Axillary Meristem Development. Budding Relationships between Networks Controlling Flowering, Branching, and Photoperiod Responsiveness[1]

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Morphology in many animals is preordained during embryonic development and remains unchanged by environment. In contrast, vast differences in phenotype can occur in plants of identical genotype in different environments. Being sessile organisms, plants must rely on morphological and physiological plasticity to cope with a variable environment. The basis for this ability is the maintenance of numerous pluripotent stem cell clusters called meristems.

During embryonic development, plants produce a shoot and a root apical meristem. It is unclear whether axillary meristems are produced de novo in leaf axils or whether they are derived from the apical meristem of the primary shoot (Grbiæ and Bleecker, 2000). Variation in the timing of the initiation and development of axillary mersitems can be observed by comparing the rosette crucifer, Arabidopsis, with the caulescent legume, pea (*Pisum sativum*). Arabidopsis has delayed axillary meristem initiation, causing some nodes to be devoid of axillary meristems (Grbiæ and Bleecker, 2000). Subsequent development of these meristems may also be delayed such that a pronounced axillary bud is often not observed. Pea develops axillary meristems at most nodes along its stem, and development usually proceeds apparently uninhibited up to the stage of a dormant bud that consists of several undeveloped leaves and internodes.

The nodes at which axillary meristems and/or branches occur along the stem are influenced by photoperiod in pea (Arumingtyas et al., 1992; Napoli et al., 1999) and Arabidopsis (Grbić and Bleecker, 2000; Stirnberg et al., 2002). The formation of basal branches in pea is enhanced under short photoperiods (Fig. 1, A and B). Bud outgrowth at upper nodes in pea often occurs at the onset of flowering and may also be, directly or indirectly, under photoperiod control. Although the formation of branches in Arabidopsis is somewhat constrained until the floral transition, this species shows similar photoperiod responses in the node of axillary bud initiation and development to those observed for branching in pea (Grbić and Bleecker, 2000; Stirnberg et al., 2002).

Determination of the axillary meristem as either vegetative or floral is a key step in regulating plant architecture and involves interactions among genotype, environmental cues, and endogenous phytohormone-like signals. Once the identity of an axillary meristem is determined, a further developmental program acts locally to maintain that determination and to prescribe organ identity within the axillary structure. However, long and short-range signals control not only the specification and maintenance of meristem identity but also organ outgrowth, thus exerting a major influence on whether axillary meristems reach their potential to form a mature branch or an inflorescence-bearing fruit.

The suitability of pea for investigating long-distance signaling makes it a valuable tool for elucidating the coordinate regulation of axillary meristem development. Its long internodes separating nodes in vegetative and reproductive zones and its ample root size for xylem sap extraction make it suitable for endogenous and exogenous phytohormone studies at several developmental stages. Moreover, in contrast to Arabidopsis, which does not respond to exogenous auxin after decapitation (Cline, 1996), pea shows a typical strong apical dominance phenotype and does respond to auxin after decapitation (e.g. Beveridge et al., 2000). Whereas grafting has only recently been successfully applied to Arabidopsis (Turnbull et al., 2002), pea is readily amenable to many different graft unions, allowing the production of genetic chimeras without the complication of adventitious rooting.

The phenotypes of various mutants discussed herein indicate that apical and axillary meristems are, to some extent, independently regulated. Genetic and physiological analysis of flowering time and shoot

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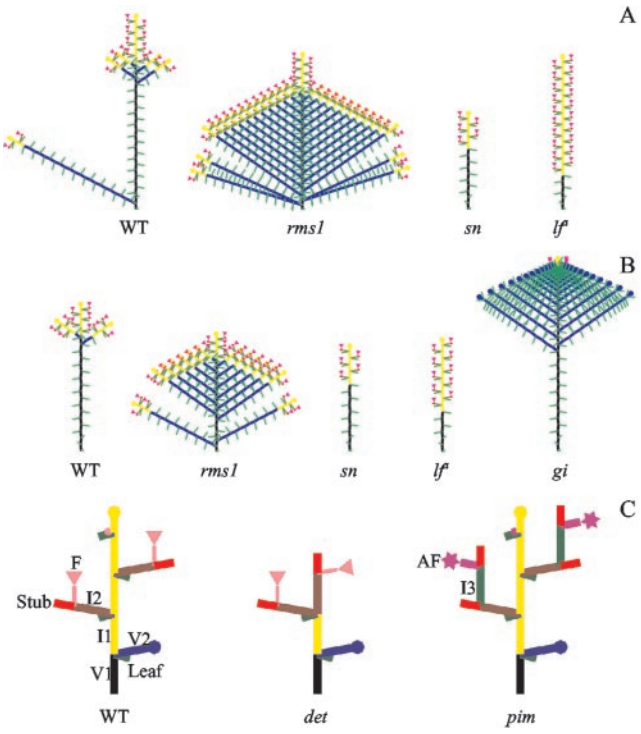

