MAR13-2012-020265

Abstract for an Invited Paper
for the MAR13 Meeting of
the American Physical Society

The interplay between invasion and proliferation in tumor cell navigation[1]

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Tumor cells can employ different cellular and molecular modes of invasion. The two main phenotypic mechanisms are: 1. *Amoeboid* (or "path finder") cells that can squeeze through small gaps in the ECM (extracellular matrix). 2. *Mesenchymal* (or "path generator") cells that are more rigid and can decompose the ECM to pass through. In addition there is interplay between energy directed to more rapid motility vs. energy used for proliferation. Understanding the relative contributions of these distinct mechanisms and the balance between motility and proliferation to the efficiency of metastatic cancer migration is fundamental to the therapeutic targeting of cancer. We present a conceptual and modeling framework for the analysis and assessment of the success rate, time-to-target, and survival probability of amoeboid vs. mesenchymal modes. Similarly, we contrast invasion with and without proliferation. We treat the complex ECM geometry as a maze and employ semi-realistic modeling of cell motility. Our approach includes metabolic and timing degrees of freedom. The theoretical studies were compared with experimental efforts of cell navigation in specially designed microfluidic devices.

[1]Center for Theoretical Biological Physics sponsored by the NSF (Grant PHY-0822283) Rice University, The Tauber Family Foundation and the Maguy-Glass Chair in Physics of Complex Systems at Tel Aviv University.

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