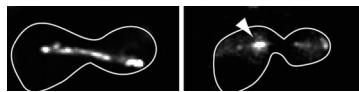


Passengers operate the brakes



Compared to control cells (left), yeast lacking the CPC protein Sli15 (right) accumulate more of the kinesin Cin8 at their spindle midzones.

Members of the chromosome passenger complex (CPC) regulate kinesin motors to control how fast the mitotic spindle elongates, [Rozelle et al.](#) report.

The CPC consists of the mitotic kinase Aurora B and its associated regulatory proteins. The complex localizes to kinetochores at the beginning of mitosis and regulates their attachment to the mitotic spindle. The CPC moves to the central spindle in anaphase, but the complex's precise function at this stage of mitosis remains unclear.

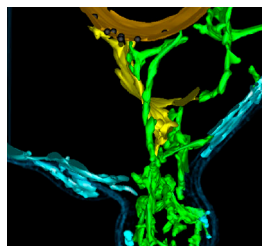
Rozelle et al. examined the anaphase spindles of budding yeast lacking various components of the CPC. Although spindles grew normally in cells lacking Aurora B itself, spindle poles moved apart more slowly in the absence of the regulatory passenger proteins

Sli15, Bir1, and CBF3. Spindle microtubules are slid apart by the kinesins Cin8 and Kip1, yet deleting either of these motor proteins accelerated spindle elongation in CPC-mutant yeast, indicating that they can also act as brakes to limit microtubule movements. The CPC may switch the motors' activities by rearranging them on the anaphase spindle—more kinesin accumulated at the spindle midzone in the absence of the CPC regulatory subunits—or by influencing their posttranslational modification.

CPC mutants took longer to complete mitosis because their slowly elongating spindles activated a signaling pathway called the mitotic exit network, which maintains cells in anaphase. Senior author Kenneth Kaplan now wants to investigate how slow-growing spindles activate this pathway and to determine whether the CPC modulates spindle length in order to ensure that chromosome segregation is complete before the cell divides in two.

Rozelle, D.K., et al. 2011. *J. Cell Biol.* doi:[10.1083/jcb.201011002](#).

How the ER shapes up and ships out



Peripheral ER structures include a central cisterna (yellow), tubules (green), and a plasma membrane-associated network (blue).

West et al. reveal the diversity of endoplasmic reticulum (ER) structures present in yeast and describe how these structures pass from the mother cell into the bud.

The peripheral ER is a continuous network of membrane tubules and cisternae that extends from the nuclear envelope. West et al. looked at ER structure using electron tomography to reconstruct the organelle's three-dimensional organization.

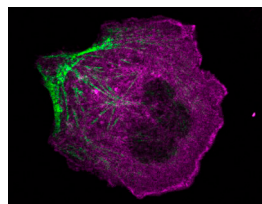
The researchers saw three types of ER. ER tubules branched from the nuclear envelope throughout the cytoplasm, connecting distinct ER domains and contacting other organelles. A single central

cisterna stretched from the nuclear envelope toward the developing bud. The rest of the ER was tightly associated with the plasma membrane as a network of both tubules and cisternae. Plasma membrane-associated ER was unable to enter the bud directly. Instead, the central cisternal and tubular ER domains extended into the bud and then spread out to re-associate with the daughter cell cortex.

ER membranes are partly shaped by two protein families called reticulons and Yop1. Yeast lacking these proteins lost all curved ER structures, with most of the organelle forming a single flat cisterna close to the plasma membrane. Yet curved ER tubules were still pulled into mutant buds before flattening back out, indicating that reticulons and Yop1 are required to maintain, but not generate, ER membrane curvature. Author Gia Voeltz now wants to investigate the ER's contacts with other organelles, particularly its extensive association with the plasma membrane.

West, M., et al. 2011. *J. Cell Biol.* doi:[10.1083/jcb.201011039](#).

Myosin II gets polarity back to front



The Rac GEF βPIX (magenta) mainly localizes to the cell front but is excluded from adhesions associated with actomyosin bundles (green) at the rear.

Vicente-Manzanares et al. describe how myosin II isoforms ensure that a migrating cell knows its back from its front.

The leading edge of a migrating fibroblast is the site of rapid actin polymerization and adhesion turnover, whereas the rear of the cell contains stable actin bundles coupled to large adhesions. This polarized arrangement is controlled by myosin II, although how this motor protein and actin-crosslinker generates front-back asymmetry is unclear.

Vicente-Manzanares et al. found that one particular myosin II isoform—MIIA—initially specifies the cell rear by assembling actin clusters that the researchers called “proto-bundles.” But a different isoform—MIIB—was required to stabilize and

enlarge these clusters into the actomyosin bundles typically found at the back of migrating fibroblasts. Microtubules weren't needed to form these bundles, but the leading edge was no longer positioned directly opposite the cell rear in the presence of the microtubule-depolymerizing drug nocodazole.

Cell-matrix adhesions associated with MIIB-dependent actin bundles were larger and more stable than adhesions at the cell front, but their adhesive signaling was suppressed. Key adaptor proteins like paxillin and p130(Cas) were largely unphosphorylated at rearward adhesions, and proteins that stimulate the small GTPase Rac weren't recruited as strongly, limiting actin polymerization and membrane protrusion at the cell rear. Rac was activated by adhesions at the cell front, however, in order to stimulate leading edge protrusion.

Author Miguel Vicente-Manzanares now wants to investigate how myosin II controls adhesive signaling. One explanation may be that it regulates the tension that adhesions are subjected to.

Vicente-Manzanares, M., et al. 2011. *J. Cell Biol.* doi:[10.1083/jcb.201012159](#).