is that the high metabolic cost of the Na^+ and Ca^{2+} pumps in darkness results from the rod being depolarised: when stimulated by light, the rod hyperpolarises and Na^+ and Ca^{2+} channels close. With their closing, the extent of which is proportional to light level, the influx of Na^+ and Ca^{2+} ions declines, fewer ions need to be pumped out to maintain steady state, and energy expenditure plummets. Even though rods and cones are similarly expensive in the dark — and for similar reasons [11,12] — illumination of cones never closes all of the outer segment channels, even at highest light intensities [13]. This means that in bright light the Na^+ pumps of cones need to work harder to maintain steady state. In addition, recent experiments indicate that the biochemistry of transduction uses more ATP in cones than in rods. This extra energetic cost makes cones more 'expensive' than rods.

This remarkable fact — that rods are cheaper than cones — has profound implications for our understanding of the evolution of vision. As is becoming increasingly clear, the energy costs associated with maintaining neural tissues are significant [5,14,15] and have been a major selective pressure during the evolution of nervous systems, not the least the senses [3,6,15]. As Darwin certainly would have appreciated, better performance is likely in larger sensory organs with greater numbers of neurons. But in natural selection this benefit must always be weighed against the cost, since more neurons consume a greater proportion of the animal's total energy budget. Thus, the cheaper cost of running rods in bright light may explain why the vertebrate duplex retina evolved, why in most mammals (even diurnal ones) the rods greatly outnumber the cones, and why in diurnal species these relatively few cones are usually restricted to a small region of the retina (the fovea). By having two sets of photoreceptors adapted to different light levels, with one set (comprising the majority of receptors) consuming little energy for half of the day, the total cost and performance of vision over a 24-hour period can be optimised.

Energy arguments may also explain why vertebrate photoreceptors hyperpolarise in response to light. Insect photoreceptors also consume a considerable amount of ATP in darkness, and for the same reason as a rod or cone: to bias the synaptic transmitter release into a sensitive region of its range. But in contrast to rods and cones, insect photoreceptors depolarise in response to light and the energetic costs increase with light intensity [6] (Figure 1B). The benefit they gain from this investment is the ability to resolve rapid contrast changes in bright light [6]. But now, seen in the light of photoreceptor costs, it is perhaps not surprising that nocturnal arthropods, which have evolved elaborate strategies to optimise vision at night [16,17], also restrict retinal illumination during the day, by employing pupil mechanisms [18], by reducing the volume of their phototransductive membranes before dawn [19] or by simply retreating to a dark hide.

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Department of Cell & Organism Biology, Zoology Building, University of Lund, Helgonavägen 3, S-22362 Lund, Sweden.
E-mail: Eric.Warrant@cob.lu.se

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Leaf Development: Untangling the Spirals

How do plants position their leaves and flowers around the stem in such regular patterns? Auxin is well established as an essential regulator. Now, the modification of a structural cell wall component is shown to have a dramatic impact.

Naomi Nakayama and Cris Kuhlemeier

New leaves and flowers form in ordered patterns, a process called phyllotaxis [1,2]. The most common type is spiral phyllotaxis, in which the lateral organs are initiated in an equiangular spiral with a higher order organization of overlapping spirals in opposite directions. Phyllotactic patterning takes place in the shoot apical meristem, a dome of tissue at the tip of the stem, which contains stem cells that supply cells for continuous organogenesis. New lateral organs always emerge at the flank of the meristem in the peripheral zone, where

