Seminar/Talk

Rupture strength of living cell monolayers

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The ability of tissues to sustain mechanical stress and avoid rupture is a fundamental pillar of their function. Rupture in response to physiological levels of stress can be undesired, for example resulting from disease or genetic mutations, or be an integral part of developmental processes, such as during blastocoel formation in mouse or leg eversion in flies. Despite its importance, we know very little about rupture in cellularised tissues because it is a multi-scale phenomenon that necessitates comprehension of the interplay between mechanical forces and biological processes at the molecular and cellular scales. Using a combination of mechanical measurements, live imaging and computational modelling, we characterise rupture in epithelial monolayers. We show that, despite consisting of only a single layer of cells, monolayers can withstand surprisingly large deformations, often accommodating several-fold increases in their length before rupture. At large deformation, epithelia increase their stiffness multiple-fold in a process controlled by a supracellular network of keratin filaments. Perturbing keratin organisation fragilises monolayers and prevents strain stiffening. Using computational approaches, we show that, although the kinetics of adhesive bond rupture ultimately control tissue strength, tissue rheology and the history of deformation prior to failure set the strain and stress that the tissue reaches at the onset of fracture. Our data paint a picture of epithelia as versatile materials that combine resistance to shocks with deformability when subjected to low strain rates.

Thursday, January 16, 2025 11:00am - 12:00pm
Moonstone Bldg / Ground floor / Seminar Room F (I24.EG.030f)

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