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Fully Parabolic Keller-Segel Model for Chemotaxis with Prevention of Overcrowding

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Resumen

In this paper we study a fully parabolic version of the Keller-Segel system in presence of a volume filling effect which prevents blow up of the L^∞ norm. This effect is sometimes referred to as *prevention of overcrowding*. As in the parabolic elliptic version of this model (previously studied in [BDFDS06]), the results in this paper basically infer that the combination of the prevention of overcrowding effect with a linear diffusion for the density of cells implies domination of the diffusion effect for large times. In particular, first we show that both the density of cells and the concentration of the chemical vanish uniformly for large times, then we prove that the density of cells converges in L^1 toward the Gaussian profile of the heat equation as time goes to infinity, with a rate which differs from the rate of convergence to self similarity for the heat equation by an arbitrarily small constant ('quasi sharp rate').

1. Introduction

In this work we shall deal with the following parabolic system modeling *chemotaxis with prevention of overcrowding*

$$\begin{cases} \rho_t = \varepsilon\Delta\rho - \div(\rho(1-\rho)\nabla S) \\ S_t = \Delta S - S + \rho \end{cases} \tag{1}$$

Here ρ models the density of cells, S is the concentration of the chemical substance (chemoattractant). The parameter $\varepsilon > 0$ models the diffusivity of the cells. The present model is posed on the whole space $\mathbb{R}^N$ with $L^1 \cap L^\infty$ initial data for both ρ and $W^{1,1} \cap W^{1,\infty}$ for S. In the sequel we shall present a brief overview of results in the literature concerning chemotaxis models, by justifying the variants included in (1).

Chemotaxis is the phenomenon by which cells move under the influence of chemical substances in their environment. It has been known and widely studied since first descriptions were done by T.W. Engelmann and W.F. Pfeffer for bacteria in 1881 and 1884, and H.S. Jennings for ciliates in 1906. First mathematical models based on partial differential equations arose from the works of C.S. Patlak in 1953, who derived similar models with applications to the study of long-chain polymers (cf. [Pat53]) and E.F. Keller and L.A. Segel in 1970, who proposed a macroscopic model for aggregation of cellular slime molds (cf. [KS70]). Afterwards, several transport phenomena in biological systems have been labeled with the term *chemotaxis*, such as the bacteria *Escherichia coli*, or the amoebae *Dyctiostelium discoideum*, or endothelial cells of the human body responding to angiogenic factors secreted by a tumor. The main feature of these systems (in a very simplified form involving only two species) is the motion of a species ρ being biased by linear diffusion modeling random motion, with a diffusivity $\varepsilon > 0$ and by the gradient of a certain chemical substance S, whereas the flow of S features secretion/degradation mechanism without cross–diffusion. More precisely, one usually deals with solutions to the Cauchy problem on $\mathbb{R}^N$ for the system

$$\begin{cases} \rho_t = \varepsilon \Delta \rho - \operatorname{div}(\rho \chi(\rho, S) \nabla S) \\ S_t = \Delta S + r(\rho, S). \end{cases} \tag{2}$$

In system (2), the secretion/degradation mechanisms for S are contained in the term $r(\rho, S)$. A typical form is the linear one $r(\rho, S) = \alpha\rho - \beta S$ with $\alpha, \beta > 0$. The term $\chi(\rho, S)$, called *chemotactic sensitivity*, is very important in this context. In many situations it turns out that the expression of $\chi(\rho, S)$ determines the final outcome of the competition between diffusion (dispersion of particles) and singular aggregation phenomena (concentration to deltas) at the level of ρ (cf. the works of Jäger–Luckaus [JL92], Nagai [Nag95], Herrero–Velazquez [HV96] among others). In the case $\chi(\rho, S) \equiv$ constant, the above system has been extensively studied, especially in its parabolic–elliptic variants with the second equation in (2) replaced by $0 = \Delta S + \rho - S$ or by Poisson's equation $-\Delta S = \rho$. In particular, it is well known (cf. [DP04]) that the 2 dimensional Keller Segel system

$$\begin{cases} \rho_t = \Delta \rho - \operatorname{div}(\rho \chi \nabla S) \\ 0 = \Delta S + \rho \end{cases} \tag{3}$$

(with $L^1_+ \cap L \log L(\mathbb{R}^2)$ data for ρ) features a χ–dependent critical threshold m^* for the total mass of ρ determining finite time blow–up or global existence (blow–up for initial mass larger than m^*, global existence otherwise). Related results are contained in [CPZ04, Per04, BDP06, CP08] and in the recent preprint [CC08] for the parabolic case. For further references about all the several versions of the Keller–Segel system and related models, we refer to the review papers by Horstmann [Hor03, Hor04] and the references therein.

