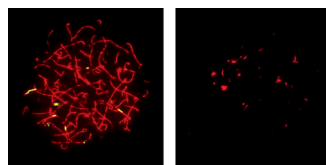


Cohesin sticks to it during meiosis



Homologous chromosomes synapse in normal mouse spermatocytes (left) but not in spermatocytes lacking cohesin components REC8 and RAD21L (right).

Cohesin doesn't shirk its duties during mammalian meiosis after all. Llano et al. reveal that the protein complex forms part of a structure that enables homologous chromosomes to pair up.

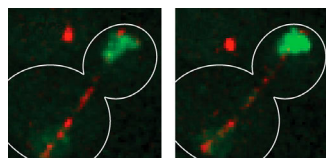
During mitosis, the cohesin complex holds sister chromatids together with a ring-like structure. During meiosis in

yeast, cohesin has an additional function in forming axial elements, adhesive strips that help homologous chromosomes line up opposite one another. This juxtaposition enables homologues to swap genetic material through crossing over and to repair damaged DNA through homologous recombination. Yeast lacking one meiosis-specific component of cohesin, the kleisin REC8, can't

construct axial elements. But what happens in mammals is unclear, probably because our genomes have a gene for a second meiotic kleisin. Mouse spermatocytes lacking either REC8 or the other kleisin, RAD21L, can generate axial elements, suggesting that cohesin isn't necessary for their construction.

To clarify cohesin's job in mammalian meiosis, Llano et al. crossed mice to produce animals that were missing REC8 and RAD21L. The mice appeared healthy. However, they were sterile, and their cohesin complexes were faulty, lacking two other components besides REC8 and RAD21L. Spermatocytes from the animals were missing axial elements, and their homologous chromosomes didn't pair up at all. Although cells from these mice formed the double-strand breaks that permit DNA repair, they couldn't mend these breaks because a key repair enzyme, RAD51, didn't arrive at the chromosomes. Overall, the researchers say, the results suggest that cohesin is necessary for axial element construction in mammals. Llano, E., et al. 2012. *J. Cell Biol.* <http://dx.doi.org/10.1083/jcb.201201100>.

Why yeast mothers and daughters don't share



As anaphase proceeds (left to right), Ace2 (green) remains in the bud.

Ace2 helps a newly formed yeast bud establish its identity. Boettcher et al. reveal what prevents this protein from leaking into the mother cell while it's still attached to the bud.

Like a teenager, a yeast bud has to distinguish itself from its parent. The transcription factor Ace2 helps drive this process by flipping on a set of genes only in the bud. During anaphase, Ace2 slips from the cytoplasm into the nucleus of the dividing cell, but it only lingers in the portion of the nucleus that's inside the bud. Why Ace2 doesn't diffuse into the mother's side of the nucleus is unclear.

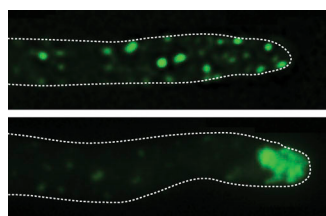
One possible explanation for the asymmetry in the nucleus is that Ace2 clings to chromosomes on the bud side, but

Boettcher et al. ruled out this option using photobleaching experiments. The team then traced a nuclear protein called TetR that can cross the bridge that connects the parting mother and daughter nuclei, testing the effects of mutations that alter the width and length of this bridge. They found that the bridge's dimensions were crucial to the amount of exchange. For example, a mutation that shortened the bridge allowed more TetR to pass into the mother's nucleus. Experiments with Ace2 showed a similar relationship between bridge size and the amount of nuclear "leakage."

The researchers' computer models suggest that there doesn't need to be a diffusion barrier inside the bridge to explain their results. Rather, the bridge's long, narrow shape curbs movement to the other side, confining Ace2 to the bud.

Boettcher, B., et al. 2012. *J. Cell Biol.* <http://dx.doi.org/10.1083/jcb.201112117>.

Lis1 cuts its work short



Endosomes (green) move in normal fungi (top) but stay put if Lis1 is absent (bottom).

Egan et al. clarify the role of the dynein co-factor Lis1 in cargo transport.

The molecular motor dynein ferries cargoes toward the minus ends of microtubules, typically toward the nucleus. The role of Lis1 has been controversial. Although researchers agree that Lis1 helps dynein position nuclei and centrosomes within the cell, they disagree about whether Lis1 aids dynein in other situations and how it influences the motor protein. In particular, whether Lis1 continues to interact with dynein once the cargo has started rolling is uncertain.

Egan et al. took a closer look at Lis1's actions by tracking the movement of fluorescently labeled cargoes in the long hyphae of

the fungus *Aspergillus nidulans*. They found that Lis1 was essential for relocating not only nuclei, but also endosomes and peroxisomes. That Lis1 takes part in shipping such diverse cargoes raises the possibility that it participates in transportation of all dynein cargoes, the researchers say.

In fungi lacking Lis1, dynein failed to associate with endosomes and peroxisomes, which therefore mostly stalled and amassed at the tips of hyphae. But the team noticed that the few endosomes that got moving traveled at a normal pace, suggesting that Lis1's role involves starting rather than sustaining movement. In normal cells, more than 90% of moving endosomes were attached to dynein, but less than 2% of them were associated with Lis1. The results support the notion that Lis1's job entails loading cargo onto dynein rather than transporting it.

Egan, M.J., et al. 2012. *J. Cell Biol.* <http://dx.doi.org/10.1083/jcb.201112101>.