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Modeling Interactions among Migration Growth and Pressure in Dynamics

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Abstract

Although the analysis of some of the biochemical aspects related to the signaling pathways involved in the spread of tumors has advanced notably in recent times, their feedback with the mechanical aspects is a crucial challenge for a global understanding of the problem. The aim of this paper is to try to illustrate the role and the interaction between some evolutionary processes (growth, pressure, homeostasis, elasticity, or dispersion by flux-saturated and porous media) that lead to collective cell dynamics and defines a propagation front that is in agreement with the experimental data. The treatment of these topics is approached mainly from the point of view of the modeling and the numerical approach of the resulting system of partial differential equations, which can be placed in the context of the Hele-Shaw-type models. This study proves that local growth terms related to homeostatic pressure give rise to retrograde diffusion phenomena, which compete against migration through flux-saturated dispersion terms.

Keywords: Cell motility; Flux-saturated; Hele-Shaw model; Mathematical modeling; Mechanical feedback; Numerical simulation; Porous media; Tumor dynamics

Introduction

This paper seeks to address the analysis of the interaction among pressure, growth and cell migration in avascular tumors. We propose a thermodynamically consistent approach that links the theory of finite growth with a novel migration term based on flow-saturated mechanisms that allows controlling the dispersion front of the tumor, both from a qualitative point of view defining the characteristics of the front, and quantitatively since the speed of the tumor can be regulated from experimental data.

According to data published, 19.3 million new cases were detected and 10.0 million deaths occurred in 2020. Due to the relevance of the issue together with the technical difficulties of obtaining data from in vivo experiments, mathematical modeling appears to be a useful tool to predict and anticipate tumor development. Some papers highlight the challenges surrounding the dynamics of cancer and review mathematical models that attempt to respond to these concerns. From one side, research has been focused, on a large degree, on models that control growth and treatments through biochemical interactions that identify morphogens and target genes involved in deregulation and growth associated with tumor processes. In addition, the processes of migration and growth entail a reorganization of the collective distribution of cells, even a substantial change in their form when, for example, the epithelial–mesenchymal transition occurs. These processes might produce a substantial change in the elastic properties and aggregation forces and, therefore, in the intracellular and extracellular pressure that affects the cell structure and the environment, both from the biomechanical and biochemical points of view.

In the last few decades, the biology–mechanics interaction has been confirmed as a key player in tumor growth. One of the first mechanical pieces of evidence was supported in 1997 when Helmlinger et al. reported measurements of adenocarcinoma spheroids embedded in agarose gels matrices with different concentrations (different levels of stress). They found that spheroids proliferation was inhibited according to increasing gel concentrations. Further experiments proved stress distribution also affected the shaping and patterning of tumor spheroids. Nonetheless, the inhibitory effect that pressure produces on spheroids has been demonstrated to be reversible, disappearing if stress differences vanish. This fact may be explained because pressure seems to cause a quiescent state of cells in which there is a balance between duplication and a certain degree of pressure until stress ceases.

Thus, there is clear evidence that cells sense and respond to mechanical forces which regulate biochemical cascade and tumor fate through mechanotransduction. Mathematical models need to consider both biochemical and biomechanics agents that interact in the processes of cellular rearrangement, growth and migration in order to be realistic and to have an accurate predictive character.

The relationship between mathematical models and biology is relatively recent, although prominent authors such as Fick, Fisher, Keller, Segel, and Murray have contributed to drawing attention to the mutual scientific interest for both sciences. This field opens new frontiers to research, particularly in a problem of social relevance such as cancer disease. The mathematical biomechanical approach emerged strongly in the mid-1990s by the name of finite strain theory to consider the growth-stress interplay. The latest studies have investigated how external pressure of the surrounding tissue could limit tumor evolution through an empirical law, a mechanical function that affects mobility and proliferation. Moreover, poroelasticity arose to explain in part the cooperation of two main regions of tumor tissue: the solid tumor and the extracellular matrix. This connection has been mainly modeled by Darcy's law, as proposed in, giving rise to Hele Shaw-type models.

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