

Bract reduction in Cruciferae: possible genetic mechanisms and evolution

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Summary: This review is an attempt to analyze possible ways of bractless inflorescence formation in Cruciferae. Function of genes which are supposed to play a certain role in a process of bract reduction/development – *LFY*, *AP1*, *AP2*, *BOP1*, *BOP2*, *JAG*, *FUL/AGL8*, *SOC1/AGL20*, *BRA* – is discussed with concentration on the structure of flowers and inflorescences, based on the results of genetic analysis (including data on gene expression). The potential of these genes in the evolutionary process of bract reduction is hypothesized.

Keywords: inflorescences without bracts, Brassicaceae, Cruciferae, developmental genetics, evolution, inflorescence morphology

Recent progress in plant developmental genetics has led to an improved understanding of the genetic control of development of complex morphological structures. Comparative studies in different plant species gave rise to some valuable suggestions on the evolutionary pathways of the regulation of plant development (e.g. KRAMER & IRISH 2000; BARKOULAS et al. 2008). Extensive genetic and molecular studies on *Arabidopsis thaliana* (L.) Heynh., the model object of plant genetics, made the family to which it belongs – Cruciferae – probably the best experimental system for investigation of molecular basis of morphological evolution (BOWMAN 2006). One of the most important morphological traits of Cruciferae is the formation of indeterminate racemose inflorescences in which flowers are not subtended by bracts (SAUNDERS 1923; Figs. 1a, 2). The process of bract reduction is related to the regulation of cell division (LONG & BARTON 2000); in many species the formation of so called ‘cryptic’ bracts is observed. They are initiated but suppressed at later stages of development and their presence may be derived only from the presence of stipules (ARBER 1931; KUSNETZOVA et al. 1993) or from the specific profile of gene expression (LONG & BARTON 2000; BOSCH et al. 2008). The genetic mechanisms of this suppression are still unclear. It is postulated that the reduction of bracts occurred in a common ancestor of the whole family Cruciferae (SAUNDERS 1923; BAUM & DAY 2004). In the closely related family of Cleomaceae, which has greater variation in floral and inflorescence morphology, both, bracteate and abacteate forms, are present and the loss of bracts is treated as a derived trait occurring independently in several lineages (ILTIS 1957). Thus, the study of the genetic control of bract reduction may help to understand the processes of morphological evolution at family level. In this article the genetic mechanisms of bract reduction in the model plant *Arabidopsis thaliana* and possible roles of genes controlling this process in the evolution of inflorescence will be discussed.

Bract is by definition a leaf developing on the inflorescence and subtending a flower. This term, however, is often interpreted more broadly and applied to any inflorescence-associated leaves or to any leaf with active axillary meristem (IRISH & SUSSEX 1990; DINNENY et al. 2004). Here and

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Table 1: Genes, mutations affecting bract development.

Gene	Phenotype	Function	Product	Reference
<i>TERMINAL FLOWER1</i>	Accelerated transition to flowering, decrease of number of flowers, terminal flower formation. Basal flowers are subtended by bracts.	Maintenance of inflorescence meristem. Negative regulation floral meristem identity genes at the centre of the shoot apex.	Phosphatidylethanolamine binding protein	SHANNON & MEEKS-WAGNER 1991; SCHULTZ & HAUGHN 1993; OHSHIMA et al. 1997
<i>LEAFY</i>	Flowers are transformed into shoot or possess a part of shoot characteristics: spiral phyllotaxis, loss of identity by floral organs, branching and development of bracts. Delayed transition to flowering.	Integration of signals from different pathways of flowering initiation and control of floral meristem identity. Positive regulation of floral organ identity genes.	Transcription factor	SCHULTZ & HAUGHN 1991; WEIGEL et al. 1994
<i>APETALA1</i>	Loss of sepal and petal identity; instead of perianth organs 6–8 vegetative leaves are formed. Secondary floral meristems often develop in the axils of these leaves. Transition to flowering is delayed.	Transition to flowering; sepal and petal identity (A class gene). Positive regulator of <i>LFY</i> , <i>LFY</i> and <i>API</i> constitute a positive feedback system.	MADS-domain containing transcription factor	IRISH & SUSSEX 1990; MANDEL et al. 1992; BOWMAN et al. 1993
<i>APETALA2</i>	In weak alleles (e.g. <i>ap2-1</i>) perianth organs loose their identity and transform into vegetative leaves with active axillary meristems (like in <i>ap1</i>). The number of such leaves is less than in <i>ap1</i> mutants (2–4). In strong alleles the number of floral organs is reduced and all floral organs acquire identity of reproductive organs.	Negative regulation of <i>AGAMOUS</i> (gene which controls stamen and carpel identity and floral meristem termination), sepal and petal identity.	AP2-domain containing transcription factor	KUNST et al. 1989; JOFUKU et al. 1994

