

Occludin (red) and Claudin-1 (green) overlap at the tight junctions (yellow).

Tight junctions loosen up

Tight junctions are more flexible than researchers realized, as [Shen et al.](#) show. The team discovered that proteins are constantly entering and leaving these connections between epithelial cells. The continual remodeling might allow organisms to adjust the permeability of cell layers.

In locations such as the skin and the lining of the intestines, epithelial cells snuggle up to each other to form tight junctions. More than 30 proteins cluster at tight junctions, including ZO-1, which settles on the inner surface of the cell

junctions, suggesting that proteins enter and leave the junctions or that the entire structure changes position.

To distinguish between these possibilities, Shen et al. used fluorescence recovery after photobleaching and a related technique, fluorescence loss in photobleaching, to track ZO-1, occludin, and claudin-1. ZO-1, they found, was a restless protein that continually cycled between tight junctions and the cytoplasm. Occludin was also a traveler, but it got around through a different mechanism. The protein moved within the junction, the team discovered. It could also enter and leave the junction while remaining within the membrane. Claudin-1, by contrast, was the homebody of the trio, largely staying put.

The work indicates that cells are constantly tinkering with the tight junctions, removing and adding proteins. The researchers speculate that the body alters the lineup of proteins in the junctions to control how much can pass through epithelial layers. For example, after a meal the intestine might loosen the junctions to allow absorption of more nutrients. **JCB**

Shen, L., et al. 2008. *J. Cell Biol.*
doi:[10.1083/jcb.200711165](https://doi.org/10.1083/jcb.200711165).

membrane and links the junction to the cytoskeleton. Two other tight junction proteins, occludin and claudin-1, crisscross the membrane. The standard view of tight junctions was that they are static, with the proteins pretty much locked in place.

The researchers had previously observed a subtle flow of tagged occludin molecules in tight

Turning back the clock for Schwann cells

Myelinating Schwann cells have an ability every aging Hollywood star would envy: they can become young again. [Parkinson et al.](#) have pinned down a protein that returns the cells to their youth, a finding that might help researchers understand why myelin production falters in some diseases.

Wrapped around neurons in the peripheral nervous system, Schwann cells can “dedifferentiate” into a state in which they can’t manufacture myelin. Reverting to an immature type of cell speeds healing of injured nerves. Researchers knew that the protein Krox-20 pushes immature Schwann cells to specialize and form myelin, but they didn’t know what prompts the reversal. One suspect was a protein called c-Jun, which youthful Schwann cells make but Krox-20 blocks.

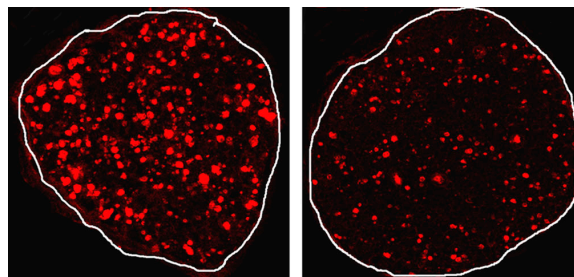
Parkinson et al. cultured neurons

with Schwann cells whose c-Jun gene they could activate. Turning on the gene curbed myelination, suggesting that c-Jun prevents young Schwann cells from growing up. c-Jun also prodded mature Schwann cells to become youthful again, the researchers discovered. Schwann cells that are separated from neurons normally dedifferentiate, but the team found that the cells remained specialized if c-Jun was missing. They suspect that c-Jun works in part by activating Sox-2, as this protein also inhibits myelination.

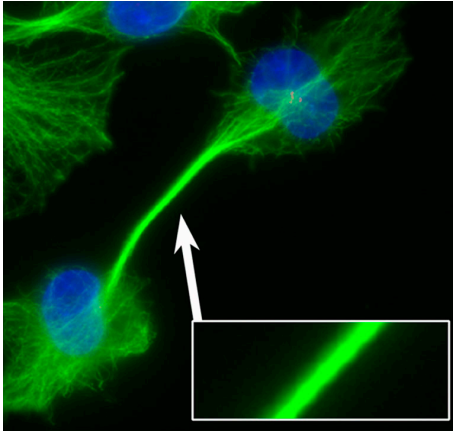
The researchers now want to investigate whether c-Jun is involved in illnesses where myelin dwindles, such as Charcot-Marie Tooth disease and Guillain-Barre syndrome. The re-

sults might also provide clues about multiple sclerosis, in which immune attacks destroy myelin in the central nervous system. Unlike Schwann cells, oligodendrocytes, the myelin makers in the central nervous system, can’t revert to an immature state. Whether c-Jun affects oligodendrocyte differentiation isn’t known. **JCB**

Parkinson, D.B., et al. 2008. *J. Cell Biol.*
doi:[10.1083/jcb.200803013](https://doi.org/10.1083/jcb.200803013).