Figure 1. Schematics of WT and mutant shoot architecture in pea. Representative phenotypes of WT (e.g. Torsdag), *rms1*, *sn*, and *lf*[a] plants are shown under 8-h (A) or 18-h (B) photoperiods. B, Mutant *gi* phenotypes are also included under 18 h. C, The development of inflorescences in WT and in the mutants *det* and *pim*. WT shoot development begins with a first-order vegetative meristem (V1; black) that initiates second-order vegetative axillary meristems (V2; blue) at each node. In response to photoperiod and mobile signals, the V1 meristem transits to a first-order inflorescence meristem (I1; yellow). The I1 meristem initiates a second-order axillary meristem (I2; brown). I2 meristems initiate floral meristems (F; pink) and terminate in a stub (red). Additional inflorescence branching (I3; green) in *pim* precedes formation of aberrant flowers (AF; purple). Leaves are shown as short green lines.

architecture mutants in garden pea has identified three interacting networks (Fig. 2). Two of these, the vegetative and floral meristem networks, are devoted specifically to axillary meristem identity and/or subsequent development. A third, the photoperiod network, controls the developmental strategy of the whole plant in response to daylength and coordinately regulates both vegetative traits and flowering. This *Update* will discuss these regulatory networks with an emphasis on the involvement of long-distance signals.

VEGETATIVE MERISTEM DEVELOPMENT NETWORK

In many plants, and particularly in weakly branching monopodial plants such as pea, rapid outgrowth of axillary buds after decapitation allows the plant to maintain vigorous growth under competitive conditions and may be essential to provide replacement sites for reproductive development. However, it is also clear that this response must be regulated, because indiscriminate bud outgrowth could quickly lead to deleterious shading and might also divert resources away from reproductive structures developing elsewhere on the plant. This implies communication among axillary buds and between axillary buds and the shoot tip. Gene expression and protein profiling studies in pea have revealed that removal of the shoot tip induces axillary buds to enter a transition state between dormancy and growth (Fig. 2A) and that whether buds subsequently revert back to the dormant state or proceed to sustained growth is influenced by the state of other buds (Stafstrom et al., 1998; Shimazo-Sato and Mori, 2001).

Until recently, the most widely accepted hypothesis on the role of systemic signals in regulating bud outgrowth was that auxin derived in shoot tips and young leaves acts indirectly to inhibit branching by decreasing cytokinin supply to buds (e.g., Cline, 1994). Central to recent progress to expand this simplistic hypothesis has been the identification of mutants that differ from the wild type (WT) primarily due to enhanced development of vegetative axillary meristems. The most comprehensive genetic and physiological analysis of shoot branching in plants has been performed with the *ramosus* series of branching mutants in pea (Arumingtyas et al., 1992; Rameau et al., 2002). Studies with these *rms* mutants have revealed a more complex regulatory network by demonstrating involvement of long-distance signals in addition to auxin and cytokinins (Beveridge et al., 2000; Fig. 2A).

Nonallelic mutants *rms1* to *rms5* exhibit increased branching at basal and aerial nodes, whereas *rms6* mutants branch at basal nodes only. The *rms* mutants enhance rather than override the ontogenetic variation in tendency for bud outgrowth exhibited by WT plants. As in WT plants, the pattern of bud outgrowth in mutants *rms1* to *rms5* remains strongly influenced by photoperiod, with a decrease in basal branching and an increase in aerial branching under long days (LD) compared with short days (SD; Fig. 3; Arumingtyas et al., 1992).