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Cont. Table 1:

Gene	Phenotype	Function	Product	Reference
<i>BLADE-ON-PETIOLE 1, 2</i>	Single mutants <i>bop1</i> and <i>bop2</i> display alterations in lateral organ development, first of all increased proliferation of their proximal parts. In double mutants this trait is more intensively expressed and bracts also develop.	Regulation of meristem activity in proximal parts of leaves (suppression of leaf blade development in the region where petiole is formed), negative regulation of <i>JAG</i> .	Proteins containing ankyrin repeats and a BTB/POZ domain	NORBERG et al. 2005; HEPWORTH et al. 2005
<i>AGAMOUS-LIKE8/FRUITFUL, AGAMOUS-LIKE20/SUPPRESSOR OF OVEREXPRESSION OF CONSTANS 1</i>	In single mutants transition to flowering is delayed. Double mutants develop bracts.	Regulation of flowering time; bract suppression.	MADS-domain containing transcription factors	GENNEN et al. 2005
<i>JAGGED</i>	Dominant mutants develop bracts. Recessive mutants display alterations in lateral organ development.	Control of lateral organ morphogenesis.	Zinc finger C ₂ H ₂ -type transcription factor	OHNO et al. 2003; DINNENY et al. 2004
<i>BRACTEA</i>	Mutants develop bracts and terminal flower. Cell differentiation and response to photoperiod is also altered.	Bract reduction, maintenance of proliferative activity of shoot apical meristem.	Product unknown	EZHOVA & PENIN 2001

further the term 'bract' will be used in its narrow sense. Those structures that satisfy the broad definition of bracts, but do not satisfy the narrow, will be referred to as 'pseudo-bracts'.

Arabidopsis mutants developing pseudo-bracts

There are several mutants of *A. thaliana* (single and double) that form bracts (Tab. 1). One of the first described is a mutant *leafy* (*lfy*) (SCHULTZ & HAUGHN 1991; Figs. 1b, 2). *LFY* gene controls transition to flowering and the establishment of flower meristem. In *lfy* mutants the flowers are partially or completely transformed into vegetative shoots. These shoots often develop in the axils of bracts. Fertile flowers that are sometimes formed in *lfy* mutants may also be subtended by bracts. Thus, the inflorescence of *lfy* may be formally regarded as bracteate (WEIGEL et al. 1992). This fact gave rise to the suggestion that one of the functions of *LFY* gene is a suppression of bract formation or rather this function is a new one, arisen in the evolution of Cruciferae (COEN & NUGENT 1994). The latter is derived from the comparison with *Antirrhinum majus* L., the species from another family, Scrophulariaceae, where bracts are present in wild type without disruption of *LFY* function. However, the phenotype of *lfy*, in which the formation of bracts correlates with the acquisition of vegetative traits by the flowers, does not contradict to other interpretation of

