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OPEN COMPLEX DYNAMICS IN CELL MECHANOBIOLOGY AND THE PROBLEM
 OF CANCER

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Cells reveal the structural organization to promptly adapt their mechanobiological environment in realization of fundamental vital cellular functions. The mechanobiological properties of living cells are mediated by the cytoskeleton (CSK) representing a dynamic network of filamentous proteins composed of actin filaments, microtubules, and intermediate filaments. Plastic deformation as the unique mechanism of the defects induced momentum transfer and the structural memory provides specific CSK organization in mechanobiological environment. The cell plasticity can be considered as the leading mechanism providing the vital CSK properties, including the cell self-organization up to the cell division. The cell division being the vitality ground has also natural links to the defects behavior that provides the evolutionary controlled cell division (due to the preceded plasticity) or the fragility due to the pathological CSK changes leading to the cell fragmentation as the spontaneous division and proliferation. The duality of defects in the realization of the vital cellular functions (plasticity, damage, damage-failure transitions) is stimulating for the consideration of the CSK structure as out-of-equilibrium system with defects taking into account the fundamentals of defects in the matter properties as the localization of the symmetry groups [1].

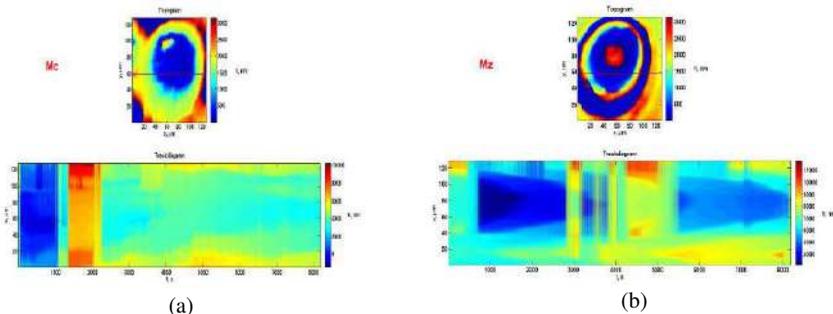


Fig. 1. Topogram (top) and the track diagram (bottom) of the HCT116 cancer cell (a) and the TTC healthy cell (b).

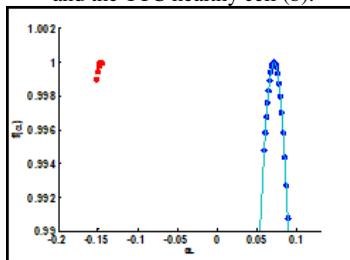


Fig. 2. Typical multifractal spectra of CPM data: “blue” is the normal breast cell, “red” is the cancer breast cell (carcinoma).

Multiscale mesoscopic approach in the simulations of biological systems with defects (biological molecules, cell and tissue) are analyzed in the mechanobiology statement to link the qualitative changes of behavior of mesoscopic systems as the specific type of criticality (structural-scaling transitions). The mechanobiology in the combination with molecular genetic approach could

Секция 1. Современные задачи и новые приложения физической мезомеханики материалов с иерархической структурой

provide the actual direction for objectification of pathological tendencies in the living cells in the case of cancer. The methodologies, numerical and experimental techniques coming from mechanobiological approach and combined with multi-scale signal processing are the ground of the advanced concepts of biological systems evolution related to the role of collective phenomena. "In-situ" study of the cell mechanobiology by the coherent phase microscopy (CPM) with the following definition of "meaningful" collective degrees of freedom allows the determination of dynamic stability of biological systems, including tissue, and they qualitative changes with "damage" accumulation with an application to cancer progression. Study of the nonlinear CSK dynamics was conducted analyzing the time series of phase thickness fluctuations after the Coherent Phase Microscopy (CPM) in the cell "cross-sections" containing the nucleus, the nucleolus, cytoplasm. High spatial and temporal resolution of the CPM pattern allowed the analysis of dynamic processes in living cells using CPM topograms and track diagrams in different cell cross-sections, Fig. 1. The application of the WTMM method allowed the demonstration of the links of temporal correlations of finite-amplitude phase thickness fluctuation, dynamics of collective modes of open complexes and qualitative different CSK dynamics that are characteristic for the cell plasticity and fragility. The phase thickness fluctuations display the fat-tail distributions, the log-normal and the power laws, with multi- and monofractal singularity spectrum. The multifractal singularity spectrum in the case of the cell plasticity reflects the temporal sequences of the phase thickness fluctuation in the presence of mentioned open complex "singular" collective modes (breathers, auto-solitary, blow-up). The monofractal singularity spectrum and the power law of the phase thickness fluctuation are the consequence of the shifting of the CSK dynamics into the area of the attractor with the blow-up open complex dynamics, that leads to the spontaneous CSK fragmentation (the cell fragility), Fig.2. The open complex dynamics, which follows to the structural-scaling transition, allows the interpretation of the normal and cancerous cell evolution scenario. The structural CSK susceptibility to both scenario is given by the values of structural-scaling parameter charactering the nonlinearity (metastability) of the epigenetic landscape and corresponding open complex kinetics. The pathological changes of the CSK structure in the presence of the "monofractal" blow-up open complex dynamics leads to the cancer progression [2].

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