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FLOWERING NEWSLETTER REVIEW

Update on the genetic control of flowering in garden pea

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Abstract

The garden pea has been a model for the genetics of flowering for several decades and numerous flowering loci have been identified, but until recently little was known about the molecular nature of these loci. This paper presents an update on recent work on the molecular genetics of flowering in pea, outlining progress in gene and mutant isolation, expression analyses, grafting and other physiological studies, and candidate gene assessment. Work so far has led to the identification of the *LATE1* and *DNE* loci as orthologues of *Arabidopsis GIGANTEA* and *ELF4*, respectively, and candidate genes for several other loci are being evaluated. Expression analysis of an expanded *FT-like* gene family suggests a more complex role for this group of genes. These results provide the first insight into the circadian clock, photoperiod response mechanism, and mobile signals in pea, and identify both conserved and divergent features in comparison with *Arabidopsis*.

Key words: Circadian clock, flowering, *Medicago*, mobile signals, pea, photoperiod, photoreceptor.

Introduction

The legume family is a large, diverse, and economically important plant group that includes a number of important food, feed, oil, and fodder crops. Both daylength and temperature have an important influence on flowering and growth habit of many legume crop species and responsiveness to these factors is an important production trait. Genetic variation for flowering has been documented in many legume species, with several flowering loci known in the short-day plants (SDP) soybean (*Glycine max*) and common bean (*Phaseolus* spp), and at least one Mendelian locus or major-effect QTL identified in many others, including the long-day plants (LDP) chickpea (*Cicer arietinum*), lentil (*Lens culinaris*), lupin (*Lupinus* spp), clover (*Trifolium* spp), and barrel medic (*Medicago truncatula*) (Sarker *et al.*, 1999; Cogan *et al.*, 2006; Millan *et al.*, 2006; Phan *et al.*, 2007; Pierre *et al.*, 2008). However, among legumes, garden pea (*Pisum sativum*) is the species for which the genetic control of flowering is best understood. This is perhaps, in part, the legacy of Mendel, but is also due to the impetus provided by the availability of mutants derived from radiation breeding in Eastern Europe during the 1950s and 1960s. More than 20 flowering-related loci have been identified in pea from natural and induced genetic variants, and there is a substantial body of older literature on the physiological studies of flowering and mobile flowering signals (Haupt, 1969; Murfet, 1977; Murfet and Reid, 1993; Weller *et al.*, 1997).

Most of this older 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 (Franklin *et al.*, 2005; Sung and Amasino, 2005; Gardner *et al.*, 2006; Imaizumi and Kay, 2006; Turck *et al.*, 2008). This information, together with the availability of extensive sequence databases in a number of model legumes (Sato *et al.*, 2007) and the well-documented synteny between pea and *Medicago* (Kaló *et al.*, 2004; Aubert *et al.*, 2006) has

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opened up a number of avenues for the molecular analysis of flowering in pea.

Work on flowering at the University of Tasmania recommenced in 2002, with the isolation of numerous flowering gene homologues and new flowering mutants. After an initial period of slow progress during which these resources were generated, results of this work are now beginning to emerge. Earlier updates appeared in FNL 36 and FNL 40, and this update will provide an overview of recent developments and work currently in progress. Numerous reviews of older work are available (Murfet, 1977, 1985; Murfet and Reid, 1993; Weller *et al.*, 1997) and can be consulted for further information.

