glucan phosphate, and biologically inert β-glucans such as laminarin [9].

There is a notable difference between Dectin-1 and the Toll-like receptors (TLRs) in terms of their ability to respond to soluble factors. The TLR family of pattern recognition receptors sense soluble microbial factors and are activated by dimerisation of intracellular signalling domains, resulting in stimulation of inflammatory responses. Although the TLRs are highly sensitive, their signalling is also tightly regulated through control of their localisation and trafficking, and at transcriptional, post-transcriptional and post-translational levels [10,11]. On the other hand, the regulation of Dectin-1 signalling is not very well understood. Unlike many C-type lectin receptors, it does not appear to have a paired inhibitory receptor to regulate its function. It is possible that the unusual ITAM may be a means of regulation. Although there are parallels between Dectin-1 signalling and receptors that signal via traditional ITAMs, there are also differences. For instance, it is thought that Syk binds two singly phosphorylated ITAMs on adjacent clustered Dectin-1 receptors [2], a feature which may result in weaker or 'regulated' signal transmission compared with traditional ITAM signalling where a single Syk family kinase is recruited to one doubly phosphorylated ITAM.

The phagocytic synapse proposes a model mechanism for the specific detection of factors associated with a microbial surface, as opposed to those shed from distantly located organisms, and it may also be a method of direct regulation of Dectin-1 and other phagocytic receptors. To a certain extent it resembles the immunological synapse between the TCR and peptide–MHC complexes, an interaction which is critical at later stages of an immune response. The phagocytic synapse, which is more important in early immune responses, may be an evolutionary precursor of the immunological synapse. It will be interesting to see how this model applies to other pattern recognition receptors involved in innate immune responses.

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Phyllotaxis: In Search of the Golden Angle

How are the regular patterns of organs established along a plant stem and how are the transitions between different patterns regulated? Now genes of the *PLETHORA* family have been shown to modulate these transitions by fine-tuning the mechanisms of polar transport of auxin, a key effector of organogenesis.

Jean-Christophe Palauqui and Patrick Laufs

The regularity of the arrangement of leaves, flowers or floral organs in plants, a phenomenon called phyllotaxis, can not only be easily observed by anyone, but it is also the source of inspiration for scientists since the classical antiquity [1]. Depending on the position and number of organs that are formed on a given point along the stem (a node), different pattern types can be distinguished. For instance, if leaves grow one by one, each at a constant angle from the previous one, the phyllotaxis is called distichous when the angle is 180° or spiral when the divergence angle is close to the golden angle, about 137.5° [2]. When two evenly spread organs are formed on a node, the phyllotaxis is decussate, and whorled when three or more organs arise simultaneously. Although phyllotactic patterns tend to be stable, they are affected by environmental factors and may change during the development of the plant. For instance, in the model plant *Arabidopsis thaliana*, the embryonic leaves and the two first vegetative leaves show a decussate pattern before switching to a spiral phyllotaxis for later vegetative leaves and flowers, and finally to a whorled pattern for floral organs (Figure 1). Now, in this issue of *Current Biology*, Prasad, Grigg *et al.* [3] reveal a role for genes of the *PLETHORA* family in the control and the transition of phyllotaxis.

Phyllotaxis emerges in the meristems, specialized structures that combine a self-renewing group of undifferentiated cells in their centre with continuous initiation of primordia from their periphery in a spatially and temporally controlled pattern [2]. To understand the basis of the phyllotaxis it is necessary to decipher the mechanisms behind the regular pattern of organ initiation in the meristem.

