RESEARCH NOTE

Interaction between *COCHLEATA* and *UNIFOLIATA* genes enables normal flower morphogenesis in the garden pea, *Pisum sativum*

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Introduction

The simple stipules, leaflet and tendril bearing imparipinnately compound leaf blades and zygomorphic flowers, produced on stems of the diploid ($2n = 14$; $\geq$ 5000 Mbp nuclear genome) papilionoid Fabaceae plant *Pisum sativum,* are serving as unique and highly informative models for the dissection of plant developmental programmes. The growing information has revealed that the processes of stipule, leaf and flower morphogeneses are genetically interconnected in *P. sativum* (Hofer *et al.* 1997; Yaxley *et al.* 2001; Wang *et al.* 2008; Kumar *et al.* 2009).

Some important regulatory steps in the pathways of stipule and leaf development have been defined. Each vegetative and flower bearing node produces two stipules, one on each side of the leaf. Formation of a stipule at a node is autonomous of that of the leaf and other stipule at that node. Stipule primordium is initiated by the function of *COCHLEATA* (*COCH*) gene. Synergistic action of *COCH* gene and *STIPULE REDUCED* (*ST*), another major gene, promotes growth and development of the primordium into mature stipule (Yaxley *et al.* 2001; Kumar *et al.* 2009). Leaf comprises of petiole and its leaf blade. The leaf blade rachis, extension of petiole, bears one to three pairs of leaflets on the petiole side (proximal domain), one to four pairs of tendrils on the farther side of petiole (distal domain) and a terminal tendril (terminal domain). Leaf blade development is interactively controlled by functions of several major genes, including *UNIFOLIATA* (*UNI*), *STAMINA PISTILLOIDA* (*STP*), *TENDRIL-LESS* (*TL*), *AFILA* (*AF*) and *MULTIFOLIATE-PINNA* (*MFP*) (Gourlay *et al.* 2000; DeMason and Schmidt 2001; Yaxley *et al.* 2001; DeMason 2005; Mishra *et al.* 2009). The proximodistal growth of leaf blade rachis, sequential separation of leaflet and tendril primordia on the growing rachis are mediated by the *UNI* and *STP* functions. The *AF* and *TL* and *MFP* functions allow the formation of leaflet and tendril in the proximal, distal and terminal domains, respectively. Repression of *AF* on *UNI* does not allow rachis to ramify and thus normal leaf blade development is controlled (Mishra *et al.* 2009). *COCH* prevents stipule from assuming leaf blade-like compound architecture by repressing the *UNI* led leaf blade pathway, from each of the stipule domains (Kumar *et al.* 2009); stipules tend to become compound and leaf blade like in *coch* plants. *UNI* is an orthologue of *LEAFY* (*LFY*) gene and *STP* that of *UNUSUAL FLORAL ORGANS* (*UFO*) of *Arabidopsis thaliana* (Hofer *et al.* 1997; Lee *et al.* 1997; Gourlay *et al.* 2000; Taylor *et al.* 2001; Champagne *et al.* 2007). The *TL* and *UNI* products are transcription factors (Hofer *et al.* 1997, 2009); properties of the products of *COCH*, *ST*, *AF* and *MFP* genes remain to be revealed. Two kinds of *uni* mutant alleles have been used in the genetic analysis of leaf blade morphogenesis– the null alleles designated as *uni* and the promoter affected *unifoliata-tendrilled acacia* (*uni-tac*) allele; the *uni-tac* allele expresses intact *UNI* product albeit at about one sixth of the level of the wildtype pea (Hofer *et al.* 1997; Gourlay *et al.* 2000; DeMason and Schmidt 2001; DeMason and Chawla 2004a, b). It is for this reason that *uni-tac* plants produce compound leaf blades in which proximal domain is intact, distal is abridged to one or two pairs of tendrils and the terminal tendril is replaced by a leaflet, in contrast with a simple leaflet as leaf blade in which all three domains are merged in *uni* plants.

