The observation that the *Arabidopsis LFY* gene can cause transgenic aspen plants to flower many years earlier than they normally would, illustrates how conserved the function of the floral meristem-identity genes is. Moreover, it suggests that modification of their expression might be useful in non-agricultural applications, such as modifying horticultural species to create novel plant morphologies or to regulate their flowering time, or in promoting the flowering of trees so that they can be rapidly cross-pollinated to produce new varieties. □

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DEVELOPMENTAL BIOLOGY

New role for tropomyosin

Daniel St Johnston

THE interests of cell biologists and developmental biologists often pursue separate paths, with the former tracing the goings-on inside a single cell while the latter follow the interactions between cells that lead to complex patterns. One area where these paths look like converging is in anterior–posterior axis formation in *Drosophila*, in which the polarity of the whole organism is defined by the localization of two messenger RNAs, *bicoid* and *oskar*, to opposite ends of a single cell[1,2]. The mislocalization of either mRNA causes pattern defects in the resulting embryos, so these phenotypes can be used to detect mutations in the genes required for the polarized transport of mRNA within the oocyte. Now, for the first time, such a screen has identified a component of the cytoskeleton as an unexpected participant: on page 524 of this issue, Erdélyi *et al.*[3] describe how mutants in non-muscle tropomyosin block almost all transport of *oskar* mRNA from the anterior of the oocyte to the posterior pole.

Tropomyosins form highly α-helical coiled-coil dimers that bind along actin filaments. In skeletal muscle, a troponin-dependent repositioning of tropomyosin inhibits the interaction between myosin and actin, thereby regulating muscle contraction. The role of tropomyosins in other cell types is less clear because these cells lack troponin. Non-muscle-tropomyosins are usually associated with the most stable actin structures within a cell, such as stress fibres, and *in vitro* studies suggest that these isoforms may both stabilize actin filaments and regulate the movement of myosin motors along them[4].

This dual role is supported by genetic data from yeast. Mutations in one of the two tropomyosin genes in *Saccharomyces cerevisiae* cause a loss of stable actin cables and a defect in polarized growth and secretion[5], and the *Schizosaccharomyces pombe cdc8* tropomyosin gene is required for cytokinesis[6]. It is therefore a surprise to find that the first non-muscle tropomyosin mutants in a higher eukaryote should specifically impair the transport of a single mRNA, particularly as *Drosophila* seems to have only one non-muscle tropomyosin gene[7]. The original mutants isolated by Erdélyi *et al.*[3] merely reduce the expression of non-muscle tropomyosin, but the lethal alleles generated by the imprecise excision of a P-element are likely to be null mutations. Nevertheless, these mutants do not block mitosis in the female germ line, nor the formation of stable actin structures such as the stress fibres in the nurse cells.

It seems, then, that in *Drosophila* at least, non-muscle tropomyosin has a much more limited and specific function than we expected. *Drosophila* may have another non-muscle tropomyosin gene, however, and this possibility needs to be ruled out before it can safely be concluded that non-muscle tropomyosins do not play a major role in actin organization.

The requirement for tropomyosin in *oskar* mRNA localization is also a surprise to those of us who work on axis determination in *Drosophila*, for several results have suggested that *oskar* mRNA is transported along microtubules to the posterior of the oocyte. First, *oskar* mRNA localization requires intact microtubules, as it is abolished by treating ovaries with the microtubule-destabilizing drug colchicine[8]. Second, dynein and a transgenic kinesin–β-galactosidase fusion protein, which are microtubule motors, both colocalize with *oskar* mRNA to the posterior of the oocyte, suggesting that the microtubules are polarized along this axis[8,9]. Furthermore, when the oocyte develops a mirror-symmetric anterior–posterior axis, as in protein kinase A mutants, these motor proteins still co-localize with *oskar* mRNA, but this time to the centre of the oocyte[8,10].

Given all this evidence for microtubule-based transport, how can the requirement in *oskar* mRNA localization for tropomyosin and, by implication, the actin cytoskeleton be explained? One possibility, which can probably be discounted, is that tropomyosin mutants block *oskar* mRNA transport by altering the microtubule organization, as both dynein and kinesin–β-galactosidase localize normally in mutant oocytes[3].

An attractive alternative is that an actin-based transport system at the anterior of the oocyte delivers *oskar* mRNA to the microtubules for transport to the posterior. This model of local traffic along actin and long-distance transport along microtubules is consistent with the tropomyosin-mutant phenotype — most *oskar* mRNA could get trapped at the anterior because it cannot be transported along F-actin or because the actin is misorganized, but the small amount that does reach the microtubules can be transported all the way to the posterior pole. There is also a precedent for transport particles that can move along both actin and microtubules: single vesicles can switch between the two motility systems in extruded squid axoplasm[11].

There are still many questions that need answering before the role of tropomyosin in *oskar* mRNA transport can be resolved. For example, it would be nice to know the distribution of tropomyosin in the oocyte, and the arrangement of actin at the anterior pole. Nevertheless, the new results show that *oskar* mRNA localization is surprisingly complicated, and that it involves both the actin and microtubule cytoskeletons. In future, the *Drosophila* oocyte should provide a powerful model system in which the tricks of developmental genetics can be used to address basic questions in cell biology. □

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