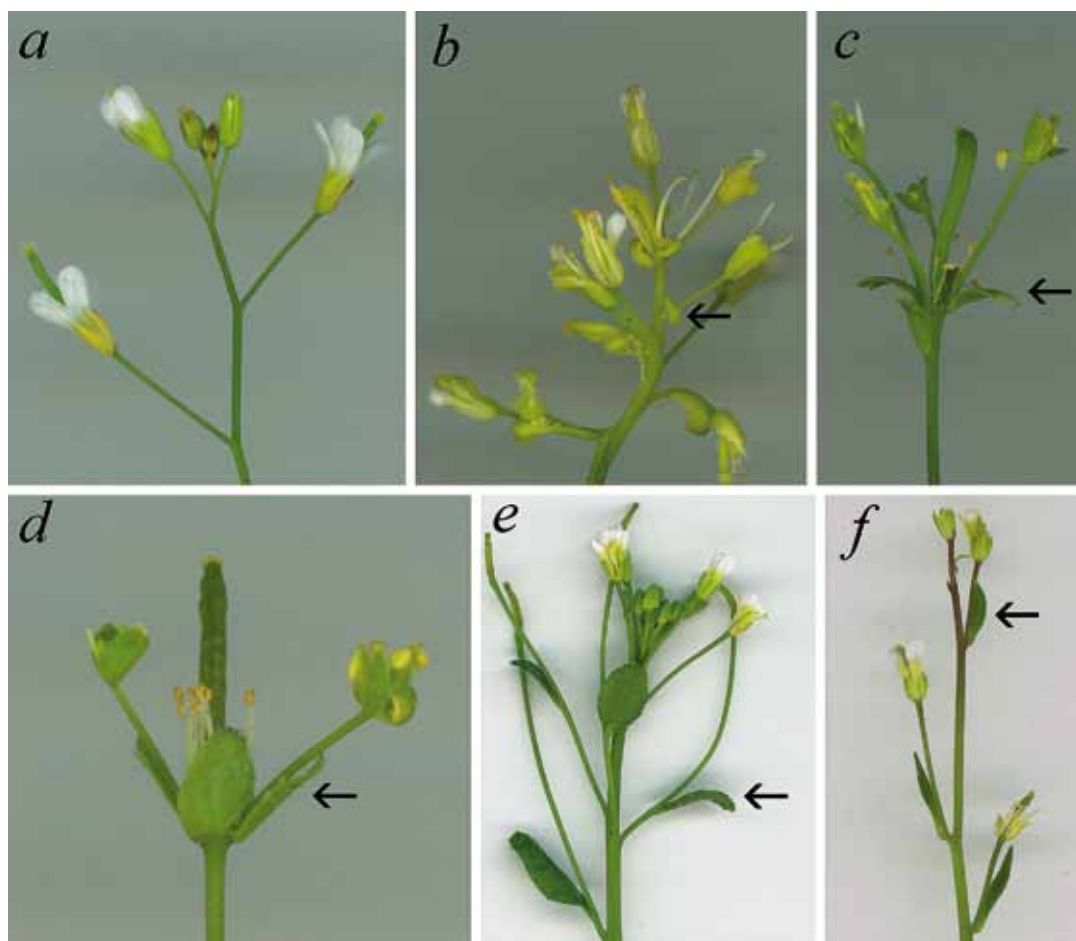


Figure 1: *Arabidopsis thaliana* wild type and mutants developing bracts. *a* – wild type, *b* – *lfy-5*, *c* – *ap1-20*, *d* – *ap2-1*, *e* – *agl8 agl20 (ful soc1)*, *f* – *bra*; *a*, *b*, *e*, *f* – inflorescences; *c*, *d* – flowers. Arrows indicate bracts.

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LFY function loss. It is assumed that bract development is the result of intensification of vegetative shoot traits in *lfy* flowers. Accordingly, bracts are the most significant characteristic for basal and apical 'flowers' that are the mostly shoot-like. Bract development in whorls of sepals and petals in a strong allele *lfy-6* also supports this interpretation. Moreover, there are some species of Cruciferae (e.g. *Sisymbrium supinum* L.) that form bracteate inflorescences without any alteration in flower development (DEVRIES 1904). In this case the re-appearance of bracts occurs without the disruption of *LFY* function that makes a suggestion on the role of *LFY* in bract reduction more questionable. In some species the reduction of bracts is delayed with respect to flower formation – e.g. in *Matthiola incana* (L.) R. Br. (SAUNDERS 1923), *Alyssum tortuosum* Waldst. &

