Development Supplement 1, 1991, 169–175
Printed in Great Britain © The Company of Biologists Limited 1991

Properties of *deficiens*, a homeotic gene involved in the control of flower morphogenesis in *Antirrhinum majus*

HANS SOMMER, WOLFGANG NACKEN, PIO BELTRAN[1], PETER HUIJSER, HEIKE PAPE, ROLF HANSEN, PETER FLOR, HEINZ SAEDLER and ZSUZSANNA SCHWARZ-SOMMER

Max-Planck Institut für Züchtungsforschung, 5000 Köln 30, Germany and [1]Instituto de Agroquimica y Technologia de Alimentos, CSIC, 46010 Valencia, Spain

Summary

***deficiens*, together with other homeotic genes, is involved in the genetic control of floral morphogenesis in *A. majus*. Mutations in this gene cause homeotic transformations of petals to sepals and stamens to carpels, thus producing male sterile flowers.**

The deduced DEF A protein shows homology to the DNA-binding domain of the transcription factors SRF of mammals (activating *c-fos*) and MCM1 of yeast (regulating mating type), suggesting that DEF A has a possible regulatory function as a transcription factor. Interestingly, DEF A belongs to a family of putative transcription factors, called the MADS box genes, whose DNA-binding domains show conserved homology. Two members of this family correlate with known morphogenetic mutants of *Antirrhinum*.

DEF A is constantly expressed during flower development in all floral organs; it is strongly expressed in petals and stamens, but only at a low basal level in the other organs. Molecular, genetic and morphological observations with *deficiens* morphoalleles indicate that transcriptional and post-transcriptional control of *deficiens* specifies and diversifies its function in space and time.

Key words: *deficiens*, *Antirrhinum majus*, homeotic gene, floral morphology.

Introduction

In higher plants, flower development follows a heritable pattern. Although the results of differentiation may appear to be different comparing, for example, the flower of a snapdragon with that of a rose or a tulip, certain universal rules are observed during the developmental process. For example, organogenesis is sequential and follows a precise spatial and temporal pattern in the initiation of organ primordia. Organs with identical functions are located in specific whorls. The sepal primordia in the first whorl are initiated first, followed by the primordia of petals, stamens and carpels in their respective whorls. The temporal and spatial pattern of organogenesis and the number of individual organs within a whorl is species-specific, and these differences contribute to differences in the phenotypic appearance of the mature flower. The spatial and temporal developmental pattern (that is the plane and number of cell divisions, and the patterns of cell elongation) of different organs differ from each other. This results in morphologically and functionally distinct structures. Thus stamen primordia, for example, differentiate into filaments carrying anthers in which pollen is produced, whereas carpel primordia differentiate into female organs (consisting of ovary, style and stigma) that are destined to produce ovules.

Morphogenesis in plants occurs during the whole, occasionally decades-long, life time. Thus it seems that organogenesis cannot be related to maternally determined positional information and, since plant cells do not change their position with respect to each other, cell migration may also not be involved in the developmental processes (Walbot, 1985). Therefore, questions concerning the constancy of sequential developmental events are particularly intriguing (Heslop-Harrison, 1963; Holder, 1979; Green, 1989). How is positional information established and what is the mechanism by which cells in adjacent whorls interpret their position and differentiate precisely into the organ they are supposed to? And since the overall organization of flowers is conserved evolutionarily in many species, are the genes controlling and governing it also conserved?

The reproducible sequence of events during flower organogenesis seems to be based primarily on temporal and spatial activity of genes. Altered or abolished gene action due to mutation in genes controlling morphogenesis results in disruption of the sequence of events and leads to abnormal development, often revealed as a homeotic alteration (Sattler, 1988; Meyerowitz *et al.*

1989). In *Antirrhinum*, many genes are known that interfere with cell differentiation at different stages of development (Stubbe, 1966; Carpenter *et al.* 1990). In some mutants, the initiation and development of flowers is arrested at an early stage; in others the symmetry, number or the form of floral organs is affected. In this article, we shall focus on a homeotic gene, *deficiens*, which interferes with the determination of floral organ identity.

Homeotic genes in the control of flower organ identity

In *Antirrhinum majus* all homeotic mutants that display incorrect organs can be assigned to three different types: type 1, in which the first and second whorls (the perianth of the flower) are affected; type 2, in which the third and fourth whorls (the reproductive organs) are altered with concomitant increase in the number of organs; and finally type 3, in which the second and third whorl is transformed (Schwarz-Sommer *et al.* 1990). These three types of mutants are also found in *Arabidopsis* (Haughn and Sommerville, 1988; Bowman *et al.* 1989) and some of them in other species as well (Meyer, 1966).

