Update on the genetics of flowering

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The biological mechanisms controlling flowering and photoperiod responsiveness have been of interest to plant biologists for nearly a century, and the genetic control of flowering in pea has been under investigation for a similar length of time. For a period in the 1970s, peas held a prominent place as a model species for physiological genetics of flowering, due largely to the efforts of Ian Murfet. In a career spanning more than forty years Ian, together with his colleagues and students, identified more than a dozen major flowering loci through analysis both of natural variants and induced mutants. He also mapped many of these loci, and used various physiological and genetic approaches to define their functions and interactions.

Most of this early work preceded the molecular era, and until recently the molecular nature of the pea flowering loci has remained largely unexplored. However, over the last decade work in arabidopsis has given major insights into the genes and genetic mechanisms controlling plant responses to photoperiod and temperature, flower development, light perception and endogenous rhythms (3, 18, 19, 24, 41). Partly as a result, there have also been significant advances in the molecular biology of flowering in several other model species, including rice, barley, wheat and tomato (9, 14, 23). This information, together with the availability of extensive sequence databases in a number of model legumes (38) and the well-documented synteny between pea and medicago (2, 20) has opened up a number of avenues for molecular analysis of flowering in pea. Over the last few years work on flowering in pea has resumed in Hobart, with the isolation of numerous flowering gene homologues and new flowering mutants (15, 16).

In this update I will summarize some of these more recent developments after first providing some background information. Numerous reviews of earlier work are available (30, 31, 37, 51) and can be consulted for further information.

1. Flowering loci

(a) LATE FLOWERING (Lf): A major flowering gene linked to *A* (now linkage group II) had been observed by many workers before the *Lf* locus was definitively described as one of four major loci contributing to the genetic variation for flowering time among existing pea cultivars (27, 29). Numerous induced *lf* mutants are now also known, and all flower earlier than their respective progenitor lines (42). The most severe mutants (Murfet's *lf-a* class) flower as early as node 6, but most cultivars appear to carry an intermediate allele in the *lf* or *Lf* class and it seems likely that the ancestral form is represented by the *Lf-d* class (29, 42). *Lf* is considered to govern the plant's "inherent lateness", because allelic variation at *Lf* does not appear to interfere with the plant's ability to respond to photoperiod.

The *Lf* locus is notable as the first of the classical pea flowering loci to be identified at the molecular level. One of three pea homologs of arabidopsis *TFL1 (TFL1c)* was identified as a candidate gene for *Lf* based on its map position, and several *lf-a* class mutants were shown to have large deletions or amino acid substitutions in *TFL1c* consistent with a complete loss of function (11). The isolation of an additional EMS mutant *(lf-22)* carrying a nonsense mutation has provided further support for this conclusion (V. Hecht, J. Weller unpubl.). In arabidopsis, mutations in *TFL1* confer both early flowering and a conversion of the indeterminate primary inflorescence to a flower (8). Although the primary inflorescence of severe *lf* mutants remains indeterminate, a small function of *Lf* in determinacy is apparent in early secondary inflorescences, which in *lf* mutants tend to terminate in an abnormal flower, instead of the normal indeterminate stub.

Although the deletion and nonsense mutants clearly demonstrate that *Lf* is *TFL1c,* variation in flowering time attributed to allelic variation at *Lf* is not always associated with mutation in the *Lf* coding sequence. For example, the isolines WL1771/1770/1769 *(Lf-d, Lf* and lf, respectively) have no

polymorphism within the coding region or introns of *Lf* but *Lf* expression does correlate with flowering across this series (11). However, the possibility that these lines may carry mutations in the *Lf* promoter region has not yet been excluded.

Despite the importance of *Lf* for flowering time, it is not known how *Lf* participates in mechanisms controlling flower transition. Grafting experiments suggest *Lf* acts in the shoot apex, as its effects are not graft-transmissible (26). Preliminary results suggest that expression of *Lf* occurs throughout the plant and does not show any marked developmental or environmental regulation (11; B. Wenden, V. Hecht, C. Knowles, unpublished), and it will be interesting to see if this is supported by more detailed studies.