The flower morphologies of *uni*, *stp* and *coch* mutants are known to be highly disturbed (Monti and Devreux 1969; Hofer *et al.* 1997; Ferrandiz *et al.* 1999; Yaxley *et al.* 2001). The four whorls of the bilaterally symmetrical pea flowers comprise of five sepals (calyx), five petals (corolla), ten stamens (androecium) and central carpel (gynoecium). The corolla has three types of petals, two small petals placed

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abaxially are fused to form the keel, two comparatively larger petals placed laterally comprise the wings (alae) and a petal of very large size placed adaxially makes the standard (vexillum). Filaments of nine stamens are fused to form a staminal tube which encloses a subsessile carpel. The 10th stamen stands alone adaxially (Tucker 1989; Ferrandiz *et al.* 1999). Flowers of *uni* and *stp* mutants have similar abnormalities and are sterile. They have no petals and stamens, many carpelloid organs and an incomplete calyx whorl and there occur supernumerary flowers in the axils of sepals (Hofer *et al.* 1997; Ferrandiz *et al.* 1999). The *coch* mutant flowers have supernumerary sepals, petals, stamens and/or carpels. The staminal tube is often ruptured and stamens are variously fused. Also present are sepal–petal, petal–stamen and stamen–carpel mosaic organs. The number of each kind of organ varies among different flowers. The *coch* plants are only partially fertile (Yaxley *et al.* 2001). The flower abnormalities indicate that *UNI*, *STP* and *COCH* genes participate both in leaf/stipule and flower developmental programmes.

In *A. thaliana, LFY* is necessary for all stages of the flower development, from initiation of flower meristem to flower differentiation (Liu *et al.* 2009; Wagner 2009); *lfy* mutants produce indeterminate flowers which combine characters of both flowers and shoot (Weigel *et al.* 1992). *LFY* and *UNI* being orthologues, expectedly, *uni* flowers mimic the indeterminacy of *lfy* flowers in *P. sativum*. The leaf blade phenotype of *uni* mutant, abridgement of compoundedness, is in sharp contrast to assumption of expansiveness in *uni* flower structure. The indeterminancy in the *uni* flowers means loss of control, on the size of flower primordium and floral organs related subprimordia, which is intact in the presence of *UNI* function. Presence of supernumerary organs in *coch* flowers also suggests loss of control on the size(s) of floral primordium(ia). Ramification in leaf blade is controlled by repressive actions of *AF*, *TL* and *MFP* on *UNI*. A question arises as how is the size of flower primordium controlled such that normal flowers are formed; which control is lost in *uni* and *coch* plants. To begin to answer these questions, we have quantitatively characterized the flower phenotypes of 20 genotypes of related background: wild-type, *af, tl, mfp, uni-tac, af tl, af mfp, tl mfp, af uni-tac, tl uni-tac, mfp uni-tac, af tl uni-tac, af mfp uni-tac, tl mfp uni-tac, af tl mfp, af tl mfp uni-tac, coch, coch tl, coch uni-tac* and *coch tl uni-tac.* In these experiments, instead of the *uni* mutant allele of *UNI* which causes sterility in flowers, the *uni-tac* allele of *UNI*, which does not lead to loss of fertility, has been deployed (Sharma and Kumar 1981). The observations described below show that in *P. sativum* normal flower development requires optimum expression of *UNI* function via negative control of any hyper-expression by repressive control of *COCH* on *UNI*.

Materials and methods

Genotypes derived from a constant genetic background (Kumar *et al.* 2009) were grown in the field of NIPGR. The growth conditions have been described earlier (Sharma and Kumar 1981; Mishra *et al.* 2009). The sampling procedure for the characterization of flowers is described in the tables 1 and 2.

Results and discussions

The observed effects of leaf blade affecting mutations, *af*, *tl*, *mfp* and *uni-tac*, singly and in all possible combinations,

Table 1. Effect of *uni-tac* mutation in interaction with *af, tl* and *mfp* mutations on flower morphology in *Pisum sativum*.

