

Bleb nucleation through membrane peeling

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Blebs are cellular protrusions arising from a local detachment of the cell membrane from the underlying actomyosin cortex. We study the nucleation of blebs by means of a simple model in which membrane-cortex adhesion is mediated by elastic linker proteins with force-dependent binding kinetics [1]. The model shows that bleb nucleation is governed by membrane peeling, namely the fracture propagation process whereby adjacent linkers sequentially unbind. By this mechanism, the growth or shrinkage of a detached membrane patch is completely determined by the linker kinetics, regardless of the energetic cost of the local detachment. We predict the critical nucleation radius for membrane peeling and the corresponding effective energy barrier. These are typically smaller than those predicted by classical nucleation theory, implying a much faster nucleation. We also perform simulations of a continuum stochastic model of membrane-cortex adhesion to obtain the statistics of bleb nucleation times as a function of the pressure on the membrane. The determinant role of membrane peeling changes our understanding of bleb nucleation, opening new directions in the study of blebs.

[1] R. Alert and J. Casademunt, Phys. Rev. Lett. **116**, 068101 (2016)