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The nonequilibrium thermodynamics and kinetics of focal adhesion dynamics KRISHNA GARIKIPATI, JOSEPH OLBERDING, MICHAEL THOULESS, ELLEN ARRUDA, University of Michigan — We consider a focal adhesion (FA) to be made up of molecular complexes consisting of ligands, integrins, and associated plaque proteins. Free energy changes drive the binding and unbinding of these complexes, thus controlling the FA's dynamic modes of growth, treadmilling and resorption via the following mechanisms: (i) work done during the addition of molecular complexes, (ii) the chemical free energy of addition of a molecular complex, (iii) the elastic free energy of deformation of FAs and the cell membrane, and (iv) the work done on a molecular conformational change. We have developed a treatment of FA dynamics as a nonlinear rate process driven by out-of-equilibrium thermodynamic driving forces, and modulated by kinetics. The mechanisms governed by the above four effects allow FAs to exhibit a rich variety of behavior, predicting growth, treadmilling and resorption. Treadmilling requires symmetry breaking between the ends of the focal adhesion, and is achieved by driving force (i) above. In contrast, the remaining mechanisms cause symmetric growth or resorption. These findings hold for a range of conditions: temporally-constant force or stress, and for spatially-uniform and non-uniform stress distribution over the FA. This treatment of FA dynamics can be coupled with models of cytoskeleton dynamics and contribute to the understanding of cell motility.

Krishna Garikipati
University of Michigan

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