Genotype[a]	Calyx (number of sepals)	Corolla[b] (standard[c])	Androecium (number of stamens)	Total number of flower organs[d]
WT, af, tl, mfp, af mfp, af tl, tl mfp, af tl mfp	5.0 ± 0	1.0 ± 0	10.0 ± 0	21.0 ± 0
uni-tac	5.0 ± 0	1.0 ± 0	9.8 ± 0.6	20.7 ± 0.7
af uni-tac	5.0 ± 0	1.0 ± 0.4 (0.9 ± 0.3)	9.5 ± 1.1	20.4 ± 1.1 (20.3 ± 1.1)
tl uni-tac	5.0 ± 0	0.6 ± 0.5 (0.5 ± 0.5)	9.0 ± 1.2	19.8 ± 1.4 (19.7 ± 1.5)
mfp uni-tac	5.0 ± 0	1.0 ± 0.2	10.0 ± 0.4	20.9 ± 0.5
af tl uni-tac	5.1 ± 0.2	0.8 ± 0.5 (0.8 ± 0.4)	9.7 ± 0.7	20.6 ± 0.6 (20.3 ± 0.6)
af mfp uni-tac	5.0 ± 0	0.9 ± 0.3	9.9 ± 0.4	20.8 ± 0.6
tl mfp uni-tac	5.0 ± 0	0.6 ± 0.5	9.2 ± 1.1	19.8 ± 0.2
af tl mfp uni-tac	5.0 ± 0	1.0 ± 0	10.0 ± 0.3	21.0 ± 0.2
Mean of *uni-tac* genotypes	5.0 ± 0.04	0.9 ± 0.2 (0.8 ± 0.2)	9.6 ± 0.4	20.5 ± 0.5 (20.4 ± 0.5)

[a]Twenty five flowers taken from five plants on three occasions, were examined for each genotype. The construction of genotypes and phenotypes of leaf blades of all the genotypes have been reported earlier. The crop growing conditions, in 2009–2010 winter (rabi) season when the flowers were scored, were also like those reported earlier (Kumar *et al.* 2009; Mishra *et al.* 2009).
[b]There was no variation for the number of wings in any of the genotypes and among all genotypes only *af tl uni-tac* demonstrated variation in keel number.
[c]Number in parentheses is of the standard petal obtained by treating aborted standard as absent.
[d]Number of carpels was one in all the flowers examined; therefore, the total number of organs includes gynoecium.

Table 2. Effect of *coch* mutation on flower morphology in *Pisum sativum*.

Genotype[a]	Calyx (number of sepals)	Corolla (number of corolla components)				Androecium (number of stamens)	Gynoecium (number of carpels)	Total number of flower organs
		Standard	Wing	Keel	Others[b]			
coch	5.8 ± 0.8	1.2 ± 0.4	2.5 ± 0.9	2.3 ± 1.1	0.2 ± 0.5	10.9 ± 2.3	1.2 ± 0.4	24.1 ± 5.0
coch uni-tac	5.4 ± 0.7	1.1 ± 0.4	2.2 ± 0.4	2.3 ± 0.6	0.1 ± 0.3	9.4 ± 1.0	1.0 ± 0	21.5 ± 1.7
t_{48}	2.9**	0.6	1.7	0.3	0.3	3.7**	1.8	3.3**
coch tl	6.2 ± 1.0	1.3 ± 0.7	2.5 ± 0.6	3.2 ± 0.9	0.3 ± 0.6	11.6 ± 2.3	1.1 ± 0.3	26.2 ± 3.3
coch tl uni-tac	5.4 ± 0.5	1.1 ± 0.6	1.8 ± 0.5	1.9 ± 0.4	0	9.7 ± 0.9	1.1 ± 0.3	21.0 ± 1.2
t_{48}	4.1**	2.0	5.8**	7.2**	1.0	4.4**	NA[c]	8.0**
Mean of *UNI-TAC coch* genotypes	6.0 ± 0.3	1.3 ± 0.1	2.5 ± 0	2.8 ± 0.6	0.3 ± 0.1	11.3 ± 0.5	1.2 ± 0.1	25.2 ± 1.5
uni-tac coch genotypes	5.4 ± 0	1.1 ± 0	2.0 ± 0.3	2.1 ± 0.3	0.1 ± 0.1	9.6 ± 0.2	1.1 ± 0.1	21.3 ± 0.4
t_{98}	4.8**	2.0*	4.8**	3.4**	0.6	5.7**	1.8	7.2**

[a]Twenty five flowers taken from five plants on three occasions were examined for each genotype. The derivation of genotypes and phenotypes of leaf-blade and stipule-blade of all the genotypes has been reported earlier (Kumar *et al.* 2009). The crop growth conditions were same as for the genotypes included in the table 1.
[b]Other corolla organs were petal-like attached to calyx or stamens, which could not be classified into the standard, wing or keel classes of corolla organs. [c]NA, not applicable. *, **, Significant at 5% and 1% probability level, respectively.

