In This Issue

Determining the replication factory settings

Saner et al. describe how neighboring DNA regions stochastically assemble into replication factories in budding yeast. A replication factory is a stretch of DNA duplicated from a single replication origin. In eukaryotes, multiple replication factories can find each other and pair up to undergo recombination. Clustering is controlled by LINC complexes, which span the nuclear envelope to couple chromosomes to the microtubule-based motor protein cytoplasmic dynein. Dynein can therefore pull chromosomes toward the centrosome on one side of the nucleus.

KASH5 helps meiotic chromosomes LINC up

Horn et al. identify a protein that helps homologous chromosomes pair up in meiosis by connecting them to the microtubule cytoskeleton. Early in meiosis, chromosomes cluster together so that homologous chromosomes can find each other and pair up to undergo recombination. Clustering is controlled by LINC complexes, which span the nuclear envelope to couple chromosomes to the microtubule-based motor protein cytoplasmic dynein. Dynein can therefore pull chromosomes toward the centrosome on one side of the nucleus.

RNA granules act as egg timers

Kotani et al. reveal how RNA granules control the timing of cyclin B translation during oocyte maturation. Fully grown oocytes initially arrest in prophase I, and mRNAs required for meiotic progression are translationally repressed. In response to maturation hormones, mRNA translation is activated so that the oocytes can progress to metaphase II, ready for fertilization. One key mRNA is the cyclin B transcript, whose translation induces germinal vesicle (nuclear) breakdown and assembly of the first meiotic spindle. Exactly how cyclin B translation is controlled is unclear, however.

Kotani et al. found that cyclin B mRNA is assembled into granules in the cytoplasm of immature zebrafish and mouse oocytes. Maturation hormones induced disassembly of these granules at the same time that cyclin B began to be translated. Researchers discovered that granule assembly was promoted by actin filaments and by the protein Pum1, which bound to cyclin B transcripts. Disrupting granule assembly—by depolymerizing actin or expressing a mutant cyclin B mRNA unable to bind Pum1—caused cyclin B to be translated sooner after stimulating oocyte maturation. Inhibiting granule disassembly, on the other hand, delayed cyclin B translation and germinal vesicle breakdown.

Lead author Tomoya Kotani says that granule assembly isn’t required to repress cyclin B translation; even in the absence of granule formation, Cyclin B isn’t produced until oocyte maturation is initiated. Instead, granules control the timing of cyclin B translation in maturing oocytes. Kotani now wants to investigate what triggers granule disassembly and translation activation and to follow the process in real time using live imaging.