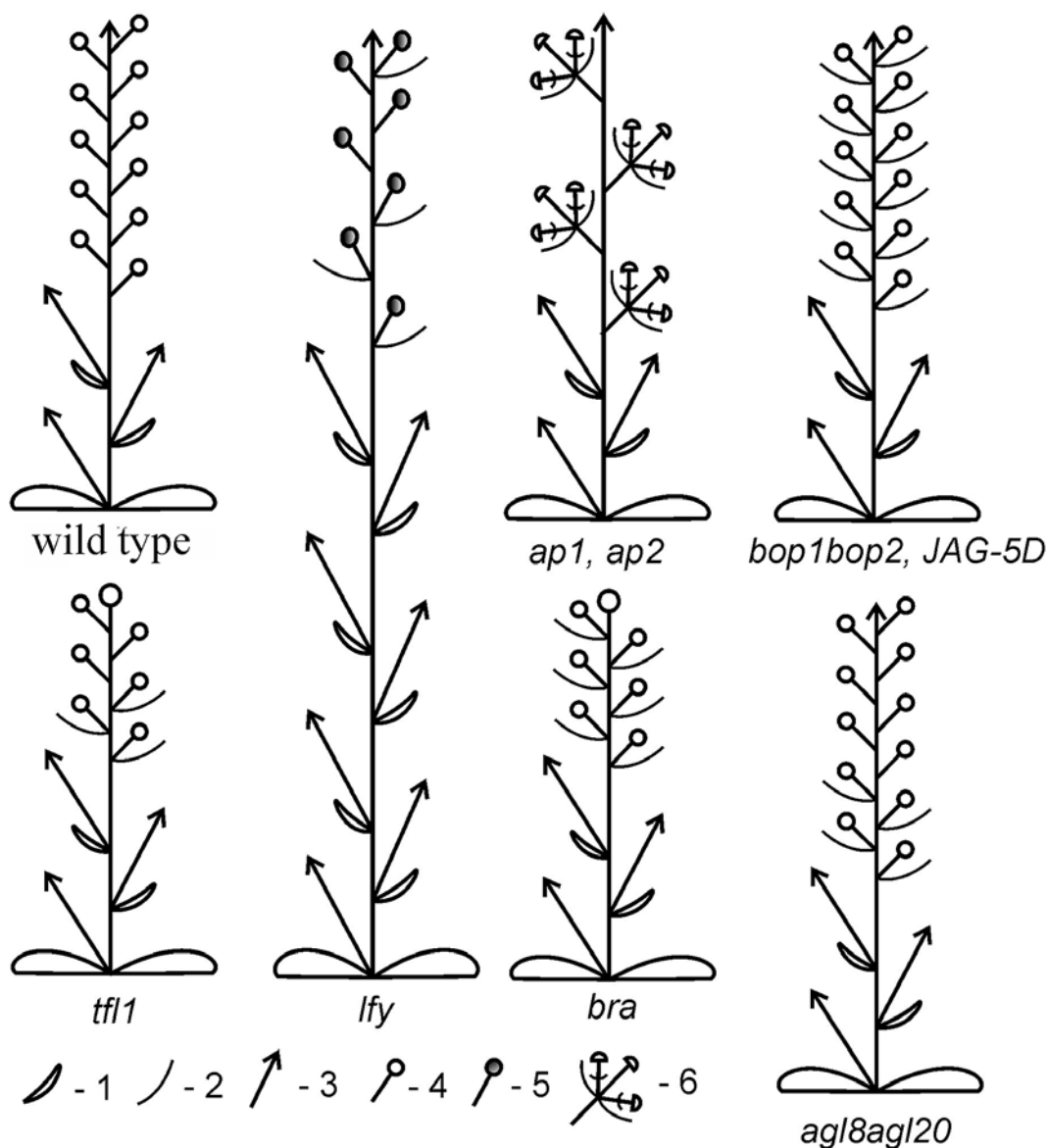


Figure 2: *Arabidopsis thaliana* wild type and mutants developing bracts, schematic representation of plant architecture. 1 – cauline leaf, 2 – bract, 3 – proliferating axis, 4 – flower, 5 – flower of *lfy* mutant, combining flower and shoot characteristics, 6 – flowers of *ap1* and *ap2* mutants, semicircles indicate reproductive organs.

