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The Genetic Regulation of Flowering Time in Pea (*Pisum sativum* L.)

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Abstract
The time of flowering is one of the primary adaptive attributes in the survival strategy of flowering plants. This feature is a significant ingredient of crop adaptation and yield. When hypothetical studies are applied on the flowering time in pea, the short day and long day circumstances of growing season are taken into consideration. In the same way, the flowering time and the first node location of the flower are straight related to each other. Over 20 loci connected to flowering time and inflorescence development have been identified in pea. A detailed comprehension of flowering mechanisms is the substance of future improvements for genetic sequence-based selection and target manipulation of genes. The aim of this review is to summarize the present knowledge of flowering time regulation in the pea.

Keywords: Adaptation, genes, locus, selection, yield

INTRODUCTION

Economically, legumes are the second best-known crop genus with regarding 27% of the world's crop output [1], and they are the 3rd topmost family of flowering plants with more than 650 genera and 18,000 species [2]. Garden pea (*Pisum sativum* L.) is the fourth most-produced crop among legumes grown worldwide. According to the most recent data of the United Nations Food and Agriculture Organization (FAO), worldwide garden pea production was approximately 19.88 million tons in 2016 from 2.59 million hectares. According to the FAO, Turkey produces 112.643 tons of garden peas annually, and it ranks as the 10th largest producer [3]. Normally in temperate climate zone of Turkey, such as Marmara and Ege region, pea grows and yields at maximum, date from end of the autumn to May or June. In some cases, the farmers expected significant yield loses in pea caused by cold or heat stress during flowering.

It is supposed that proper timing of flowering under favorable conditions have an important adaptive value for plants, in order to achieve pollination and fertilization [4]. Flowering sets the transition from vegetative stage to generative stage in plants. The genetic arrangement of flowering time is more susceptible to ecological indicators than many other agriculturally relevant traits. A critical phonological growth stage of crops is the floral initiation and timing of flowering [5]. In numerous seasonal crops, the short occurrence of hot temperatures (>32–36 °C) can greatly decrease seed formation, and hereby crop yield, if they come across with a brief critical period of only 1–3 day around the time of flowering. For this reason, the moderation of crop development will be crucial to the effects of climate alter on yield in two ways: owing to define the season length, and therefore the presence of water, radiation, and nutrient resources for growth; and by affecting the exposure of the crop to climate extremes [6].

Pea flowers, similarly most zygomorphic flowers, have a distinct corolla with three petal types, which are collocated throughout a dorsoventral axis, and indicate two types of dissymmetry: dorsoventral dissymmetry in the floral plane and organ internal dissymmetry in the floral organ plane [7].

Pea shows an indeterminate shoot growth structure and, once flowering is initiated at a certain node, called the first node of flower initiation (NFI), all following later formed nodes are floral. Therefore, the date of starting of the first flowering node is assumed to be the time of floral induction [8].

Although most pea cultivars are facultative long-day plants, some are day neutral, and others are fundamentally obligate long-day plants that may not flower at all in photoperiods shorter than 12 h if un-vernalized. The early varieties of pea generally flower at the ninth or tenth node and are insensible to vernalization and photoperiod. The late varieties, which usually do not flower under the 15th node, generally proceed as quantitative long-day plants and are vernalizable [9].

Pea (*Pisum sativum* L.) was the primary genetic model species, used to indicate central genetic notions such as dominance, segregation and independent assortment [10].

An elaborate understanding of flowering mechanisms is the basis of future improvements for genetic sequence-based selection and target manipulation of genes. Numerous works have been conducted to detect probable signaling pathways managing flowering time in pea, such as the physiological characterization of flowering mutants grown in varied peripheral conditions and grafting studies [7].

Early works on genetic regulate of flowering resolved a few loci from substantial variation among different cultivars of garden and field pea, while other loci were later identified through the definition of induced mutants and specific mutant screens.

The subject of this review is to summarize the current knowledge of flowering time regulation in the pea. This information will be shared under two main sections: a) Naturally variations, b) Induced mutants.

Naturally Variations

The occurrence of spontaneous variation in the flowering time of several pea cultivars, by the first node of flower initiation on the primary shoot of the plant, defined using phonotypical classification by Murfet. According to author, this natural variation appeared by variation in the main

flowering loci: SN (sterile nodes), LF (late flowering) and HR (high response to photoperiod) [11; 12].

SN (Sterile Nodes):

Grafting tests have found that the *SN* gene collocate the formation of an inhibitor in the cotyledons and foliaceous shoot. The inhibitor output of *SN* reserves is reduced by exposure to long photoperiods or by vernalization. The inhibitor mediates not only photoperiodic flower induction but also the photoperiodic response for the wide range of pleiotropic features such as the proportion of flower bud improving, the period of the reproductive stage, the development of basal laterals and ontogenetic transition in leaf complexity [20]. A new study has founded *SN* as pea orthologs of Arabidopsis circadian clock genes *LUX* [14, 15].

