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Editorial

Molecular and cellular mechanics

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Molecular and cellular mechanics is a fundamental field for understanding the physiology and pathology of biological systems at the nano- and microscale. It is widely recognized that cell behavior is highly dependent on its microenvironment, including the extracellular matrix and neighboring cells. Cell behaviors such as migration, differentiation, proliferation, and immunity can be regulated by modulation of both cell–matrix and cell–cell interactions. Quantitative studies of the biophysical mechanisms that drive cellular behavior will enhance our comprehension of disease pathogenesis and development, including tumors, cardiovascular diseases, and neurodegenerative diseases. The present special issue features a compilation of recent publications on molecular and cellular mechanics from diverse authors. We provide a synopsis of the contributions within this special issue.

Gu et al. [1] reviewed the fundamental features and operating mechanism of electroactive polymer-based actuators while highlighting their implementation in cell mechanical stimulation as soft actuators. They showed that the electroactive polymer-based actuators can serve as an active culture substrate for cells or tissues. With the application of electrical stimuli, these actuators can regulate the deformation modes of cells or tissues, including uniaxial/biaxial stretching, compression, periodic vibration, and cyclic attachment/detachment.

Yang and Jiang [2] studied the durotaxis and negative durotaxis behaviors of cell mobility using a unified theoretical framework: the persistent random walks model. Durotaxis refers to a type of cell migration where cells are guided by rigidity gradients and prefer to migrate up rigidity gradients, whereas negative durotaxis occurs when the cells move preferentially towards a softer matrix. The authors demonstrated that the dependence of cell motion persistence time on matrix rigidity following a biphasic pattern is adequate to produce both durotaxis and negative durotaxis, regardless of the correlation between substrate rigidity and migration speed. This study offered valuable insights into the underlying mechanisms governing the seemingly contradictory cell migrations responding to the matrix rigidity gradient.

Guan and colleagues [3] utilized a structural stiffness matrix-based model to study the linear and nonlinear mechanical responses of cell monolayers in conditions of crowding. The model depicted the cell monolayer as a series of polygons representing cells with strings and bars that mimicked the cell membrane and internal microtubules. The cells' shape and motion depend on the positional changes of the polygons' vertices and nucleus, which are determined by force balance resulting from the determination of the structural stiffness matrix. This study reveals that the epithelial monolayer demonstrates linear elastic behavior under mild crowding but exhibits nonlinear elastic behavior in cases of overcrowding. The deformation in the linear elastic regime arises primarily from cell orientation changes, whereas the nonlinear elastic behavior results from the contribution of microtubules.

Li et al. [4] studied the mechanical properties of epithelial monolayers using indentation experiments and theoretical models. They showed that Young's modulus of in vitro-cultured epithelial cell monolayers is significantly higher than that of its constituent cells in an isolated state by orders of magnitude. Additionally, the study revealed that Young's modulus of epithelial cell monolayers demonstrates nonlinear behavior depending on cell density. This study provides significant insights into the impact of cell density and intercellular interactions on the mechanical properties of cell monolayers.

Qin and colleagues [5] investigated how Nesprin-1/2 facilitates cellular migration in narrow environments by regulating nuclear deformation. As a family of nuclear envelope spectrin repeat proteins, Nesprin-1/2 is located primarily on the outer nuclear membrane and attaches to actin filaments. The authors found that Nesprin-1/2

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can regulate the expression of lamin A/C and that a loss of Nesprin-1/2 induces the ubiquitination-proteasome-dependent degradation of lamin A/C. Stable expression of Nesprin-1/2 preserves the accumulation of F-actin at the nucleus tail, thus facilitating the transmission of pushing forces and promoting cell migration through confined spaces.

Liang and colleagues [6] conducted a study on the proliferation of cancer cell clusters, which play a crucial role in tumor progression. The results showed that cells in the posterior cell zone of cell clusters had a significantly higher proliferation rate than those in the anterior cell zone. These observations are linked to the activation of the Ras-ERK1/2-YAP pathway. The study also revealed the significance of the cytoskeleton for cell proliferation, as the absence of myosin inhibited both cell proliferation and nuclear entry activation of ERK1/2-YAP. These findings advance our understanding of cancer cell cluster proliferation and help identify potential targets for anticancer drugs focusing on reducing cancer cell mobility.

Han and colleagues [7] developed a particle-based coarse-grained model of blood cells, including white blood cells, red blood cells, and platelets, to examine the effects of fibrinogen on blood rheology and red blood cell aggregation. The study showed that an elevation in fibrinogen concentration can cause a substantial rise in plasma viscosity. The simulations revealed how interactions among blood cells affect blood flow and how the properties of blood flow, such as blood velocity, influence the cell–cell interactions among blood cells. This study enhances our understanding of cell–cell interactions impacting microvascular blood flow. At the same time, it emphasizes the significance of fibrinogen-induced modifications in plasma viscosity alongside blood cell aggregation and adhesion as risk factors for microvascular complications in patients with COVID-19.

Wang and Li [8] conducted all-atom molecular dynamics simulations on the conformational transitions of full-length integrin incorporated in the membrane. The simulations involved attaching ligand fibronectin to the integrin head and applying a pulling force to the ligand. The results demonstrated that mechanical force can induce a conformational change in the integrin embedded in the membrane.

We hope that this issue can generate insightful discussions on molecular and cellular mechanics and inspire further studies in the field.

Data availability No data associated in the manuscript.

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