Tracks for cellulose

Cellulose synthase (CESA) tracks along paths coincident with microtubules, say Alex Paredez, Chris Somerville, and David Ehrhardt (Carnegie Institution, Stanford, CA). The resulting parallel cellulose fibrils constrain cell expansion so that plants elongate primarily along a single axis.

A transmembrane CESA complex takes cytoplasmic substrates and turns them into 36 extracellular glycan chains. At some distance from the complex, the extruded chains crystallize into a cellulose microfibril.

CESA’s relationship to plant cortical microtubules has been difficult to determine given the microtubules’ dynamic nature. Through rapid treadmilling and turnover, the microtubules bump into each other and realign, thus helping create a parallel array that is perpendicular to the axial direction of plant growth. In static pictures CESA was often nowhere near a microtubule, leading some to suggest that CESA was channeled between microtubule tracks rather than interacting with them directly.

Using live, single-particle imaging, however, the Stanford group saw that CESA was often coincident with microtubules, tracked along the microtubules, and reoriented in response to re-orientation of the microtubule arrays by light.

When a microtubule treadmilled away from CESA, the CESA complex kept going in a straight line as defined by the now-absent microtubule. This is consistent with the team’s belief that most if not all of the motive force comes from cellulose polymerization rather than a cytoskeletal motor. Extruded cellulose microfibrils bond to other cell wall polymers, so it is the CESA that must move forward as more cellulose is created. Any link between CESA and microtubules is yet to be determined. JCB

Reference: Paredez, A.R., et al. 2006. Science. doi:10.1126/science.1126551.

Flow makes vein

Leaf veins, just like roots and shoots, use flows of the plant hormone auxin to drive their patterning, say Enrico Scarpella (University of Alberta, Canada), Thomas Berleth (University of Toronto, Canada), and colleagues.

Models for leaf vein patterning have been based on either auxin flows or reaction–diffusion systems. The flow models had a hard time explaining how loops would arise. But “all the modeling so far has been based on the final pattern,” says Berleth. “Now flow can be reconciled with closed networks, although other types of cellular interactions may also play a role.”

His team used the auxin efflux protein AtPIN1 as an early marker of vein formation. Expression was found at a series of “convergence points” at the leaf margin, from where it led down paths that foreshadowed first the leaf’s main central vein and then other major veins connected to the main vein.

Auxin is presumed to flow from leaf margin to the interior, consistent with AtPIN1’s polarization to the parts of the cells closest to the main vein. Loops could also form, however, because certain cells had bipolar AtPIN1, allowing auxin flow to the main vein via two alternate routes. Auxin turns on AtPIN1 expression, thus refining and reinforcing the pattern.

Interfering with auxin flow disturbs vein formation and spacing. Vein placement seems to depend on the positions of convergence points, but those may be defined both by auxin’s positive feedback on its own flow and on undefined genetic circuits. JCB

Reference: Scarpella, E., et al. 2006. Genes Dev. 20:1015–1027.

Lamins as youth elixir

Ageing looks and feels like it is multifactorial: everything falls apart independently. But now Paola Scaffidi and Tom Misteli (National Cancer Institute, Bethesda, MD) report that multiple hallmarks of cellular aging can be reversed by eliminating one aberrant splicing product of lamin A.

The lamins form a structural cage on the interior surface of the nucleus. Lamin A has a long tail that is first farnesylated and then chopped off. In the 50 or so patients known to have the premature aging syndrome Hutchinson–Gilford Progeria (HGPS), an aberrant splicing event creates a lamin A that gets farnesylated but not cleaved.

The NCI team now shows that normal cells also have a small amount of this aberrant splice product. Although neither the splice product nor its protein product accumulate to higher levels with age, their effects do. As in HGPS cells, older cells have decreased heterochromatin and other nuclear markers, and increased markers of unrepaired DNA damage. Many of these changes were reversed by an oligonucleotide that eliminated the aberrant splice product.

Normal lamin A is found both at the nuclear periphery and within the nucleoplasm. But the aberrant splice product retains its farnesylation, and therefore gloms itself, and normal lamin A, onto the nuclear envelope. It is not clear how this leads to the many problems, although another group has suggested that lamin defects trigger a checkpoint that assesses nuclear envelope integrity. This, or some other mechanism that deals with the presence of the aberrant lamin product, must somehow be more sensitive in older cells. JCB

Reference: Scaffidi, P., and T. Misteli. 2006. Science. doi:10.1126/science.1127168.