ is large enough, then for any sufficiently smooth initial data there exists a classical solution which is global in time and bounded. The results of this paper improve the results of Tao and Winkler (2014) [46,51].

1. Introduction

The Keller–Segel model [1,2] is a widely studied topic, see e.g. Murray [3] for a general background and Horstmann [4] for a fairly complete survey on the Keller–Segel model and the variants that have been proposed. For a detailed description of the intrinsic scaling method and some applications, we refer to the books [5,6].

One important extension of the classical Keller–Segel model to a more complex cell migration mechanism was proposed by Chaplain and Lolas [7] in order to describe processes of cancer invasion, that consists of several important steps involving different biological mechanisms. A variety of mathematical models have been developed for various aspects of cancer invasion (see [8–11]). The variables u, v and w describe the density of the cancer cell population, the concentration of a matrix-degrading enzyme (MDE) and the concentration of extracellular matrix (ECM), respectively. Then (u, v, w) satisfies

$$\begin{cases} u_t = \nabla\cdot(\phi(u)\nabla u) - \chi\nabla\cdot(\psi(u)\nabla v) - \xi\nabla\cdot(\varphi(u)\nabla w) + f(u,w), & x\in\Omega,\ t>0, \\ \tau v_t = \Delta v + u - v, & x\in\Omega,\ t>0, \\ w_t = -vw + \eta w(1-u-w), & x\in\Omega,\ t>0, \\ \dfrac{\partial u}{\partial \nu} = \dfrac{\partial v}{\partial \nu} = \dfrac{\partial w}{\partial \nu} = 0, & x\in\partial\Omega,\ t>0, \\ u(x,0) = u_0(x), \qquad v(x,0) = v_0(x), \qquad w(x,0) = w_0(x), & x\in\Omega, \end{cases} \tag{1.1}$$

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where $\tau \in \{0, 1\}$, $\eta \geq 0$, Ω is a bounded domain in $\mathbb{R}^N (N \geq 1)$ with smooth boundary $\partial\Omega$, $\Delta = \sum_{i=1}^{N} \frac{\partial^2}{\partial x_i^2}$, $\frac{\partial}{\partial \nu}$ denotes the outward normal derivative on $\partial\Omega$, $\chi > 0$ is a parameter referred to as chemosensitivity, $\xi > 0$ is a parameter that measures the haptotactic sensitivities, $\psi(u)$ describes the chemotactic sensitivity of cell population and the growth function $f : ([0, \infty))^2 \mapsto \mathbb{R}$ is smooth and satisfies $f(0, w) = 0$ as well as

$$f(u, w) \leq a - bu^r \quad \text{for all } u, w \geq 0 \tag{1.2}$$

with some $a \geq 0$, $b > 0$ and $r > 1$. Moreover, we assume that the functions $\phi(u)$ and $\psi(u)$ fulfill

$$\phi, \psi, \varphi \in C^2([0, \infty)) \quad \text{and} \quad \psi(u) \geq 0,\ \varphi(u) \geq 0 \text{ for all } u \geq 0. \tag{1.3}$$

Moreover, in order to prove our results, we need to impose the conditions that there exist constants $q, p > 0$, $m \geq 1$, C_ϕ, C_ψ and C_φ such that

$$\phi(u) \geq C_\phi (u + 1)^{m-1} \quad \text{for all } u \geq 0 \tag{1.4}$$

$$\psi(u) \leq C_\psi u^q \quad \text{for all } u \geq 0 \tag{1.5}$$

and

$$\varphi(u) \leq C_\varphi u^p \quad \text{for all } u \geq 0. \tag{1.6}$$

System (1.1) (chemotaxis–haptotaxis model) (see [8,12,10,11]) has been introduced in order to explain the cancer invasion, that consists of several important steps involving different biological mechanisms. A variety of mathematical models have been developed for various aspects of cancer invasion. Mathematical modeling of cancer has emerged into a large discipline, with models developed to describe these many facets of cancer development, aiming to shed fresh impetus on tumor formation, progression and treatment.