rms Mutants Reveal Involvement of Novel Signals

Grafting studies with three of the pea mutants (*rms1*, *rms2*, and *rms5*) have demonstrated clear roles for long-distance signals in the control of bud outgrowth and have shown regulation by genes acting in shoot and/or stem and root (Beveridge et al., 1997; Morris et al., 2001). Similar results have been obtained with recently isolated Arabidopsis branching mutants, *max1* and *max3*, and the *dad1* branching mutant of petunia (*Petunia hybrida*), providing further evidence that axillary bud outgrowth is not under control of the shoot tip alone (Napoli, 1996; Turnbull et al., 2002). Grafting experiments in pea have

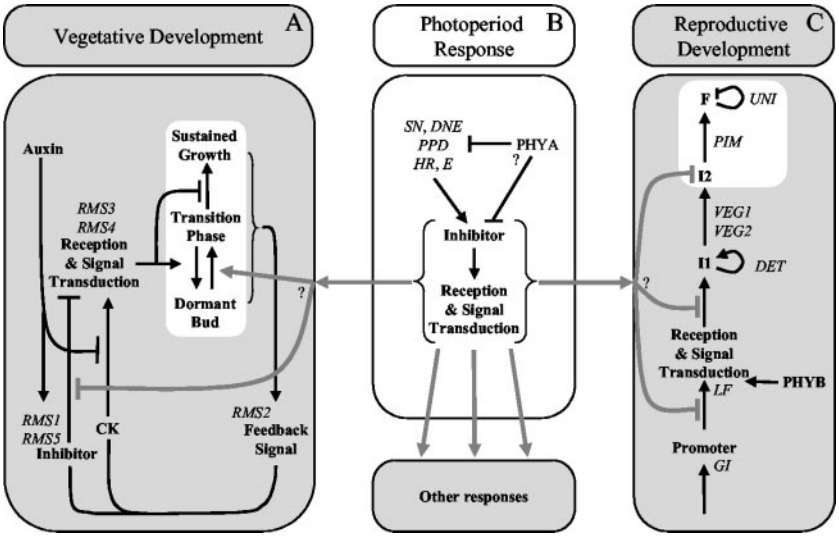

Figure 2. Regulatory networks controlling vegetative and floral meristem development in pea. The vegetative (A), photoperiod response (B), and reproductive (C) development networks are shown. Arrows between the networks show hypotheses of where the points of coordinate regulation may occur.

shown that *RMS1* and *RMS5* may act in the same biochemical pathway for a signal that acts like an inhibitor and moves only acropetally in shoots, presumably through the xylem (Foo et al., 2001; Morris et al., 2001; Fig. 2A).

The *rms* mutants have been used to determine the possible phytohormone basis of these graft-transmissible signals (for review, see Beveridge, 2000; Morris et al., 2001). The signal regulated by *RMS1* and *RMS5* is unlikely to be either cytokinin or auxin for several reasons. For example, *rms1* and *rms5* plants have greatly reduced, rather than elevated, xylem sap cytokinin concentrations (Beveridge, 2000; Morris et al., 2001). Also auxin and auxin precursors are not thought to be carried in the xylem. Moreover, auxin content in the shoot of *rms* mutants is typically elevated, not reduced, and exogenous auxin does not restore a WT phenotype to *rms* plants (Beveridge et al., 1997, 2000; Beveridge, 2000). These results indicate that *RMS1* and *RMS5* may regulate a novel signal (Fig. 2A). The recent cloning of *RMS1* after isolation of the *MAX4* sequence from the new *max* series of branching mutants from Arabidopsis and isolation of a *MAX4* homolog from *Medicago truncatula* (K. Sorefan, J. Booker, K. Haurogné, M. Goussot, E. Foo, S. Chatfield, C. Beveridge, C. Rameau, and O. Leyser, unpublished data) will be reported elsewhere and opens new avenues to test this hypothesis.

