Adducin-1 moonlights at the mitotic spindle

Adducin-1 (green) turns the mitotic spindle yellow in this mitotic cell.

Adducin-1 is an actin-binding protein that helps shape the mitotic spindle, as Chan et al. report. Researchers long thought that actin and microtubules performed different tasks during mitosis. They believed that microtubules formed the mitotic spindle that pulls chromosomes apart, whereas actin joined with myosin to produce the contractile furrow that separates the mother and daughter cells. But recent studies have revealed that the roles of the two cytoskeletons converge. For instance, actin helps situate the mitotic spindle, and microtubules help localize the cleavage furrow.

Histone shortages hold back replication forks

In a cell low on histones, nucleosomes haven’t reformed behind a replication fork (inset).

Histone deficiencies hold back replication forks, much as an automobile assembly line slows if engines are in short supply, DNA replication slackens if the cell is low on new histones, Mejlvang et al. reveal. To replicate their DNA, cells require ample amounts of nucleotides. But whether the availability of fresh histones, which package DNA into nucleosomes, also controls the rate of DNA copying is unclear. Yeast can complete S phase without new histones, researchers have found. However, studies that blocked protein synthesis in mammalian cells showed that DNA duplication falters, suggesting that a scarcity of histones can impede replication.

Mejlvang et al. addressed the issue in mammalian cells by investigating how cells sense that nucleosome assembly is incomplete. Nucleotide shortages trigger DNA damage checkpoints. But to the researchers’ surprise, histone scarcity didn’t initially trip these checkpoints, indicating that cells can wait for supplies of histones to build up without jeopardizing genome integrity. The next challenge is resolving how cells sense that nucleosome assembly is incomplete.

Thyroid hormones speed cellular aging

Cells treated with T3 (right) show more sites of DNA damage (red dots) than do control cells (left).

Thyroid hormones fire up metabolism by binding to either of two receptors, thyroid hormone receptor α (THRA) or thyroid hormone receptor β (THRB) and can induce opposing impacts on health. On the one hand, people with hyperthyroidism accumulate liver damage and have shortened life spans. On the other hand, thyroid hormone receptor blocks cancer growth and metastasis.

Zambrano et al. discovered a surprising new example of this functional overlap when they tracked the protein adducin-1. During most of the cell cycle, adducin-1’s C-terminal tail domain fastens onto actin at the cell membrane and helps brace the cortical cytoskeleton and cell–cell junctions. But Chan et al. found that during mitosis, adducin-1 attaches to the spindle with its N-terminal head domain. Adducin-1 relocates to the mitotic spindle when CDK1 phosphorylates two serines in the protein. However, it doesn’t hitch to the spindle directly. Adducin-1 couples to the microtubule-binding motor myosin-X. Chan et al. determined that this connection was crucial for the formation of the mitotic spindle. In cells lacking adducin-1, the spindles were distorted and often displayed multiple poles. How adducin-1 shapes the spindle—and whether actin is involved in that process—remains unclear.

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