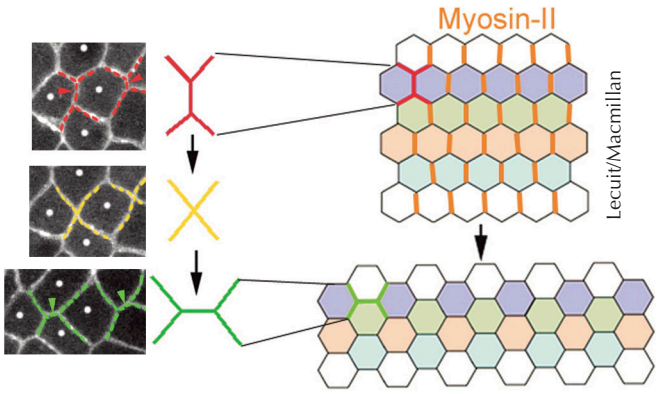
Research Roundup

Moving while sticking

Intercalation—the slotting of cells in between one another—is an established method for converting short and fat into long and slim. This is all very well in slippery, adhesion-sparse mesenchymal tissues. But many such extension events occur in epithelial tissues, where cells are glued together by adhesion complexes. Claire Bertet, Lawrence Sulak, and Thomas Lecuit (IBDM, Marseille, France) now report that epithelial intercalation relies on some strategic tugging by myosin that remodels junctions.

The French group expected that extension in fly embryos would involve a few individual motile cells nosing between other stationary cells. Instead they saw "a global and ordered reorganization," says Lecuit. Borders between cells that lay anterior and posterior to each other contracted to a point. Perpendicular expansion of this point generated a border between dorsal and ventral cells, thus pushing the anterior cell more anterior and the posterior cell more posterior. Extension was therefore achieved by a geometrical shuffling of the original hexagonal arrangement.

Myosin II was concentrated near the shrinking (anterior–posterior) membranes and reduced near the expanding (dorsal–ventral) membranes. In embryos with less myosin II, junctions froze and intercalation failed. Myosin need only destabilize adhesion proteins at anterior–posterior membranes, as junction proteins are naturally very dynamic.



Contraction and expansion of membranes reorganizes cells as the tissue extends (top to bottom). Anterior is left; posterior is right.

It is not known how myosin is concentrated preferentially near anterior and posterior membranes. Genes that define anterior–posterior polarity of the fly embryo are needed; they may exert this effect by either local (cell-to-cell) or global (gradient) messages.

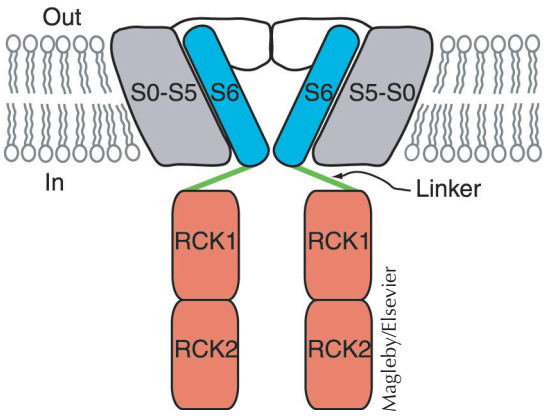
These polarity cues result in an intercalation method that, says Lecuit, maintains "the balance between stability and dynamics." It can account for a nearly twofold extension. If more extension is needed, for a gut tube or an arm, then cell shape changes and oriented cell division may come into play. ■

Reference: Bertet, C., et al. 2004. *Nature.* 429:667–671.

How a channel springs open

The gating mechanism of a K^+ channel includes a perfect spring, according to Xiaowei Niu, Xiang Qian, and Karl Magleby (University of Miami, Miami, FL).

The group studied the BK channel from mice. This channel is opened by changes in both voltage and intracellular Ca^{2+} concentration. Depolarization drives a positively charged section of the protein outwards and thus drags apart the S6 channel domains that form the channel's gate. Addition of Ca^{2+}, by contrast, widens the diameter of an intracellular gating ring, once again pulling apart the S6 gates.



The linker between the RCK gating ring and the S6 gate acts as a perfect spring.

The two control mechanisms converge on the same target—positioning of the S6 gates—so one control mechanism can be used to probe the mechanism used by the other. The authors changed the length of the linkers that run from gating ring to gates, and tested how much voltage was needed to open the new channel variants.

Shortening the linkers made it easier to open the channel with voltage changes, probably because the diameter of the gating ring is fixed, so shortening the linker pulls the S6 gates outwards toward the gating ring. Lengthening the linker made it more difficult to open the channel with a voltage change. There was a linear relationship between the changes in linker length and required voltage, indicating that the linker-gating ring complex is acting as a perfect, Hookean spring.

The same linear relationship did not hold up when channels with different linker lengths were probed with varying Ca^{2+} levels. "With calcium the gating ring becomes an active machine and changes in shape," says Magleby.

Magleby hopes to confirm the proposed movements with fluorescence proximity probes. But already, says Niu, "you don't have to look at the channels as blobs of protein. You can have mechanical models that really work." ■

Reference: Niu, X., et al. 2004. *Neuron.* 42:745–756.