The most striking common feature of the three types of homeotic mutants is that they all affect organs of two adjacent whorls. To alter the fate of organs in two adjacent whorls, expression of homeotic genes would have to be established in the meristematic progenitor cells of the future organs, prior to the development of the organ primordia. Thus homeotic genes could be involved in both the establishment of positional information and in its interpretation. But in homeotic mutants, organ primordia are formed at the correct position as compared to wild type (Hill and Lord, 1989; Sommer *et al.* 1990), although these primordia differentiate into incorrect organs. Development of these transformed organs follows the time-course of the wild-type organs in the affected whorl. Further, the kinds of concomitant homeotic transformations of organs is limited. For example, sepal-like development of petals (sepalody) is always acompanied by feminization of stamens (carpellody), although stamens have the potential to undergo petal-like development (petalody) as well. This may indicate that positional information, at least in part, is established before homeotic genes act and that the expression and function of homeotic genes is under transcriptional control. In addition, several non-allelic mutants display similar phenotypes. For example, five homeotic genes *deficiens*, *globosa*, *viridiflora*, *femina* (Stubbe, 1966) and *sepaloidea* (Carpenter and Coen, 1990) belong to type 3 genes. This indicates that these genes may control each other or that they interact with each other in the control of other (target) genes.

Morphological, genetic and molecular analysis of the *deficiens* gene in *Antirrhinum majus* was conducted to elucidate how the expression of this gene is established and how its function is regulated.

Morphological observations on three morphoalleles of *deficiens*

Mutants of the *deficiens* gene were isolated and genetically and morphologically characterized at the beginning of this century (Klemm, 1927; Stubbe, 1966). *deficiens* is a homeotic gene, mutants of which display sepaloid petals and carpelloid stamens. Three classical mutant alleles are known which differ morphologically in the extent of organ transformation (Fig. 1). These so-called morphoalleles allow morphological features to be correlated with molecular functions (see later).

deficiens^{globifera} reveals the most extremely transformed phenotype. The petals in the second whorl are almost indistinguishable from sepals. The stamens in the third whorl are feminized, thus they develop as carpels and bear fertile ovules in the mature flower. The genuine female organ either does not develop at all or its fuses with the third whorl organs. Observation of young *globifera* flower buds with the scanning electron microscope indicates that the initiation of organ primordia is not affected by the mutation (Sommer *et al.* 1990).

The flowers of morphoalleles, like *nicotianoides* and *chlorantha*, display chimeric features. Their second whorl organs are sepaloid and they are reduced in size and greenish in colour as compared to the wild-type petals. Feminization of stamens is revealed by various types of chimeric structures. For example, stamens of *chlorantha* strongly resemble wild-type organs, but bear ovules, whereas stamens of *nicotianoides* display carpellody, as indicated by many morphological characters (broadened and shortened filaments, stigmatic tissues, hairs), but on some third whorl organs no ovules are formed. Generally the tendency for transformation is higher for organs that develop at the upper (adaxial) part of the flower than in the lower (abaxial) part. Interestingly, the morphological characteristics of the morphoalleles can be influenced by changing environmental conditions and also by introducing the alleles into different genetic backgrounds.

The function of *deficiens* in petals and stamens can be dissected. In a newly isolated *deficiens* morphoallele (*deficiens*-23) the petals display near wild-type form, whereas the stamens are transformed to carpels and form a structure similar to those in *globifera* flowers (Fig. 2).

These observations with different *deficiens* morphoalleles indicate diversification and specification of *deficiens* function in different organs and also in different parts of an organ.