(b) Photoperiod-specific repressors of flowering: As in the case of Lf, the effects of allelic variation at the *STERILE NODES (Sn)* locus have probably been under study for more than a century, but it was only with the use of controlled-photoperiod conditions that the existence of this locus could be clearly demonstrated (4, 27). Mutant *sn* plants flower early under both LD and SD but, in contrast to *lf* mutants, are unable to respond to photoperiod and when grown in SD display the short reproductive phase and rapid reproductive development typical of WT plants in LD. Mutants at two other loci, *DIE NEUTRALIS (Dne)* and *PHOTOPERIOD (Ppd)* have early-flowering, day-neutral phenotypes similar to *sn* (1, 21, 44). The origin of the original *Sn/sn* allelic difference is obscure, but three additional induced *sn* alleles have now been identified(1; S.E. Jones, J. Vander Schoor, J. Weller unpublished).

Comparisons with the arabidopsis system suggest that the majority of early-flowering photoperiod-insensitive mutants have primary defects in maintenance of the circadian clock. We are currently examining the expression of circadian clock gene homologues in *sn, dne* and *ppd* mutants and find that all three show defects in rhythmic gene expression under light/dark cycles and constant conditions (V. Hecht, L.C. Liew, unpublished). In parallel with these physiological studies, we are refining map positions for all three loci (1, 21, 48) and examining relationships with candidate circadian clock genes in corresponding regions.

Throughout the 1960s and 1970s, a variety of different grafting experiments was used to explore how the *Sn* locus might influence the transmission of graft-mobile flowering signals. The majority of grafts were performed epicotyl to epicotyl, effectively examining transmission from roots/cotyledons to shoot apex. A small delay in flowering of *sn* scions induced by WT stocks was interpreted to suggest that *sn* impaired production of a mobile inhibitor (4, 27). Similar conclusions were later reached for *dne* and *ppd* mutants (21, 44). However, in all experiments of this type a strong promotion of WT scions by early mutant stocks was also observed making it equally plausible that the mutants possess elevated levels of a mobile floral stimulus.

More recently, we have been re-examining this question in grafts with leafy stocks possessing 4-5 true foliage leaves. In this system the influence of the cotyledons has declined and flowering of the scion is primarily determined by the influence of the stock leaves. We observe substantial promotion of flowering in WT scions by early mutant stocks, but no significant inhibitory effect of WT stocks on early mutant scions (L.C. Liew and J. Weller, unpublished), consistent with the view that genes of this nature predominantly act through regulation of a mobile flowering stimulus.

(c) *HIGH RESPONSE (Hr):* *Hr* was another of the four major loci initially characterized by Ian Murfet (28). Like *Sn, Dne* and *Ppd,* the dominant *Hr* allele inhibits flowering mainly under SD. In an otherwise WT background, this inhibition may be so strong as to confer a near-obligate requirement for long days. This suggests that *Hr* can be viewed as a photoperiod response gene, and evidence from grafting experiments suggest that leafy *hr* stocks can strongly promote flowering in *Hr* scions and that *Hr* may act through the same mobile signal as *Sn* (34, 35). One possibility is therefore that *Hr,* like *Sn, Dne* and *Ppd,* may have defects in rhythmic gene expression and a primary role in the photoperiod response pathway.

However, another possibility is that *Hr* may be analogous to *FRIGIDA (FRI)* and *FLOWERING LOCUS C (FLC)* in arabidopsis. These loci are typically discussed as mediators of the vernalization

response (41) and are not generally considered as part of the arabidopsis photoperiod pathway, although they do influence the photoperiod response through a dramatic delay in flowering under SD. Genes in the *FLC* clade have not yet been conclusively identified in pea or in any model legume despite the existence of extensive EST and genomic databases. On the other hand, *FRI* homologues are known from a range of species, and orthologues of both *FRI* and *FRIGIDA-LIKE1 (FRL1)* are present in medicago (15). Updated medicago mapping data suggests that *MtFRI* and *MtFRLa* both map on chromosome 3, and that position of *MtFRI* corresponds to the approximate location of *Hr* in pea LGIII (J. Weller, unpublished data).