Genetic and molecular evidence has established a central role for gradients of a phytohormone, auxin, in organogenesis at the meristem. Peaks of auxin mark the position of the incipient organ primordia and local application of auxin can trigger organ formation [4–6]. These gradients of auxin are established and refined by the activity of membrane-localized transporters, which can either direct auxin influx or efflux, creating cell-to-cell polar auxin transport [4,6,7]. In particular, polarization of the PIN1 auxin efflux transporter plays a key role: inactivation of the *PIN1* gene or pharmacological inhibition of auxin efflux transport with a drug called NPA abolishes organogenesis, and auxin flux modeling based on subcellular PIN1 distribution efficiently predicts auxin distribution in the periphery of the meristem [5,8]. Gradients of auxin are translated into coordinated changes in gene expression and signaling networks required for proper organogenesis [9]. This includes activation of cell wall modifying enzymes such as expansins or pectin methyl-esterases (PME) that may contribute to differential growth associated with organ formation through modification of cell wall properties [10,11]. Interestingly, local application of expansins or PME is able to trigger the formation of ectopic organs and modifies phyllotaxis [10,12], but it remains currently unknown how this in turn feeds back to auxin signaling in the meristem.

A central issue of the organ patterning process in the meristem is the feedback provided by older organs on the position of new emerging organs. Both micro-surgery experiments in which the effects of the displacement of older organs on pattern formation was studied and modeling approaches indicate that preexisting primordia inhibit new organogenesis in their neighborhood [13,14]. This inhibition may result from drainage of auxin by the nearby organ primordia that act as sinks. Alternatively, physical constraints may also contribute to the propagation of phyllotaxis in the meristem [15]. Recently, PIN1 sub-cellular polarisation was shown to respond to mechanical signals, thus providing a possible link between mechanics and auxin signaling [16]. Although it is clear that the expression of *PIN1*, the dynamic subcellular localization and the activity of PIN1 are important for controlled morphogenesis in the meristem, the factors controlling *PIN1* expression remain largely unknown.

In their study, Prasad, Grigg *et al.* [3] shed new light on the control of phyllotaxis and clearly demonstrate that genes that belong to the *PLETHORA* (*PLT*) family of transcription factors play a role in the control of foliar and floral phyllotaxis. Thus, this gene family, which was already known to be involved in the regulation of the stem cell niche and cell differentiation in the root meristem [17,18], is now shown to play a role in the shoot apical meristem as well. Strikingly, triple *plt3plt5plt7* mutants are delayed in their transition from decussate to spiral phyllotaxis during the vegetative phase and display a defective phyllotactic pattern in the inflorescence with successive flowers diverging by about 180 or 90°.

Because organ initiation and thus phyllotaxis is subordinated to polar auxin transport, they investigate the connections between phyllotaxis defects of *plt* mutants and *PIN1* expression. In fact, partially reducing *PIN1* expression or blocking PIN1-dependant polar auxin transport mimics the vegetative phyllotaxis defects of *plt* triple mutants. Furthermore, the weak phyllotaxy defects of *plt* double mutants are strongly increased when *PIN1* gene dosage is reduced or following NPA treatments. This provides strong evidence that PLT and PIN1 act together to control the changes in phyllotaxis, and suggests that PLT-induced spiral phyllotaxis involves a PIN1-dependent mechanism.

Does the effect of PLT on phyllotaxis rely on a PLT controlling *PIN1* gene expression? To answer this question, the authors show that the strong accumulation of PIN1 protein at the site of incipient primordia observed in wild-type plants is no longer present in the *plt* triple mutant. Instead, PIN1 protein is evenly distributed in the peripheral zone, although subcellular polarization was not obviously perturbed. Then they provide arguments in favor of transcriptional control of *PIN1* gene expression by PLT5, suggesting that a local enhancement of *PIN1* gene expression mediated by PLT proteins contributes to a normal phyllotactic pattern. Indeed, specific expression of PIN1 in incipient primordia partially rescued the defective phyllotaxis of the *plt* triple mutant.