Instability of *deficiens*^{globifera}

Transposable elements are genetic entities that can change their location within the genome. They can be inserted into a gene, and they can also excise and subsequently insert again somewhere else. The insertion of a transposon generates a mutation in the affected gene. Subsequent excision may restore gene

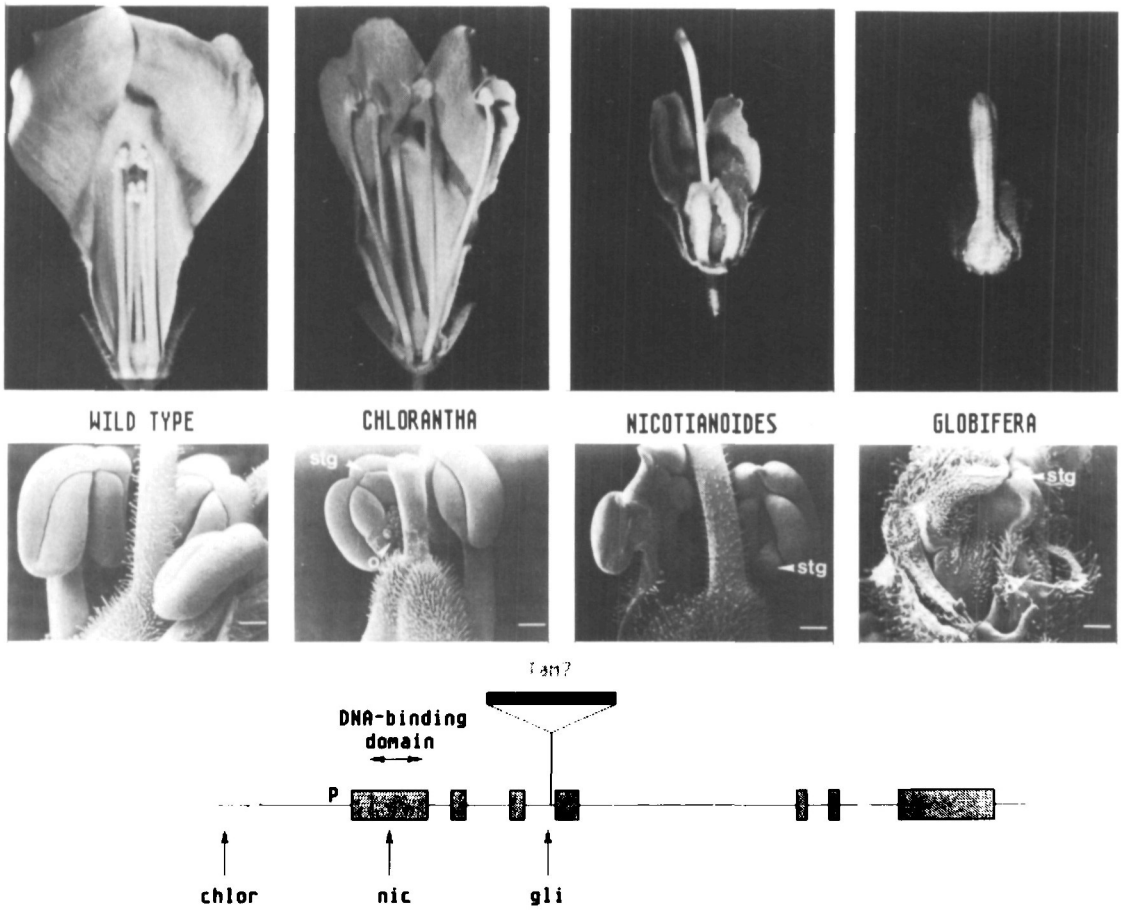

Fig. 1. Flower phenotypes of mutant alleles of the *deficiens* locus of *Antirrhinum majus*. The genotype of each flower is shown below the photographs. To uncover the structure of the inner whorls some sepals and the lower lobe of the corolla were removed. The scanning electron micrographs in the middle show the structure of inner whorl organs (stamens and gynoecium) of young flower buds of wild-type and mutant plants. stg=stigmatic tissue, o=ovules. Bars, 0.5 mm. The structure of the *deficiens* transcription unit is shown at the bottom. Shaded boxes represent exons and P indicates the promoter of the gene. The first exon contains a conserved DNA-binding domain (shown in detail in Fig. 4). Arrows point to the site of mutation determined in the *deficiens* alleles. Tam7 is a transposable element whose excision is responsible for the genetic instability of the *globifera* allele.