Mutant recessive *sn* plants flower early in both long day and short day conditions. But these plants are unable to respond to photoperiod and when grown in short days display the brief generative stage and quick reproductive growth typical of wild-type plants in long days [16].

According to Barber, a single dominant gene, *SN*, regulate not only a high flowering node but also a capability to respond to both photoperiod and vernalization and that long days and vernalization behave competitively to reduce the flowering node [17].

The vernalization response is not observed if the expression of the *Sn* gene is hindered by exposure of the seedlings to sustained light from the beginning of germination. However, Reid and Murfet supported the opinion that long days or cold temperatures cause repression of *Sn* activity rather than demolition of flower inhibitor as suggested by Barber [18].

LF (Late Flowering):

The second locus, late flowering (*LF*), prohibits flowering in both long and short days conditions. This locus was defined over 10 years ago as a different homolog of *TFL1* [19]. The *LF* locus is considered as the primary of the classical pea flowering loci to be defined at the molecular level [16]. Variations at the *LF* locus occur regardless of environmental conditions [20].

A lot of allelic versions of *LF* are known, with the inclusion of both spontaneous occurring and induced mutant alleles [14]. Genotypes in which the dominant *LF* gene is deleted or inactivated by nonsense mutation display extremely early, photoperiod-insensitive initiation of flowering [19].

It regulates the transition to flowering without affecting photoperiod responsiveness. There are three pea homologues of Arabidopsis. One of them (*TFL1c*) was defined as a nominee gene for *LF* based on its map location, and several robust recessive allele mutants were demonstrated to have wide deletions or amino acid replacements in *TFL1c* consistent with a full loss of function [19].

Despite the significance of *LF* for flowering time, it is not known how it attends in mechanisms regulating flower transition. *LF* locus acts in the shoot apex, and its' influences are not graft-transmissible [16].

Examination of spontaneous variation identified four allelic classes: *lf-a*, *lf*, *Lf*, *Lf-d* with extension dominance conferring increasing first node of flower initiation and therewith postpone the time of flowering. These alleles characterize distinctions in the inherent minimum node of flower initiation, with rate of 5, 8, 11, and 15 nodes for *lf-a*, *lf*, Lf and Lf-d, respectively [21]. The large majority of the induced mutant alleles that have later been defined fall almost into one of these classes [20].

HR (High Response):

The third locus, high response (*HR*), induce early flowering in short day conditions and decrease, but do not eliminate, the photoperiod response [14]. This loci firstly characterized by Murfet [12].

The dominant Hr allele restrict flowering principally under short day conditions. In an otherwise wild-type genotype, this restriction may be so potent as to confer a near-obligate necessity for long days These alleles are found mostly in field and forage cultivars [16]. This long-day deficiency is unfastened to a quantitative response in plants carrying recessive *hr*. Grafting and photoperiod-transfer attempts demonstrate that inhibitor production in short days maintains high in dominant *HR* plants for a long-term, but drops quickly in recessive plants after approximately four weeks of growth [20]. For this reason, this locus considered as either a photoperiod response gene or as similar to Frigida (*FRI*) and Flowering Locus C (*FLC*) in Arabidopsis by some authors [16].

The plurality of garden pea cultivars has a spring habit which is conferred by recessive alleles at the *HR* locus [22]. Two new studies have founded *HR* as pea orthologs of Arabidopsis circadian clock genes *ELF3* [14; 15].

The examination of sequence variation proposes a large distribution of the hr allele through domesticated pea germplasm and a significant and ancient role for this mutation in the spring-flowering habit [15].

Identification of the *HR* locus is express of interest and should ensure significant comprehension into the origin of the spring habit in pea, and comparisons with other legumes [16].

E (Early Initiating):

The *E* locus was defined from diversity among modern cultivars. This locus governs inhibitor production in the cotyledons. The dominant *E* allele induces an important increase of flower initiation in a recessive *lf* or *lf-a* background, but does not influence the general photoperiod susceptibility of the plant. The variation between dominant E and recessive e is not expressed phenotypically in a wild-type (*LF SN hr*) structure, and for this reason, it is not normally essential to consider the genotype at the *E* locus. However, analysis of the interaction between the *E* and *LF* loci has demonstrated beneficial in describing the threshold character of the flowering process in pea [20].

The least well-understood naturally variant loci are *EARLY (E)*. Dominant alleles of *E* govern early start of flowering in some genetic structures, but this effect displays complex interactions with other loci and missing penetrance. The latest recognition of the main influence on quantitative trait loci (QTL) for flowering time in a chromosomal location similar to *E* [22; 15] may help in its further molecular characterization [14].