on the flower morphology are shown in table 1. It will be seen that on an average basis, the total number of organs in the flowers of *uni-tac* genotypes was about 8% lower than that in the *UNI* (or *UNI-TAC*) genotypes. In the *uni-tac* flowers, there was occasional deficiency of one or more stamen(s) and absence of a petal, the standard. Usually, the adaxial stamen was found missing. The other andorecium related abnormalities included fusion of two or more stamens and presence of petalous stamen(s) (figures 1, b–d, g & h). Besides the absence of standard (figure 1b), partial abortion of standard (figure 1, d–f) was also visualized. The known sequence of events during differentiation of normal flowers are (Tucker 1989; Ferrandiz *et al.* 1999): production of an abaxial sepal primordium, two primordia for lateral sepals, an abaxial primordium for the keel petals and stamens, a carpel primordium, two lateral primordia for wings and stamens and lastly, an adaxial primordium for the standard petal and stamens. It seems from the observations, summarized in table 1, that from about 6% of the *uni-tac* mutant flower initials, the adaxial primordium was formed defectively such that the standard was partially or fully aborted and adaxial stamens were absent or malformed. Other primordia were formed normally or were only marginally defective. These observations mean that *UNI* is required in optimal concentrations for flower differentiation to occur normally and deficiency of *UNI* product, as expected in *uni-tac* plants on account of mutation in the *UNI* gene promoter decreasing the expression of *UNI* (or *UNI-TAC*) gene, leads to the formation of flowers with lowered complexity. Thus it emerges that leaf blades as well as flowers of *uni-tac* mutants are less complex than the counterparts borne on *UNI* plants.

Among the *uni-tac* genotypes, the degree of disturbance in the development of corolla standard and androecium whorl were observed to be generally higher in those genotypes whose compound leaves are known to harbour higher degree of dissection than in *uni-tac* genotype. These observations indicate the involvement of *AF*, *TL* and *MFP* genes in the flower differentiation process.

The quantitative effects of *coch* mutation on the expression levels of organs in various flower whorls are shown in table 2. It will be noted that in conformity with the observations of Yaxley *et al.* (2001), the *coch* flowers in general bear more than normal number of organs in all the flower whorls. There did not appear to be any particular intra-whorl or inter-whorl patterns in the formation of supernumerary organs; the extra structures were formed at different organ position in varying numbers in different flowers. For example, flowers contained 5 to 7 sepals, 1 to 4 standard petal(s), 2 or 3 wing petals, 2 to 4 keel petals, 10 to 20 stamens and/or 1 or 2 carpel(s). A variety of mosaic organs were also formed: sepal-cum-petal, petal-cum-stamen(s), affecting filament sizes and subgrouping among stamens. When two carpels were present, they were distinctly developed or identically developed and fused. Apparently, there was incremental effect of *coch* mutation on the meristem sizes of all the different floral primordia. Formation of mosaic organs

Figure 1. Flower morphology in the wildtype and *unifoliata-tendrilled acacia* (*uni-tac*) genotypes of garden pea *Pisum sativum*. (a) Wildtype flower: entire flower and calyx whorl of five sepals, corolla whorl comprising of adaxial standard, lateral wings and abaxial keels, androecium consisting of a cluster of nine stamens whose filaments are fused and a stand alone stamen and gynoecium consisting of a carpel are shown. (b–h) Various kinds of abnormalities that were noted in *tl uni-tac* flowers (b–e) and *af tl uni-tac* flowers (f–h) are shown; the normal whorl(s)/organ(s) are not shown. (b) The standard petal, the stand alone stamen and a stamen from stamenal cluster are absent; (c) two stamens from the cluster are absent; (d) the standard is malformed and stand alone stamen and two from the stamen cluster are absent; (e) the standard is partially aborted; (f) the standard is malformed and a keel–stamen mosaic organ is present; (g) three stamens of the cluster are fused; and (h) all stamens are in a cluster within which four stamen are fused of which one is petalous.

suggested that the *coch* mutation somehow changed the identity of organs specified by each kind of the floral primordia. The comparison of the *coch UNI* and *coch uni* genotypes, with and without *tl* allele, showed that there were about 11.8% more organs in the *coch UNI* flowers than the organs present in *coch uni-tac* flowers. Or, *uni-tac* mutation decreased the complexity of *coch* flowers. This means that *UNI* is under the negative control of *COCH*. Apparently, such a control step ensures optimal expression of *UNI* as required in the normal flower differentiation. Any overexpression of *UNI*, such as, that occurring in *coch* mutants, would increase the meristem sizes of floral organ primordia leading to abnormal flower differentiation resulting in the formation of supernumerary organs or indeterminacy. The indeterminate floral phenotype of null mutants in *UNI* gene or *uni* mutants suggests that there must also exist an extra-*UNI* pathway(s) of floral differentiation which remain repressed in the presence of *UNI* (*UNI-TAC*) in the *UNI COCH*, *UNI coch*, *uni-tac COCH* and *uni-tac coch* genotypes. Because the floral phenotype of *uni* and *lfy* mutants is similar, *LFY* must also function as repressor of the aberrant pathways of flower development in *A. thaliana* (Schultz and Haughn 1991; Huala and Sussex 1992; Weigel *et al.* 1992).