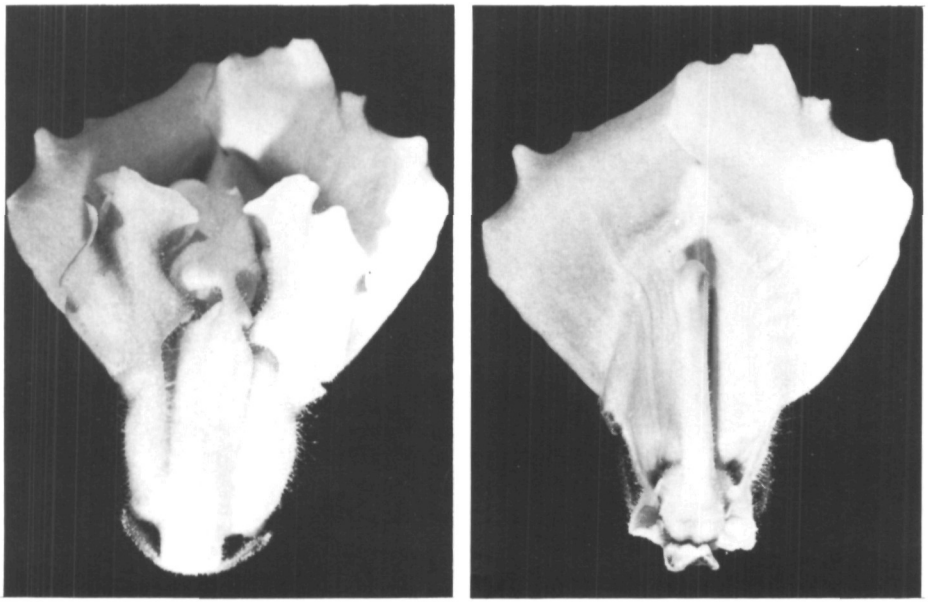

Fig. 2. Phenotype of the *deficiens*-23 allele. The photograph on the left shows the lower petals which were removed in the photograph on the right to reveal the structure of the reproductive organ.

activity (reversion). Genes whose expression is altered by the insertion of a transposon are thus genetically unstable. Transposons can be utilized to isolate genes by transposon tagging, and also to generate new mutants and mutant alleles (Carpenter *et al.* 1990). Unstable alleles and their germinal revertants are particularly useful for the cloning and identification of genes.

Excision may occur at virtually any time during development and is revealed by somatic sectors of revertant tissue in the mutant. The mosaic character of mutant and revertant sectors allows some insight into the mode of action of the unstable gene. The analysis of somatic excision events in the unstable *deficiens*^{globifera} mutant led to the following results.

(1) Excision events that occur very late in development of the sepaloid petals restore petaloid features in a clonal manner (Carpenter *et al.* 1990; Sommer *et al.* 1990) indicating that *deficiens* acts cell-autonomously.

(2) Due to early, but not heritable, somatic excision, single second whorl organs may display a sepaloid and a petaloid sector separated by a sharp boundary which extends from the base of the chimeric organ to its tip. This may indicate that cell groups within a primordium are autonomously differentiating into a given part of the organ.

(3) Early excisions may restore the whorl of petals without simultaneously affecting the developmental fate of the feminized third whorl. However, stamens or stamenoid characters in the third whorl are never restored without concomitant restoration of petals on the second whorl. This indicates that initiation of stamen development requires early *deficiens* gene function.

In conclusion, early and late *deficiens* gene functions are dissectable and different in the second and third whorls. During early petal development *deficiens* perhaps controls cell division, and thus contributes to the form of mature petals. Lack of late functions is reparable, because petaloid characters in the second whorl reappear when *deficiens* expression is restored. In contrast, stamen differentiation in the third whorl depends on early *deficiens* function that cannot be compensated for later in development.

Occasionally somatic excision events that are not heritable in the germinal progeny of the plant can be stabilized and become somatically heritable. These plants are periclinal chimera because they carry mutant and revertant cell sectors. The vegetatively propagated progeny of such plants display flowers (Fig. 3) with uniform and altered morphology. The green rims of petals and their unique form suggest that only part of the petal primordium consists of revertant cells. The stamens of such stable somatic revertants are carpelloid and reveal variably shortened and broadened filaments which carry ovules. Occasionally stamens with wild-type morphology appear in some flowers whose petals still show the altered features described above. Thus the boundary between revertant and mutant tissue between the first and the second whorl seems to be well defined, whereas the boundary between the second and third whorl is less precisely defined. These observations could indicate that there is a difference in precision with which meristematic cell layers contribute to the development of petal and stamen primordia.

