Actin’s origins

A newly determined structure of a filament-forming protein from bacteria is eerily similar to that of eukaryotic actin.

The bacterial protein, MreB, was known to affect cell shape in bacteria; *Escherichia coli* that lack MreB are spherical rather than rod-shaped. In March of this year, Jeffery Errington and colleagues (University of Oxford, UK) reported that filamentous helical structures containing MreB form close to the cell surface.

Fusinita van den Ent and colleagues (MRC Laboratory of Molecular Biology, Cambridge, UK) leapt on this finding. Beginning in March, they showed that purified MreB protein could form filaments. They then quickly crystallized and then solved the structure of MreB. They find that MreB and actin share structures containing MreB form close to the cell surface.

Remaking the heart

In recent years, stem cells and progenitor cells have been shown to perform remarkable tricks of transformation, from blood to brain, and skin to nerve. But the concept remains of differentiated cells as fixed personalities. Now Giulio Cossu (University of Rome, Italy), Elisabetta Dejana (FIRC Institute of Molecular Oncology, Milan, Italy) and colleagues have found that a differentiated endothelial cell line can transdifferentiate into contractile cardiac muscle—a feat that may have implications for treating heart attacks.

The endothelial cells do not transdifferentiate spontaneously, but require the assistance of cocultured cardiomyocytes. A small fraction of the cocultured endothelial cells begin expressing cardiac markers, and become electrically coupled with the cardiomyocytes. Injection of endothelial cells into heart tissue that has been starved of oxygen results in the formation of cardiac muscle by some of the injected cells.

This phenomenon makes Dejana think that it might be possible to repair tissue damaged by heart attacks. “You have a major problem when you have heart failure because cardiomyocytes cannot divide,” she says. Injected endothelial cells, and even the endothelial cells from microvessels around an oxygen-starved region, might replace dead and dying cardiac muscle cells. But in both cases Dejana believes that the transdifferentiation process must be understood so that it can be improved upon. “Now the major challenge,” she says, “is to make it more efficient.”

Reference: Condorelli, G., et al. 2001. *Proc. Natl. Acad. Sci. USA*. 98: 10733–10738.

Beware the Borg

Too much of a Borg can be a bad thing. Just how and why it is bad may give clues to the function of septin proteins.

Ian Macara (University of Virginia, Charlottesville, VA) and colleagues have found that certain mammalian Borg proteins, first characterized as Cdc42 effectors, can also bind to septins. An excess of the Borgs induces the formation of long and thick septin filaments, and expression of the septin-binding domain of a Borg protein clusters all visible septins into one perinuclear spot in the cell.

The Rho GTPase Cdc42, which has been implicated previously in cytokinesis, vesicular transport, and cell polarity, inhibits septin binding to Borgs. Macara suspects that Cdc42 may inhibit another, unknown factor that normally unfolds Borg proteins so that their septin-binding domains are exposed.

The complete disruption of septin organization by a Borg protein fragment might give clues to normal septin function. As yet, the only cellular disruption that Macara has noted is some problems with cytokinesis, which would be consistent with prior yeast genetics and septin antibody experiments in mammalian cells. Septins in cytokinesis may form a docking site for addition of new membrane. The details of this process, or why it requires a protein that can form filaments, remain a mystery.

Reference: Joberty, G., et al. 2001. *Nat. Cell Biol*. 3:861–866.