# In This Issue

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## Proteins keep Cdc55 in its place

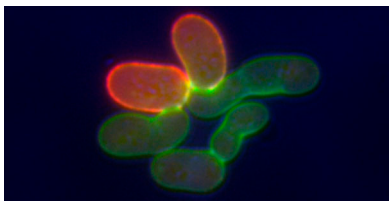


IMAGE COURTESY OF VALENTINA ROSSIO

**Yeast buds lacking Zds1 and Zds2 (green) are abnormally elongated because they delay mitosis.**

Rossio and Yoshida reveal how cells control the location of the mitosis-regulating protein Cdc55.

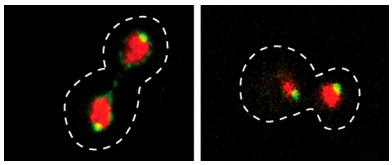
Cdc55 is a key component of the protein phosphatase 2A (PP2A) complex, which helps determine when cells begin and end mitosis. Two other proteins, Zds1 and Zds2, keep the complex under control. They switch on PP2A to nudge the cell into mitosis and switch it off to spur the cell to exit mitosis. Rossio and Yoshida wanted to investigate how this regulation occurs.

The researchers tracked the locations of Zds1, Zds2, and Cdc55 in budding yeast cells. Zds1 and Zds2 stayed exclusively in the cytoplasm, whereas Cdc55 could also enter the nucleus. To determine whether Cdc55's location altered its effect on mitosis, Rossio and Yoshida created mutant versions of the protein that remained either in the nucleus or the cytoplasm. Cells that harbored the cytoplasm-only Cdc55 form started mitosis normally, but yeast that made the exclusively nuclear version procrastinated, delaying the beginning of mitosis.

In the absence of Zds1 and Zds2, Cdc55 built up in the nucleus, where it prolonged mitosis by hindering the release of Cdc14, which normally helps push the cell into G1. The researchers think that Zds1 and Zds2 play a dual role. They trap Cdc55 in the cytoplasm, where it can prompt the cell to initiate mitosis. And because the proteins prevent Cdc55 from moving on to the nucleus, they allow the cell to exit mitosis.

Rossio, V., and S. Yoshida. 2011. *J. Cell Biol.* doi:10.1083/jcb.201101134.

## A histone finds a traveling companion



**Mps3 (green) normally localizes to the INM [left, circular pattern around the DNA (red)], but mutant Mps3 stays away (right).**

The H2A.Z histone has a second job, Gardner et al. have discovered. It escorts an inner nuclear membrane (INM) protein into the nucleus.

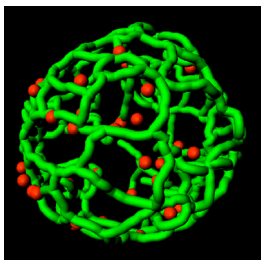
The yeast INM protein Mps3 belongs to a protein family, the SUN proteins, that positions chromatin within the nucleus during mitosis and meiosis. Mps3 helps maneuver telomeres and DNA segments containing double-stranded breaks close to the INM. But how does Mps3 get to the INM? Researchers had assumed that newly synthesized Mps3 diffuses from the ER into the INM. If so, the protein could also diffuse back out, suggesting that it needs a mechanism to keep it in place. Gardner et al. investigated whether the H2A.Z histone, which binds to Mps3, serves as a tether for the protein.

The team discovered that Mps3 couldn't reach the INM in cells where H2A.Z was absent, suggesting that H2A.Z shepherds Mps3 into the nucleus. Several lines of evidence indicated that H2A.Z performs this task independently of its chromatin-based functions. For example, chromatin-free H2A.Z can still link up with Mps3. On the other hand, Gardner et al. found that, in the presence of a mutant Mps3 unable to bind to H2A.Z, the histone was still incorporated into chromatin and continued to function in transcription, DNA damage repair, and chromosome organization.

Gardner et al. suspect that the two proteins meet in the cytoplasm and travel to the nucleus together, although whether H2A.Z tethers Mps3 in the INM is unclear. The researchers say that histones in other organisms may also serve as INM protein escorts.

Gardner, J.M., et al. 2011. *J. Cell Biol.* doi:10.1083/jcb.201011017.

## Plant virus gets the bends



**This 3D reconstruction of a yeast cell's cortical ER network (green) shows where TGBp3 (red) localizes.**

A protein that targets to curved sections of the ER enables a plant virus to spread between cells, Wu et al. show.

Animal viruses typically ride from cell to cell inside vesicles, but plant viruses spread via plasmodesmata, passageways that connect adjacent cells. Cortical ER tubules pass through the plasmodesmata, serving as highways for infection. Plant viruses often rely on so-called movement proteins to help them get around. In the group of viruses known as Potexviruses, the movement protein TGBp3 clusters with another movement protein, TGBp2, in the curved ER tubules near plasmodesmata. How the proteins home in on this location and whether their presence in the tubules is essential for infection wasn't clear.

Wu et al. tracked TGBp3 in yeast cells, which have a similar ER network as plants but lack plasmodesmata. They found that TGBp3 sports a sorting signal, a sequence of amino acids that directs the protein to the curved ER tubules. The sorting signal helps the virus spread: plants infected by viruses with mutations in the sequence stayed healthy or suffered only mild symptoms.

The researchers also found that the virus' third movement protein and its capsid, or protective protein coat, hook up with TGBp2. The virus' RNA might also connect to the TGBp3–TGBp2 complex. Curved ER tubules could be a staging point for the virus, where all of the viral components convene before moving on to the next cell. Researchers now need to determine how the virus triggers plasmodesmata to open up and let it through.

Wu, C.-H., et al. 2011. *J. Cell Biol.* doi:10.1083/jcb.201006023.

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