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Cytoskeletal organization by motor and polymerization forces

GIJSJE KOENDERINK, FOM Institute AMOLF

Cells need to constantly change their change to perform vital functions, such as growth, division, and movement. Dysregulation of cell shape can have severe consequences such as cancer. Our goal is to resolve physical mechanisms that contribute to cell shape control. For this purpose, we study simplified experimental model systems reconstituted from purified cellular components. In this talk, I will give two examples of our recent work. The first example concerns active contractility of the actin cortex, which lies underneath the cell membrane and drives shape changes by means of myosin motors. Using in vitro models, we studied how myosin motors and actin filaments collectively self-organize into force-generating arrays. I will show that motors contract actin networks only above a sharp threshold in crosslink density. We discovered that right at this threshold, the motors rupture the network into clusters that exhibit a broad distribution of sizes, as expected in filamentous networks near a percolation threshold. The second example I will discuss concerns cell shape polarization directed by interactions between the actin and microtubule (MT) cytoskeletons. A prominent example is the guidance of MT growth along F-actin bundles towards specific targets, i.e. focal adhesions. It has been suggested that MT end-tracking proteins (+TIPs) that also bind F-actin are responsible for this process. We built an in vitro system involving a simplified actin-MT crosslinker molecule and could show that the interaction between MT ends and actin is sufficient to capture and re-direct MT growth along actin bundles. By keeping MT growth tightly coupled to F-actin, this mechanism allows linear arrays of actin bundles to act as templates for MT organization. Instead, when interacting with single actin filaments, MT ends become the dominant organizing factor, exerting forces that align, pull and even transport actin filaments in the direction of MT growth. We conclude that actin and MTs can influence each other's organization through coupling by +TIP proteins.