A sliced nerve retains more myelin if c-Jun is absent (left) than it does in controls (right).



Dividing cells that lack Tektin 2 fail to separate.

Cutting the cord

Like parents sending their child off to college, a dividing cell has to let its offspring go. A microtubule-organizing protein helps ensure that the daughter cell breaks free, [Durcan et al.](#) report.

When chromosomes begin to pull apart during mitosis, the microtubules in the middle of the cell bunch up into a structure known as the central spindle. As the new cell begins to pinch off, it remains connected to its parent by a tether, the midbody, which contains the central spindle and doesn't part until after division is complete. Microtubules in developing flagella and cilia are arranged with the help of a protein called Tektin 2. [Durcan et al.](#) were therefore curious whether this protein performs a similar job in the midbody tether.

To determine the protein's function, the scientists slashed its levels with siRNA. Although Tektin 2–lacking cells seemed to divide normally, they often reunited. For a closer look at what occurred in the central spindle, the team treated cells with blebbistatin, which prevents dividing cells from pinching in two. Microtubules in the central spindle normally form a bundle, but they were jumbled in blebbistatin-treated cells dosed with Tektin 2 siRNA.

The team concluded that Tektin 2 promotes cell separation by bundling the microtubules of the central spindle. How Tektin 2 achieves this feat isn't clear, but they hypothesize that it sets the length of microtubules in the central spindle, controlling how much overlap occurs between microtubules from the would-be parent and offspring cells. **JCB**

[Durcan, T.M., et al. 2008. *J. Cell Biol.* doi:10.1083/jcb.200711160.](#)

RNA polymerase doesn't make deliveries

Researchers are divided over whether RNA polymerase tracks down genes that are ready to be transcribed or whether the enzyme remains in place. [Xu and Cook](#) add to the evidence that a gene has to relocate to one of a few sites in the nucleus to be transcribed.

One possibility is that the RNA polymerase, the DNA-reading enzyme, moves from gene to gene, transcribing each in situ. The alternative is that active RNA polymerase molecules remain stationary in “factories,” which genes visit to be transcribed. Some results support the existence of these factories, but the issue remains controversial.

[Xu and Cook](#) tackled the question by inserting two plasmids that carried different promoters and identifier genes into monkey cells. The plasmid DNA fused to form minichromosomes that the host's RNA polymerase can read. The researchers then tracked these minichromosomes to determine where they were being transcribed. Instead of spreading around the nucleus, they clumped at only about 20 sites, the team found. Minichromosomes with the same promoter ended up in the same location, even though their genes differed. But minichromosomes with different promoters separated. Intron-carrying and intron-lacking minichromosomes went to different locations, although the reason for this separation is mysterious.

The findings offer further backing for the presence of transcription factories, the team says, and suggest that the factories specialize to handle certain types of genes. The researchers now want to determine how many kinds of transcription factories a cell contains and what directs genes to particular ones. **JCB**

[Xu, M., and P.R. Cook. 2008. *J. Cell Biol.* doi:10.1083/jcb.200710053.](#)

Ribosomes rebuffed

New results from [Kawahara et al.](#) reveal how a translation-blocking protein performs its job. The protein makes a nuisance of itself, getting in the way of another protein that is essential for starting translation.

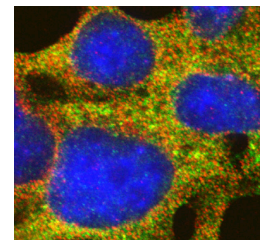
The researchers had previously discovered that the protein, Musashi-1, prevents neural stem cells from differentiating by stalling translation of mRNA for m-Numb, which helps orchestrate cell specialization.

But how Musashi-1 halts translation was a mystery. Other translation-blocking proteins thwart the initial step of the process by which the small ribosomal subunit latches onto a newly minted mRNA strand. Two vital proteins for completing this step are the eukaryotic initiation factor eIF4G and the poly (A) binding protein (PABP). They connect and help bring the small ribosomal subunit into position.

[Kawahara et al.](#) showed that Musashi-1 also hooks up with PABP. A mutant Musashi-1 lacking the PABP-recognizing sequence could not attach to PABP or halt translation. The team also found that Musashi-1 competed with eIF4G to bind to PABP.

To determine whether this competition hampers translation, the researchers measured how well ribosomes bound to mRNA in Musashi-1's presence. The small ribosomal subunit settled onto the mRNA just fine. However, Musashi-1 hampered the attachment of the large ribosomal subunit, which is also necessary to make a functional ribosomal complex. Because a variety of stem cells make Musashi-1, it might be a key factor for controlling stem cell differentiation, according to the researchers. **JCB**

[Kawahara, H., et al. 2008. *J. Cell Biol.* doi:10.1083/jcb.200708004.](#)



Musashi-1 (green) and PABP (red) cluster on cells.