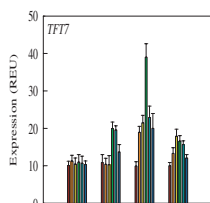


## Budding gene-ius

The variety of life forms amongst herbaceous angiosperms is well illustrated in the paper by **Alvarez *et al.* (Palmerston North, New Zealand, pp. 953–963)** dealing with branching patterns in *Lotus japonicus*. This species often has a ‘straggly’ appearance, at least partly due to its unusual branching pattern. It is well known that lateral branches arise in the axils of leaves. This may include seed leaves—cotyledons—although in many species the meristems in cotyledonary axils are inactive or rudimentary. However, in *L. japonicus* it is the axillary meristems of cotyledons that are major

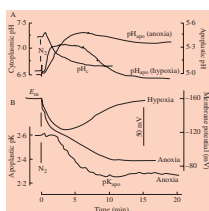
determinants of the branching pattern. Not only are these meristems active, but they also give rise to additional (accessory) meristems that are themselves active. Extensive lateral shoot formation thus originates from the cotyledonary axils. The authors have studied several aspects of the formation and activity of these meristems; we concentrate here on genetic control. The origin of axillary meristems is still uncertain: do they arise *de novo* in the axils, or are they derived originally from the shoot apical meristem (SAM) (the ‘detached meristem’ hypothesis)? Study by *in situ* hybridization of the expression of the *L. japonicus* version of the gene *SHOOT-MERISTEMLESS* (*STM*) suggests very strongly that during embryogenesis axillary meristems are indeed derived from the SAM and continue to express *STM* during the life of the plant. The accessory meristems then arise between the axillary bud and the cotyledon in part of the zone in which *STM* is expressed. However, the reason that these particular axillary meristems continue to throw off further meristems is not clear, although, as the authors state, the *Lateral Suppressor* (*LS*) gene may well be involved. Finally, the discovery of a mutant, *super-accessory-branches*, *sac* (which exhibits increased lateral branch formation from the axils of all leaves, not just cotyledons) provides a further tool for investigating this fascinating phenomenon.



## Numbers stack up for stressed tomatoes

I often tell my students that they can work out what an enzyme does from its name. However, when it comes to structural and regulatory proteins this simple idea may not always work. For example, a family of regulatory phosphoserine binding proteins are known as 14-3-3 proteins: the name comes from the numbers of particular structural domains in each molecule. They are already known to be involved in the regulation of many cellular activities, probably including stress responses. It is this topic that has been the subject of study by **Xu and Shi (Nanjing and Beijing, pp. 965–974)**. The

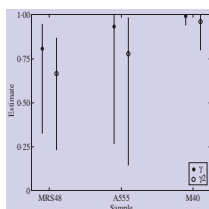
authors have firstly identified the members of the gene family encoding 14-3-3 proteins in tomato (*Solanum lycopersicum*). There are 12 genes in all, falling into two major groups. They have then subjected young plants to three different stresses: salt stress (exposure to 100 mM NaCl), potassium deficiency and iron deficiency. Expression levels in roots of the 12 genes were then determined by quantification by real-time RT-PCR of the mRNA concentrations before and after the application of the stresses. The authors point out the advantages of this technique for studying expression of several genes simultaneously, especially if any of the genes are expressed at low level, a point with which those of us who use this technique will readily agree. Their analysis showed that the 12 genes were expressed at very different levels in control roots: mRNA concentrations ranged from ‘not detectable’ to ‘high’. There were also differential responses to the stresses: eight of the genes were upregulated in response to K deficiency, one in response to Fe deficiency and four in response to salt stress. Only one gene, *TFT7*, was upregulated under all three stresses. These data support the view that 14-3-3 proteins are involved in stress responses and raise the possibility of cross-talk between signalling pathways involved in responses to widely differing stresses.



### Basic instinct—protons pulled back as air supply fails

Higher plants are essentially aerobic organisms. A complete lack of oxygen (anoxia) leads to a failure in mitochondrial electron transport. The subsequent impact on cytoplasmic processes is quite well documented. However, it is the contention of **Hubert Felle (Geissen, Germany, pp. 1085–1093)** that studies of this topic have ignored the apoplast. This is a very important compartment, having multiple functions that include transport, storage, defence and a limited range of enzyme catalysis.

So, what happens to the apoplast under low oxygen tension? Firstly, using non-invasive micro-probe techniques, the author showed that treatment of barley seedlings with fusicoccin, which stimulates the plasma membrane  $H^+$  pump, results, as expected, in hyper-polarization of the plasma membrane and an acidification of the apoplast. Cyanide, which inhibits mitochondrial electron transport, by contrast reduced  $H^+$  pumping, leading to a depolarization of the plasma membrane and alkalinization of the apoplast. However, the loss of  $H^+$  ions from the apoplast was not enough to explain the decrease in cytoplasmic pH, suggesting that  $H^+$  also entered the cytoplasm from another source (e.g. vacuole). In leaves exposed to anoxic or hypoxic conditions, responses were very similar to those seen with cyanide, but under hypoxia both membrane potential and apoplastic pH started to recover after a few minutes. A key question now arises: are these changes local or are they systemic? Plant shoots were maintained in air while the roots were kept anoxic under nitrogen. Very interestingly, after a delay, the apoplastic pH increased, as it did if roots were treated with cyanide. Signalling from root to shoot caused the leaf cell apoplasts to respond as if leaf cells were anoxic. However, this did not happen within the 2 h if the roots were simply flooded, possibly indicating that the oxygen tension in the roots did not, in the time of the experiment, drop low enough to trigger export of an ‘anoxia signal’.



### A chip off the very old block

A few years ago Alan Cooper, then director of the Henry Wellcome Ancient Biomolecules Centre (<http://abc.zoo.ox.ac.uk>), gave a research seminar here in Exeter. The seminar was totally fascinating, not only because he spoke about the range of information to be gained by the study of ancient DNA, but also because he described the rigorous precautions that must be taken to exclude any modern DNA contamination, including that emanating from the researchers themselves: it takes very little of the wrong DNA to give a misleading result. This point has been taken seriously by the international

research team of **Liepert *et al.* (pp. 1107–1111)**. They point out that very little work on ancient DNA has been done with plants, despite the potential of plant remains and sub-fossils to give useful information on plant distribution and human history. The authors have therefore set out to establish the ‘gold standard’ for studying ancient plant DNA. Samples of ancient wood were sent to several laboratories working with ancient DNA; for each source of wood, different code numbers were used for the different laboratories: not only were lab personnel unaware of the identity of the sample, but they were also unable to compare notes based on sample numbers. Two primer pairs were designed for PCR, such that the DNA bands obtained enabled researchers to identify the trees from which the wood samples were obtained. Preparation of the samples for DNA extraction and analysis of the DNA were done under the rigorous conditions mentioned above. Only results that were replicated in different laboratories were accepted as authentic. From the results it is clear that useful DNA samples can be extracted from 1000-year-old wood. Following Cooper’s advice about ancient DNA, ‘*do it right or not at all*’, has placed these authors in an excellent position to study ancient plant DNA in a broad range of research areas.

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