In This Issue

Actin branches out in yeast

Many of the same proteins control actin polymerization in budding yeast and mammalian cells. But budding yeast researchers—hampered by an emphasis on genetics and an inaccessible yeast cytoplasm dense with ribosomes and glycogen granules—have often lagged behind in providing structural descriptions based on electron microscopy (EM). This has fueled suspicions about whether the study of yeast actin is relevant to human systems.

Now, Young et al. (page 629) get the structural ball rolling with an EM characterization of yeast cortical actin patches, which are the most prominent actin structures in budding yeast. They find significant structural parallels between the yeast and mammalian systems, suggesting that the smaller yeast structures may be a handy model for tackling problems of actin dynamics.

The team partially purified patches from cells expressing GFP-labeled capping protein. Once cells were lysed, the researchers stabilized and cross-linked the actin patches and used correlated fluorescence and electron microscopy to visualize them.

Actin filaments were arrayed in a branched fashion, with the branch placement and angles characteristic of Arp2/3 complex-induced branching seen in mammalian cells. The authors conclude that a modified form of the dendritic nucleation model, which is used to explain actin dynamics at the leading edge of motile mammalian cells, does apply to yeast actin dynamics. The relevance of this model to yeast had been questioned because the concentration of actin protein in yeast appeared to be too low to support dendritic nucleation.

Yeast actin patches are known to move as they drive endocytosis. In the isolated patches, the branching was equal in all directions, but patches isolated directly from the cortex of living cells (both wild type and mutant) may reveal what process provides directionality.

Less kinesin, more condensation

Kinesin motors drag cargos, including chromosomes, but do not normally reshape those cargos. But on page 613, Mazumdar et al. demonstrate that a human chromokinesin HKIF4A is needed to establish the correct condensation state of chromosomes.

Chromokinesins are localized along chromosome arms and are thought, at least in some cases, to act as part of the polar wind: they walk along microtubules away from centrosomes, thus dragging their chromosome cargos toward the middle of the mitotic spindle. The authors depleted HKIF4A from human fibroblast cells using antibodies and RNAi. They observed numerous mitotic defects including misaligned chromosomes, incomplete chromosome separation during anaphase, and disorganized spindles. The resulting daughter cells had a high rate of aneuploidy.

When the authors visually examined the chromosomes in RNAi-treated cells, they saw that chromosomes were hypercondensed relative to those in control cells. This was not due to an extended mitosis, as chromosomes of cells just entering mitosis were also hypercondensed. Furthermore, they found that HKIF4A interacts with the condensin complexes responsible for chromosome condensation.

Cells lacking HKIF4A had a diffuse rather than the normal axial localization of some condensin complex proteins. Thus, HKIF4A, having localized to chromosomes so that it can perhaps act as part of a polar wind, may use that localization to recruit or otherwise organize the condensins so that they can do their job. The more provocative possibility is that the kinesin motor itself is used to power a condensation event—a possibility that can be tested by injecting a kinase-dead mutant. The group also hopes to understand whether the segregation errors are triggered by the condensation problems, lack of motor function, or both.