MODELING CANCER ONSET AND DEVELOPMENT

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Abstract

Tumorigenesis has been described as a multistep process, where each step is associated with a genetic alteration, in the direction to progressively transform a normal cell and its descendants into a malignant tumor [1, 2]. In this work, it is proposed an ODE model for cancer onset and development, considering three cell populations: normal cells at a tissue; premalignant mutant cells, with proliferative advantage but high apoptosis rate; and tumor mutant cells, which, besides the proliferative advantage, have evaded apoptosis and are aggressive to tissue. The model assumes a Hill function to describe the transition from precancer population to cancer population. For boundary subsystems, all limit sets are globally characterized. The comparison of backward and forward bifurcations obtained indicate that aggressive tumors have an extra protection against elevation of apoptotic rate, being more resistant to chemotherapy. Mathematical analysis of full model dynamics was also treated in details and revealed the ocurrence of bistability and three-stability. The results implicate that the presence of aggressive tumor cells opens way to survival of less adapted cells, while, under other conditions, the mutation from premalignant cells to cancer cells can sustain the tumor permanence in a scenario at which the tumor would not survive without the premalignant cells. Finally, numerical simulations are performed with parameter values based on real data, and the necessary time to cancer reach detectable size from a single mutant cell is estimated in relation with some parameters.

References

[1] R.A. Weinberg *The biology of cancer*. Garland Science, New York (2013)

[2] D. Hanahan and R.A. Weinberg, *Hallmarks of cancer: the next generation*, Cell **144** no. 5 (2011), pp. 646-674.