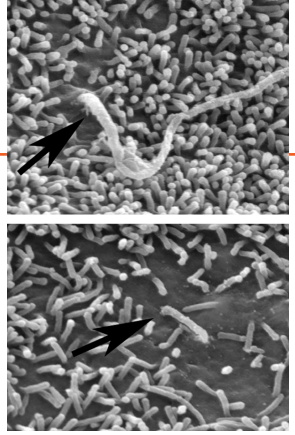

A cell without Par3 (bottom) sports a petite cilium (arrow).

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For long, lush cilia, try Par3

A protein that induces cells to create tight junctions also helps the primary cilium grow, as Sfakianos et al. show. Although the details remain murky, the protein, Par3, helps lengthen the structures by connecting a molecular motor that travels along the cilium to proteins that are embedded in the cilia membrane.

Researchers know that Par3 teams up with three other proteins, but they don't know all of its effects. Some studies suggest that Par3 helps induce adhesive tight junctions between epithelial cells. Other work indicates it sets up cell polarity in neurons by defining the axon. Sfakianos et al. have identified yet another function for Par3.

The team used RNAi to quash the protein. Although cells lacking Par3 still established tight junctions, they took extra time to form. The cells seemed to polarize normally, suggesting that Par3 isn't necessary to complete this process. However, loss of Par3 fouled up construction of the cilium. Normal cells grew lengthy cilia, but cells lacking Par3 could only manage puny filaments.

Par3 hooks up with a molecular motor called Kif3a, which helps haul new cilium building blocks to the growing tip. Without this interaction, cilia were stumpy. But cilia were absent when cells were missing another protein called Crumbs3, which settles in the membrane along the cilium. The team showed that Par3 uses Crumbs3's PDZ-binding domain to maneuver Crumb3 into position. So Par3 might spur cilium elongation by tying the membrane protein to motor proteins that slide along the cilium. The next step for the researchers is to determine how these links guide fresh components to the end of the cilium. JCB

Reference: Sfakianos, J., et al. 2007. *J. Cell Biol.* 179:1133–1140.

Making room for muscle

A mysterious version of the protein calcineurin turns out to be a healer that helps refurbish damaged muscle, as Lara-Pezzi et al. report. The molecule promotes cell division and drives away immune cells that can obstruct repair.

Sparked by rising calcium levels, calcineurin flips on transcription factors that control everything from immune responses to heart development to muscle cell differentiation. The two halves of the protein, CnA and CnB, come in several forms. Scientists discovered a new version of CnA, known as CnAβ1, nearly 20 years ago, but they knew little about its function.

Now, the researchers show that, with CnAβ1, undifferentiated muscle cells divide more quickly and are less likely to specialize. Calcineurin usually exerts its influence by activating the NFAT transcription factors. CnAβ1, however, activated a different signaling pathway and blocked the transcription factor FoxO.

CnAβ1 also sped regrowth of damaged muscle, the team shows. In mice that had received an injection of a muscle-destroying poison, boosting CnAβ1 levels caused an increase in the number of active muscle stem cells and a thickening of regenerating muscle fibers. CnAβ1 also trimmed the number of macrophages at the injury site and limited accumulation of fresh extracellular matrix.

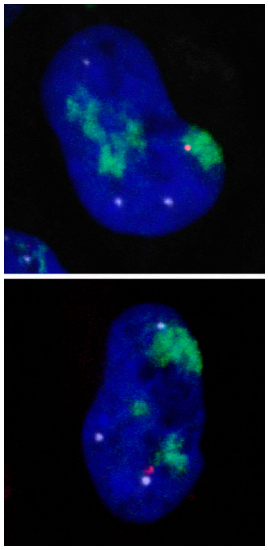
Thus CnAβ1 helps muscle heal by encouraging cell division, calming inflammation, and limiting scarring, leaving more room for new muscle cells. The next question, the scientists say, is whether the variant is important for other fast-dividing cells, such as stem cells and tumor cells. JCB

Reference: Lara-Pezzi, E., et al. 2007. *J. Cell Biol.* 179:1205–1218.

Hey, DNA, get over here

Active genes sidle up to Cajal bodies with help from actin, Dundr et al. report. The study is the first to show directed movement of mammalian genes that are being transcribed.

Interphase chromosomes jiggle, but they usually remain within so-called territories. Particular DNA segments, however, can travel substantial distances. One situation that might involve DNA movement is the liaison between active genes and Cajal bodies, which harbor small nuclear RNAs (snRNAs) for splicing. Cajal bodies often show up near working genes for snRNA and histone proteins, although researchers didn't know whether Cajal bodies form near these genes or whether the partners move toward each other.



A DNA stretch with active genes (red) closes in (top to bottom) on a Cajal body (white).

To find out, Dundr et al. inserted into HeLa cells an artificial chromosome carrying 16 copies of an snRNA gene. The team tracked the positions of the chromosome and Cajal bodies after the genes started transcription. The two components cozied up, the researchers found. The Cajal bodies were sluggish, remaining in roughly the same place. The DNA, by contrast, was responsible for most of the movement, particularly during a final lunge that began around six to seven hours after gene activation. In total, it traveled about two to three microns.

The researchers also found that a tether of RNA linked an active gene to the Cajal body, indicating that the newly made strand was feeding directly into the structure. Disrupting actin, Dundr et al. showed, prevented the movement, suggesting that actin helps haul certain active genes to Cajal bodies. The question of why this movement occurs remains unanswered. JCB

Reference: Dundr, M., et al. 2007. *J. Cell Biol.* 179:1095–1103.