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# Stability of planar traveling waves in a Keller–Segel equation on an infinite strip domain

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### Abstract

We consider a simplified model of tumor angiogenesis, described by a Keller–Segel equation on the two dimensional domain  $(x, y) \in \mathbb{R} \times S^{\lambda}$  where  $S^{\lambda}$  is the circle of perimeter  $\lambda$ . It is known that the system allows planar traveling wave solutions of an invading type. In case that  $\lambda$  is sufficiently small, we establish the nonlinear stability of traveling wave solutions in the absence of chemical diffusion if the initial perturbation is sufficiently small in some weighted Sobolev space. When chemical diffusion is present, it can be shown that the system is linearly stable. Lastly, we prove that any solution with our front condition eventually becomes planar under certain regularity conditions. © 2018 Elsevier Inc. All rights reserved.

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#### 2

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## 1. Introduction and main theorems

The formation of new blood vessels (angiogenesis) is the essential mechanism for tumour progression and metastasis. A simplified model of tumor angiogenesis can be described by a Keller–Segel equation [8,14,20]. In this paper we consider the equation on the two dimensional cylindrical domain  $(x, y) \in \Omega = \mathbb{R} \times [0, \lambda]$  with a front boundary condition in *x* and the periodic condition in *y*, both specified later,

$$\partial_t n - \Delta n = -\nabla \cdot (n\chi(c)\nabla c),$$
  

$$\partial_t c - \epsilon \Delta c = -c^m n$$
(1.1)

with  $m \ge 1$ , where  $n(x, y, t) \ge 0$  denote the density of endothelial cells,  $c(x, y, t) \ge 0$  stands for the concentration of the chemical substance or the protein known as the vascular endothelial growth factor (VEGF) and  $\chi(\cdot) : \mathbb{R}^+ \to \mathbb{R}^+$  is a decreasing chemosensitivity function, reflecting that the chemosensitivity is lower for higher concentration of the chemical. The system (1.1) includes the both zero chemical diffusion ( $\epsilon = 0$ ) and non-zero chemical diffusion ( $\epsilon > 0$ ) cases.

Endothelial cells forming the linings of the blood vessels are responsible for extending and remodeling the network of blood vessels, tissue growth and repair. Tumors or cancerous cells are also dependent on bloods supply by newly generated capillaries formed toward them, where the process is called endothelial angiogenesis. Modeling endothelial angiogenesis, the biological implication is that the endothelial cells behaves as a invasive species, responding to signals produced by the tissue.<sup>1</sup> Accordingly the system (1.1) is given the front condition at left-right ends such that

$$\lim_{x \to -\infty} n(x, y, t) = n_{-} > 0, \quad \lim_{x \to \infty} n(x, y, t) = 0, \tag{1.2}$$

$$\lim_{x \to -\infty} c(x, y, t) = 0, \quad \lim_{x \to \infty} c(x, y, t) = c_+ > 0.$$
(1.3)

We choose the *x*-axis by the propagating direction.

A *planar* traveling wave solution of (1.1) is a traveling wave solution independent of the transversal direction y such that

$$n(x, y, t) = N(x - st), \quad c(x, y, t) = C(x - st)$$
 (1.4)

with a wave speed s > 0. From now on, we consider only planar traveling waves (N, C) satisfying the above boundary conditions (1.2) and (1.3), and moreover we assume

$$N'(\pm\infty) = C'(\pm\infty) = 0. \tag{1.5}$$

(We denote  $\lim_{x\to\infty} N(x)$  by  $N(\infty)$  in short.)

In this paper we study the stability of a planar traveling wave solution (N, C) of (1.1) for  $\epsilon \ge 0$  case assuming  $\chi(c) = c^{-1}, m = 1$ ,

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 $<sup>^{1}</sup>$  In Bruce Alberts et al. [1], it is summarized as follows: "Cells that are short of oxygen increase their concentration of certain protein (HIF-1), which stimulates the production of vascular endothelial growth factor (VEGF). VEGF acts on endothelial cells, causing them to proliferate and invade the hypoxic tissue to supply it with new blood vessels."

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