**Research Roundup**

**Sudden bacterial death syndrome**

Just before they lyse, bacteria infected with lambda phage have no idea what is about to hit them, according to Angelika Gründling, Mike Manson, and Ry Young of Texas A&M University (College Station, TX). Gründling watched bugs spin around their single tethered flagella as an indicator that their membranes were still energized. The spinning stopped abruptly a few seconds before the cells lysed, thus contradicting the assumptions of one model for lysis timing. That model was born from the observation that membrane energy poisons induce lysis prematurely. Energy poisons, some suggested, were merely accelerating a normal process of gradual proton leakage. In this model, phage-produced holin proteins would cause the leak, until a critical reduction in proton motive force (pmf) across the membrane triggered any remaining holins to form into pores and finish off the bug.

Gründling’s new observations suggest that there may indeed be a critical level of pmf, but that there is no significant leakage early on that would gradually lead the cell to this critical level. Instead, holins increase in concentration until a pore spontaneously precipitates in the membrane. The catastrophic proton leak that results initiates the formation of many other pores, leading to the bug’s rapid demise.

This triggering mechanism leads not only to rapid and complete lysis, but may also reduce competition from other phage. As new phage attach to a cell, they cause a transient drop in pmf. This drop may trigger holin aggregation, thus killing the cell before the invader has time to start competing with the resident phage.


**A nurse for bicoid**

*Bicoid* mRNA is an anterior determinant for fly development that is produced in nurse cells before moving into the neighboring oocyte. In the simplest models, the mRNA was thought to use polarized microtubules to move to the anterior of the fly oocyte.

Now William Theurkauf and colleagues (University of Massachusetts Medical School, Worcester, MA) show that such a simple model will not work, because the mesh of oocyte microtubules is largely unpolarized. Instead, *bicoid* mRNA picks up transport factors in the nurse cells, before entering the oocyte and using those factors to mediate anterior localization, possibly on a polarized subset of microtubules. Without the factors, the mRNA can move on microtubules but its movement is undirected.

Byeong Cha in the Theurkauf lab began his experiments by injecting in vitro transcribed fluorescent *bicoid* mRNA into the center of oocytes. He found that the mRNA moved to whatever region of the oocyte cortex that was closest, including anterior and lateral areas. *Bicoid* mRNA enters oocytes through large ring canals in the anterior, so Theurkauf says “our initial bias was that it was just being trapped” as it entered.

But further experiments showed that fly oocytes do a more complete job than simple trapping. Cha found that injection of the fluorescent mRNA into nurse cells, followed by recovery of that injected mRNA and reinjection into the center of oocytes, resulted in full anterior localization. Theurkauf and Cha suggest that factors picked up by *bicoid* in the nurse cell allow the resultant complex to move along a subset of polarized microtubules that are hidden within the bulk of the non-polarized microtubule array.

Based on mutant analysis, one protein that must be transferred from nurse cells is Exuperantia. Theurkauf is now searching for other such proteins. One approach, pursued in collaboration with Paul Macdonald’s group (University of Texas at Austin), involves defining the cis elements that *bicoid* mRNA needs to pick up its factors in the nurse cell, and then using these sequences to fish out the binding proteins.

Formin a link to microtubules

A protein more often linked to effects on the actin cytoskeleton has been implicated in the polar stabilization of microtubules. Gregg Gundersen (Columbia University, New York, NY) and colleagues show that mDia1 and mDia2, two formins related to fly Diaphanous and budding yeast Bni1, can induce the formation of stable detyrosinated microtubules that may help polarize cells. Gundersen had earlier found that the proteasome is required for efficient transcription elongation, and colleagues (University of Texas Southwestern Medical Center, Dallas, TX) recently reported that the 19S regulatory particle of the proteasome is recruiting the proteasome. Stephen Johnson (Cold Spring Harbor Laboratory, Cold Spring Harbor, NY) and colleagues (Cold Spring Harbor Laboratory, Cold Spring Harbor, NY) and colleagues, who present what they hope will be the beginning of a lengthy study of the interactions between heterotypic cell types.

Transcription gets a licence

Ubiquitination of some transcriptional activators may be necessary both to make them functional activators and to signal their destruction, according to a recent study from William Tansey (Cold Spring Harbor Laboratory, Cold Spring Harbor, NY) and colleagues. Tansey suggests that ubiquitination is a temporary licence for transcription that preprograms destruction into the very activation process, thus keeping activators under tight control.

One thing that ubiquitin may be doing, not necessarily related to actual destruction, is recruiting the proteasome. Stephen Johnson and colleagues (University of Texas Southwestern Medical Center, Dallas, TX) recently reported that the 19S regulatory particle of the proteasome is required for efficient transcription elongation, perhaps in some kind of chaperone role.

As for destruction, Tansey does not know how tightly it is coupled with transcription. “Are transcription factors truly a disposable thing—a one-shot deal?,” he asks. Further studies are needed to answer this question and to determine just how many different transcription factors are licensed by this mechanism.

References:
http://www.sciencemag.org/cgi/content/abstract/1062079