Actin mesh hinders microtubules

Like an iron curtain, a mesh of actin keeps microtubules in place in the fly oocyte, according to Katja Dahlgaard, Daniel St Johnston, and colleagues (University of Cambridge, UK). When the mesh comes down, microtubules are washed into a new orientation by free-flowing cytoplasm.

Microtubule orientation in the fly oocyte points transported mRNAs for polarity-inducing proteins in the right direction. The microtubule array starts off nucleated from the anterior end of the oocyte and extending toward the posterior. At late oogenesis, the array is redistributed to lay flat against the edge of the oocyte. At the same time, the cytoplasm of the oocyte begins to churn like a washing machine.

This rearrangement and churning occurs prematurely in mutants of the actin-associated proteins Cappuccino and Spire, resulting in polarity problems. Dahlgaard was imaging oocytes in an attempt to understand how actin or its regulators control the microtubule network. Since microtubules are so fragile, she was fixing her samples as quickly as possible. Her speed revealed a glimpse of a fleeting actin mesh throughout the oocyte cytoplasm. Stabilization of this mesh prevented microtubule reorganization and streaming.

The actin mesh was missing in the Cappuccino and Spire mutants. It was also dismantled in wild-type flies at late oogenesis, when streaming begins. The authors suggest that the mesh physically hinders streaming, which is probably created by kinesin’s tugging of large cellular structures such as organelles. Slow kinesin mutants negated the need for a mesh to prevent premature streaming.

Streaming cytoplasm probably aligns microtubules in the direction of flow and pushes them to the cortex, aligning more kinesin traffic and further bolstering streaming. The mesh might physically block microtubules so they can’t turn or reach the cortex. Or it might increase cytoplasmic viscosity, thereby slowing kinesin. And St Johnston has yet another hypothesis. “I favor the idea that the mesh is somehow anchoring organelles as kinesin is trying to move them.” To address the issue, he says, “we first need to find what’s being transported to generate flow.”


Promoters enter plant–pathogen fray

The evolutionary arms race between plants and pathogens has moved into new territory—gene promoters—according to two new articles. In the first, Sabine Kay, Ulla Bonas (Martin-Luther-Universität, Halle, Germany), and colleagues reveal that a bacterial protein mimics eukaryotic transcription factors. Plants, in turn, coopt that bug protein to activate defensive genes, according to Patrick Römer, Thomas Lahaye, and colleagues (Martin-Luther-Universität, Halle, Germany).

One plant’s killer is another’s simple nuisance; this difference can be encoded by a single gene. For instance, Xanthomonas strains that inject a protein called AvrBs3 into plant cells cause severe disease in several crop plants. But plants that carry the Bs3 gene resist those particular bugs by actively killing off infected cells before the pathogen spreads too much. The mechanistic basis of this resistance was not known.

Kay et al. now show that the AvrBs3 protein helps bacteria reprogram the plant cell’s metabolism by directly activating plant genes as would a plant transcription factor. AvrBs3 bound directly to the promoter of a master regulator of plant cell size known as upa20. Its encoded protein—also a transcription factor—caused cells to swell, which probably helps squeeze the bacteria out of infected tissue and into new terrain. The group also identified the promoter sequence in the plant gene bound by the bacterial activator.

Römer et al. then showed that some plants take advantage of AvrBs3’s transcriptional abilities. They found that the plant resistance gene, Bs3, contained the promotor sequence identified by Kay et al. This trick made bacterial AvrBs3 activate transcription of the bug’s killer: accumulation of Bs3—an unusual flavin monooxygenase—triggered cell death.

“As far as I know,” says Bonas, “this is the first example of a resistance factor that is a promoter element rather than a protein.”

Gurken (large black dots) and dynein (circled) are found in static sponge bodies in the dorso-anterior portion of the fly oocyte.

Turning motor into anchor

A motor in the fly oocyte is turned into a static anchor to hold its polarity-inducing cargo in place, say Renald Delanoue, Bram Herpers, Jan Soetaert, Ilan Davis (University of Oxford, UK), and Catherine Rabouille (University Medical Centre Utrecht, Netherlands). The switch from motor to anchor is thrown by an RNA-binding protein called Squid.

