Pseudocerus bifurcus, behaviours such as penis-fencing are favoured to avoid receiving sperm [19]. Thus, the opposite pattern of a universal preference for playing the male role can also emerge.

Nevertheless, the work of Anthes *et al.* [5] is exceptional in providing definitive evidence for sperm trading in hermaphroditic sexual reproduction. Moreover, this work provides clear evidence of male 'mate choice' in the form of selective sperm donation to 'honest' partners. Alone, such features should earn this study a place in the text books; more so since it also provides a rare unequivocal example of conditional reciprocity being employed to escape the tragedy of the commons in biology.

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Plant Meristems: Mobile Mediators of Cell Fate

How do transcription factors control the fates of cells that express them? One class of plant transcription factors has recently been shown to function by regulating the synthesis of cytokinin and gibberellin hormones — mobile molecules more usually associated with long-distance signalling.

Andrew Hudson

Cell fates at the apex of plant shoots are controlled by homeobox transcription factors of the KNOX-I family. *KNOX-I* genes act as selectors of meristem cell identity; their activity is needed to distinguish cells of the shoot apical meristem (SAM) from those of leaves (Figure 1A), and ectopic *KNOX-I* expression can confer SAM-like identity on leaves. For any transcription factor that controls cell identity, one major question is how that identity is realised through regulation of target genes. Two papers [1,2] published recently in *Current Biology* report evidence that two plant hormones, gibberellin and cytokinin, together mediate the KNOX-I control of SAM cell identity.

Control of cell fate in the SAM has long been known to involve *KNOX-I* genes. *KNOX-I* expression is characteristic of the SAMs of diverse land plants [3] and is lost from peripheral cells as they are specified as leaf initials (Figure 1B). For example, activity of the *KNOX-I* gene *SHOOT MERISTEMLESS* (*STM*) is needed to prevent cells of the *Arabidopsis* apex expressing leaf genes and differentiating, giving rise to embryos without SAMs [4]. Conversely, ectopic *STM* expression in developing leaves confers characteristics of the peripheral SAM and is sufficient to specify complete SAMs when expressed ectopically with the distantly related transcription factor WUSCHEL, which promotes central cell identity [5].

Earlier work [6] had shown that STM is needed in the SAM to maintain low gibberellin levels and inhibit expression of the *GA20-ox1* gene, which encodes a rate-limiting enzyme of gibberellin biosynthesis. *GA20-ox1* expression is normally confined to leaves, where gibberellin levels are high, but exluded from the apex by STM activity. Two lines of evidence suggested that repression of *GA20-ox1* by STM is functionally relevant. Firstly, the interaction is likely to be direct — KNOX-I protein can bind a regulatory sequence in the GA-20 oxidase gene of tobacco [7]. Secondly, the effects of KNOX-I activity are partly dependent on an ability to respond to gibberellin. For instance the *spindly* (*spy*) mutation, which mimics high gibberellin levels by allowing a constitutive gibberellin response [8], enhances the effects of weak *stm* mutations and

