Hox signals death for neuronal precursors

The number of neurons in an adult fly is determined by a death-inducing blast of a homeodomain protein, based on results from Bruno Bello, Frank Hirth, and Alex Gould (National Institute for Medical Research, UK).

Plasmodesmata are the conduits for intercellular communication in plants, unique intercellular organelles that establish cytoplasmic and ER continuity between neighboring cells. Jung-Youn Lee, William Lucas (University of California, Davis, CA), and colleagues now identify a selective gatekeeper for many non–cell-autonomous proteins (NCAPs). Lee et al. figured that some NCAPs might bind to plasmodesmal proteins, so they used an affinity column based on an NCAP called CmPP16 and a cell wall fraction highly enriched for plasmodesmal proteins to identify NCAPP1.

Rec’d and repaired

DNA replication stalls when the polymerase encounters lesions in the DNA, but recovers soon after lesion repair. In a recent work, Justin Courcelle and colleagues (Mississippi State University, Mississippi State, MS) examine what happens to the replication fork during this downtime. The results show that maintaining the correct fork structure depends on recombination proteins that may help to prevent illegitimate strand exchanges.

Courcelle’s group used two-dimensional gel electrophoresis to examine the shapes of a replicating bacterial plasmid. Advancing replication forks yielded the expected Y-shaped structure. But UV-induced lesions stalled the replication fork and produced X-shaped structures. These structures represent the nascent DNA backing up from the apex of a Y-shaped fork. The stalled structures were processed by RecQ and RecJ and maintained by RecA and RecF, which are the same proteins that promote homologous DNA pairing during recombinational processes.

A mid-replication stall is “like catching a cell with its pants down,” according to Courcelle. “It can’t live as one and a half cells for eternity,” he says. Unchecked free DNA ends are recombinogenic. Fork stabilization by these Rec proteins may be essential for preventing unwanted mitotic recombination and its potentially cancerous consequences. In addition, fork regression and Rec binding probably delays replication long enough for repair enzymes or SOS polymerases either to repair the lesion or to replicate past it. Whether RecA and RecF recruit repair enzymes or simply maintain an open fork remains to be determined.

Promoting passage in plasmodesmata

As expected for a plasmodesmal function, colocalization of NCAPP1 and CmPP16 was observed at the ER near the orifice to plasmodesmata. A dominant–negative form of NCAPP1 altered trafficking of the NCAPs CmPP16, LEAFY, and the movement protein of Tobacco mosaic virus, but left trafficking of other NCAPs unaffected. Thus, NCAP movement is a regulated process, similar to nuclear pore trafficking.

Reference: Courcelle, J., et al. 2003. Science. 10.1126/science.1081328.

Reference: Lee, J.-Y., et al. 2003. Science. 299:392–396.