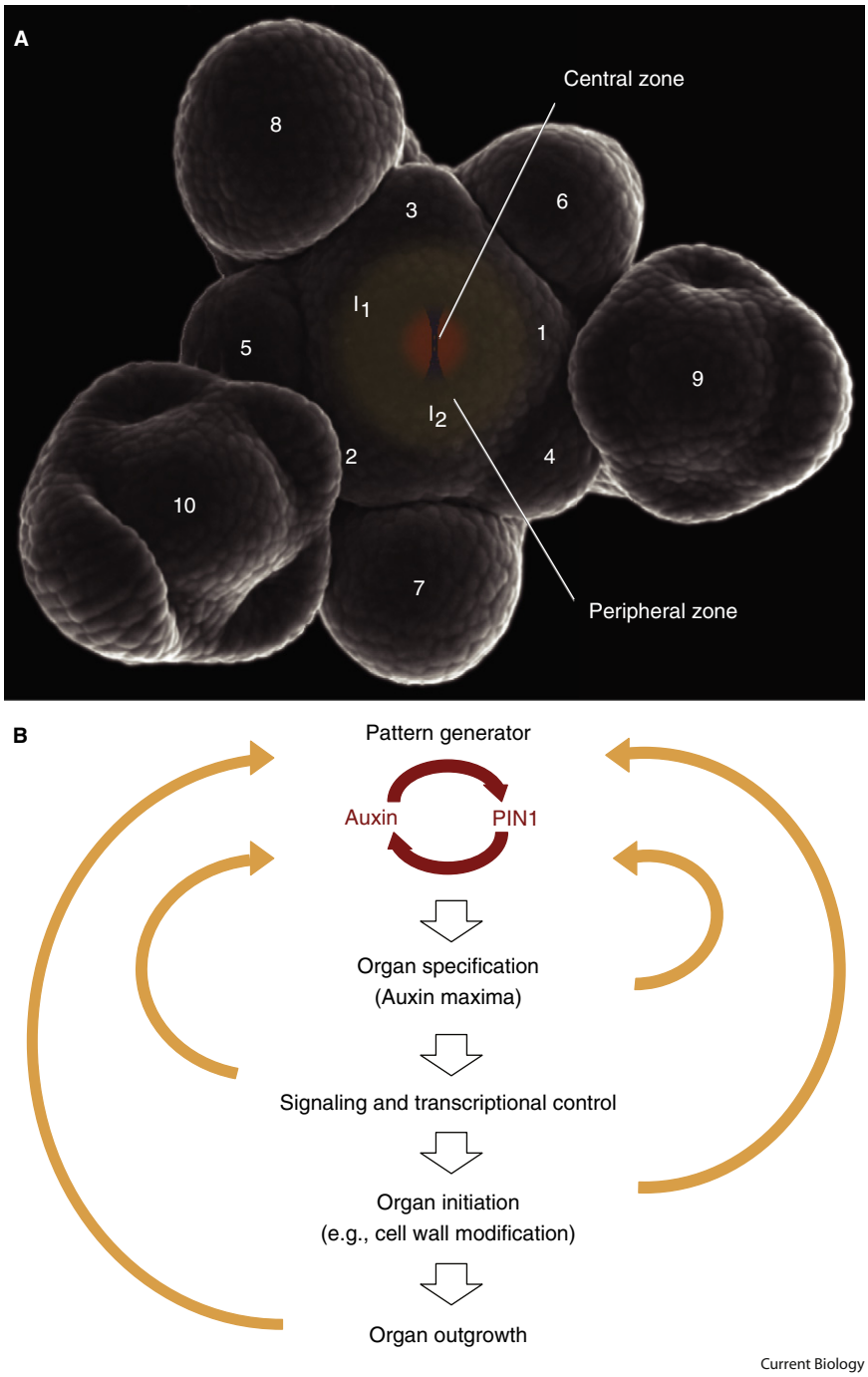

Figure 1. Pattern generation by auxin transport and feedback loops.

(A) An *Arabidopsis* inflorescence shoot apical meristem. The central zone contains the stem cells; their descendants in the peripheral zone are competent to form organs. The individual flowers (labeled 10 through 1 in order of appearance) form in a spiral phyllotaxis with divergence angles of approximately 137°. I_1 and I_2 predict the positions of incipient primordia (scanning electron micrograph by Soazig Guyomarc'h). (B) A conceptual representation of auxin-based regulation of phyllotaxis. A positive feedback loop between auxin and its transporter PIN1 creates a local auxin maximum and depletes auxin around it. The auxin maximum induces the activation of a signaling cascade, which in turn induces local wall modification. Wall modification is a prerequisite for localized outgrowth. The orange arrows indicate hypothetical feedback controls at all levels. Peaucelle *et al.* [13] demonstrate that the wall modifying enzyme PME is necessary for organ formation and that PME misexpression alters phyllotaxis.

cells are competent for differentiation and responsive to organogenic cues (Figure 1A). How plants can position their organs with such mathematical precision has tickled curious minds past and present, and much effort has been made to elucidate the mechanisms behind the pattern formation.

The heart of the phyllotactic patterning system is a pattern generator. Its primary output is a spatially and temporally restricted signal, which, through a cascade of molecular events, causes organ formation (Figure 1B). Evidence has accumulated that the phytohormone auxin is the primary output signal and that a positive feedback loop between auxin and its transporter PIN1 can pattern phyllotaxis [3–6]. Auxin accumulation precedes leaf specification and is necessary and sufficient for leaf initiation; suppression of auxin transport abolishes organogenesis and results in naked, radially symmetric meristems, while local application of auxin is sufficient to restore it [4,7,8]. Reactivation of auxin transport can re-establish normal phyllotaxis from the organless meristem, indicating that the auxin/PIN1 loop is capable of *de novo* pattern formation [8]. The auxin maxima are interpreted by transcriptional regulators and signaling molecules, which translate the primary output into activation of the organ development program.

The simple concept of a pattern generator with downstream signaling and effector genes is complicated by the feedbacks that operate in the system. A critical feedback is the negative influence of pre-existing primordia: they inhibit new organogenesis in their vicinity. Developing organs are thought to be sinks for auxin; they help prevent auxin accumulation around them and thereby enable pattern formation [4]. Other feedbacks are necessary both to stabilize the system and to enable it to respond to external stimuli. For instance, auxin-dependent transcription factors not only induce downstream genes but also feedback on the pattern generator by regulating the expression of PIN1 [9]. In principle, any factor involved in organ initiation or formation could alter phyllotaxis.

Further downstream effectors can also feedback on the central pattern