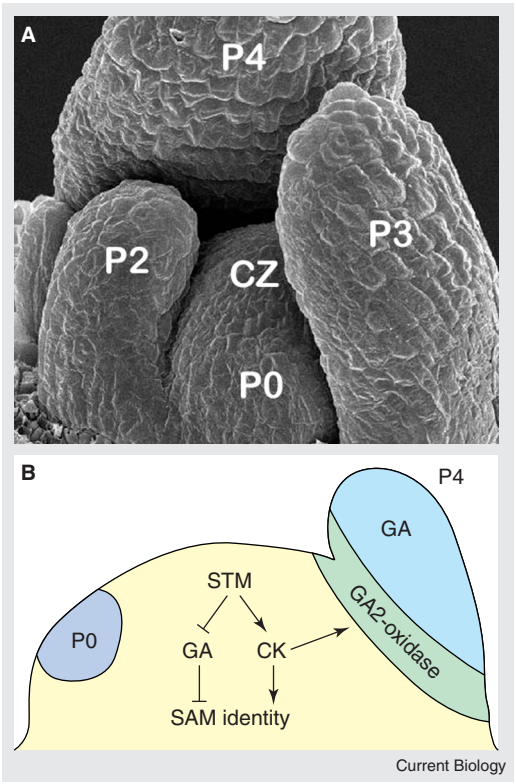

Figure 1. The shoot apical meristem.
(A) A shoot apical meristem (SAM) of *Arabidopsis*. The progeny of cells in the central zone (CZ) assume identity of leaf initials (P0) as they pass to the periphery and subsequently grow out as leaf primordia (P2–P4, in order of age). Cells between leaf primordia give rise to the stem tissues that separate leaves of the mature shoot. (B) A model for STM function. *KNOX-I* transcription factor genes, such as *STM* depicted here in yellow, are expressed in SAM cells but not in cells fated to form leaves, shown in blue. STM promotes non-leaf identity by repressing gibberellin synthesis and promoting cytokinin synthesis. The resulting low gibberellin, high cytokinin environment promotes SAM cell identity. In developing leaves, expression of gibberellin biosynthetic enzymes leads to high gibberellin levels. Before gibberellin can reach the SAM it is deactivated by GA2-oxidase, expressed at the SAM–leaf boundary and promoted by cytokinin from the SAM. (Photo courtesy of Paulo Piazza and Miltos Tsiantis.)

conversely represses the effects of ectopic *KNOX-I* expression [5].

The ability of *KNOX-I* genes to promote SAM identity by imposing low levels of gibberellin is consistent with the ability of gibberellin to promote expansion of differentiating cells and to discourage formation of shoots in tissue culture. The role of STM cannot, however, simply be regulation of gibberellin synthesis. For example, increased gibberellin response in the *spy* mutant does not cause a shoot meristemless phenotype equivalent to loss of STM activity [8]. STM is therefore likely to regulate other targets needed for SAM identity.

Several lines of evidence had previously implicated another class of plant hormones — cytokinins — as an additional target of KNOX-I control. For example, ectopic *KNOX-I* expression increased cytokinin levels in leaves of several species and could cause typical cytokinin responses, including delayed senescence [9]. Are cytokinins therefore involved in mediating control of the SAM by *KNOX-I* genes? This question was addressed recently by two groups working independently [1,2]. Both exploited transgenic plants expressing a hybrid STM protein that could be activated ectopically by applying a synthetic steroid. They first confirmed that STM increased cytokinin levels when activated in leaves. They then used a transgene that is responsive to cytokinin signals to monitor cytokinin activity *in situ*, finding high levels in the SAM of wild-type plants and ectopic activity in leaf primordia induced to express STM. Increased activity appeared to involve increased cytokinin synthesis, because STM caused rapid up-regulation of two genes encoding isopentenyl transferase (IPT), an enzyme that catalyses the final stage of cytokinin biosynthesis.

Yanai *et al.* [2] reasoned that, if promoting cytokinin activity is an important aspect of STM function, cytokinin should substitute for STM activity. This proved to be the case — applying cytokinin or expressing an IPT gene from the *STM* promoter allowed null *stm* mutants to produce functional SAMs. Conversely, Jasinski *et al.* [1] reasoned that weak *stm* mutants should have intermediate levels of cytokinin and therefore be sensitive to further reductions in cytokinin responses caused by loss of the cytokinin receptor WOODENLEG (WOL) [10]. As predicted, loss of WOL activity strongly enhanced the phenotype of weak *stm* mutations.