(d) Photoperiod-specific promoters of flowering: In arabidopsis, several mutants with a LD-specific late-flowering phenotype were among the first flowering mutants isolated (22) and the corresponding genes have all turned out to be important components of the photoperiod response mechanism (14, 18, 33). We have therefore been particularly interested to find mutants of this type in pea. The *phytochrome A (phyA)* mutants were first identified by their defective de-etiolation responses to far-red light, and subsequently shown to flower late in LD with additional phenotypes that are essentially a phenocopy of WT plants grown in SD (increased basal branching, delayed senescence) (50). Mutations in the *PHYA* gene are necessary for the promotion of flowering in response to photoperiod extensions rich in red light but have little effect on the response to blue light (32, 52). A dominant, hypermorphic *phyA* mutant, *phyA-3D,* was also identified in seedling screens, with an early flowering phenotype in SD similar to the sn, *dne* and *ppd* mutants (53).

More recently, we have isolated a number of other mutants with phenotypes similar to *phyA* (16). Like *phyA* mutants, *late bloomer 1 (late1)* mutants flower late in LD and have the general appearance of SD-grown WT plants. Mutant *late1* plants also have defects in rhythmic expression of circadian clock genes, suggesting that *Late1* may have a primary role in clock function (16). Consistent with these roles, *Late1* is the pea ortholog of arabidopsis *GIGANTEA* (16), which has a central role in circadian clock function and additional, independent effects on photoperiodic flowering (25). The late flowering phenotype of *late1* mutants is rescued by grafting to leafy WT stocks in LD, indicating that *LATE1,* like *Sn, Dne* and *Ppd,* acts through regulation of a mobile flowering stimulus (16).The *LATE BLOOMER 2* locus has a mutant phenotype similar to *phyA* and *late1,* but has yet to be further characterized.

(e) *Gigas:* The *Gigas* locus is currently defined by two recessive mutant alleles. In SD both *gigas* mutants flower later (10 to 20 nodes) than their respective WT, but otherwise show little phenotypic difference. In LD, *gigas* mutants also show delayed flowering, but have a striking phenotype distinct from photoperiod mutants *phyA* and *late1.* Mutant *gigas* plants develop normally until around the time WT plants flower, and then undergo a striking "vegetative shutdown" in which internodes become shorter and thinner, and the axillary buds buds at these nodes are released. The main shoot may eventually produce one or two flowering nodes, but in other cases flowers may only be formed on lateral branches, and in the strongest expression of the phenotype, the plants may never flower (7, 43). Expression of the *gigas* phenotype is also influenced by light quality and temperature, and mutants are more likely to flower in response to supplementation with light of low R:FR ratio (1; J. Weller, unpublished), under higher irradiances (43), or at lower ambient temperatures (J. Weller and J. Vander Schoor, unpublished).

Grafting of *gigas* mutant scions to WT stocks can result in a significant promotion of flowering (7, 43), leading to the suggestion that *Gigas* is involved in production of a mobile floral stimulus. However, as the LD phenotype of *gigas* is distinct from *phyA* and *late1,* it seems likely the *Gigas*-dependent mobile signal does not mediate all aspects of the photoperiod response but is limited to the initiation of flowering. One possibility is that the *Sn, Dne, Ppd* and *Late1* genes all act through *Gigas* to regulate the same mobile flowering stimulus, but it is possible that they may also affect other, *Gigas*-independent, mobile signals. New grafting experiments are underway to examine this question.

Comparative mapping in pea and medicago locates *Gigas* near a cluster of medicago genes similar to the arabidopsis *FT* gene (16). The arabidopsis *FT* gene has an important role in integration of flowering signals and mobile signalling from leaf to apex, and in light of grafting results the possibility that *Gigas* corresponds to an *FT*-like *(FTL)* gene in pea remains attractive. Isolation of pea *FTL* genes is in progress.

(f) Other flowering loci: Two other genes with a primary role in light responses also have effects on flowering. Mutations in the *PhyB* gene confer early flowering phenotype that is primarily apparent in SD. However, a null *phyB* mutation is epistatic to both *phyA* and *late1* mutants in LD, showing that *phyB* can act to delay flowering in both LD and SD (16, 52). This also suggests that *PhyA* and *Late1* genes promote flowering in LD by opposing a *PhyB*-dependent inhibition. Unlike *sn, dne* and *ppd* mutants, *phyB* mutations only affect the node of flower initiation and do not markedly alter other pleiotropic aspects of the photoperiod response.