Kit., *Arabidopsis toxophylla* Busch (PENIN et al. 2005) – here the first flowers are subtended by bracts. This suggests that the genetic mechanism of bract reduction is not directly related to those of flower development and that for bract reduction higher level of activity of genes responsible for transition to flowering or longer time of their action is needed. This is also supported by the fact that basal flowers of mutants characterized by the accelerated transition to flowering (e.g. *terminal flower1*) are subtended by bracts (SCHULTZ & HAUGHN 1993; Fig. 2). True bracts subtending basal flowers also develop in wild type *Arabidopsis* plants if they are transferred from a non-inductive (short day) to inductive (long day) photoperiod (HEMPEL et al. 1998).

Another two genes where mutations lead to bract formation have also been known since the end of the '80s. These are *APETALA2* (*AP2*) and *APETALA1* (*AP1*) (KUNST et al. 1989; IRISH & SUSSEX 1990). In *ap1* and *ap2* weak alleles bracts that resemble wild type cauline leaves but subtend secondary flowers are formed on the axes terminated by the reproductive organs (in *ap2* strong alleles only reproductive organs are formed), i.e. on the floral axes on the place of the perianth (Figs. 1c, d, 2). Reproductive organs in these mutants are indistinguishable from those of the wild type flowers. The bracts are not formed on the main inflorescence axis (in contrast to *lfy* mutants). Thus, in *ap1* and *ap2* a new combination of characters not typical for wild type *Arabidopsis* arises in a zone where the perianth is formed in the wild type. One part of these characters (leaf formation) corresponds to the paracladial zone of inflorescence and another (development of flowers) to the main inflorescence zone (Tab. 2). Thus, the formation of bracts in the zone of the perianth in *ap1* and *ap2* is not due to the suppression of bract development by *AP1* and *AP2*, but to the inactive mechanism that is responsible for bract suppression in the inflorescence. It has been postulated (HAUGHN & SOMMERVILLE 1988) that the leaf is a ground state for the fate of developing organ primordia in the zone of the perianth. As *AP2* and *AP1* are genes that confer sepal and petal identity to lateral organs, the development of leaves takes place in the absence of their activity.

Mutants developing true bracts

In recent years several mutants forming 'true bracts' – leaves subtending normal flowers or flowers with slight alterations from the wild type – were identified. These are double mutants for genes *BLADE-ON-PETIOLE 1* (*BOP1*) and *BLADE-ON-PETIOLE 2* (*BOP2*) (NORBERG et al. 2005), *AGAMOUS-LIKE 8* (*AGL8*; also known as *FUL*) and *AGAMOUS-LIKE 20* (*AGL20*, also known as *SOCI1*) (GENNEN et al. 2005, LIU et al. 2007, LIU et al. 2008). Moreover, the bracts are formed in plant carrying dominant mutation *jag-5D* in *JAGGED* (*JAG*) gene (DINNENY et al. 2004) or recessive mutation in *BRACTEA* (*BRA*) gene (EZHOVA & PENIN 2001, PENIN et al. 2007). In these mutants bracts are formed on main inflorescence axis (Figs. 1e, f, 2) as in *lfy* mutants, but the flowers are not converted into vegetative shoots, what is characteristic for *lfy*.

In wild type *Arabidopsis* the genes *BOP1* and *BOP2* are expressed in the proximal part of lateral organs determining their shape. These genes do not allow the leaf blade to expand into the region where the petiole is formed. In double mutant *bop1 bop2* leaves are sessile. In triple mutant *bop1 bop2 lfy* the leaves subtending shoot-like 'flowers' are more expanded (NORBERG et al. 2005). The authors treat this phenotype as an intensification of this character and conclude that *BOP1*, *BOP2* and *LFY* act together in the suppression of bracts. However, taking into consideration that the floral traits of the 'flowers' of triple mutant are very weakly expressed, this phenotype

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Table 2: Comparison of structure of 'perianth' zone in *ap1* and *ap2* mutants with perianth and inflorescence zones of wild type, illustrating hybrid nature of this zone. Characters, specific for each zone, are marked out as follows: paracladial zone – *italic*; main inflorescence – **bold**; perianth – underlined.