Interactions of SN, LF, HR, E

Allelic variations at the *HR, SN, LF*, and *E* loci interact to define a highly broad range of flowering times in plants in non-inductive situations. This range extends from the genotype "lf sn" which may flower as early as node 7 and is entirely insensitive to photoperiod, to genotype "*LF SN HR e*" which flowers relatively late under long day and may

not flower at all under short day [23;15] most mutagenesis programs have been governed in spring-flowering (hr) cultivars, and in some conditions in lines that also keep "sn" or *lf* alleles. Many of them are also probably to carry derived alleles at the *E* locus. Mutants isolated from these programs, therefore, carry a minimum one extra mutation affecting flowering time, and potentially as many as four [14].

Murfet defined the relation of four classes based on their behavior of flowering characters. These classes namely ED (early developing), El (early initiating), L (late) and LHR (late high response) have been studied and four major genes "*_Lf* (= S1)", "E", "*Sn* (= S2)" and "*Hr_*" have been found to arrange between class diversity and to some content within-class variation. However, there is also an argument of polygenic systems which in opposition seem to conduct usually to within-class variation but sometimes to between-class variation as in the case of the penetrance modifiers of "*Sn*" [11;12].

In this classification system [13];

- ***"lf e sn hr"*** : Early developing (flowers about node 10) and day-neutral.
- ***"lf E Sn hr"*** : Early initiating (the node of first initiated flower is early and day-neutral but the flowering time is delayed in short days as a result of retarded development or abortion of the lower flower buds).
- ***"lf e Sn Hr"*** : Late high response (it flowers with the "Late" plants in long days but is very extensively delayed (25-50 nodes) in warm short day conditions.
- ***"Lf e Sn hr"***; ***"If E Sn hr"***; ***"If e sn H"*** : basically early developing but Hr confers early initiating tendencies.

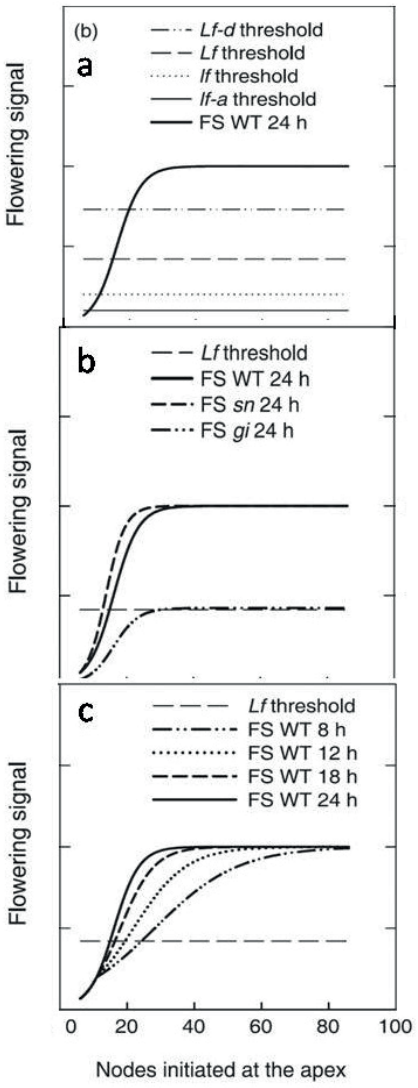

Figure 1: Based on the standard model hypothesis, the computational model imitates the level of flowering signal over time. Flowering is started when flowering signal reaches the flowering threshold, which is characterized by the LF allele (lf-a, lf, Lf, Lf-d; represented by horizontal lines) [8].

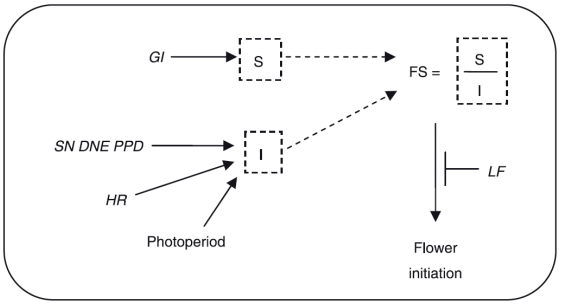

Figure 2: Schematic exhibition of the regulation of flowering in pea: a mobile flowering inhibitor (I) and mobile flowering stimulus (S) are integrated as a flowering signal (FS), which is equal to the ratio S/I. [8].

Induced Mutants

Dieneutralis (DNE) and ***Photoperiod (PPD)*** have an act similar to *SN*, as recessive *dne* and *ppd* mutants display early flowering in short day conditions. In the existence of recessive *hr*, these mutations confer perfect photoperiod in susceptibility for flowering and other traits [27].