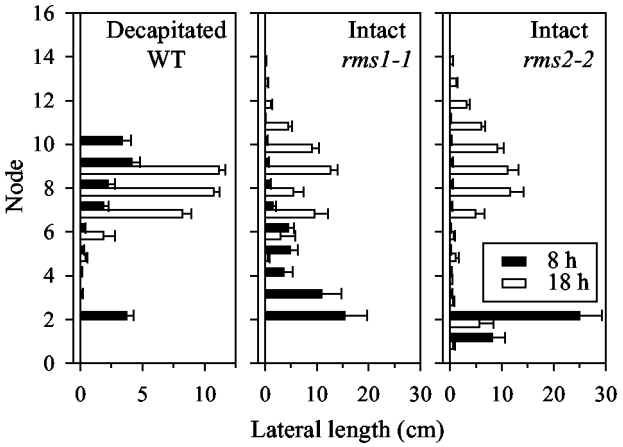

Figure 3. Lateral lengths at nodes of decapitated WT (pea cv Parvus) and intact *rms1-1* and *rms2-2* plants (left to right). Plants received 8 h of natural daylight followed by darkness or light extension to 18 h supplied by a 1:1 mixture of fluorescent (40-W, white) and incandescent (100-W) lights providing an intensity of 25 to 30 μmol m^{-2} s^{-1} at the pot top. Intact WT plants did not produce laterals greater than 1 cm. Nodes are counted from the cotyledonary node as node 1. Decapitation was performed below the highest expanded leaf, 7 d before scoring. All plants were scored on d 36 when intact mutant plants under LD and SD had 13 to 14 and 11 leaves expanded, respectively. The first flower opened under LD at node 16. Data are presented as mean ± SE; n = 5–6.

The sequence of an additional *MAX* gene, *MAX2*, has been reported and encodes an F-box protein that may be involved in signal transduction (Woo et al., 2001; Stirnberg et al., 2002). The obvious candidates for a pea ortholog of *MAX2* are therefore genes such as *RMS3* or *RMS4* that appear to act mostly in the shoot and that are proposed to control the response to signals involved in branching control (for review, see Beveridge, 2000; Fig. 2A).

What Is the Role of Auxin?

The auxin inhibition of bud outgrowth in decapitated plants appears to require the long-distance signal regulated by *RMS1* (Fig. 2A). Decapitated *rms1* mutant shoots can only respond to exogenous auxin when grafted to WT rootstocks (Beveridge et al., 2000). Moreover, *RMS1* may be auxin regulated because *RMS1* expression drops after decapitation and is restored by exogenous auxin (E. Foo, C. Beveridge, and C. Rameau, unpublished data).

The possibility that other phytohormones or environmental cues may directly or indirectly regulate

RMS1 expression, RMS1 protein stability, precursor availability, or product degradation and therefore affect levels of the shoot-to-root signal regulated by *RMS1*, should be investigated. This could reveal how bud outgrowth may be regulated via long-distance signals that do not cause auxin-related pleiotropic effects.

In pea, the *bushy* mutant and several late flowering mutants under certain conditions (Fig. 1B and see below) have a pleiotropic highly branched and dwarfed phenotype (Beveridge et al., 2001; Symons et al., 2002). In contrast to *rms* mutants, which have vigorous shoot tip growth and do not have depleted auxin levels, these pleiotropic phenotypes are related to weak shoot tip growth and may be at least partly attributed to reduced endogenous auxin levels (Beveridge et al., 2001; Symons et al., 2002).

In addition to the auxin-regulation of *RMS1*, autoregulation of bud outgrowth may also involve auxin-independent modulation of xylem sap cytokinin content via a shoot-to-root signal. Several of the *rms* mutants show strongly reduced xylem sap cytokinin concentration. Graft combinations of WT and *rms3* or *rms4* reveal that the branching phenotype of the shoot is associated with the rate of cytokinin export from the roots, regardless of the root genotype, implying involvement of a shoot-to-root feedback signal (Beveridge et al., 1997; Beveridge, 2000; Fig. 2A). This shoot-to-root signal is unlikely to be indole-3-acetic acid, because indole-3-acetic acid levels and transport are not greatly affected in these genotypes. *RMS2* may regulate the feedback signal because *rms2* plants have elevated xylem sap cytokinin content, and double mutants show that *rms1* and *rms5* do not cause reduced xylem sap cytokinin content in the presence of *rms2* (Beveridge et al., 1997; Morris et al., 2001).