Character/Zone	Paracladial zone of inflorescence (corresponds to 'early inflorescence' zone in SCHULTZ & HAUGHN 1993) in wild type plants, <i>ap1</i> and <i>ap2</i> mutants	Main inflorescence (corresponds to 'late inflorescence' zone in SCHULTZ & HAUGHN 1993) in wild type plants, <i>ap1</i> and <i>ap2</i> mutants	Zone of perianth in wild type plants	Zone of perianth in <i>ap1</i> and <i>ap2</i>
Activity of axillary meristem	<i>active</i>	active	<u>not active</u>	active
Type of axillary meristem (vegetative or floral)	<i>vegetative</i>	floral	<u>not active</u>	floral
Leaf reduction	<i>no reduction</i>	reduction	<u>no reduction</u>	<u>no reduction</u>
Type of phyllome	<i>vegetative (cauline leaf)</i>	N/A	<u>perianth organ</u>	<i>vegetative (bracts, similar to cauline leaves)</i>

should probably be treated as increase in size of cauline leaves, characteristic for *bop1 bop2*, as in case of *LFY* activity loss.

Dominant mutation in *JAG* gene – *jag-5D* – as well as the constitutive expression of this gene under the control of Cauliflower Mosaic Virus 35S promoter leads to the formation of bracts, whereas the absence of *JAG* activity suppresses bract development in the zone of perianth formation in *ap1 jag*, *ap2 jag*, *lfy-6 jag* and partially suppresses their development on main inflorescence axis in *lfy-6 jag*. Therefore, it was suggested that *JAG* is necessary for bract development (OHNO et al. 2004; DINNENY et al. 2004). Besides, in *jag* mutant lateral organs are abnormally shaped, in particular, they are smaller and narrower than in wild type (OHNO et al. 2004). *BOP1* and *BOP2* negatively regulate *JAG* activity, confining its spatial expression to distal parts of lateral organs (HEPWORTH et al. 2005). In double mutant *bop1 bop2* expression of *JAG* is increased, while in dominant mutant *bop1-6D* its expression is decreased (NORBERG et al. 2005), that may also evidence the involvement of *JAG* in bract development. However, triple mutants *bop1 bop2 jag* form bracts as well as double mutants *bop1 bop2* – by the absence of *JAG* activity disruption of *BOP1* and *BOP2* activity does not cause bract reduction. As far as in the genome of *A. thaliana* there is a gene similar to *JAG* (it is called *JAGGED-LIKE* – *JGL*, also known as *NUBBIN* – *NUB*) and acting partially redundant with it at least in flower development (DINNENY et al. 2004, 2006). It was suggested that the phenotype of triple mutant *bop1 bop2 jag* is a result of action of *JGL* (NORBERG et al. 2005). This assumption does not explain, however, why *JGL* does not compensate loss of *JAG* function in double mutants *jag ap1*, *jag ap2* and *jag lfy*. In addition, the absence of *JAG* expression in 'bracts' on the main inflorescence axis in *lfy-6* mutants is also unexplained. The

presence of *JAG* expression in the secondary axes, where the inflorescences develop in the zone of perianth, may be explained the same way as in *ap1* and *ap2* single mutants.

