

## Oscillating wildly

Bacterial cell division is restricted to the middle of the cell. This, say Kyoko Suefuji, Regina Valluzzi, and Debabrata Ray-Chaudhuri (Tufts University, Boston, MA), can be explained by polymerization events that oscillate between the two ends of the cell.

The polymerization process forms filaments of MinD at one end of the cell, which sequester MinC from the middle of the cell, thus leaving the bacterial cell division protein FtsZ to do its job. An additional component, MinE, forms a cap on the MinCD crescent so that the inhibitor, MinC, cannot reach the central FtsZ.

These proteins must inhibit division at both ends, and they do so by oscillating from one end of the cell to the other every 50 seconds. In several existing models, self-assembly is a key part of this oscillation. Joe Lutkenhaus (University of Kansas, Kansas City, KS) has recently seen self-assembly of MinD on lipid vesicles, with diffraction patterns suggesting a regular structure.

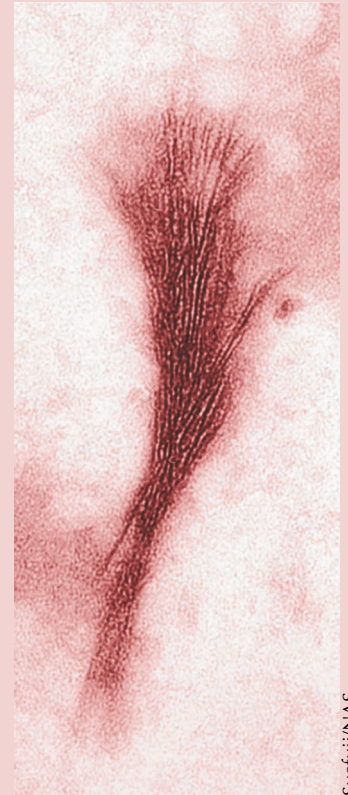
But the Tufts team is the first to visualize MinD filaments directly. Assembly was dependent on ATP (MinD is an ATPase), and filaments were much longer and thicker

when vesicles were added. MinE bound to the filaments and increased their length and width even further, but also led to bundle disassembly that was dependent on ATP hydrolysis. As the bundles disassembled, they frayed preferentially at one end.

“MinE is clearly doing two things at one time—bundling the MinD filaments and turning them over,” says RayChaudhuri. In its negative role, MinE may both halt the progression of polymerization toward the cell center and begin chewing away at the existing filaments. As MinD is liberated from one end, it may polymerize at the only MinE-free site: the other end of the cell. These new MinD polymers then attract MinE, and the cycle begins again.

Although MinD is not quite a bacterial version of the microtubule, RayChaudhuri can see parallels. He plans to use motor-like assays to test whether MinE’s depolymerization activity is polar. And he says that, like microtubules, “this system clearly evolved as a mechanism to search and explore cellular space.” ■

Reference: Suefuji, K., et al. 2002. *Proc. Natl. Acad. Sci. USA*. 10.1073/pnas.262671699.



Suefuji/NAS

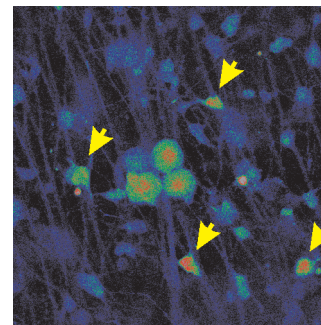
MinD filament bundles get frayed by MinE treatment.

## A trigger for myelination

Some cells can induce the production of their own protective blanket. The signal comes from active neurons in the central nervous system (CNS), and triggers the differentiation of oligodendrocyte precursor cells (OPCs)—thus yielding the cells that cover the neurons with a protective sheath of myelin. Now, Beth Stevens, Douglas Fields, and colleagues (National Institutes of Health, Bethesda, MD) have shown that the neurons accomplish this task by releasing a simple metabolite, adenosine.

The finding could be critical for possible stem cell treatments for the myelinating disease multiple sclerosis. “The problem [with stem cell treatments] is not getting the stem cells; the problem is getting the cells to differentiate at the right stage,” says Fields. Adenosine looks like a promising stop signal for OPCs. And, as a further enticement, Fields showed that a brief exposure to adenosine might be enough to set cells on the right path.

Fields added a wide variety of biomolecules to OPCs and found that adenosine alone could reduce proliferation and induce everything from differentiation markers to a differentiated morphology and myelination. Adenosine receptor antagonists, meanwhile, prevented the changes in OPC proliferation and morphology normally caused by active neurons.



Fields/Elsevier

Oligodendrocytes (arrows) are activated by signals from active neurons (center).

These results contrasted with the group’s earlier findings with Schwann cells, which provide myelination for the peripheral nervous system (PNS). For Schwann cells it is ATP that is active but with an opposite effect: the ATP arrests maturation. This may give the PNS axons time to mature before they are surrounded by myelin.

The regulation of the two systems will take some time to decipher. ATP and adenosine not only have different effects on OPCs and Schwann cells, but one metabolite can be converted to the other via extracellular enzymes. Fields thinks the decoding effort will be worthwhile. “As neuroscientists we are all focused on rapid communication,” he says, “but all cells communicate, and this is one of the most ancient systems.” ■

Reference: Stevens, B., et al. 2002. *Neuron*. 36:855–868.