These experiments supported the hypothesis that STM acts in the SAM by repressing gibberellin levels and increasing cytokinin levels (Figure 1B). Jasinski *et al.* [1] further tested the effects of mimicking a leaf-like hormonal environment by overexpressing an enzyme that degrades cytokinin and using the *spy* mutation to increase gibberellin signalling. Decreasing cytokinin levels led to a shoot meristemless phenotype only in a *spy* mutant background. Thus regulation of gibberellin and cytokinin levels can account for the role of STM in promoting SAM identity in embryogenesis, although STM might have other direct targets later in development. Other potential targets of direct KNOX-I control include genes involved in the synthesis of the cell wall polymer, lignin [11]. The enhanced effects of low cytokinin in a *spy* mutant background also suggested that gibberellin and cytokinin are regulated independently by STM, and not, for example, that STM decreases gibberellin which in turn increases cytokinin.

Jasinski *et al.* [1] did, however, find evidence for a subtle interplay between the two hormones involving genes (*GA2ox*) encoding GA2-oxidase, an enzyme responsible for deactivating gibberellin. As for an orthologue in rice [12], the *Arabidopsis GA2ox* genes are expressed at the base of leaf primordia where they might prevent gibberellin synthesised in leaves reaching the SAM. Expression of one *GA2ox* gene was promoted by cytokinin; its expression domain was expanded by applying cytokinin and reduced in the cytokinin receptor mutant *wol*. Therefore cytokinin might promote destruction of gibberellin at the SAM-leaf boundary by

increasing GA2ox activity. This interaction is suggested as a mechanism that could help refine the boundary between cells with SAM or leaf identity — a process which is expected to be particularly important when the molecules mediating cell fate are mobile.

Gibberellin and cytokinin have antagonistic effects in a number of processes — suggested to reflect convergence of cytokinin and gibberellin signals on the SPY protein [13] or incompatibility in the effects of cytokinin on cell division and gibberellin on cell expansion [8]. Such antagonism could further discourage specification of cells with intermediate identities at the SAM-leaf boundary. One of the many questions raised by these findings is how a high concentration of cytokinin, which can affect leaf development [14], is itself restricted to the SAM.

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Evolution: A Study in Bad Taste?

Bitter tastes are among the most salient of life's experiences — who can forget one's first encounter with dandelion milk or a stout beer? Studies of the genes underlying these tastes are providing new perspectives on human origins and health.

Stephen Wooding

Bitter-taste sensitivity, of course, begins on the tongue. Concentrated at the back of the tongue, on disc-like structures called circumvallate papillae, specialized bitter-taste receptor cells await contact with potentially bitter compounds. Upon exposure to an appropriate ligand, these receptor cells depolarize, generating a signal that is conveyed via the facial and glossopharyngeal nerves to the brain (Figure 1A). In principle, any mechanism that stimulates this neural pathway will lead to the sensation of bitter taste; however, recent studies have highlighted the importance of a small group of G-protein-coupled receptors encoded by the *TAS2R* (also called *T2R*) gene family [1,2].

In humans, this family includes roughly 25 functional genes and eight pseudogenes, each roughly a kilobase in length, found in three clusters on chromosomes 5, 7 and 12. The protein products of these genes are concentrated at the apex of bitter-taste receptor cells, near the taste pore, where they are positioned to bind bitter ligands as they wash past, dissolved in saliva (Figure 1A). Upon ligand binding, these receptors catalyze a series of reactions leading to the efflux of intracellular calcium, and the cascade of events leading to taste perception begins (Figure 1B).

Considerable effort has been directed at identifying ligands for these receptors, and a range of compounds have been identified that are capable of activating TAS2R10, TAS2R14, TAS2R16, TAS2R38, TAS2R43, and TAS2R44 and TAS2R61 [3–7]. These studies have produced a variety of interesting surprises. The artificial sweetener saccharin, for instance, activates TAS2R43 [6]. More striking, however, is the observation that an inordinate fraction of the compounds that activate the TAS2Rs are secondary compounds produced by plants. Further, many of these compounds are toxic, used by plants as means of defense against herbivores. TAS2R10, for instance, binds strychnine [3], the well-known toxin found in plants in the genus *Strychnos*, and TAS2R14 binds α-thujone,