The *light-independent photomorphogenesis 1 (lip1)* mutant was isolated as a spontaneous mutant showing a constitutively de-etiolated appearance even when grown in complete darkness (12). In this respect *lip1* is similar to the *COP/DET/FUS* mutants of arabidopsis, and has been shown to carry a complex duplication/ rearrangement in the pea *COP1* ortholog (40). The original *lip1* mutant arose spontaneously in a genetic background (nominally cv. Alaska) carrying an *sn* mutation, which masked any effects of *lip1* on flowering. However, after selection away from *sn* and introgression into the cv. Torsdag background it has become evident that *lip1* mutants are somewhat early flowering in SD and show a reduced photoperiod response similar to sn, dne, *ppd* and the *phyA-3D* mutant (J. Weller unpublished).

Among a wide range of flowering mutants obtained from recent screens, we have identified two other new *LATE BLOOMER* loci, *Late3* and *Late4.* The *late3* and *late4* mutants have a novel flowering phenotype characterised by extremely late flowering and a delay in the compound leaf transition under both SD and LD (J. Weller and J. Vander Schoor, unpublished). Mutants do not commence flowering until after node 35 and thereafter abort flower initials, fail to set pods, and occasionally show vegetative reversion. Some pods do eventually form at later reproductive nodes, but show very weak growth and yield few seeds. The *late3* and *late4* mutants continue to grow almost indefinitely in a cool environment if free from disease, and exhibit a massively extended reproductive phase. Although nearly sterile, they do not display the vegetative shutdown seen in *gigas* mutants, flower-sterile mutants or WT plants from which flowers have been removed. Nor do they exhibit basal branching or other SD characters in LD like *phyA, late1* and *late2* mutants. Instead, *late3* and *late4* produce strong aerial lateral branches later in development. Preliminary evidence indicates that the *late3* and *late4* phenotypes are neither rescuable nor transmissible through grafting.

Several other flowering loci, including *E, Lw* and *Dm,* have been described in various earlier reports and reviews (31, 37, 49), but no new information about these loci has become available since the last review (37). A role for the *Aero* locus in flowering has also recently been reported (47). However, with the exception of early work on *E* (27, 31), the relationship of these loci with other flowering genes has not been explored.

2. Inflorescence identity loci

The pea inflorescence is a compound raceme, and its development has been discussed in several reviews (5, 39). A number of mutants affecting inflorescence and floral development have now been characterized at the molecular level. The *unifoliata (uni), proliferating inflorescence meristem (pim)* and *stamina pistilloida (stp)* mutants predominantly affect the floral meristem, and the *Uni, Pim* and *Stp* genes correspond to the arabidopsis *LFY, AP1* and *UFO* genes, respectively (6, 17, 45, 46). Although all three mutants also have additional defects in development of the secondary inflorescence, they undergo a clear transition to flowering at a similar node to WT and produce peduncles clearly distinct from vegetative shoots. They therefore seem able to correctly specify both primary and early secondary inflorescence

development. This implies the existence of additional, earlier-acting genes that also participate in secondary inflorescence development, and several such loci are known.

The *Det* locus has a negative role in secondary inflorescence development, acting to prevent expression of the secondary inflorescence program in the primary inflorescence meristem. This role is analogous to that of arabidopsis *TFL1,* and *Det* is now known to encode another of the three *TFL1* homologs *(TFL1a)* in pea (11). The genetic interactions and molecular consequences of *det* mutations have yet to be explored.

Three other loci have a positive role in secondary inflorescence development. The *VEGETATIVE1 (Veg1*) locus (formerly *VEGETATIVE; Veg)* is represented by a single mutant allele. Homozygous mutant plants never produce flowers, and must be maintained through the heterozygote (13). Despite their failure to flower, *veg1* mutant plants grown in LD clearly undergo a vegetative shutdown similar to *gigas* (7, 36), suggesting that the photoperiod response mechanism is intact but the conversion of vegetative to primary inflorescence meristem is blocked. Comparative mapping in pea and medicago has located *Veg1* near two MADS box genes that are homologues of arabidopsis *FRUITFULL* and *SEPALLATA1* (15).

