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Senescent cells in growing tumors STEFANO ZAPPERI, National Research Council of Italy, IENI-CNR, Milano, Italy, CATERINA A.M. LA PORTA, Department of Biomolecular Science and Biotechnology, University of Milano, Italy, JAMES P. SETHNA, LASSP, Department of Physics, Cornell University, Ithaca NY
— Tumors are defined by their intense proliferation, but sometimes cancer cells turn senescent and stop replicating. In the stochastic cancer model in which all cells are tumorigenic, senescence is seen as the result of random mutatations, suggesting that it could represent a barrier to tumor growth. In the hierarchical cancer model a subset of the cells, the cancer stem cells, divide indefinitely while other cells eventually turn senescent. Here we formulate cancer growth in mathematical terms and obtain distinct predictions for the evolution of senescence in the two models. We perform experiments in human melanoma cells which confirm the predictions of the hierarchical model and show that senescence is a reversible process controlled by survivin. We conclude that enhancing senescence is unlikely to provide a useful therapeutic strategy to fight cancer, unless the cancer stem cells are specifically targeted.

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