Isolation of genes and mutants

We started with the assumption that the conservation of flowering pathways might support a candidate gene approach to the identification of some, if not all pea flowering loci. As a first step in this approach, the isolation and mapping of many different pea homologues of *Arabidopsis* flowering-related genes were reported (Hecht *et al.*, 2005). This work has continued and many additional flowering-related gene homologues have been identified, isolated, and mapped in pea and/or *Medicago*, including *PHYE*, *FRI*, *FRLa*, *SVP*, *PRR37*, *PRR59*, *TIC*, *TEJ*, *FHY3*, *SHP*, *STK*, *SPA1/2*, *CDF1/2, LUX*, and *FD* (V Hecht, LC Liew, F Sussmilch, J Weller, unpublished data). Not surprisingly, almost all *Arabidopsis* flowering-related genes are represented in legumes in some form, although, as might be expected, there are many gene families that differ in size between legumes and *Arabidopsis*. This is conveniently illustrated by the three photoreceptor families active in flowering which are either larger (cryptochromes) or smaller (phytochrome and ZTL/FKF families) in pea and *Medicago* than in *Arabidopsis* (Hecht *et al.*, 2005). A MADS-domain gene clearly belonging to the *FLC* clade has still not yet been identified, although one recent report places sequences from soybean within this group (Reeves *et al.*, 2007). However, *Medicago* EST and genome sequencing is still continuing, and it is possible more sequences may yet come to light. In parallel, phenotypic screening has also been conducted for new flowering loci, focusing initially on loci needed for the promotion of flowering, which were underrepresented in the older mutant collection. These screens have resulted in the isolation of over 20 new mutants and so far have defined six new loci, *LATE BLOOMER 1* (*LATE1*) to *LATE6*, in addition to new alleles at many known loci.

Photoperiod response loci

Two classes of photoperiod response mutants are known in pea; early day-neutral mutants that behave under short-day (SD) conditions as if grown under long days (LD), and late day-neutral mutants that behave under LD as if grown under SD. One of the features that distinguishes these mutants as photoperiod-response mutants is the fact that they affect all photoperiod-responsive aspects of growth and not just the induction of flowering. The existence of early-flowering photoperiod-insensitive forms has been known for over 80 years, and genetic analysis showed that this trait was conferred by recessive alleles at a locus termed *SN* (Murfet, 1971*a*). Two other loci, *DNE* and *PPD*, are known from induced mutants (King and Murfet, 1985; Arumingtyas and Murfet, 1994). Somewhat surprisingly, our new flowering screens have so far not identified any new mutants in this class, and have returned only new alleles of *sn* and *ppd*. However, a similar early day-neutral phenotype is also conferred by two other mutants with primary defects in light signalling; a dominant hypermorphic *phyA* mutant (Weller *et al.*, 2004) and a mutant for the pea *COP1* orthologue *LIP1* (Sullivan and Gray, 2000; JL Weller, unpublished data).

Photoperiod response mutants in the late day-neutral class were not represented among the older collection of pea mutants, but several loci of this type have subsequently been identified (Weller *et al.*, 1997; Hecht *et al.*, 2007). Under phytotron conditions, null mutants for the phyA photoreceptor are late-flowering and unresponsive to photoperiod, indicating that phyA is the predominant photoreceptor mediating the effects of LD (Weller *et al.*, 1997, 2001). However, *phyA* mutants do show a promotion of flowering by far-red and blue light photoperiod extensions indicating a subsidiary role for other photoreceptors, including phyB and possibly cry2 (Weller *et al.*, 2001; Platten *et al.*, 2005). Mutants in the *LATE1* gene have a LD phenotype similar to *phyA,* but differ in their effects on seedling de-etiolation, causing only mild hyposensitivity to R and FR (Hecht *et al.*, 2007). In a manner generally consistent with the role of its *Arabidopsis* orthologue *GIGANTEA*, *LATE1* also influences diurnal and circadian rhythms of several clock genes including *LHY*, *TOC1*, and *ELF4* (Hecht *et al.*, 2007). The identity of two other loci *LATE2* and *LATE6* is not yet known, but mapping data have ruled out several potential candidate genes, including *CRY2a*, *CRY2b*, *FKF*, *PRR59*, and the four known Group I *CO*-like genes (V Hecht, LC Liew, J Weller, unpublished data).

As in the case of these late mutants, an early photoperiod-insensitive mutant phenotype could also result from a defect in light signalling or in the circadian clock. This is clearly the case for *phyA-3D* and *lip1*. Unlike these mutants, the *sn*, *dne*, and *ppd* mutants have no gross photomorphogenic defects, but have clear defects in rhythmic gene expression under light/dark cycles and constant conditions (V Hecht, LC Liew, unpublished data). In parallel with physiological studies of these mutants, the map positions for all three loci have been refined and relationships with candidate circadian-clock-related genes in corresponding regions have been examined. These studies recently identified *DNE* as *PsELF4* (LC Liew *et al.*, unpublished data). They have also ruled out several genes as candidates for *SN* and *PPD*, including *LHY*, *ELF3*, *TIC*, *FHY3*, and several *PRR* genes, but the relationship to other genes (*TOC1*, *LUX*, *TEJ*) remain untested.

