MEMBRANE TRAFFICKING

# Building sites

In most eukaryotic cells, coatomer protein (COP)II-coated vesicles, which carry cargo from the endoplasmic reticulum (ER) to the Golgi, bud from discrete sites called transitional ER (tER) sites. Although the existence of these sites has been documented for some time, how they are generated and maintained has remained elusive. However, work from Ben Glick and colleagues in the yeast *Pichia pastoris* has begun to yield some insights.

Unlike its better-studied relative *Saccharomyces cerevisiae*, which lacks tER sites, *P. pastoris* is similar to mammalian cells in that it has well-organized tER structures from which COPII vesicles bud. It has therefore been a useful model organism in which to study tER organization. Previous work from this group had indicated that tER sites assemble by self-organization and, to understand this process better, the authors focused on Sec12.

Sec12 is a tER-localized protein and it is the guanine nucleotide-exchange factor for the GTPase Sar1, which is required to initiate the budding of COPII-coated vesicles. They found that Sec12-containing tER sites are stable entities, but that individual molecules of Sec12 in the tER sites rapidly exchange with Sec12 in other ER regions. In addition, they showed that the association of Sec12 with tER sites is saturable, which indicates that, rather than being targeted by self-association, Sec12 localizes to these sites through an as-yet-unidentified determinant.

In *S. cerevisiae*, the Sec12 protein localizes throughout the ER and *S. cerevisiae* Sec12 is similarly localized when it is expressed in *P. pastoris*. The authors could therefore identify the targeting determinant for tER sites in *P. pastoris* Sec12 by doing a series of domain-swap experiments using Sec12 from the two yeast species. These experiments showed that both the cytosolic and lumenal domains of *P. pastoris* Sec12 are important for its tER localization. In a key experiment to test whether Sec12 localization determines the site of COPII-vesicle budding, the authors found that delocalized Sec12 affected neither the geography of vesicle budding nor the integrity of tER sites. In other words, Sec12 does not have to be at tER sites to catalyse vesicle budding. This result is consistent with what has been noted for mammalian Sec12, which is also not concentrated at tER sites.

Nonetheless, these data support the idea that a Sec12-independent scaffold exists at tER sites. Future work will be necessary to identify components of this scaffold, which the authors speculate could include proteins that target Sec12, as well as the peripheral ER membrane protein Sec16, to tER sites. A molecular definition of tER sites will also help determine whether these sites are crucial for regulating the spatially restricted budding of COPII vesicles.

*Sowmya Swaminathan*
*Associate Editor*, Nature Cell Biology

**References and links**

**ORIGINAL RESEARCH PAPER** Soderholm, J. *et al.* The transitional ER localization mechanism of *Pichia pastoris* Sec12. *Dev. Cell* **6**, 649–659 (2004)

**FURTHER READING** LaPointe, P., Gurkan, C. & Balch, W. E. Mise en place — this bud's for the Golgi. *Mol. Cell* **14**, 413–414 (2004)

**WEB SITE**
**Benjamin Glick's laboratory:**
http://mgcb.bsd.uchicago.edu/index3.html?content=faculty/bGlick/index.html

# IN BRIEF

CELL CYCLE

### The *Caenorhabditis elegans* centrosomal protein SPD-2 is required for both pericentriolar material recruitment and centriole duplication.

Pelletier, L. *et al. Curr. Biol.* **14**, 863–873 (2004)

The authors identified SPD-2, a component of the *Caenorhabditis elegans* centrosome (the main site of microtubule nucleation in animal cells). SPD-2 localized to the centrioles (of which there are two per centrosome) and accumulated in the pericentriolar material (PCM) during mitosis. This pattern coincided with that of SPD-5, which was shown to be required for SPD-2 accumulation and SPD-2-mediated PCM recruitment. SPD-5, however, was dispensable for the role of SPD-2 in centriole duplication.

GENE TRANSCRIPTION

### Genome-wide localization of the nuclear transport machinery couples transcriptional status and nuclear organization.

Casolari, J. M. *et al. Cell* **117**, 427–439 (2004)

The authors found that, in budding yeast, most nuclear transport factors and nuclear-pore-complex (NPC)-associated proteins bound preferentially to transcriptionally active genes, the majority of which contain sites that bind the transcriptional regulator Rap1. By contrast, the Ran guanine-nucleotide exchange factor Prp20 bound inactive genes. Transcriptional activation caused genes to relocate to the NPC and dissociate from Prp20.

TECHNIQUES

### Homogeneous detection of unamplified genomic DNA sequences based on colorimetric scatter of gold nanoparticle probes.

Storhoff, J. J. *et al. Nature Biotech.* 30 May 2004 (doi:10.1038/nbt977)

Storhoff *et al.* described a rapid colorimetric method that detects zeptomole quantities of nucleic acids without the need for target amplification. Gold-particle-labelled DNA probes recognize their target in solution and their nanoparticle scatter is detected by spotting the solution onto a glass microscope slide and exciting it with white light in the plane of the slide. Complexed probes scatter yellow or orange light, whereas free probes scatter green light.

MORPHOGENESIS

### Myosin-dependent junction remodelling controls planar cell intercalation and axis elongation.

Bertet, C., Sulak, L. & Lecuit, T. *Nature* **429**, 667–671 (2004)

A simple mechanism that enables cells to intercalate in the plane of the epithelium during germ-band elongation in *Drosophila melanogaster*, despite the presence of adherens cell–cell junctions, is presented. The authors found that myosin II localized in a planar polarized manner at cell–cell junctions, and that this enabled the junctions to disassemble and be remodelled, and cells to intercalate.