When $w \equiv 0$, the PDE system (1.1) is reduced to the classical Keller–Segel system with logistic source, that is,

$$\begin{cases} u_t = \nabla \cdot (\phi(u)\nabla u) - \chi \nabla \cdot (\psi(u)\nabla v) + f(u, v), & x \in \Omega,\ t > 0, \\ \tau v_t = \Delta v + u - v, & x \in \Omega,\ t > 0, \\ \dfrac{\partial u}{\partial \nu} = \dfrac{\partial v}{\partial \nu} = 0, & x \in \partial\Omega,\ t > 0, \\ u(x, 0) = u_0(x), \qquad v(x, 0) = v_0(x), & x \in \Omega. \end{cases} \tag{1.7}$$

The Keller–Segel models have become one of the best study models in mathematical biology, and throughout the main issue of the investigation was whether the solutions of the models are bounded or blow-up (see e.g., Cieślak et al. [13–15], Calvez and Carrillo [16], Horstmann et al. [4,17,18], Osaki [19,20], Painter and Hillen [21], Perthame [22], Rascle and Ziti [23], Winkler [24–26]).

In the absence of the logistic source (i.e. $f \equiv 0$) for problem (1.7), the results appear to be rather complete. In particular, to $\phi(u) \equiv 1$, (1.7) with $\psi(u) = u$, leading to the classical chemotaxis model, has successfully been investigated up to now: It is known that the model has only bounded solutions if $N = 1$ [20]; if $N = 2$, there exists a threshold value for the initial mass that decides whether the solutions can blow up or exist globally in time [27,17,28]; when $N \geq 3$, there is no such threshold [14,28–30]. Especially, in [17], Horstmann and Wang showed that the solutions are global and bounded provided that $\psi(u) \leq c(u + 1)^{\frac{2}{N} - \varepsilon}$ for all $u \geq 0$ with some $\varepsilon > 0$ and $c > 0$; on the other hand, if $\psi(u) \geq c(u + 1)^{\frac{2}{N} + \varepsilon}$ for all $u \geq 0$ with $\varepsilon > 0$ and $c > 0$, $\Omega \subset \mathbb{R}^N (N \geq 2)$ is a ball, and some further technical conditions are satisfied, then the solutions become unbounded in finite or infinite time. Moreover, for more general $\phi(u)$, there are also many results concerning the question whether the solutions are bounded or blow-up (see e.g. Tao and Winkler [31], Ishida et al. [32], Winkler [30], Cieślak and Stinner [14,33]). In [31], Tao and Winkler proved that if $\frac{\psi(u)}{\phi(u)} \leq c(u + 1)^{\frac{2}{N} + \varepsilon}$ for all $u \geq 0$ with some $\varepsilon > 0$ and $c > 0$, then the corresponding solutions are global and bounded provided that $\phi(u)$ satisfies some another technical conditions. Recently, Ishida et al. [32] have improved the results of [31] in the case of degenerate diffusion on a bounded non-convex domain.

When $\chi = 0$, the PDE system (1.1) becomes the haptotaxis-only system

$$\begin{cases} u_t = \nabla \cdot (\phi(u)\nabla u) - \xi \nabla \cdot (\varphi(u)\nabla w) + f(u, w), & x \in \Omega,\ t > 0, \\ \tau v_t = \Delta v + u - v, & x \in \Omega,\ t > 0, \\ w_t = -vw + \eta w(1 - u - w), & x \in \Omega,\ t > 0, \\ \dfrac{\partial u}{\partial \nu} = \dfrac{\partial v}{\partial \nu} = \dfrac{\partial w}{\partial \nu} = 0, & x \in \partial\Omega,\ t > 0, \\ u(x, 0) = u_0(x), \qquad v(x, 0) = v_0(x), \qquad w(x, 0) = w_0(x), & x \in \Omega. \end{cases} \tag{1.8}$$

Global existence theories for this system were explored in [34–36], whereas the boundedness and asymptotic behavior of solution were studied in [37]. The above results rule out the possibility of blow-up of solutions to this haptotaxis-only