Such discrepancies regarding *JAG* function are probably caused by the fact that while discussing it, the authors deal with the genetic control of bract development and with the genes required for this process. The function of *JAG* is postulated on the base of the fact of bract absence in double mutants *jag ap1* and *jag ap2*, though, as it was noted above, the bracts of *ap1* and *ap2* are pseudo-bracts, not homologous to those developing on the main inflorescence axis. It should be also noted that during the evolution of the family Cruciferae the bracts have been lost and their accidental re-appearance in some species should be treated as a reversion to an ancestral character state, not as a development of a new character (DEVRIES 1904). Moreover, in an early stage of *Arabidopsis* ontogenesis lateral organs (leaves) develop, forming the rosette and cauline leaves, and only after transition to flowering, they reduce. Thus, we can conclude that the developmental program for bracts is by default 'switched on' and only after transition to flowering it is 'switched off'. Considering the role of *JAG* from such viewpoint, one may suggest alternative scheme of its action in bract reduction. On the base of phenotype of triple mutants *bop1 bop2 jag*, that form bracts in the absence of *JAG* activity, I suggest that this gene specifies expression of *BOP1* and *BOP2* controlling bract reduction or regulating them in some other way. In triple mutant, if *BOP1* and *BOP2* are inactive, the absence of their regulator activity does not result in any additional effects. In this case there is no need to introduce in the scheme of genetic regulation of bract development an additional factor – *JGL* gene. If the hypothesis is true, the phenotype of quadruple mutant *bop1 bop2 jag jgl* will be similar to those of *bop1 bop2* and *bop1 bop2 jag* – i.e. it will develop bracts. Construction of this mutant will allow testing it. In case of ectopic expression of *JAG* in plant carrying dominant mutation *jag-5D* the activity of *BOP1* and *BOP2* is blocked and leads to the development of bracts on main inflorescence axis. The development of bracts in the zone of perianth formation in *ap1*, *ap2-1* and *lfy-6* is caused by this zone not being completely transformed into inflorescence, but partially retaining the profile of gene expression characteristic for the perianth (except for the genes controlling organ identity). In this case the activity of *JAG* in such modified 'perianth' prevents suppression of lateral organ development in that zone. The reduction of bracts in *ap1 jag* and *ap2 jag* is caused by the absence of *JAG* activity, *BOP1* and *BOP2* activity, and suppressing the development of lateral organs. Partial reduction of bracts on main inflorescence axis in double mutant *jag lfy-6* (compared with *lfy-6* single mutant) seems to be related to the general defects of lateral organ development in *jag* (the vegetative leaves and floral organs in *jag* mutant are also abnormally shaped (OHNO et al. 2004)), but not to any bract-specific action of this gene. Thus, *JAG* does not act directly in bract development, but regulates *BOP1* and *BOP2* which are suppressors of bract development. The change in expression of these genes may lead to the reduction of bracts in Cruciferae ancestors.

Less is known about the molecular mechanisms leading to bract development in other mutants. In recessive mutant *bra* many alterations of shoot and leaf structure have been observed. These alterations include not only bract development but also formation of terminal flower, disruption of trichome development and smaller size of mutant plants (EZHOVA & PENIN 2001; PENIN et al. 2007). The two latter effects are most probably caused by the disruption of the process of cell differentiation. Bract development in this mutant is not related to its action on the expression of genes that control bract suppression (Penin, Budaev, unpubl. data), i.e. *BRA* does

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not regulate these genes. It may, however, be regulated by these genes and act in a process of bract reduction, for example, by the regulation of cell division. Alternatively, *BRA* may be involved in an independent pathway of bract reduction. For two other genes, *AGL8* and *AGL20* (double mutant *agl8 agl20* basal flowers are subtended by bracts) the interaction with genes controlling bract reduction is not known too. Both of these genes are involved in transition to flowering (GENNEN et al. 2005; LIU et al. 2007; LIU et al. 2008) as well as *TFL1* mutants develops bracts, and it is possible that their disruption results in the increase of the delay between bract reduction and the acquisition of floral identity by lateral meristems but does not involve the mechanism of bract suppression itself.

Conclusion

The bracts on the main inflorescence axis in *Arabidopsis thaliana* are formed as a result of three types of alterations: I) involving genes *BOP1*, *BOP2* and *JAG* – II) involving *AGL8* and *AGL20* – III) involving *BRA*. The interrelation between these genes is not yet revealed; they may represent independent pathways of suppression of bract development. The existence of several genetic pathways, i.e. changes leading to bract reduction, may account for the independent loss of bracts in different lineages of Cleomaceae, as postulated by ILTIS (1957). Such convergent evolution of inflorescences is of great interest for further studies. It evidences that the formation of a new character may be mediated by a large number of genes, including those not interacting directly with each other. Analogous situation is characteristic for genes controlling perianth development in different groups of angiosperms – the formation of morphologically similar structures is mediated by the action of different genes (RONSE DE CRAENE 2007). Further study of the genetic control of bract reduction may help to elucidate the mechanisms of morphological evolution in angiosperms.

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