

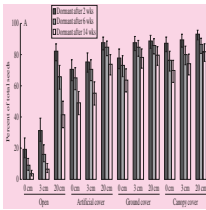
Fat SAM tests positive for hormone overdose

The changes in morphogenesis required to generate a strongly fasciated stem are very dramatic and it is of great interest that they may be caused by mutation of a single gene. Such is the case with sunflower, *Helianthus annuus*, where one such gene is known as *stem fasciated* (*stf*), as described by **Fambrini et al. (Pisa and Bologna, Italy, pp. 715–730)**. Mutations are recessive, implying that they cause loss of the function(s) controlled by the wild-type gene. The authors have set out to find, by comparing mutant with wild type, what this gene does, using morphological, histological and biochemical analyses. The phenotype of the mutant raises expectations that the shoot apical meristem (SAM) is likely to be distorted, leading to stem thickening and probably also to disturbance of the normal pattern of primordia formation. In the authors' very extensive study these expectations were indeed met. The vegetative SAM of the mutant was larger than that of the wild type; there was some distortion of one of the cell layers in the central zone, and the nuclei of both the peripheral and central zones were enlarged. Normal phyllotaxis was lost (perhaps because of the shape of the SAM) and, more surprisingly, the plastochron was shortened so that leaves were produced more frequently. Very interestingly, *stf* plants were significant over-producers of auxin, even allowing for the greater size of the apex. Further, decapitation of *stf* plants did not abolish apical dominance. The implication is that over-production of auxin in the apex led to higher concentrations further down the stem. However, the data did not bear this out: there are clearly other factors at work here. *stf* is thus a very interesting gene with effects on, amongst other things, the shape and size of the SAM, on tissue patterning and organogenesis in the SAM, and on the hormonal physiology of the apex.



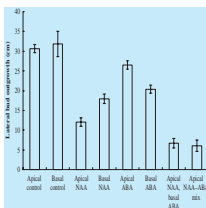
Obscure compounds, dark glands, healing balms

Polyketides and their derivatives hardly feature in our mainstream biochemistry teaching, but these compounds, derived essentially via condensation of acetate units, constitute in many plant species a significant proportion of the 'secondary metabolites'. Despite being relatively little known, polyketides have had their place in history: the lethal compound in hemlock extract, used to execute Socrates, is the polyketide coniine. However, these compounds have more benign uses. Extracts from different plants have been used in traditional medicine and preparations from *Hypericum perforatum* (St John's wort) are used extensively as a complementary remedy for depression. Active components are hypericin and pseudohypericin, in the naphthodianthrone group of ketide derivatives. The medicinal use of these compounds has led **Zobayed et al. (Chiba, Japan, pp. 793–804)** to investigate their distribution in the plant, their likely site of synthesis, and the relationship between accumulation and photosynthetic rates. The results show clearly that the distribution within the plant of hypericin/pseudohypericin is closely correlated with the presence of dark glands, one of several types of secretory structure possessed by *H. perforatum*. The denser the dark glands, the higher is the concentration of hypericin/pseudohypericin. Conversely, organs with very few dark glands contain very little of these compounds. Dark glands themselves differ little in content; it is the number of dark glands that matters. The densest array of dark glands is on the stamens, consistent with earlier findings that flowers are a rich source of hypericin/pseudohypericin. Further, in addition to indicating that dark glands are the likely organs of secretion of hypericin/pseudohypericin, the occurrence of the immediate precursor of hypericin suggests that at least the final stages of biosynthesis take place in the dark gland cells. Finally, the density of dark glands and hence the concentration of hypericin/pseudohypericin in leaves increases when the net photosynthesis rate increases, indicating a relationship between C fixation and hypericin synthesis.



The steamy, shady side of seed success and survival

One of the aspects of angiosperm biology that fascinates me is the great range of variation in seed physiology. Here I focus on the research of **van Klinken *et al.* (Indooroopilly, Queensland, pp. 875–883)**, working with the leguminous shrub *Parkinsonia aculeata*, seeds of which exhibit physical dormancy. The seeds are very hard with a palisade layer in the seed coat that is both tightly packed and water-repellent. Although dormancy breakage in hard-seeded species is not well understood, the authors propose, based on the ecology and distribution of *P. aculeata*, that wet heat is the main factor in this species. This would mean that dormancy is broken during the wet season in the plant's native habitat. To test this idea, a comprehensive seed burial experiment was carried out (depths from 0 to 20 cm) under different shade conditions for different lengths of time. The mean air temperature was 28 °C (giving a mean temperature at the soil surface of 43.6 °C in open ground; this value decreased with both soil depth and shading). Over 1000 mm of rain fell in the 14-week period, most of it in the first 6 weeks; the soil was at 100 % field capacity between weeks 2 and 6, but fell to 27 % between weeks 6 and 14. Analysis of the germination data showed very clearly that, as predicted, wet heat is the key factor in breaking dormancy. Thus, dormancy was broken very quickly in seeds 'buried' at 0 cm in open ground but less quickly as depth of burial increased. Shading had a major inhibitory effect on dormancy breakage so that in both ground-cover and canopy-cover sites, most of the seeds were still dormant after 14 weeks, the actual figure increasing with depth of burial. These features thus ensure wet-season germination and provide both a system for detecting gaps in the canopy and a capacity for seed banking.



The ups and downs of hormone treatment

Many of us will remember those simple student experiments on apical dormancy, using decapitated *Phaseolus coccineus* plants and blobs of lanolin paste containing auxin. We concluded that auxin derived from the apex and transported down the stem normally suppressed lateral shoot growth. However, we now know that it is more complicated; indeed, even now, there are details that remain to be elucidated, as so clearly presented by **Morris Cline and Choonseok Oh (Columbus, OH, pp. 891–897)**. Firstly there have been suggestions that it is ABA, synthesized or released in response to auxin and transported acropetally (towards the apex), that inhibits lateral shoot growth. However, other authors have rejected this view and postulate instead that an as yet unidentified, carotenoid-derived inhibitor, transported acropetally, is the real inhibitor. The loss of apical dominance in mutants that lack carotenoid-cleaving dioxygenase is cited as further evidence for this view. The present authors have therefore carried out classical decapitation and hormone-replacement experiments with *Ipomoea nil* (morning glory), *Solanum lycopersicum* (tomato) and *Helianthus annuus* (sunflower). In all three species, auxin could replace the apical bud in inhibiting outgrowth of laterals, as in our classical experiments. Indeed, there was even some inhibition of lateral shoot growth if auxin was applied basally; this was particularly marked in *S. lycopersicum*. Apical application of ABA to decapitated shoots did not restore apical dominance but there was moderate inhibition of lateral shoot growth if ABA was applied basally in *I. nil* and *S. lycopersicum*. Indeed, in the former, the effects of basally applied ABA and apically applied auxin were additive. ABA therefore does indeed move acropetally but in these experiments can at most only partially replace the inhibitory effects of auxin. A role for a third player, the unknown carotenoid derivative, remains a viable possibility, in addition to which the variation between species is a feature of note.

Professor J. A. Bryant
 University of Exeter, UK
 E-mail j.a.bryant@exeter.ac.uk