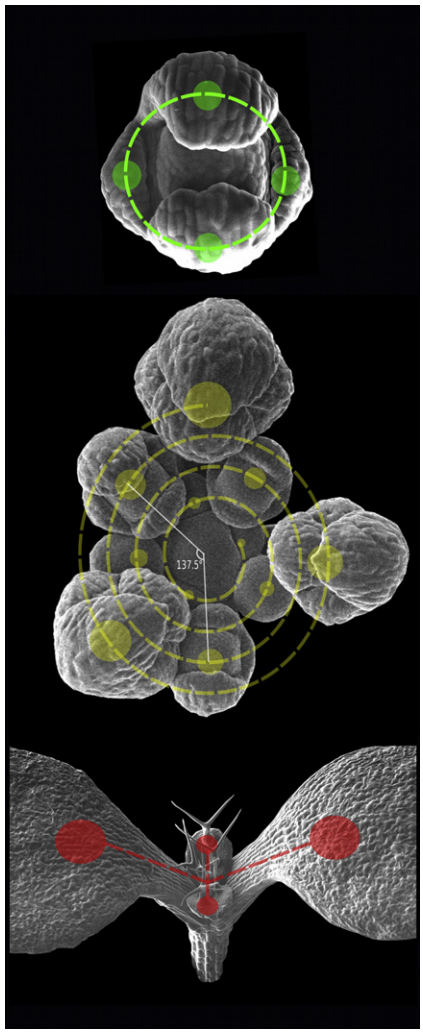

Figure 1. Phyllotaxis transitions in *Arabidopsis thaliana*.

The two embryonic leaves and the first pair of vegetative leaves arise in a decussate pattern (bottom). In the inflorescence, flowers arise along a spiral, separated by a divergence angle close to the golden angle (137.5°, middle). Finally, in the flower, floral organs show a whorled pattern (4 emerging sepals are shown in the upper panel).

This study raises a number of questions: how does a rather uniform expression of the PLETHORA genes contribute to the local accumulation of PIN1 in young primordia? Does this involve crosstalk between PLT, PIN1 and auxin? The observation that, in the root, the expression of *PLETHORA* genes is regulated by PIN proteins and more generally by auxin [17,19] supports this hypothesis. Alternatively, it is possible that the effect of

PLETHORA genes is at least partially mediated by local changes in growth patterns and mechanics. The meristem of *plt* triple mutants is slightly smaller than in the wild type [3] and members of the *PLETHORA* clade have been shown to control growth [20], suggesting that *PLETHORA* genes may modify growth and mechanical forces within the meristem. Since changes in mechanics can modify PIN1 polarity [16], and hence auxin distribution, which in turn can modify PIN1 expression level, the link between PLT and PIN1 may be indirect, despite the fact that increased PIN1 transcript levels are observed 4h after PLT5 activation [3]. Elucidating the mechanism underlying PLT-mediated control of phyllotaxis will be challenging and likely depend on quantitative descriptions and modeling of PLT expression, PIN1 levels and polarization, auxin distribution, growth and mechanics.

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Olfactory Coding: Giant Inhibitory Neuron Governs Sparse Odor Codes

Electrophysiological investigations in locusts have revealed that the sparseness of odor representations, in the brain region expected to mediate olfactory learning, is shaped by a unique inhibitory neuron.

Nitin Gupta and Mark Stopfer

Brain mechanisms have evolved to gather and organize sensory information. This information does not flow passively from the outer environment through neural circuits, coming to rest as memories or actions. Rather, information is encoded, processed, and dramatically transformed in myriad ways as it travels through the brain, providing multiple advantages to the animal. For example, in many species and brain areas, sensory stimuli elicit dense bursts of action potentials from neurons in peripheral structures, but sparser firing in more central structures [1–3]. Working in the well-characterized olfactory system of the locust, Papadopoulou *et al.* [4] have recently uncovered an influential new participant in the process by which neural representations become more sparse — a singular, giant GABAergic neuron that regulates the output of tens of thousands of cells.

In the first olfactory processing center of the locust, the antennal lobe, any given odor elicits torrential bursts of action potentials, arranged in complex patterns, from a large portion of the projection neurons which transmit olfactory information further downstream (Figure 1A). But, in the mushroom body — an area that immediately follows the antennal lobe and participates in olfactory learning — odors elicit very few spikes in just a small fraction of the 50,000 intrinsic neurons, the Kenyon cells. Thus, as information moves from the antennal lobe to the mushroom body, its coding format changes from dense to sparse. Several neural mechanisms contribute to establishing and maintaining sparseness [5,6]. Within the antennal lobe, local circuitry establishes an oscillatory rhythm that synchronizes the firing of projection