THE MICROMECHANICS AND PHYSICS OF CANCEROUS CELLS: WHAT ARE THE PHYSICAL HALLMARKS OF CANCER METASTASIS?

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The mainstream research in cancer biology focuses on the genetic and molecular signatures underlying the disease. An alternative and perhaps more physics-oriented approach is to study cancer from a perspective of an entire cell treated as a complex system characterized by a set of micromechanical properties and cell-motility characteristics. In my lecture, I will talk about both of these aspects. In the context of micromechanics, I will first describe a new cell-tomography technique which allows for the reconstruction of 3D shapes of cells subject to well-defined, geometrically regular microconfinements. The reconstructed shapes allow for the derivation of an analytical micromechanics model according to which cell's mechanical properties are determined by the deformability of the cell membrane and cortical actin and by the limited compressibility of the nucleus. This model does not involve any arbitrary parameters and is universal in the sense that it reproduces the shapes, volumes and surface areas of cells of different types under different experimental conditions. Unfortunately, analysis of cancerous vs. non-cancerous cells indicates little – if any – differences in their micromechanical properties. Thus, in search for the features distinguishing malignant from non-malignant cells the second part of the talk will focus on the ways in which cells move – again, over well defined microenvironments allowing for quantitative analyses. As I will show, non-cancerous and non-metastatic cells migrate in ways fundamentally different from metastatic cells. While non-metastatic cells are simple, diffusive random walkers, their non-metastatic variants employ space-searching strategies used by animal predators. In this spirit, metastatic "cellular predators" can be viewed as navigating optimally through human body in search for suitable conditions to thrive (as in the "seed and soil"). Importantly, in doing so they desynchronize their cytoskeletal dynamics and "lose" a characteristic time scale present in non-metastatic cells. These observations prompt a hypothesis that cancer metastasis entails "desynchronization" of a cytoskeletal "clock".

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