Cell division: **Plant-like properties of animal cell cytokinesis**

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Recent evidence that a syntaxin is required for cytokinesis in *Caenorhabditis elegans* embryos suggests that the mechanism of cell division in plant and animal cells may be more similar than previously imagined.

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Past studies of cytokinesis in plants and animals have suggested that two key processes, contractile ring function and membrane addition at the cell surface, contribute to the partitioning of daughter cells at the end of mitosis [1]. Genetic and molecular studies of cytokinesis in vertebrate, insect, yeast and slime mold systems have provided ample *in vivo* evidence that the actin cytoskeleton and a contractile ring are critical for cell division [2]. Genetic studies in plants have demonstrated a requirement for membrane addition, though not cortical contraction [3]. Now, a study of cytokinesis in the nematode *Caenorhabditis elegans*, published recently in *Current Biology* [4], has shown that a syntaxin — a type of protein known in other contexts to play a part in specific membrane fusion — is necessary for cell division. This new

more generally for the fusion of all secretory vesicles at the cell surface.

The identification and analysis of temperature-sensitive alleles of *syn-4* may be necessary to dissect these different requirements. Meanwhile, examining the localization of transmembrane proteins expressed in the early embryo, such as the Delta and Notch homologs Glp-1 and Apx-1, might indicate whether *syn-4* is required more generally for vesicle transport. It will also be interesting to learn whether microtubules are involved in targeting new membrane addition in *C. elegans* embryos, and whether the midzone microtubules that become constricted by the contractile ring at the end of cytokinesis are in some ways similar to the phragmoplast of dividing plant cells.

Intriguingly, a protein that from its sequence is likely to be a member of the kinesin family of motor proteins is localized to the spindle midzone in early *C. elegans* embryos [10,11]. This protein is called Zen-4 or CeMKLP1, and inactivation of the *zen-4* gene was found to result in a late defect in cytokinesis, with cleavage furrows regressing only after substantial ingression. While the cytokinesis defect in *zen-4* mutant embryos might be caused indirectly by spindle defects, it also is possible that Zen-4/MKLP-1 is required to target membrane vesicles to the cleavage furrow late in cytokinesis, promoting the final separation of daughter cells at the spindle midzone eT*ı˝0.26 Twı˝(s Twı˝Twı˝(simil,alloihter cells)Tjı˝T*nr cellsslate in)Tthe final sdeT*ı˝ect3G Tw ˝T* mired more geoThe fi27d more generally