Tangled DNA tightens chromosomes

ramming your vacation wardrobe into your luggage is a breeze compared with the packing job cells perform. Before they can divide, they have to scrunch long DNA molecules into tiny chromosomes. The tangles that form in DNA molecules help this chromosome compression, Kawamura et al. report. The team studies newt chromosomes—they are large and convenient to observe—that are packing masters. Jammed into a 10-micron-long chromosome is a meter-long DNA molecule. Proteins such as condensins bunch up the DNA. They aren’t the whole story, however, since protein-degrading enzymes don’t spur chromosomes to completely unravel. The DNA itself is tangled, and these knots might also help the chromosome stay taut, Kawamura et al. hypothesized.

SENP6 cuts SUMOs down to size

Kinetochore formation involves some SUMO wrestling. Two opposing proteins involved in the SUMO pathway control assembly of the structures, Mukhopadhyay et al. reveal.

Mitosis stalled in cells lacking SENP6, and chromosomes often didn’t line up properly during metaphase. Those defects also appear in cells that are missing the CENP-H/I/K complex, which helps insert other molecules into the forming kinetochore. When SENP6 was absent, CENP-H and CENP-I vanished from the inner part of the kinetochores. So did other proteins that the CENP-H/I/K complex helps put in place, such as CENP-O.

Further experiments suggested that the missing CENP-I had been destroyed. Removing RNF4 or disabling the proteasome, which demolishes ubiquitin-tagged molecules, caused CENP-I levels to return to normal. The overall picture is that SENP6 permits kinetochore assembly by preventing RNF4 from ubiquitinating CENP-I. Why cells adopt this indirect mechanism to control ubiquitination isn’t clear. CENP-I might require SUMOylation to do its job, the researchers speculate.

Neighbors limit cell renovations

No cell is an island, especially when it’s embedded in an epithelial layer of a fruit fly embryo. As Martin et al. reveal, forces transmitted among surrounding cells restrict how the cell can change shape during development.

Mitosis stalled in cells lacking SENP6, and chromosomes often didn’t line up properly during metaphase. Those defects also appear in cells that are missing the CENP-H/I/K complex, which helps insert other molecules into the forming kinetochore. When SENP6 was absent, CENP-H and CENP-I vanished from the inner part of the kinetochores. So did other proteins that the CENP-H/I/K complex helps put in place, such as CENP-O.

Further experiments suggested that the missing CENP-I had been destroyed. Removing RNF4 or disabling the proteasome, which demolishes ubiquitin-tagged molecules, caused CENP-I levels to return to normal. The overall picture is that SENP6 permits kinetochore assembly by preventing RNF4 from ubiquitinating CENP-I. Why cells adopt this indirect mechanism to control ubiquitination isn’t clear. CENP-I might require SUMOylation to do its job, the researchers speculate.

Mitosis stalled in cells lacking SENP6, and chromosomes often didn’t line up properly during metaphase. Those defects also appear in cells that are missing the CENP-H/I/K complex, which helps insert other molecules into the forming kinetochore. When SENP6 was absent, CENP-H and CENP-I vanished from the inner part of the kinetochores. So did other proteins that the CENP-H/I/K complex helps put in place, such as CENP-O.

Further experiments suggested that the missing CENP-I had been destroyed. Removing RNF4 or disabling the proteasome, which demolishes ubiquitin-tagged molecules, caused CENP-I levels to return to normal. The overall picture is that SENP6 permits kinetochore assembly by preventing RNF4 from ubiquitinating CENP-I. Why cells adopt this indirect mechanism to control ubiquitination isn’t clear. CENP-I might require SUMOylation to do its job, the researchers speculate.

Mitosis stalled in cells lacking SENP6, and chromosomes often didn’t line up properly during metaphase. Those defects also appear in cells that are missing the CENP-H/I/K complex, which helps insert other molecules into the forming kinetochore. When SENP6 was absent, CENP-H and CENP-I vanished from the inner part of the kinetochores. So did other proteins that the CENP-H/I/K complex helps put in place, such as CENP-O.

Further experiments suggested that the missing CENP-I had been destroyed. Removing RNF4 or disabling the proteasome, which demolishes ubiquitin-tagged molecules, caused CENP-I levels to return to normal. The overall picture is that SENP6 permits kinetochore assembly by preventing RNF4 from ubiquitinating CENP-I. Why cells adopt this indirect mechanism to control ubiquitination isn’t clear. CENP-I might require SUMOylation to do its job, the researchers speculate.