

Preface: molecular, cellular, and tissue mechanobiology

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Mechanics plays a crucial role in a diversity of biological processes at different length (from molecules, cells, tissues, organs to organisms) and time scales. As a rapidly growing field across the interfaces of mechanics, biology, and medical engineering, mechanobiology aims to identify the mechanical and biological responses of cells and tissues of mechanical factors (e.g., stress, strain, and substrate stiffness) and their underlying mechanisms at different scales; to correlate the physiological and pathological growth, adaptation, remodeling, and degradation of tissues and organs with their mechanical forces, properties, and environments; and to provide mechanics-based approaches and techniques for diagnosis and therapy. To this end, mechanobiology integrates the experimental and theoretical methods of biology and mechanics to investigate the physiological and pathological responses of biological molecules, cells, and tissues to mechanical factors at different levels. Mechanobiology not only provides insights into a wealth of biological processes at the molecular, cellular, and tissue levels but also cues to developing novel therapeutic strategies for diseases such as cancer.

In particular, cell mechanobiology (or cytomechanobiology) has attracted much theoretical and experimental efforts

devoted toward clarifying how cells sense their environment and adequately respond in terms of shape, migration, proliferation, differentiation, and survival. Cells can not only sense and respond to mechanical stresses but also regulate the mechanical properties of the surrounding extracellular matrix (ECM). For example, the integration of physical and biochemical factors within the microenvironment can modulate the differentiation of stem cells; naive mesenchymal stem cells can specify lineage and commit to specific phenotypes depending on the elastic modulus of ECM. Compressive stress can direct cancer cells toward an invasive phenotype and enable coordinated migration of these malignant cells.

This thematic issue of *Acta Mechanica Sinica* contains six invited articles in the field of mechanobiology. The editors hope that it will bring new interests and inspirations to the research of molecular, cellular, and tissue mechanobiology.

Cells use a dynamics actin network to drive their motility, polarization, and morphogenesis. Considering the molecular-level processes of actin polymerization, branching, capping, and depolymerization, Gong et al. [1] developed a computational framework to investigate the stochastic and cooperative behaviors of growing actin networks in overcoming obstacles. They discovered that the characteristic sizes of obstacles change the protrusion power per unit length but do not influence the orientation distribution of actin filaments in the growing network.

The regulated transport of molecules of different sizes across cell membranes usually involves the formation of nanopores. For example, Bu et al. [2] performed all-atom molecular dynamics and bias-exchange metadynamics simulations to reveal the molecular mechanisms of water pore formation. They showed that a water pore of a few nanometers in diameter can form on a cell membrane subjected to an electric field through which water molecules can pass through

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the membrane. They provided some atomic details of how an electric field influences the movement of water molecules and lipid head groups during water pore formation.

The force regulation and coordination of molecular motors are crucial for the elongation and contraction of skeletal muscles. Wei et al. [3] studied the role of adenosine triphosphate (ATP) in regulating the coordination among multiple molecular motors. A mechanics model of sarcomeres was used to show how the ATP concentration affects the motor force regulation during skeletal muscle contraction. They found that there could be negative cross-bridges resisting contraction within the sarcomere, and a higher ATP concentration might decrease the resistance force from negative cross-bridges by promoting their timely detachment.

Epithelial morphogenesis not only integrates many physiological activities such as cell proliferation and apoptosis, but also entails collective cell mechanics. So that Lin et al. [4] presented a dynamic cellular vertex model to study the morphomechanics of a growing epithelial monolayer. The feedback between cell proliferation and mechanical stresses was introduced into the model to elucidate how the feedback at the cellular level tailors the macroscopic features at the tissue level. They highlighted the critical role of mechanical factors in the growth and morphogenesis of epithelial tissues.

To date, it remains unclear how dental neurons sense hot and cold stimuli. To find out about this, Lin et al. [5] investigated the relationship between temperature distribution, tooth structure deformation, dentinal fluid flow, and the time course of dental neuron response under thermal stimulation. They used biophysical models to demonstrate that both hydrodynamic theory and neuron theory contribute to dental thermal pain sensations. Their results support a central role of mechanosensitive ion channels and the associated hydrodynamic hypothesis.

Osteoporosis is accompanied by the reduction of bone quantity and is a major cause of fractures in the spines of older persons. By applying the progressive loading technique, Li et al. [6] experimentally studied how osteoporosis induced by estrogen depletion influences the evolution of post-yield microdamage accumulation and plastic deformation in vertebral bodies. They found that osteoporosis could increase the vertebral fracture risk probably through microdamage accumulation and plastic deforming degradation.

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