The circadian clock

Pea and *Medicago* contain orthologues of all the major clock genes known from *Arabidopsis*. The only exception is *CCA1*, which has not been isolated from pea and is so far not represented in publicly-accessible sequence databases for any temperate legume. The basic patterns of rhythmic expression under light/dark cycles are conserved between pea and *Arabidopsis*, with *LHY* peaking in the morning and other genes (*LATE1*/*GI*, *TOC1*, *PRR*, *DNE*/*ELF4*) peaking in the evening (Hecht *et al.*, 2007; LC Liew *et al.*, unpublished results). These rhythms are photoperiod responsive, with peaks under LD (16L:8D) cycles occurring roughly 4 h later than in SD (8L:16D) cycles.

Surprisingly, the rhythms seen under photoperiod cycles are not maintained when plants are moved into continuous light (V Hecht, LC Liew, unpublished results). This is different from *Arabidopsis*, where, under the same regime, most of these genes show strong circadian rhythms. However, after transfer to darkness, circadian rhythms are apparent for *LHY*, *LATE1*, and *DNE*, although somewhat weaker than rhythms seen under photoperiod cycles (LC Liew, unpublished results). The damping of rhythmic expression under constant light can also be relieved if plants are subsequently transferred to constant darkness.

The *HIGH RESPONSE (HR)* locus

Similar to many other temperate species, the ancestral forms of *Pisum* are adapted to overwinter in the vegetative state, and show strong suppression of flowering by SD and/or a strong vernalization requirement. Selection of spring varieties in which these requirements are relaxed has occurred in many species and has enabled a shift from winter to summer cropping in several legume crops including pea, lentil, and chickpea (Kumar and Abbo, 2001).

The majority of garden pea cultivars have a spring habit which is conferred by recessive alleles at the *HR* locus (Lejeune-Hénaut *et al.*, 2008). Dominant *HR* alleles act to inhibit flowering predominantly under SD, and are found mainly in field and forage cultivars. In *HR* lines inhibition may be so strong as to confer a near-obligate requirement for long days or vernalization. *HR* can therefore be considered as either a photoperiod response gene or as similar to *FRIGIDA (FRI)* and *FLOWERING LOCUS C* (*FLC*) in *Arabidopsis*. These loci are typically discussed as mediators of vernalization and are not generally considered as part of the *Arabidopsis* photoperiod pathway, although they do also affect the circadian clock and influence the photoperiod response through a dramatic delay in flowering under SD (Sung and Amasino, 2005; Salathia *et al.*, 2006). Orthologues of both *FRI* and *FRIGIDA-LIKE 1* (*FRL1*) are present in pea and *Medicago* (Hecht *et al.*, 2005), and *PsFRI* is linked to *HR* (I Lejeune, C Rameau, J Weller, unpublished results).

However, an alternative possibility is that *HR*, like *SN*, *DNE*, and *PPD*, may have a more specific defect in rhythmic gene expression. Recent preliminary results suggest that this is indeed the case (B Wenden, LC Liew, unpublished results), and that introgression of dominant *HR* alleles into our standard wild-type line can restore the robust circadian rhythms of clock gene expression under continuous light. This difference suggests several genes with a role in light input to the clock as potential candidates for *HR*, including *ELF3*, *TIC*, *LUX*, and *FHY3*. It also indicates that most standard cultivars of garden pea used in developmental studies have a significant defect in light input into the circadian clock. This will clearly need to be kept in mind in future studies, and introgression of mutants into an *HR* background is underway in order to assess their effects fully. It is interesting that analogous situation occurred in *Arabidopsis*, where most early studies were carried out in spring annual lines containing recessive alleles at the *FRI* and *FLC* loci. Identification of the *HR* locus is clearly of interest and should provide important insight into the origin of the spring habit in pea, and comparisons with other legumes.

