Available online at www.sciencedirect.com
ScienceDirect
Physica A 387 (2008) 4033–4052
PHYSICA A
www.elsevier.com/locate/physa

Jeans type analysis of chemotactic collapse

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Received 20 August 2007
Available online 16 February 2008

Abstract

We perform a linear dynamical stability analysis of a general hydrodynamic model of chemotactic aggregation [P.H. Chavanis, C. Sire, Physica A 384 (2007) 199]. Specifically, we study the stability of an infinite and homogeneous distribution of cells against "chemotactic collapse". We discuss the analogy between the chemotactic collapse of biological populations and the gravitational collapse (Jeans instability) of self-gravitating systems. Our hydrodynamic model involves a pressure force which can take into account several effects like anomalous diffusion or the fact that the organisms cannot interpenetrate. We also take into account the degradation of the chemical which leads to a shielding of the interaction like for a Yukawa potential. Finally, our hydrodynamic model involves a friction force which quantifies the importance of inertial effects. In the strong friction limit, we obtain a generalized Keller–Segel model similar to the generalized Smoluchowski–Poisson system describing self-gravitating Langevin particles. For small frictions, we obtain a hydrodynamic model of chemotaxis similar to the Euler–Poisson system describing a self-gravitating barotropic gas. We show that an infinite and homogeneous distribution of cells is unstable against chemotactic collapse when the "velocity of sound" in the medium is smaller than a critical value. We study in detail the linear development of the instability and determine the range of unstable wavelengths, the growth rate of unstable modes and the damping rate, or the pulsation frequency, of the stable modes as a function of the friction parameter and shielding length. For specific equations of state, we express the stability criterion in terms of cell density.

Keywords: Nonlinear mean field Fokker–Planck equations; Generalized thermodynamics; Chemotaxis; Gravity; Long-range interactions

1. Introduction

In biology, many microscopic organisms (bacteria, amoebae, endothelial cells,...) or even social insects (like ants) interact through the phenomenon of chemotaxis [1]. These organisms deposit a chemical (pheromone, smell, food,...) that has an attractive[1] effect on the organisms themselves. Therefore, in addition to their diffusive motion, they move preferentially along the gradient of concentration of the chemical they secrete (chemotactic flux). When chemotactic attraction prevails over diffusion, this process can lead to a "chemotactic collapse" (see Ref. [2] for a review) resulting in the aggregation of the organisms. In this way, some structures can form like clusters (clumps) or even network patterns (filaments). Therefore, the chemotactic interaction can explain several features of the morphogenesis

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[1] The case of repulsive chemotaxis due to a "poison" can also be of interest and will be considered in a future contribution.

0378-4371/$ - see front matter
doi:10.1016/j.physa.2008.02.025

of biological colonies. The chemotactic aggregation of biological populations is usually described in terms of the Keller–Segel model [3]. This is a parabolic model consisting of two coupled differential equations. The first equation is a drift-diffusion equation describing the evolution of the concentration of cells and the second equation is a reaction-diffusion equation with terms of source and degradation describing the evolution of the concentration of the secreted chemical. This model ignores inertial effects and assumes that the drift velocity of the organisms is directly induced by a chemotactic "force" proportional to the concentration gradient of the chemical. The Keller–Segel model can reproduce the formation of clusters (clumps) by chemotactic collapse [4–19]. This reflects experiments on bacteria like *Escherichia coli* or amoebae like *Dictyostelium discoïdeum* exhibiting pointwise concentration [3]. However, parabolic models fail to describe the formation of network patterns (filaments). These filaments are observed in the experiments of capillary blood vessels formation [20]. They correspond to the spontaneous self-organization of endothelial cells during vasculogenesis, a process occuring during embryologic development. In order to account for these structures, more general models of chemotaxis have been introduced [21–23]. They have the form of hydrodynamic (hyperbolic) models taking into account inertial effects. These models can reproduce the formation of filaments that are interpreted as the beginning of a vasculature. This phenomenon is responsible for angiogenesis, a major factor for the growth of tumors [24]. Interestingly, these filaments are analogous to the large-scale structures in the universe that are described by similar hydrodynamic equations [25,26].

Recently, we have introduced a general kinetic model of chemotactic aggregation based on generalized stochastic processes, non linear mean field Fokker–Planck equations and generalized thermodynamics [23]. From these kinetic equations, we have derived a hydrodynamic model of the form

$$\frac{\partial\rho}{\partial t}+\nabla\cdot(\rho\mathbf{u})=0, \tag{1}$$

$$\frac{\partial\mathbf{u}}{\partial t}+(\mathbf{u}\cdot\nabla)\mathbf{u}=-\frac{1}{\rho}\nabla p+\nabla c-\xi\mathbf{u}, \tag{2}$$

$$\frac{\partial c}{\partial t}=D_c\Delta c-kc+h\rho. \tag{3}$$

It involves a pressure force $-\nabla p$, where $p=p(\rho)$ is a barotropic equation of state that can take into account several effects like anomalous diffusion or the fact that the particles do not interpenetrate. It also involves a friction force $-\xi\mathbf{u}$ which measures the importance of inertial effects. For $\xi=0$, we recover the hyperbolic model introduced by Gamba et al. [21]. For $\xi\to+\infty$, we can neglect the inertial term in the momentum Eq. (2) leading to $\xi\mathbf{u}\simeq-\frac{1}{\rho}\nabla p+\nabla c$ (overdamped limit). Substituting this relation in the equation of continuity (1), we obtain the generalized Keller–Segel model

$$\frac{\partial\rho}{\partial t}=\nabla\cdot\left[\chi\,(\nabla p-\rho\nabla c)\right], \tag{4}$$

$$\frac{\partial c}{\partial t}=D_c\Delta c-kc+h\rho, \tag{5}$$

where $\chi=1/\xi$. Interestingly, this model of chemotaxis is similar to a model of self-gravitating Langevin particles [27] described by the damped Euler–Poisson system

$$\frac{\partial\rho}{\partial t}+\nabla\cdot(\rho\mathbf{u})=0, \tag{6}$$

$$\frac{\partial\mathbf{u}}{\partial t}+(\mathbf{u}\cdot\nabla)\mathbf{u}=-\frac{1}{\rho}\nabla p-\nabla\Phi-\xi\mathbf{u}, \tag{7}$$

$$\Delta\Phi=S_dG\rho. \tag{8}$$

For $\xi=0$, it reduces to the barotropic Euler–Poisson system [28] and for $\xi\to+\infty$, we obtain the generalized Smoluchowski–Poisson system

$$\frac{\partial\rho}{\partial t}=\nabla\cdot\left[\frac{1}{\xi}\,(\nabla p+\rho\nabla\Phi)\right], \tag{9}$$