Actin senses self-incompatibility

Actin depolymerization is a death trigger for poppy pollen, according to Thomas et al. (page 221), that prevents self-fertilization. Some plants prefer not to be fertilized by pollen grains that are genetically similar to themselves. These plants express self-incompatibility (S) proteins. A match between the pistil’s S-proteins and the pollen’s S-allele triggers a rapid calcium influx, actin filament destruction, and pollen cell death.

Several years ago, the researchers noticed that the amount of actin depolymerization far exceeded that required to inhibit pollen tube growth. The persistent actin destabilization also resembled that associated with animal cells about to undergo programmed cell death (PCD).

The authors now find that blocking actin depolymerization prevents pollen cell death in self-incompatible plants. Inducing actin depolymerization, on the other hand, activated pollen cell death. The dying cells showed the same pattern of DNA fragmentation and caspase-like activity as did pollen killed by self-incompatibility.

Changes in actin dynamics have been linked to PCD in animal and yeast cells, and a variety of noxious external stimuli triggers actin rearrangement in plants. Actin may thus serve as a sensor for extreme environmental stress to initiate PCD or other changes in cell behavior. As even a short exposure to depolymerizing drugs killed pollen cells, a brief change in actin dynamics seems to be all that is needed to kick off signaling cascades. JCB

Funky FAK proliferation

Focal adhesion kinase (FAK) transduces adhesion forces into pro-proliferation signals in a variety of cell types. Now, Pirone et al. (page 277) show that FAK also inhibits proliferation when cells lack adequate attachments. The two-sided modulation of proliferation by FAK suggests that adhesion is not a simple on–off switch. Rather, the cell monitors and modulates adhesion in a more graded fashion.

Current dogma suggests that more adhesion leads to more FAK activation and thus proliferation, and that loss of FAK activity would prevent proliferation. But Pirone et al. found that cells lacking FAK or expressing a dominant-negative form of the protein (FRNK) proliferated constitutively, whether they were cultured in high or low adhesive conditions.

Further analysis showed that FAK-null cells or those expressing FRNK had higher levels of RhoA expression than control cells. The increased RhoA led to more cytoskeletal tension and the formation of additional focal adhesions. Wild-type FAK thus seems able not only to sense adhesive forces, but also to limit their generation somehow.

A kinase-dead mutant restored normal proliferation responses to adhesion in FAK-null cells. Thus, unlike its proliferation-inducing function, FAK’s ability to inhibit cell division does not require its kinase activity. JCB

Linking secretion and cell cycle

The end of mitosis triggers the export of the Chs2p chitin synthase from the ER to the bud neck, report Zhang et al. on page 207. The Chs2p chitin synthase lays down the primary septum, which divides mother and daughter yeast cells. Previous work showed that Chs2p arrives at the neck in late telophase, but what controls the timing was unknown.

Zhang et al. found that the timing of Chs2p localization to the neck correlated with the destruction of the mitotic kinase, Clb2p. Mutations in the mitotic exit network, which normally destroys Clb2p, prevented chitin synthase localization, though the myosin that constricts the neck localized normally. Direct inactivation of Clb2p restored chitin synthase localization, suggesting that the loss of mitotic kinase activity activated the synthase’s movement.

The chitin synthase was restricted to the ER during metaphase when Clb2p activity was at its peak, but premature destruction of Clb2p triggered synthase movement to the neck. This relocation required the secretory pathway. The transport of other secretory pathway cargos was not affected by Clb2p activity. The destruction of the mitotic kinase thus specifically ensures that the septum forms at the end of mitosis when cell separation is necessary. Zhang et al. are now looking for the mechanism that links mitosis and secretion. JCB