The photoperiod response mechanism

It now seems clear that many of the known pea photoperiod response mutants affect the circadian clock or light perception. There has also been interest in examining whether these genes affect flowering through the well-characterized *CO*/*FT* pathway. Based on our gene isolation efforts and on available sequence data, it appears that pea and *Medicago* have four group I *CONSTANS*-like (*COL*) genes and five *FT*-like (*FTL*) genes (Hecht *et al.*, 2007; V Hecht, J Weller, unpublished results). Among the *COL* genes, the most similar gene to *CO* is *COLa*, but this gene is clearly not a *CO* orthologue. Under LD photoperiods, its diurnal expression rhythm has a peak at dawn and is therefore more similar to *Arabidopsis COL1* and *COL2* than to *CO* (Hecht *et al.*, 2007). Interestingly, expression of *COLa* is also not obviously misregulated in either *dne* or *late1* mutants (Hecht *et al.*, 2007; LC Liew, V Hecht, unpublished results), whereas in *Arabidopsis* both *elf4* and *gi* mutants show marked misregulation of *CO* (Suarez-Lopez *et al.*, 2001; Doyle *et al.*, 2002). *COLb* and *COLc* expression is also not affected by *LATE1* or *DNE*. We therefore have little evidence so far to suggest that *COL* genes have a role in the LD promotion of flowering, but are now addressing this question directly by isolation of specific *COL* mutants in both species using new platforms for reverse genetics (Dalmais *et al.*, 2008; Tadege *et al.*, 2008).

In the case of the *FTL* genes, we have a bit more to work with. Of the five known genes, *FTLc* is the most divergent, whereas the other four form two closely-related pairs. These genes are also physically located in two clusters, suggestive of an origin through tandem duplication. The first of the pea genes we isolated, *FTLe*, is expressed specifically in leaf tissue under LD, and this coincides with the timing of floral induction (V Hecht, C Knowles, unpublished results). The induction of *FTLe* in LD is impaired in the *late1* mutant (Hecht *et al.*, 2007), but does not show abnormal expression

in the *dne* mutant in SD (LC Liew, unpublished results), suggesting that this gene cannot be responsible for the early flowering of *dne*. It also suggests that, although *DNE* and *LATE1* are both part of the clock, they may be coupled to *FTL* genes via distinct mechanisms. The expression patterns of other *FTL* genes differ markedly in timing, tissue specificity, and photoperiod dependence (V Hecht, unpublished results); differences that hint at a different, more complex network for the integration of flowering pathways. Preliminary evidence also suggests that unlike *FTLe*, other *FTL* genes are misregulated in both *dne* and *late1* mutants. We are currently conducting functional analyses of individual *FTL* genes in order to understand how they act and interact.

However, an additional perspective is already provided by mutants at the *GIGAS* locus. These mutants are phenotypically distinct from the late photoperiod-response mutants, in that they show/retain strong growth responses to LD, but flower late or not at all with an associated profusion of aerial branches (Beveridge and Murfet, 1996; Weller *et al.*, 1997). The *gigas* mutants also flower later than WT in SD, but with an overall phenotype similar to WT (Taylor and Murfet, 1994; Beveridge and Murfet, 1996). Because the more severe mutant allele, *gi-2*, blocks flowering specifically under LD, it confers what is essentially an obligate short-day habit. Flowering of *gigas* mutants in LD can be restored/promoted by grafting to leafy WT stocks, indicating that *GIGAS* may contribute to a mobile flowering stimulus (Beveridge and Murfet, 1996), and suggesting *FT*-like genes as possible candidates. This possibility was strengthened by the observation that *GIGAS* maps near *FTL* genes in the middle of linkage group V (Hecht *et al.*, 2005), and recent molecular analysis suggests that the two known *gigas* alleles affect the *FTLa*/*FTLb* cluster (V Hecht, unpublished results). If *GIGAS* is confirmed as an *FTL* gene, this would identify an *FTL* gene with a role restricted to the induction of flowering, and not required for the photoperiod control of other growth traits. It remains to be determined whether the photoperiod responsiveness of *gigas* mutants depends on other *FTL* genes or is independent of the *FTL* family.