A second locus *VEGETATIVE2 (Veg2)* has yet to be described in a primary research paper, but descriptions of two mutant alleles are available (30, 31). The stronger of the two alleles confers a non-flowering phenotype similar to *veg1.* However, a weaker allele, *veg2-2,* displays an unique phenotype that reveals the role of this gene in secondary inflorescence development. Commencing at the node of flower initiation in WT, axillary branches of *veg2-2* plants are released, and produce a series of axillary structures varying more-or-less continuously from normal lateral branches at lower nodes to normal secondary inflorescences and flowers at higher nodes. In intermediate lateral structures, flowers may be produced directly from nodes as in a normal secondary inflorescence, but there is a failure to suppress leaf formation and to terminate apical growth. Recent data confirm that *Veg2* is located on the bottom half of linkage group I (J. Weller and I. Murfet, unpublished).

We recently identified a third locus in this group, *LATE BLOOMER 5 (Late5).* The single known *late5* mutant allele shows similarities to the weak *veg2-2* allele, resulting in late flowering, partial loss of secondary inflorescence identity, and floral abnormalities. Although not allelic with *Veg2,* preliminary results also locate *Late5* to the bottom of group I (J. Weller and S. Davidson, unpublished). Interestingly, the corresponding region in medicago includes homologues of the arabidopsis genes *FD* and *SVP* (J. Weller and V. Hecht, unpublished), and we are currently examining the relative map positions and relationships of these genes.

3. Isolation, mapping and expression analysis of flowering genes

We previously reported the isolation and mapping of many different pea homologues of arabidopsis flowering-related genes (15). This work is continuing and additional flowering related gene homologs identified, isolated and mapped in pea and/or medicago include *PHYE, FRI, SVPb, PRR3/7, PRR5/9, TIC, FHY3, SHP, STK, SPA1/2* and *CDF1/2, LUX, FTLd/e* and *FD* (V. Hecht, L.C. Liew, C. Knowles and J. Weller, unpublished data). Where relevant we are now examining the transcriptional regulation of many of these genes in studies of circadian rhythms, light and temperature responses, mobile signalling and inflorescence development.

Of particular relevance to photoperiodic flowering are the *CONSTANS (CO)* and *FLOWERING LOCUS T (FT)* gene families, both of which appear to have undergone differential expansion compared to their arabidopsis counterparts (15, 16). New comparative mapping data suggest locations for the four pea Group I CO-like *(COL)* genes *(COLa-COLd)* in LGV, LGII, LGIII and LGIV, respectively. Interestingly *COLa,* the most similar pea gene to *AtCO,* shows a different diurnal expression pattern than *AtCO,* and is not regulated by *Late1,* suggesting that it may have a different role than *AtCO* (16).

Similarly, the *FT* family in arabidopsis contains two genes *(FT* and *TSF)* but there appear to be at least five in pea and medicago (V Hecht, J Weller unpublished). By inference from medicago the pea genes are expected to be located in two clusters, in the middle and bottom of LGV. At least one of these genes is

specifically expressed in expanded leaves under LD (C Knowles, V Hecht unpublished) and this expression is greatly reduced in the *late1* mutant (16). This suggests that pea *FTL* genes may have broadly conserved roles, and we are now carrying out detailed expression studies of *FTL* genes in a wide range of different conditions and mutant backgrounds.

4. Conclusions

Our results so far have already indicated that a number of changes are necessary to previous working models for flowering in pea. Recent comparative studies in a range of species suggest a broad conservation of flowering mechanisms (9, 14, 18, 41), and we have found it useful to move to a comparative model based generally on arabidopsis. In arabidopsis, the FT protein acts as a mobile flower-promoting signal that integrates light, daylength, circadian clock, ambient temperature and vernalization inputs (3, 10, 33), and the molecular phenotypes of most pea mutants seem to fit at least generally with such a model. However, that is not to say that are no significant differences between the pea and arabidopsis flowering systems. In fact, it is already clear that there are several points of difference—concerning, for example, the roles of CO- and *FT*-like genes, the pleiotropic nature of the photoperiod response, the specification of the secondary inflorescence, and the nature of the vernalization mechanism.

It seems likely that mapping, expression studies and physiological analyses will soon help to identify the molecular basis for many of the mutants collected in Hobart over the past forty years. We hope that this will help us to understand the mechanisms regulating flowering in pea, and in particular, to give us insight into those mechanisms that are divergent or perhaps even unique in pea. This should in turn yield valuable information for the genetic analysis of flowering in related legumes such as lentil, medicago, clover and chickpea.

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