Earlier, *COCH* has been implicated in the suppression of *UNI*-led leaf blade pathway in the stipuleblade domains (Kumar *et al.* 2009). Together with the present results, it emerges that *COCH* downregulates *UNI* in the stipuleblade developmental as well as in the flower differentiation

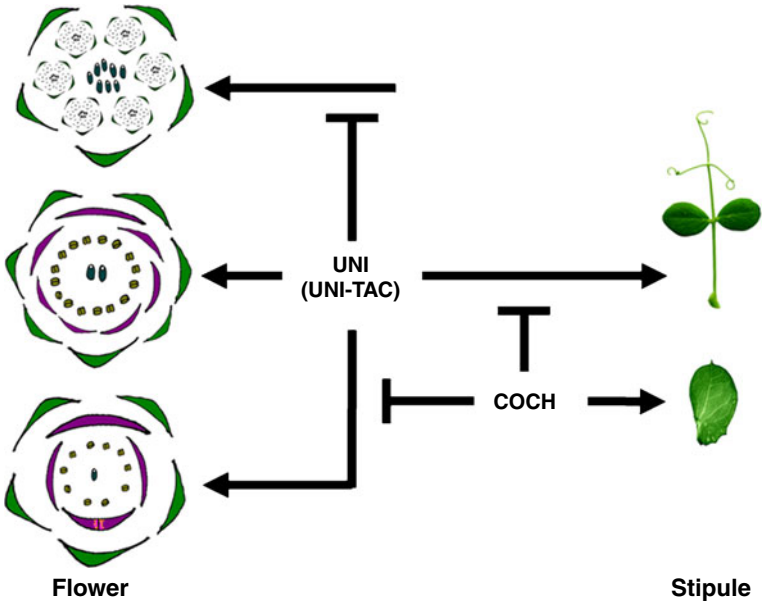

Figure 2. Diagrammatic representation of the functions of *COCHLEATA* (*COCH*) and *UNIFOLIATA* (*UNI* or *UNIFOLIATA-TENDRILLED ACACIA* (*UNI-TAC*)) genes in the flower and stipule development in *Pisum sativum*. In the absence of *UNI*, indeterminate flowers, which combine features of flower and inflorescence and lack corolla and androecium, are formed. *UNI* is required essentially for the formation of flowers which contain calyx (sepals), corolla (petals), androecium (stamens) and gynoecium (carpels). In the absence of *COCH*, indeterminate flowers which have extra sepals, petals, stamens and/or carpels are formed. Downregulation of *UNI* by *COCH* is required for the normal flower differentiation to occur, which allows the organ distribution in flowers that is typical of the species. *COCH* and *UNI* also act in the formation of another lateral organ, the stipule. In the absence of *COCH*, *UNI*-led compound stipules are formed which have the morphology of leaf blade. *COCH* is required for downregulating *UNI* in stipules, thereby formation of simple stipules is promoted and pathway for compound stipules gets suppressed. Arrows mean activity and horizontal bars reflect downregulation.

processes. Negative control of *UNI* in stipules allows them to grow as simple organs preventing them from becoming compound organs and that in flowers allows normal progression of flower growth and development, negating the formation of any supernumerary organs (figure 2). It is possible that *COCH* is involved in fine-tuning of the expression of *UNI* in all the lateral organs of *P. sativum*.

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References

Champagne C. E. M., Goliber T. E., Wojciechowski M. F., Mei R. W., Townsley B. T., Wang K. *et al.* 2007 Compound leaf development and evolution in the legumes. *Plant Cell* **19**, 3369–3378.

DeMason D. A. 2005 Extending Marx's isogenic lines in search of *Uni* function. *Pisum Genet*. **37**, 10–14.

DeMason D. A. and Schmidt R. J. 2001 Roles of the *uni* gene in shoot and leaf development of pea (*Pisum sativum*): phenotypic characterization and leaf development in the *uni* and *uni-tac* mutants. *Int. J. Plant Sci.* **162**, 1033–1051.