Several other interesting features of *gigas* mutations are coming to light. Firstly, the non-flowering LD phenotype of the strong *gigas* allele *gi-2* can be overcome by the *phyA* and *late1* mutations (J Vander Schoor and J Weller, unpublished results), consistent with the fact that these mutations confer a constitutive SD phenotype, and *gigas* mutants do flower in SD. Conversely, the double mutant *dne gi* does not flower under LD or SD conditions (LC Liew, J Weller, unpublished results). Expression of the *gigas* phenotype in LD is also influenced by light quality and temperature, and mutants are more likely to flower under higher irradiances (Taylor and Murfet, 1994), at lower ambient temperatures (J Weller, J Vander Schoor, unpublished results) or in response to supplementation with light of low R:FR ratio (Beveridge and Murfet, 1996; J Weller, unpublished results). Consistent with this last response, loss of phyB can also override the *gigas* non-flowering LD phenotype (J Weller, J Vander Schoor unpublished results).

Grafting experiments and mobile signals

Previous models considered the photoperiod response in pea to result from down-regulation of a mobile floral inhibitor in LD (Murfet, 1977, 1985; Weller *et al.*, 1997). As described in an earlier update for the *Flowering Newsletter* (Weller, 2005), this question has been re-examined in a range of new grafting experiments with both early and late photoperiod response mutants, and it is concluded that the major graft-transmissible effects on flowering that were observed were consistent with the action of a mobile stimulus (Hecht *et al.*, 2007; LC Liew, J Weller, unpublished results). In addition, grafting experiments with a *dne late1* double mutant show that *late1* can block the graft-transmissible effects of *dne* in stock leaves, implying that both *DNE* and *LATE1* act through the same mobile flowering stimulus, and that *LATE1* acts downstream of *DNE* (LC Liew, J Weller, unpublished results). However, in similar experiments with a *dne gi* double mutant, the *gi* mutation has no effect on the ability of *dne* stocks to promote flowering across a graft union (J Weller, LC Liew, J Vander Schoor, unpublished results). One interpretation of this result is that a second, *GIGAS*-independent mobile flowering stimulus is also produced in *dne* mutant leaves.

The *LATE FLOWERING (LF)* locus

The *LF* locus affects the transition to flowering without influencing photoperiod responsiveness, and is notable as the first of the classical pea flowering loci to be identified at the molecular level. One of three pea homologues of *Arabidopsis TFL1* (*TFL1c*) was identified as a candidate gene for *LF* based on its map position, and several strong *lf* mutants were shown to have large deletions or amino acid substitutions in *TFL1c* consistent with a complete loss of function (Foucher *et al.* 2003). The isolation of an additional EMS mutant (*lf-22*) carrying a nonsense mutation has provided further support for this conclusion (V Hecht, J Weller unpublished results). 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 WL1771/1770/1769 isolines carry different alleles at the *LF* locus and differ substantially in flowering node, but show no polymorphism within the coding region or introns of *LF* (Foucher *et al.*, 2003). However, *LF* expression does correlate with flowering in these lines, and the existence of functionally significant polymorphisms in the *LF* promoter region has not yet been excluded.

Despite the importance of *LF* for flowering time, it is not known how it participates in mechanisms controlling flower transition. The effects of *LF* are not graft-transmissible (Murfet, 1971*b*), suggesting that it acts in the shoot apex. Preliminary results suggest that expression of *LF* occurs throughout the plant and does not show any marked developmental or environmental regulation (Foucher *et al.*, 2003; B Wenden, V Hecht, J Weller, unpublished results),

and it will be interesting to see if this is supported by more detailed studies. Null *lf* mutants show an additive interaction with photoperiod response mutants, but are completely epistatic to *gigas* mutants (J Weller, J Vander Schoor, unpublished results). This interaction is interesting in light of the proposed mechanisms for the interaction of *FT* and *TFL1* in *Arabidopsis* (Hanzawa *et al.*, 2005; Ahn *et al.*, 2006), and is consistent with the possibility that loss of *LF* may result in constitutive activation of the *GIGAS*-dependent branch of the flowering pathway.

Inflorescence identity loci

The pea inflorescence is a compound raceme, and its development has been discussed in several reviews (Singer *et al.*, 1999; Benlloch *et al.*, 2007). 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 (Hofer *et al.*, 1997; Taylor *et al.*, 2001, 2002; Berbel *et al.*, 2001). Although all three mutants 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 correctly to 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 *DETERMINATE* locus has a negative role in secondary inflorescence development, acting to prevent expression of the secondary inflorescence programme in the primary inflorescence meristem. This role is similar to that of *Arabidopsis TFL1*, and *DET* is now known to encode another of the three *TFL1* homologues (*TFL1a*) in pea (Foucher *et al.*, 2003). The genetic interactions and molecular consequences of *det* mutations have yet to be explored. However, it was recently found that unlike *LF*/*TFL1c*, expression of *DET* is strongly up-regulated at the time of the floral transition (F Sussmilch, V Hecht, unpublished results).