How to avoid finite time blow–up of cells has been the aim of an extensive research in the last years. This issue is motivated both by the attempt of constructing an 'approximate' notion of solution preventing blow up for any initial mass on the one side, and by modeling issues related with *volume filling effects* occurring when the density of cells becomes very large on the other side. There are mainly two ways to prevent blow up of ρ. The first one introduces a volume filling effect at the level of the diffusion of cells, replacing $\Delta\rho$ by a nonlinear diffusion term $\Delta\rho^\gamma$ with $\gamma > 1$. This modification of the model (cf. [Kow05,

CC05]) allows to define a global solution $\rho(t) \in L^1 \cap L^\infty$ for all $t > 0$ no matter how large the initial mass is. The second way to prevent blow up consists in modifying the chemotactical sensitivity. Among the possible ways to do that (cf. [HPS07, BDP06]), we mention the one suggested by Hillen and Painter in [PH03, HP01], which considers $\chi(\rho) = \rho_{max} - \rho$ for a certain $\rho_{max} > 0$ representing the maximum allowed density (ρ_{max} can be taken equal to 1 for simplicity). Basically, in this model cells stop aggregating when the density reached a maximum allowed value. An extensive mathematical theory for this model with prevention of overcrowding has been performed first in [DS05] on bounded domains and then in [BDFDS06], where also a variant with nonlinear diffusion has been considered in order to stop any mobility mechanism (including diffusion) at a certain density. Both [DS05] and [BDFDS06] concern with the parabolic–elliptic model. More recent results on volume filling effect have been also achieved in [HW05, Cie08, CW08]). In this paper we try to generalize some of the results in [BDFDS06] to the fully parabolic model (1). In particular, we aim to prove large time decay of solutions and the large time self–similar behavior of the density of cells. This last issue is extremely non trivial, because of the strong coupling between the two species. In order to perform this task, we use a diffusive time dependent scaling and a variant of the relative entropy method going back to [AMTU00, CJM+01]. The major difficulty with respect to the parabolic–elliptic case treated in [BDFDS06] is the fact the cells' interaction energy cannot be expressed in the form of a nonlocal interaction potential as in that case, since S has now an evolution of its own. Similar issues have been recently faced too in [CC08], where a similar model has been endowed with a suitable energy functional involving ρ and S.

2. Summary of the main results

Here we summarize the results presented here have appeared in [DFR08]. There, section 2 is devoted to the existence theory. First the existence and uniqueness of solution locally in time for any initial condition is proved, and then some a priori estimates which will be used to prove the existence of a global solution for (1) are provided.

In section 3 we concern about the long time behavior of the solutions and establish decay rates in $L^2(\mathbb{R}^N)$ and $L^\infty(\mathbb{R}^N)$ for both the cells density and the chemical. More precisely, it is shown that for some $\lambda > 0$

$$\|\rho(t)\|_2 + \lambda\|\nabla S(t)\|_2 \leq C(t+1)^{-\frac{N}{4}} \tag{4}$$

and

$$\|\rho\|_\infty = \mathrm{O}(t^{-\frac{1}{2}}) \text{ and } \|S\|_\infty = \mathrm{O}(t^{-\frac{1}{2}}) \text{ as } t \to +\infty. \tag{5}$$

A key technical result about the decay of the L^2 norm of ρ and ∇S must be imposed. Here (and hence in all the following results), the condition

$$\varepsilon > 1/4$$

on the diffusivity constant is needed. Roughly speaking, this condition ensures that the parabolic cross–diffusion operator on the r.h.s. of system (1) is uniformly elliptic with respect to $\rho \in [0, 1]$ and $S \geq 0$. Whether this condition is necessary to have a large time decay (and consequently a self–similar behavior) for ρ is still an open problem even in the (simpler) parabolic–elliptic case described in [BDFDS06].

Finally, in section 4 the asymptotic self–similar behavior of the solutions is studied by time dependent scaling and by proving convergence to a stationary state in the new variables. Namely, it is shown that for the following time translated self-similar gaussian solution of the Heat equation

$$\rho^\infty(t) = \frac{C_M}{(4\pi(2t+1))^{1/2}} e^{-\frac{|x|^2}{2(2t+1)}}.$$

and any arbitrarily small $\delta > 0$,

$$\|\rho(t) - \rho^\infty(t)\|_1 \leq C(t+1)^{-\frac{1-\delta}{2}} \tag{6}$$

for all $t > 0$.

The results in section 2, as well as the $L^2(\mathbb{R}^N)$–decay of the cells' density and the concentration of the chemoattractant, (4), are valid in any dimension N. For the decay of the L^∞–norms of ρ and S and for the large time self–similar behavior of ρ, we shall need to restrict ourselves to the case $N = 1$.

The main result of this paper deals with the self–similar behavior of ρ as $t \to +\infty$. Such result strongly relies on the a–priori decay estimate of the L^∞ norms of ρ and S, which are valid only in the case $N = 1$. However, we remark here that the proof of this result is also valid in case $N > 1$ provided one can prove that ρ and S satisfy suitable decay estimates for large times.

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