The *phytochrome A (phyA)* mutants were preliminary rcognized by their insufficient de-etiolation responses to far-red light, and later shown to flower late in long day conditions with further phenotypes that are in fact a phenocopy of wild-type plants grown in short day [16;20]. Mutations in the *phyA* gene are required for the promotion of flowering in response to photoperiod elongations rich in red light. However, it has little impact on the response to blue light [28; 29]. The *phyA-3D* which a dominant, hypermorphic *phyA* mutant, was recognized in seedling screens, with an early flowering phenotype in short day conditions similar to the *sn*, *dne* and *ppd* mutants [30; 16]

Mutations in the ***phytochrome B (phyB)*** gene manage early flowering phenotype that is principally comprehensible in short day conditions. However, a null *phyB* mutation is epistatic to both *phyA* and late1 mutants in the long day, displaying that *phyB* may behave to postpone flowering in both short day and long day conditions [24; 29]. This also commits that *phyA* and *Late1* genes encourage flowering in the long day by opposing a *phyB*-dependent restriction. Unlike ppd, sn and dne mutants, *phyB* mutations simply influence the node of flower initiation and do not markedly modify other pleiotropic appearences of the photoperiod response [16]

Recent studies of recessive mutants at the **Gigas (GI)** locus have confirmed the presence of the floral stimulus and are yielding helpful information about its function. [20] Mutant *gi-1* plants flower later than the wild-type under all circumstances investigated to date, and display an increased response to photoperiod and vernalization. Except for the large delay in flowering, the aspect of the *gi-1* mutant in short days is relatively normal and is similar to that of lines carrying *HR* [20] .

Grafting of *gigas* mutant scions to wild-type stocks may ensue in a important increase of flowering [31], leading to the proposition that *gigas* is related in the production of a mobile floral stimulus. However, as the long day phenotype of *gigas* is different from *phyA* and *late1*, it seems probably the *gigas*-dependent mobile signal does not intervened all appearances of the photoperiod response but is restricted to the initiation of flowering [16].

The other locus has a positive act in secondary inflorescence development. The ***VEGETATIVE1* (*VEG1*)** locus (previously VEGETATIVE; VEG) is indicated by a single mutant allele. If plants carry homozygous genes, they never produce flowers and must be maintained through the heterozygote [33]. In spite of their collapse to flower, *VEG1* mutant plants grown in long day obviously spend a vegetative shutdown similar to gigas [34, 31], proposing that the photoperiod response mechanism is untouched but the rotation of vegetative to principal 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* [24; 32].

The other locus ***VEGETATIVE2 (VEG2)*** has yet to be defined in the main research paper, but the definitions of two mutant alleles are existent [35]. The vigorous of the two alleles confers a non-flowering phenotype similar to *VEG1*. However, a feeble allele, *VEG2-2*, shows an unmatched phenotype that brings to light the act of *VEG2* in secondary inflorescence development. Starting at the node of flower initiation in wild-type plants, axillary branches of *VEG2-2* plants are released, and generate a series of axillary structures altering more-or-less incessantly from normal lateral branches at lower nodes to normal secondary inflorescences and flowers at higher nodes. In middle lateral structures, flowers may be produced straight from nodes as in a normal secondary inflorescence. However, there is a defeat to forestall leaf formation and to finish apical growth [36].

The *late bloomer 1 (Late1)* mutants have the common aspect of short day-grown wild-type plants. These mutants flower delayed in long day conditions. These mutants flower delayed in long day conditions. Mutant late1 plants also have separates in rhythmic expression of circadian clock genes, suggesting that *Late1* may have a main role in clock function [24; 16].

Two other mutants have been identified, ***Late3*** and ***Late4*** by Weller and Vander Schoor. The *Late3* and *Late4* mutants have a new flowering phenotype defined by highly late flowering and a retardation in the complex leaf transition under both sort day and long day. Mutants do not start flowering until after node 35 and later cancel flower initials.

Some pods do finally formed later fertile nodes, but display very weak growth and yield few seeds [14].

The single ***Late Bloomer 5 (Late5)*** mutant allele to be defined until now displays similarities to the weak *VEG2*-2 allele, resulting in late flowering, partial loss of secondary inflorescence identicalness, and floral uncommonness. However, in opposition to the *VEG2-2* mutant the *Late5* the flowering phenotype is temporary, fascinating only the first flowering node. Despite *Late5* is not allelic with *VEG2*, both loci map to the bottom of the linkage group I in a region where homologs of the Arabidopsis genes *FD* and *SVP* are also located. The relative map positions and relationships among these genes are currently being investigated [32].

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