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 (Gottschalk, 1979). Despite their failure to flower, *veg1* mutant plants grown in LD clearly undergo a vegetative shutdown similar to *gigas* (Reid and Murfet, 1984; Beveridge and Murfet, 1996), 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* (Hecht *et al.*, 2005).

A second locus *VEGETATIVE2* (*VEG2*) has yet to be characterized in a primary research paper, but descriptions of two mutant alleles are available (Murfet, 1992; Murfet and Reid, 1993). The stronger of the two alleles confers a non-flowering phenotype similar to *veg1*. However, a weaker allele, *veg2-2*, displays a unique phenotype that reveals the role of *VEG2* 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 (Murfet and Reid, 1993).

A third locus has recently been identified in this group, *LATE BLOOMER 5* (*LATE5*). The single *late5* mutant allele to be identified so far shows similarities to the weak *veg2-2* allele, resulting in late flowering, partial loss of secondary inflorescence identity, and floral abnormalities. However, in contrast to the *veg2-2* mutant the *late5* inflorescence phenotype is transient, affecting only the first flowering node. Although *LATE5* is not allelic with *VEG2*, both loci map to the bottom of linkage group I in a region where homologues of the *Arabidopsis* genes *FD* and *SVP* are also located (F Sussmilch, V Hecht, S Davidson, J Weller, unpublished results). The relative map positions and relationships among these genes are currently being examined.

Conclusions and future prospects

Our results indicate that changes to previous genetic models for flowering in pea (Murfet, 1985; Reid *et al.*, 1996; Weller *et al.*, 1997) are clearly necessary, and it has been more constructive to move to a comparative model based generally on *Arabidopsis* (Fig. 1). In *Arabidopsis*, the FT protein acts as a mobile flower-promoting signal that integrates light, daylength, circadian clock, ambient temperature, and vernalization inputs (Turck *et al.*, 2008) and the molecular phenotypes of most pea mutants seem to fit at least generally with such a model. However, it is also becoming clear that there are numerous points of difference concerning, for example, the roles of *CO*- and *FT*-like genes, the pleiotropic nature of the photoperiod response, regulatory interactions within the circadian clock, 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 our group over the past 40 years. This should provide significant new insight into the mechanisms regulating flowering in pea, and identify those that are divergent or

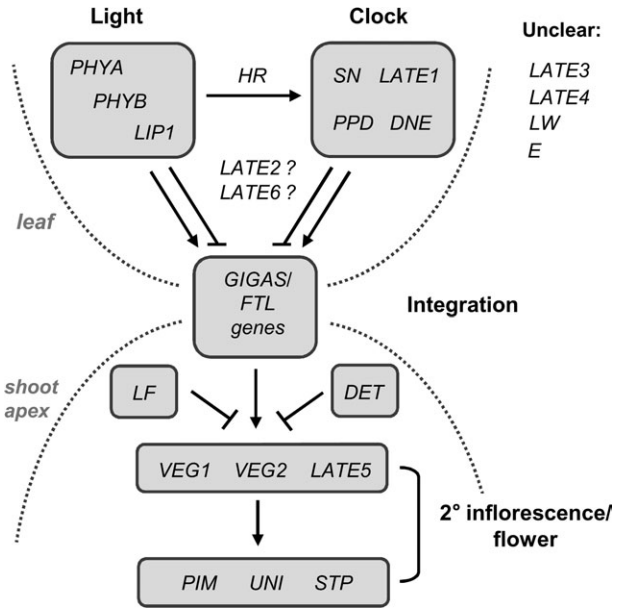

Fig. 1. Model for genetic control of flowering in pea, summarizing the roles and interactions of known pea flowering loci.

perhaps even unique. It will also yield valuable information for the genetic analysis of flowering and related growth traits in other important food and forage legumes including lentil, clover, and chickpea.

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