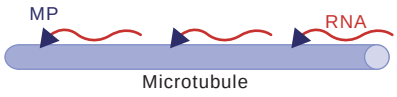

Figure 2 Model of how viral MP (dark blue) and viral RNA (red) are associated with plant microtubules during viral infection. MP is bound along the length of a microtubule as a result of mimicking part of the tubulin molecule. Each molecule of MP is associated with a strand of vRNA.

passage through the cell cycle or in response to environmental cues? How might the MP manage to extricate itself from a microtubule once it is at the mouth of the plasmodesma, given how ultra-stable the microtubule–MP complex seems to be?

In the greater scheme of things, these findings concerning virus-encoded microtubule-binding proteins may be helpful for stimulating our thinking about plant MAPs in general. Perhaps there are endogenous MAPs that can integrate into the microtubule lattice. If these also stabilize microtubules against depolymerization, they may be lost during attempts to isolate MAPs biochemically, as such attempts often make use of rounds of polymerization and depolymerization of tubulin. In spite of their opportunistic and damaging behaviour, plant viruses may have done us a favour by opening a window on a potentially new type of plant MAP.

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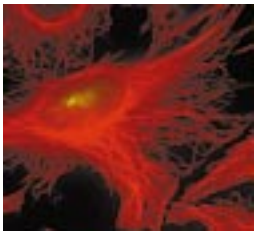
Signalling for stability

In most animal cells, microtubules are focused on the centrosome, an organelle near the nucleus that organizes the microtubule cytoskeleton into a radial array. As a result, they exhibit a property known as 'dynamic instability', involving continuous growth and shortening at their plus ends (away from the centrosome). Removal of the centrosome can cause a shift from dynamic instability to a behaviour called 'treadmilling', in which the uncapped minus ends are continuously depolymerized, leading to an increase in the concentration of microtubule monomer that gives rise to persistent growth at plus ends. Treadmilling occurs when the rate of plus-end growth matches that of minus-end shortening.

In terminally differentiated cells, such as neurons and polarized epithelial cells, the microtubule cytoskeleton is not focused on the centrosome. However, these microtubules do not treadmill but rather tend to exhibit dynamic instability. For example, microtubules in neuronal axons grow *in vivo* from their plus ends only, despite the fact that their minus ends are released from the centrosome. This has led to the proposal that such microtubules are stabilized by other putative factors, such as cytoplasmic g-tubulin.

Cell–cell contacts are crucial to the functions of neuronal and epithelial cells, and the formation of such contacts induces cell polarization. On page 797 of this issue, Chausovsky *et al.* show that the minus ends of microtubules are stabilized by signalling from cadherins, the adhesion receptors of adherens-type cell junctions. This regulation of microtubule dynamics would allow the establishment and maintenance of cell polarization by ensuring that microtubules remain



stable when released from the centrosome. Expression of cadherins can initiate cell contact and polarization in normally solitary cells, and the authors used Chinese hamster ovary (CHO) cells expressing E- or N-cadherin to monitor the effects of these receptors on microtubule organization in cells and in centrosome-free cytoplasts. They found that cadherin expression prevents the reduction in microtubule density that normally accompanies removal of the centrosome in fibroblasts. The picture shows the distribution of microtubules (red) in a CHO cell expressing N-cadherin (the centrosome is shown in green).

Chausovsky and colleagues propose that cadherins initiate a signalling pathway that influences microtubule dynamics by stabilizing minus ends, preventing treadmilling and thereby allowing net elongation. It seems that, although this signalling is direct, expression of cadherins *per se* is not sufficient for microtubule stabilization. Instead, cell–cell contacts, of which cadherins are an integral part, must be present for this process to occur. This would ensure that as cells form contacts with their neighbours, they are able to relinquish their radial microtubule arrays whilst still retaining sufficient amounts of microtubule polymer.

MICHAEL HOPKIN

Erratum — In "Evolution and function of ubiquitin-like protein-conjugation systems" by Mark Hochstrasser (*Nature Cell Biol.* **2,** E153-E157; 2000) The third sentence in the Fig. 4 legend should read: "Two catalytic cysteine residues, which are necessary for sulphur moblization in the suplhurtransferase, are retained in the ubiquitin/Ubl-conjugation systems." Figure 2b should be as shown below.