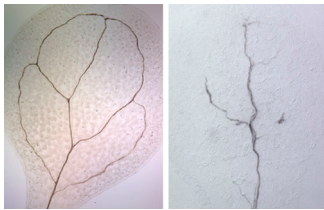


## Xylogen makes plant channels

**Z**innia are, according to a gardening website, “uncommonly beautiful” and “available in a riot of colors to satisfy any garden plan.” These plants from the daisy family are also a great model system for studying the formation of xylem—the woody channels in plants that conduct fluid and nutrients. Now, Hiroyasu Motose, Munetaka Sugiyama, and Hiroo Fukuda (University of Tokyo, Japan) have identified a protein called xylogen as an extracellular inducer of xylem formation. Like most mammalian growth factors, but unlike the chemical and peptide growth factors known to work in plants, xylogen is a large protein.



Motose/Macmillan

**Without xylogen (right), leaves form aberrant veins.**

via a wound-related response). This artificial system does not, however, say much about how xylem forms in intact plants.

The Japanese group set about purifying an activity that enhanced xylem formation. The factor was in plant extracellular fluid and turned out to be an arabinogalactan protein that they named xylogen. It contains a GPI anchor that may be cleaved.

Xylogen accumulated during xylem formation on the apical sides of differentiating cells. Motose suggests the cells secrete the molecule in a polar fashion to recruit neighboring cells directionally. Plants lacking xylogen formed discontinuous and thicker veins, suggesting that xylogen does not act alone. ■

Reference: Motose, H., et al. 2004. *Nature*. 429:873–878.

## Reprogramming by virus

**D**oes the Kaposi sarcoma (KS) herpesvirus (KSHV) replicate in lymphatic endothelial cells (LECs) or blood vascular endothelial cells (BECs)? Two groups have come up with a surprising answer to this question: neither. Instead the oncogenic virus modifies both of the two cell types to an intermediate state that better suits the virus’ replicative needs.

KS has always been known as a tumor of the endothelial system based on the reddish, bruised appearance of KS lesions. A handful of lymphatic markers are found on KSHV-infected cells, but two new studies by Hsei-Wei Wang, Chris Boshoff (University College London, UK), and colleagues and Young-Kwon Hong, Michael Detmar (Harvard Medical School, Boston, MA), and colleagues present the first comprehensive view of what these cells express.

The Boston group finds that BECs infected with KSHV induce ~70% of known lymphatic lineage-specific genes. Part of this effect is mediated by a lymphatic master regulator PROX1, earlier characterized by Hong, but other factors

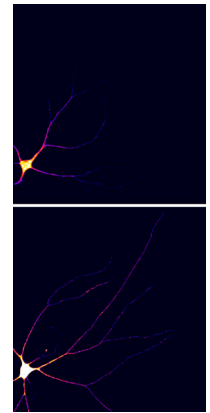
## Mini inhibition

**D**uring an action potential neurons release a huge bolus of neurotransmitter, which can alter protein synthesis in the receiving cell and reshape synapses during learning. But neurons also release single packets of neurotransmitter in a background dribble that has been largely ignored. Now, Michael Sutton, Erin Schuman, and colleagues (Caltech, Pasadena, CA) have found that these “minis” inhibit translation in dendrites. This may help neurons to start from a lower basal level, thus making the increase in translation after action potentials more dramatic.

The Caltech team “stumbled upon [the effect] by accident,” says Sutton. “The idea was to remove all synaptic activity and add it back,” while looking for changes in dendritic protein synthesis. Inhibition of action potentials reduced protein synthesis, but inhibition of both action potentials and minis led to an increase in protein synthesis.

The minis have been presumed to lack any function, so the “potential effects of minis on neuronal physiology have been largely ignored,” says Sutton. The frequency of minis does increase or decrease when synapses are strengthened or weakened, respectively, during long term potentiation (LTP) and long term depression (LTD). In theory, an increase in minis and thus decrease in protein synthesis during LTP could be a negative feedback mechanism. But in other experiments minis were necessary for the maintenance of dendritic spines during sustained blockage of action potentials. Resolving the minis’ ultimate effect will require further work in cultured cells or tissue slices. ■

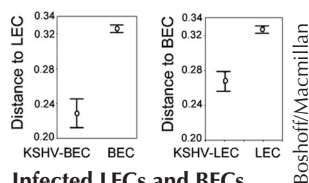
Reference: Sutton, M.A., et al. 2004. *Science*. 304:1979–1983.



Sutton/AAAS

**When minis are blocked, dendrites make more protein (bottom).**

must also be necessary. The London group independently finds that infected BECs become more like LECs, and further finds that infected LECs become more like BECs. The final result is an intermediate cellular state that is neither BEC nor LEC, but presumably suits the virus just fine.



Boshoff/Macmillan

**Infected LECs and BECs converge toward a common phenotype.**

to complete its cycle.” He suggests that transcription factors used to determine the lymphatic phenotype are probably also used by the virus to aid its reproduction. ■

References: Hong, Y.-K., et al. 2004. *Nat. Genet.* 36:683–685.  
Wang, H.-W., et al. 2004. *Nat. Genet.* 36:687–693.