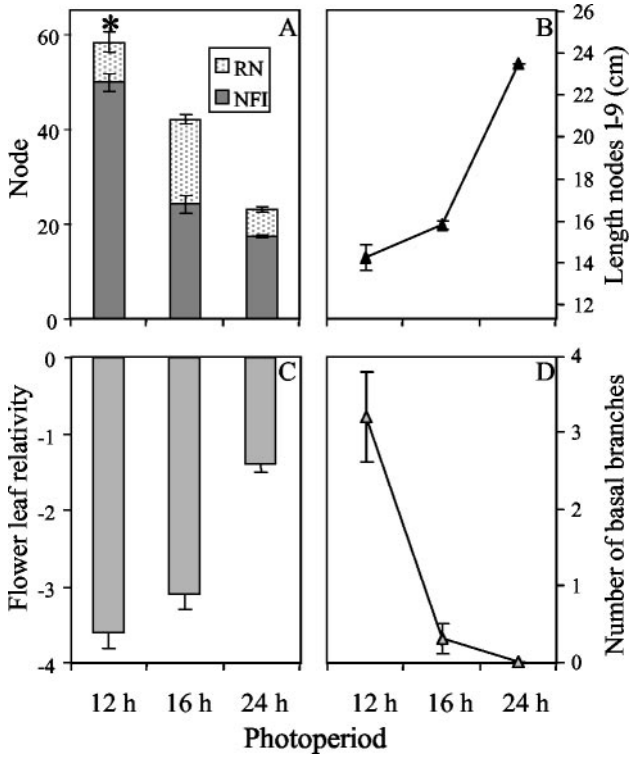

Figure 4. Typical pleiotropic effects of photoperiod response in pea. A, Node of flower initiation (NFI) and number of reproductive nodes (RN); B, length of the primary stem between nodes 1 to 9; C, node of flower opening relative to the node of the highest expanded leaf; D, number of branches at nodes 1 to 3. The highly photoperiod responsive (*SN DNE PPD HR*: HL63) line was grown under 8 h of daylight extended to 12 or 16 h with incandescent light at 55 μmol m^{-2} s^{-1} or to 24 h with incandescent light at 3 μmol m^{-2} s^{-1}. Data are means ± SE for six plants per treatment. *, Plants grown under 12 h were scored well before senescence and would have developed considerably more reproductive nodes (RN) than shown.

PHOTOPERIOD RESPONSE NETWORK

The effects of photoperiod on the initiation of flowering are well known. Perhaps less widely recognized is that, in many species, including temperate LD plants such as Arabidopsis and pea and SD plants such as common bean (*Phaseolus vulgaris*), photoperiod also affects vegetative shoot architecture (Figs. 1 and 3) and a range of other vegetative and reproductive characteristics (Wallace et al., 1993; Fig. 4).

Under inductive conditions for flowering, the resources of the plant are directed toward rapid completion of the life cycle. The growth habit under noninductive photoperiods can be understood as a strategy that prevents the plant from investing too much energy in reproduction under unfavorable conditions and prepares it to exploit a subsequent improvement in conditions by increasing the photosynthetic area and the number of sites available for reproduction. The differences in phenotype between plants grown in SD and LD cannot be explained solely by the earlier flowering and enhanced sink activity of developing flowers and fruits in plants grown under LD, because effects of photoperiod on vegetative traits can be clearly seen even in mutants that fail to initiate flowers under any photoperiod (Reid and Murfet, 1984; Kelly and Davies, 1988).

Photoperiod Response Genes Control Long-Distance Signal(s)

Although photoperiod controls many different traits, genetic analyses show that responsiveness to photoperiod depends on a common mechanism. Recessive mutations that reduce or eliminate photoperiod responsiveness (day-neutral) are known in several legumes. In most cases, these mutants are early flowering and display a "constitutively reproductive" growth habit, having lost the ability to stimulate vegetative growth and inhibit flowering under noninductive conditions. Mutants known from LD species include *sn* (Fig. 1, A and B), *dne*, and *ppd* in pea, *dn* in sweet pea, and *sn* in lentil (*Lens culinaris*;

Murfet, 1971; King and Murfet, 1985; Ross and Murfet, 1988; Sarker et al., 1999). The *ppd* mutant of common bean, a SD species, also shows a similar phenotype (Wallace et al., 1993).

