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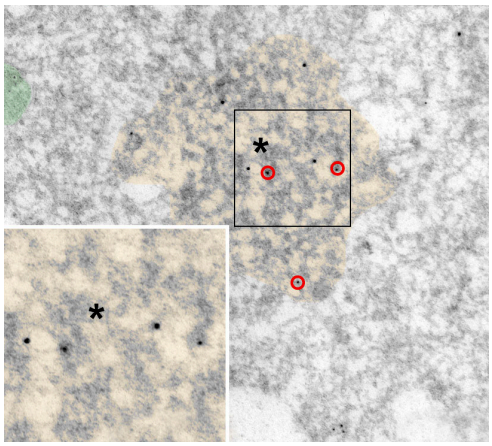


RABOUILLE/ELSEVIER

***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 reverted 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

Add 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."

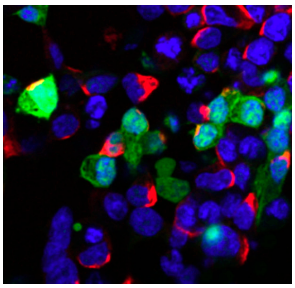
While studying proteins that interface with Arp2/3 and link actin polymerization to vesicle trafficking, the team noticed that new actin filaments still formed in extracts lacking Arp2/3. They decided it was time to fish for a new nucleator.

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



TUNNACLIFFE/NAS

A LEA protein (red) helps prevent the aggregation of polyQ proteins (green).

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.