DeMason D. A. and Chawla R. 2004a Roles for auxin during morphogenesis of compound leaves of pea (*Pisum sativum*). *Planta* **218**, 435–448.

DeMason D. A. and Chawla R. 2004b Roles of auxin and *Uni* in leaf morphogenesis of afila genotype of pea (*Pisum sativum*). *Int. J. Plant Sci.* **165**, 707–722.

Ferrandiz C., Navarro C., Gomez M. D., Canas L. A. and Beltran J. P. 1999 Flower development in *Pisum sativum*: from the war of the whorls to the battle of common primordia. *Dev. Genet.* **25**, 280–290.

Gourlay C. W., Hofer J. M. I. and Ellis T. H. N. 2000 Pea compound leaf architecture is regulated by interactions among the genes *UNIFOLIATA*, *COCHLEATA*, *AFILA* and *TENDRIL-LESS*. *Plant Cell* **12**, 1279–1294.

Hofer J., Turner L., Hellens R., Ambrose M., Matthews P., Michael A. and Ellis N. 1997 *UNIFOLIATA* regulates leaf and flower morphogenesis in pea. *Curr. Biol.* **7**, 581–587.

Hofer J., Turner L., Moreau C., Ambrose M., Issac P., Butcher S. *et al.* 2009 *Tendril-less* regulates tendril formation in pea leaves. *Plant Cell* **21**, 420–428.

Huala E. and Sussex I. M. 1992 *LEAFY* interacts with floral homeotic genes to regulate *Arabidopsis* floral development. *Plant Cell* **4**, 901–913.

Kumar S., Mishra R. K., Kumar A., Srivastava S. and Chaudhary S. 2009 Regulation of stipule development by *COCHLEATA* and *STIPULE-REDUCED* genes in pea (*Pisum sativum*). *Planta* **230**, 449–458.

Lee I., Wolfe D. S., Nilsson O. and Weigel D. 1997 A *LEAFY* coregulator encoded by *UNUSUAL FLORAL ORGANS*. *Curr. Biol.* **7**, 95–104.

Liu C., Xi W., Shen L., Tan C. and Yu H. 2009 Regulation of floral patterning by flowering time genes. *Dev. Cell* **16**, 711–722.

Mishra R. K., Chaudhary S., Kumar A. and Kumar S. 2009 Effects of *MULTIFOLIATE-PINNA, AFILA, TENDRIL-LESS* and *UNIFOLIATA* genes on leaf blade architecture in *Pisum sativum*. *Planta* **230**, 177–190.

Monti L. M. and Devreux M. 1969 *Stamina pistilloida*: a new mutation induced in pea. *Theor. Appl. Genet.* **39**, 17–20.

Schultz E. A. and Haughn G. W. 1991 *LEAFY*, a homeotic gene that regulates inflorescence development in *Arabidopsis*. *Plant Cell* **3**, 771–781.

Sharma B. and Kumar S. 1981 Discovery of one more allele of the *tac*-locus of *Pisum sativum*. *Pisum Newslett.* **1**, 21.

Taylor S., Hofer J. and Murfet I. 2001 *Stamina pistilloida*, the pea ortholog of *Fim* and *UFO*, is required for normal development of flowers, inflorescences and leaves. *Plant Cell* **13**, 31–46.

Tucker S. C. 1989 Overlapping organ initiation and common primordia in flowers of *Pisum sativum* (Leguminosae: Papilionoideae). *Am. J. Bot.* **76**, 714–729.

Wagner D. 2009 Flower morphogenesis: timing is key. *Dev. Cell* **16**, 621–622.

Wang Z., Luo Y., Li X., Weng L., Xu S., Yang J. *et al.* 2008 Genetic control of floral zygomorphy in pea (*Pisum sativum* L.) *Proc. Natl. Acad. Sci. USA* **105**, 10414–10419.

Weigel D., Alvarez J., Smyth D., Yanofsky M. F. and Meyerowitz E. M. 1992 *LEAFY* controls floral meristem identity in *Arabidopsis*. *Cell* **69**, 843–859.

Yaxley J. L., Jablonski W. and Reid J. B. 2001 Leaf and flower development in pea (*Pisum sativum* L.): mutants *cochleata* and *unifoliata*. *Ann. Bot.* **88**, 225–234.

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