**Research Roundup**

**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).

When the fly larva hatches, neurons are nearly evenly distributed along the major body axis. But in the adult, they are more numerous in the segments of the thorax than the abdomen, in part because neural stem cells (NSCs) in the thorax divide for a longer period of time. The new article describes “how division is stopped in its tracks and why this happens earlier in the abdomen than in the thorax,” says Gould.

His group has found that a pulse of an abdomen-specific Hox transcription factor, called AbdA, in late larval stage NSCs determined the final number of neurons. Rather than signaling exit from the cell cycle, AbdA limited proliferation by inducing cell death through the *grim/hid/reaper* pathway. Artificial premature expression of AbdA further decreased the number of abdominal neurons by inducing early cell death.

Ectopic expression of thorax-specific Hox genes *Antp* and *Ubx* also induced NSC apoptosis. However, these genes were not normally expressed in thoracic NSCs, thus allowing the cells to propagate longer. Gould is now looking for more players in the Hox-induced death pathway, including factors that induce AbdA expression. He is also interested in features of NSCs that make them sensitive to Hox-induced apoptosis, even while many other Hox-expressing cell types are spared.

Reference: Bello, B., et al. 2003. Neuron. 37:209–219.

**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.

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

**Promoting passage in plasmodesmata**

In plants, cell-to-cell communication is achieved through plasmodesmata, 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 this system, which they call NCAPP1.

Plasmodesmata are the conduits 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.

Lucas’ group is now purifying other potential plasmodesmal proteins that traffic NCAPP1-independent cargos.

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