The involvement of long-distance signaling in the control of photoperiod responses has been a topic of interest for several decades. Day-neutral mutants in pea and sweet pea have been used to establish a genetic basis for long-distance signaling in photoperiod responsiveness. For example, day-neutral mutants *sn*, *dne*, and *ppd* show a delay in flowering and an enhancement of vegetative vigor when grafted onto WT rootstocks, suggesting that the mutations somehow disrupt the supply of a mobile signal to the apex by interfering with its synthesis or transport (Murfet, 1985). Studies with these mutants have also reinforced the idea that the mobile signal controls multiple aspects of development, including flowering and branching (Fig. 2). Grafting experiments have also shown that the inhibitory influence of leaves declines with leaf age (Reid and Murfet, 1977).

Studies with two other genes that affect photoperiod response indicate that developmental and tissue-specific regulation of the level of the mobile signal is an important feature of the photoperiod response in pea. Dominant alleles of the *HIGH RESPONSE* (*HR*) gene occur in many primitive accessions and increase the size of the photoperiod response for flowering, mainly by delaying flowering under SD (Murfet, 1985). Results from grafting experiments suggest that rather than increasing inhibitor production, *HR* extends the time span over which leaves remain inhibitory (Reid and Murfet, 1977). Another gene, *EARLY* (*E*), decreases inhibitor production and acts only in the cotyledons. It may cause early floral initiation in some genetic backgrounds, followed by a period of inflorescence abortion or vegetative reversion after the cotyledons senesce and the photoperiod response of the shoot becomes dominant (Murfet, 1985).

Mutants have been used to investigate the role of specific photoreceptors in the regulation of long-distance signaling. Plants deficient in phytochrome A (phyA) are very similar to WT when grown under SD, but under LD, they flower late and show the full range of pleiotropic characteristics typical of WT plants grown in SD (Weller et al., 1997). Leafy *phyA* rootstocks delay flowering in WT scions under LD, confirming that phyA contributes to the down-regulation of a mobile inhibitor of flowering under LD (Fig. 2B). However, flowering of *phyA* mutants is still strongly promoted by day extensions with certain light sources, and at least one cryptochrome is probably also involved in this response (Weller et al., 2001). Phytochrome B is required for inhibition of flowering under SD, but does not affect photoperiod responsiveness for other traits, and its inhibitory effects on flowering are not graft transmissible (Weller et al., 2001; Fig. 2C).

Comparing Photoperiod Response Systems in Pea and Arabidopsis

Apart from *PHYA*, none of the pea genes involved in the photoperiod response have been cloned, and the nature of the inhibitory signal is still unknown. Further progress in understanding the functions of these genes will come from comparisons with Arabidopsis, where the photoperiod pathway is well characterized and many genes involved in the photoperiod response have been cloned. In Arabidopsis, light and the circadian clock interact to regulate the expression of genes that specifically promote or inhibit flowering (Mouradov et al., 2002). This pathway consists of genes that either promote or inhibit flowering. Among the genes that promote flowering are those encoding known photoreceptors (*PHYA* and *CRY2*), putative photoreceptors involved in light signaling to the clock (*ZTL*, *FKF*, and *LKP2*) and genes specific for the floral transition (*CO*, *FT*, *FD*, and *FE*). The genes that inhibit flowering all appear to function close to clock mechanism (*LHY*, *CCA1*, *TOC1*, *ELF3*, and *ELF4*).

The fact that most of the known photoperiod response genes in pea have an inhibitory effect on flowering (*SN*, *DNE*, *PPD*, and *HR*) raises the possibility that this system in pea might correspond to the circadian system in Arabidopsis. The Arabidopsis circadian system is implicated in the control of a wide range of processes, so this possibility is consistent with the fact that the pea genes have pleiotropic effects. Although the pea system has frequently been discussed as if it controlled a single mobile signal, it might equally represent a mechanism for the regulation of a large number of genes, some of which could be associated with long-distance signaling in specific developmental responses (Fig. 2).

FLORAL MERISTEM DEVELOPMENT NETWORK

Genetic and physiological studies indicate that the photoperiod response network interacts with networks of genes that are specific to axillary bud outgrowth or reproductive development (Fig. 2). That is, genes acting in these vegetative or reproductive specific networks do not affect photoperiod responsiveness. Like bud outgrowth, the transition to flowering involves specific long-distance signals.

Although the transition to flowering in legumes may appear to be simply the replacement of vegetative axillary meristems with reproductive axillary meristems, the primary shoot apical meristem must first make the transition from a vegetative to an inflorescence meristem (Fig. 1C). In pea, inflorescence fate can be determined as early as eight nodes before a floral meristem forms (Ferguson et al., 1991).

