Motherly loss ends cell cycle

To prevent chromosome segregation errors, cytokinesis in *Saccharomyces cerevisiae* awaits the arrival of a spindle pole body (SPB) in the bud. On page 335, Fraschini et al. show that this arrival is announced by the mother-bound SPB, which heralds the onset of cytokinesis and mitotic exit.

Mitotic exit relies on a G-protein called Tem1, which activates the destruction of mitotic cyclins.

As Tem1 is activated by Lte1, which is found in the bud, Tem1’s arrival in the bud on the SPB was thought to trigger timely mitotic exit. But another mechanism for Tem1 activation is revealed in the new study, thus partly explaining why Lte1 is not essential. Whereas Lte1 activates Tem1, a SPB-localized complex of Bub2 and Bfa1 is known to keep Tem1 inactive during spindle assembly and orientation. Timing of Bub2/Bfa1 loss from the mother-bound SPB coincides with mitotic exit and is prevented by activation of the spindle position checkpoint. The authors find that a mutant Bub2 that remains on the mother-bound SPB during telophase inhibits mitotic exit in some cells. The specific loss of Bub2 from the mother-bound pole thus contributes to mitotic exit.

Bub2’s GTPase-activating protein (GAP) activity was needed for its removal from the mother-bound SPB. Septin and kinases at the bud neck were also necessary, suggesting that the passage of one SPB through the bud neck creates a diffusible signal that travels to the mother SPB to activate the Bub2 GAP.

The authors imagine that microtubule motors or binding proteins might transmit a Bub2-activating signal along the spindle and are now testing this idea. They have already shown that loss of the microtubule plus-end binding protein Bim1 slightly decreases Bub2 loss from the mother-bound SPB, but other proteins must contribute.

Although required for correct localization, the GAP activity of Bub2 is not necessary to inactivate Tem1 in vitro. The authors feel that Bfa1 might do the actual Tem1 inactivation, whereas Bub2 recruits Bfa1 to the SPB. *JCB*

Dividing to their own beat

Multiple fungal nuclei within a common cytoplasm divide independently, based on findings on page 347. Gladfelter et al. suggest that the *Ashbya gossypii* filamentous fungus evolved a new means to control cyclin activity that might make asynchronous nuclear division possible.

Cyclin activity drives the mitotic cycle in eukaryotes. To prevent untimely mitosis, most cells degrade mitotic cyclins at telophase and show an oscillating pattern of cyclin protein levels. As cyclins can enter and exit nuclei and diffuse throughout the cytoplasm, multinucleated cells normally have synchronous nuclear divisions.

But the new report shows that *A. gossypii* nuclei divide independently of their neighbors. Nuclei at various stages of the cell cycle were found within single cells. Artificially synchronized nuclei did not remain in sync for long, suggesting that each nucleus divides on its own time frame. Such autonomy might allow the cell to grow in some spots (perhaps where nutrients are richest) without burdening the other nuclei.

This unusual nuclear independence might be possible, according to the authors, because *A. gossypii* does not rely on oscillations in cyclin levels to control mitosis. G1 and mitotic cyclins were present at every nucleus independent of its division status. And the loss of proteosome-mediated cyclin degradation did not interfere with cell cycle progression, as it does in other cells.

The group has not proven the mechanistic basis of asynchrony, but they propose that cyclin activity might be controlled by Sic1, which in budding yeast is a CDK–cyclin inhibitor. Budding yeast cells normally degrade Sic1 to escape G1, but the filamentous fungus instead seems to regulate its localization. Before division, Sic1 was found throughout the nucleoplasm. At times when cyclin activity should be highest, Sic1 was concentrated at spindle pole bodies, perhaps thereby turning it off.

Most cells have intricate and redundant mechanisms that assure precise cell cycle progression. If indeed cyclin oscillation has been lost as one such means, *A. gossypii* might have traded nuclear accuracy for autonomy. Its abundant nuclei, however, may protect the fungus from any resulting sloppiness. *JCB*