WEB WATCH

The great divide

Some subjects always get left until the end at conferences: apoptosis, dephosphorylation and cytokinesis are classic examples. This web page, hosted by Julie Canman at the University of North Carolina, was formed by a group who were determined to fight back. In their words: "We are tired of cytokinesis being an afterthought in journal clubs, and of hearing groans every time the word cytokinesis is mentioned. To ensure proper representation of our cause, we have founded the Cytokinetic Mafia".

First and foremost, the cytokinetic mafia is a journal club. Papers are chosen by mafia members on a monthly basis; you can view their choices, but I was disappointed that members' discussions of the chosen papers aren't made available. Even so, this is a good place to visit if you want to know what's catching the cytokinesis enthusiast's eye this month. Not surprisingly, aurora kinases are *de rigueur*. The mafia also highlights a relevant book each week, but again there's no evaluation of them.

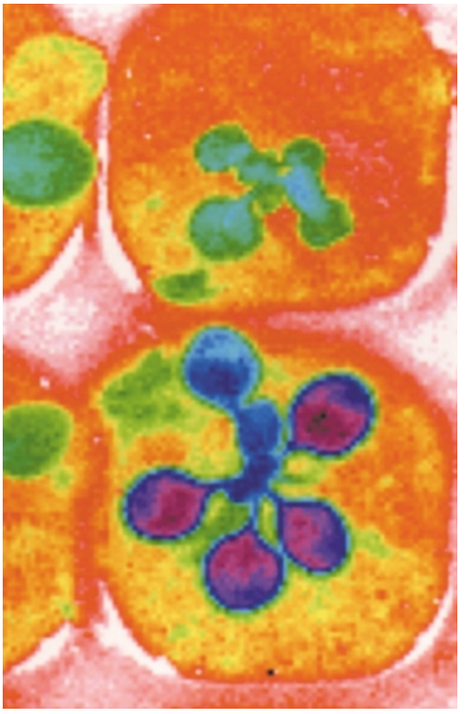
If it's movies you're after, then look no further. The movies page features dividing cells from several species, from bacteria to kangaroo rats. The asymmetric cell divisions in *Caenorhabditis elegans* are definitely worth a look, although you'll need to be patient about download times.

Last, but not least, the cytokinetic mafia is a community: you'll find lists of members, with contact details, and a comprehensive index of labs with an interest in cytokinesis. The mafia encourages new members and welcomes additions to the movie page from members and non-members alike. It's supposed to be a secret club though, so I'm half expecting to find a horse's head in my bed.

Cath Brooksbank

PLANT DEVELOPMENT

Stomatal waxing and waning



For plants, unlike animals, development is a continuing process. Whereas an adult animal's gross anatomy is pretty much fixed and determined by its genetic make-up, a plant's body is in permanent flux from the production of new organs, such as leaves and flowers. Plants use this plasticity to adapt to the different and changing environments in which they find themselves. In the 7 December issue of *Nature*, Alistair Hetherington and colleagues report that they have begun to dissect the pathway by which one such adaptation is controlled.

Perhaps the most obvious way in which plants react to environmental conditions is their phytochrome-controlled response to light. However, much subtler effects also exist. One of these is a decrease in the number of stomatal pores on leaves grown under CO_2-rich conditions.

Stomata are the main route for gas exchange in plants, but there are trade-offs to be made. A plant's demands to take up CO_2 as a raw material for photosynthesis must be balanced against its need to preserve water loss through transpiration. When CO_2 is plentiful, the pressure to conserve water is accommodated by a reduction in stomatal density. This inverse correlation has existed for over 400 million years — indeed, by counting fossil plant stomata ambient conditions can be estimated, providing clues to the causes of global extinctions. But what controls this ancient developmental response?

While investigating the results of a screen to detect tissue-specific expression in *Arabidopsis*, Hetherington and colleagues isolated a gene, which they named *HIC*, only expressed in stomatal guard cells. *hic* mutants produced leaves with more (rather than fewer) stomata in response to elevated CO_2. But functioning of the stomata was entirely normal — for example, the figure shows thermal imaging of *hic* (right) and *abil-1* (bottom left) plants. The *abil-1* plants are colder (more blue) due to increased heat loss through stomata that do not close efficiently.

Sequencing of the *HIC* gene showed it to be homologous to *KCS1*, which encodes an *Arabidopsis* 3-ketoacyl CoA synthase. KCS enzymes form part of microsomal fatty acid elongase complexes, which are involved in synthesizing the specialized waxes found in the plant's extracellular matrix. The authors showed that this is the source of HIC's developmental effects by identifying mutations of other genes involved in wax production, which also produce altered stomatal densities.

By changing the make-up of the leaf's extracellular matrix, HIC probably controls its physical properties. The regular spacing of stomata across the leaf's surface has led to the suggestion that stomata inhibit the production of other stomata in their immediate neighbourhood — perhaps by producing a diffusible suppressor of stomatal development. HIC's effect on the extracellular

PROTEIN DEGRADATION

Parkin finds a partner and a victim

Oliver Sacks' *Awakenings* familiarized many with the devastating symptoms of Parkinson's disease, and the fact that — dramatic though its initial effects may be — L-3,4-dihydroxyphenylalanine (L-DOPA) provides only temporary relief. We need a drug that targets the cause, not the consequences, of the disease. One rare cause, leading to autosomal recessive juvenile parkinsonism (ARJP), is mutation of parkin, an E3 enzyme that catalyses the transfer of ubiquitin from an E2 enzyme to proteins destined for proteasomal degradation. But what is the identity of the protein(s) whose death warrant is signed by parkin? Yi Zhang and colleagues, reporting in *Proceedings of the National Academy of Sciences*, have tracked down a substrate.

But first, they had to identify the E2 that parkin collaborates with. Database searches for parkin homologues uncovered two that interact with UBCH7 and UBCH8, so the authors reasoned that these E2s might interact with parkin. Co-immunoprecipitations showed that UBCH8 binds particularly tightly to parkin using its second RING finger domain, and that mutations that cause ARJP reduce or eliminate the interaction. *In vitro*, UBCH8 or UBCH7 allowed parkin to ubiquitylate itself — a property common among E3s. But a yeast two-hybrid screen also found another target for parkin — cell division control related protein 1 (CDCrel-1) — a protein implicated in blocking synaptic release. Wild-type parkin speeded up turnover of CDCrel-1 whereas