Commitment to a program of inflorescence development is thus separable and distinct from determination for floral development. Analysis of mutants has identified genes that may be involved in these transitions, and grafting studies are being used to separate those genes acting in long-distance signaling from those acting locally.

A Mobile Signal Is Specifically Required for Transition to Flowering

In addition to the inhibitory signal involved in photoperiod responsiveness, there is a large body of physiological evidence from many species supporting the existence of a mobile flowering stimulus (Murfet, 1971). Genetic evidence for such a promoter has more recently been demonstrated in pea (Beveridge and Murfet, 1996). Recessive mutants at the *GIGAS* (*GI*) locus can show a photoperiod response in flowering node, but show a large delay in flowering under SD, and flower late or not at all under LD. In addition to differing in flowering response to photoperiod, *gi* mutants differ from the late flowering *phyA* mutants, because vegetative traits in *gi* plants respond normally to photoperiod. *GI* is proposed to have a role in long-distance signaling (Fig. 2C) because flowering can be partially restored to *gi* mutant scions by grafting to a WT stock (Beveridge and Murfet, 1996).

Development of *gi* mutant plants in LD proceeds relatively normally until around the time of flower initiation in WT, after which the mutants appear to lose apical dominance and become highly branched at aerial nodes (Fig. 1B). The change in growth pattern occurs earlier and is more severe in an *sn* background, and it may thus reflect the natural decline in the activity of the photoperiod pathway in the absence of inflorescence development (Murfet, 1985; Beveridge et al., 2001).

A Module of Genes with Overlapping Functions Acts Locally to Specify Inflorescence and Floral Identity

Like *GI*, the *LATE-FLOWERING* (*LF*) gene appears to specifically affect the transition to flowering. Recessive mutants at the *LF* locus flower earlier than WT in both LD and SD (Fig. 1, A and B). Whereas *GI* controls a mobile stimulus, *LF* has an inhibitory effect and acts only in the shoot (Murfet, 1985). Plants carrying extreme-late, dominant *LF* alleles show a reduced apical dominance phenotype under LD that is very similar to that seen in *gi* mutants. Also, like *GI*, *LF* alleles have an essentially additive interaction with *SN*. These observations suggest that *GI* and *LF* may act in the same flowering-specific pathway (Fig. 2C). Consistent with this suggestion, a strong *lf* allele is completely epistatic to *gi* under SD (Taylor, 1997). The *GI-LF* interaction may therefore define an important point at which a mobile signal from the leaves could interact with a genetic module controlling inflorescence meristem identity in the shoot apex.

Severe mutants at the *VEGETATIVE 1* (*VEG1*) and *VEGETATIVE 2* (*VEG2*) loci never flower and yet show a photoperiod response for vegetative traits. In SD, these mutants have a relatively normal appearance, apart from the failure to initiate secondary inflorescences. In LD, they display the same aerial branching phenotype as *gi* mutants (Fig. 1B) and plants carrying extreme-late *LF* alleles. The nonflowering phenotypes of *veg1* and *veg2* plants cannot be overcome by grafting to WT, indicating that like *LF*, *VEG1* and *VEG2* act locally in the shoot apex. Both *veg1* and *veg2* are epistatic to *LF* (Reid and Murfet, 1984; Taylor, 1997), suggesting that these genes are required for *LF* function, which may be to repress *VEG1* and *VEG2* expression in newly initiated axillary meristems (Fig. 2C).

Another gene that acts locally to control inflorescence and flower development downstream of *LF* is *DETERMINATE* (*DET*). *DET* appears to maintain the indeterminacy of growth in the primary shoot inflorescence meristem (I1; see Fig. 1C; Singer et al., 1990). Development of *det* mutants proceeds normally until one or two essentially normal axillary secondary inflorescences have been produced (I2; Fig. 1C), each bearing one or more individual flowers and terminating in a stub. After this, the *det* shoot apex itself develops the characteristics of a secondary inflorescence, producing a flower and terminating in a stub (Fig. 1C), whereas the WT primary shoot inflorescence meristem remains indeterminate. This suggests that *DET* acts specifically in the main shoot apex, to suppress secondary inflorescence development, possibly by excluding *VEG1* and *VEG2* activity (Fig. 2C). The *det* mutant phenotype suggests that *DET* may be homologous to *TFL* and *CEN* from Arabidopsis and *Antirrhinum* sp., respectively (Bradley et al., 1997).