Squid mutants were originally identified by their polarity defects, which stem from mislocalized gurken mRNA. Gurken is normally transported by Dynein to the dorso-anterior portion of the oocyte. There, it is translated into a signal that instructs the overlying cells to become dorsal. Without Squid, gurken is wrongly dispersed throughout the oocyte anterior.

In the new work, Squid was shown to travel with Dynein and gurken in particles toward the dorso-anterior corner. Upon arrival, particles were transformed into dense, immobile structures called sponge bodies.

The bodies fell apart when Dynein was disrupted, suggesting that the motor becomes a static structural component. Squid was necessary for this transition; its inhibition reversed sponge bodies to transport particles. Squid might help Dynein create such a large complex that it is immobile. Or it might somehow shut off Dynein’s motor. Either way, why particles only become sponge bodies at the dorso-anterior corner is a mystery.

Davis says the fly oocyte may seem specialized, “but Dynein is universal. The components we’re studying are also likely to be relevant in the nervous system,” where mRNAs are localized and translated far from the cell body. JCB Reference: Delanoue, R., et al. 2007. Dev. Cell. 13:523–538.

New nucleator for actin

A new actin nucleator to the mix. Rashmi Ahuja, Michael Kessels, Britta Qualmann (Leibniz Institute for Neurobiology, Magdeburg, Germany), and colleagues identify Cordon-bleu (Cobl) as a nucleator of actin filaments.

“Previously, there were really only two known vertebrate actin nucleators: formins and the Arp2/3 complex,” says Qualmann. “That’s surprising if you consider the wealth of different actin structures that form.”

While studying proteins that interface with Arp2/3 and link actin polymerization to vesicle trafficking, the team noticed that new actin filaments still formed in extracts lacking Arp2/3. They decided it was time to fish for a new nucleator.

Using yeast two-hybrid analyses with those actin/vesicle linking proteins as bait, the group pulled out Cobl. Unlike Arp2/3 and its output of branched actin networks, Cobl created unbranched filaments similar to those made by formins. But formins are weak compared with Cobl, which polymerized filaments at 10-fold lower concentrations and with fewer available actin monomers.

Cobl’s physiological duties were most apparent in neurons, where it increased both neurite numbers and branching. Cobl knockdown resulted in poor branching and impaired neuronal network formation. Cobl is also seen during early development in specialized patterning cells that undergo plenty of actin building and reorganization. The group is now excited to tease out where and when Cobl is needed, how it is regulated, and whether it cooperates with other nucleators. JCB Reference: Ahuja, R., et al. 2007. Cell. 131:337–350.

Protein shields for dehydration

Many species have the ability to withstand long bouts of dehydration. New findings from Sohini Chakrabortee, Alan Tunnacliffe (University of Cambridge, UK), and colleagues reveal that a family of water-loving proteins in these organisms protects the cell from desiccation-induced protein aggregation.

Desiccation-tolerant organisms as diverse as bacteria, brine shrimp, and plants have in common a family of hydrophilic proteins known as the LEA proteins, which are necessary for survival in dry times. In the new report, a worm LEA protein is shown to inhibit aggregation of the entire worm and human proteomes during desiccation in vitro. It also prevented the aggregation that accompanies rehydration. “That experiment was a watershed,” says Tunnacliffe. “It means the proteins also work in the hydrated states.”

In fully hydrated human cells, a LEA protein hindered aggregation of polyQ-containing proteins. It also improved the cells’ ability to tolerate high salt levels, which mimic mild dehydration.

Unlike chaperones, which have defined structures, LEA proteins are natively unfolded. “Because they don’t have any structure,” says Tunnacliffe, “they don’t aggregate.” This property might allow them to work like a molecular shield, coating aggregation-prone proteins and hindering them from interacting with others of their kind. JCB Reference: Chakrabortee, S., et al. 2007. Proc. Natl. Acad. Sci. USA. doi:10.1073/pnas.0706964104.