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RESEARCH

On investigation of complexity in extracellular matrix-induced cancer dynamics under deterministic and stochastic framework

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Abstract Cancer attracts significant attention nowadays due to its rising global incidence, the complexity of its biological mechanisms, and the challenges it presents in treatment and prevention. In this context, a mathematical model provides a powerful tool for understanding this critical aspect of cancer biology. This paper introduces a novel, comprehensive mathematical model that looks into the intricate interactions between cancer cells, immune cells, cytokines, and the extracellular matrix. We derive biologically feasible equilibria and examine both local and global stability. Using Sotomayor's theorem, the occurrence of saddle-node bifurcation is established, and the Hopf bifurcation is thoroughly analyzed. We investigate codimension-2 bifurcations, such as the Bogdanov-Takens bifurcation, using normal form theory and the center manifold theorem. Furthermore, an uncertainty analysis utilizing Latin hypercube sampling is performed to evaluate the influence of parameter uncertainties on tumor growth, which is then followed by a sensitivity analysis. The model incorporates multiplicative white noise terms into the deterministic system to construct a stochastic framework. Then we identify the sufficient conditions for mean persistence and extinction for every variable. Finally, numerical simulations are conducted by adjusting the parameters to confirm the analytical findings, providing new perspectives on controlling tumor behavior.

Keywords Cancer · Extracellular matrix (ECM) · Bogdanov-Takens bifurcation · Codimension-2 bifurcations · Stochastic model · Extinction

1 Introduction

Cancer is a complex and multifaceted disease that has afflicted humanity for centuries. It manifests in numerous forms and impacts individuals worldwide, regardless of age, gender, or socio-economic status. In 2020, around 19.3 million people got diagnosed with cancer, and almost 10 million people died because of it worldwide [1]. The journey of cancer begins at the cellular level, where a normal cell, under certain conditions, loses control over its proliferation and surveillance mechanisms. This loss triggers a state of continuous division, resulting in abnormal growth-a process known as cell transformation. During this transformation, the cell acquires new genetic and phenotypic traits that disrupt its normal functionality. Crucial to this process are genetic and epigenetic modifications, which

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