Three homologs of floral meristem identity genes in other species have been characterized in pea: *PROLIFERATING INFLORESCENCE MERISTEM* (*PIM*), *STAMINA PISTILLOIDA* (*STP*), and *UNIFOLIATA* (*UNI*; Hofer et al., 1997; Taylor et al., 2001, 2002). The pea homolog of the MADS-box gene *AP1* from Arabidopsis, *PIM*, specifies floral meristem identity but has minimal influence on the phenotype of the secondary inflorescence (Fig. 2C). *pim* mutants develop a secondary inflorescence with a terminal stub, but the third-order branch develops as an inflorescence rather than a flower (Fig. 1C). Repetitive inflorescence branching leads to formation of an aberrant flower. The extent and pattern of the branching in later order branches of *pim* mutants depends on photoperiod, providing more evidence that the photoperiod system also operates at relatively late stages of flower development (Taylor et al., 2002).

The apparent independence of floral and inflorescence developmental networks is shown by the continuation of inflorescence development in the ab-

sence of the floral meristem identity function of *PIM*. However, this apparent independence may be explained by redundancy in the floral meristem developmental network, as is seen in other species. Such redundant floral meristem functions can be proposed to account for the eventual formation of flowers, albeit aberrant flowers, in *pim* mutants. *PIM* is also responsible for normal petal and stamen development, presumably through activation of downstream floral organ identity genes. Incomplete suppression of bract development in *pim* mutants is evidence that *PIM* also has a minimal role in secondary inflorescence development.

UNI and *STP* highlight the dual control developmental genes can have on vegetative and inflorescence development. The strongest *uni* mutant allele results in the production of unifoliolate leaves instead of pinnate compound leaves; *stp* mutant leaves are simplified, but to a lesser degree, compared with WT (Hofer et al., 1997; Taylor et al., 2001). Although the *UNI* homolog in Arabidopsis, *LEAFY*, is expressed in leaves during vegetative development, it does not have the profound effect on leaf development that *UNI* has in pea. The secondary inflorescence is unaffected in null *uni* and *stp* mutants, but the aberrant floral meristem first initiates a whorl of sepals and a carpel before reiterating this pattern of development (Hofer et al., 1997; Taylor et al., 2001). In contrast to the relationship among the homologous genes in Arabidopsis, *UNI* and *STP* are not required for the expression of the floral meristem identity gene *PIM* (Taylor et al., 2001).

CONCLUDING REMARKS

Long-distance signals are involved in the regulation of many aspects of axillary meristem development, including vegetative axillary meristem development and the determination and development of primary and secondary inflorescence meristems. In the control of vegetative branching, long-distance signals form an autoregulatory loop whereby the outgrowth of buds feeds back to down-regulate the outgrowth of other buds. It is interesting to note that autoregulation by long-distance signals is also an important part of the control of nodule meristem development (see Szczyglowski and Amyot, 2003; this issue). A long-awaited breakthrough in this field of research will be identification of novel long-distance signals (see Dixon and Sumner, 2003; this issue). It will also be important to understand how the major phytohormones such as auxin interact with these signals and contribute to the coordinated regulation of multiple aspects of plant development.

An enhanced rate of progress on the research of all aspects of axillary meristem development in legumes is likely to result from integrated studies among different species. For example, work in Arabidopsis suggests that mutagenesis for flowering mutants in pea, particularly photoperiod-responsive late lines is likely to lead to new loci in pea, whereas work in pea has characterized long-distance signaling mechanisms that are yet to be identified in Arabidopsis. The legume genomics projects (see VandenBosch and Stacey, 2003; this issue) will facilitate this comparative analysis by enabling rapid cloning of pea homologs of genes cloned in other species.

New opportunities to understand plant development arise from recognizing the high degree of regulation among different parts of the developmental network. Here, we have identified coordinate regulation of flowering and branching by a photoperiod response module. Studies of the properties of the entire developmental network (including modules not covered here) are likely to yield new knowledge about how perturbations in one part of the network affect other parts. Computational approaches to model and test hypotheses concerning components of the system and their interactions may be useful to conceptualize the whole system and to incorporate a rapidly increasing body of knowledge.

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