Homeotic genes encode transcription factors: the MADS-box

Recently two of the homeotic genes involved in the control of floral organogenesis have been cloned. Remarkably, DEF A, the protein encoded by *deficiens* in *Antirrhinum* (Sommer *et al.* 1990) and also AG, the protein product of the *agamous* gene in *Arabidopsis* (Yanofsky *et al.* 1990) reveal a high degree of homology to the conserved DNA-binding and dimerization domain of two known transcription factors (Fig. 4),

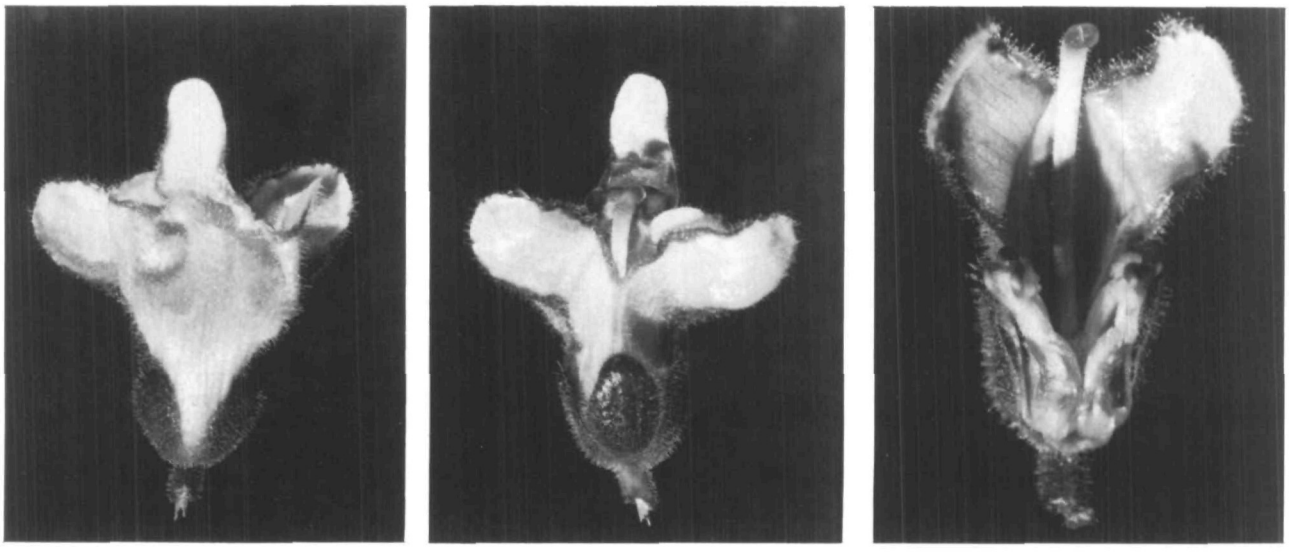

Fig. 3. Somatically stable revertant of *deficiens*^{globifera}. The lower and the upper petals of one of the uniform flowers of a plant are depicted on the left and in the middle, respectively. For the picture on the right the lower petals were removed.

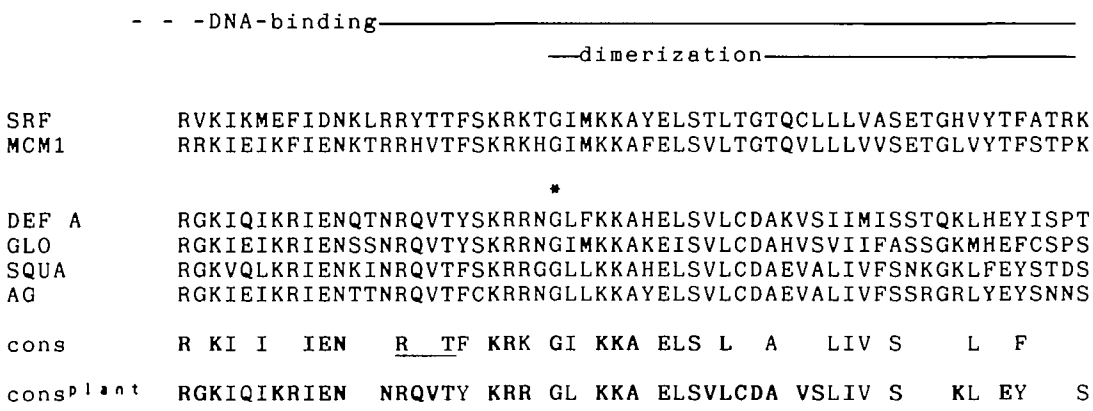

Fig. 4. Conservation of amino acids in the putative DNA-binding and dimerization domains (MADS-box) of proteins involved in the control of differentiation in mammals (SRF), yeast (MCM1) and plants (DEF A, GLO, SQUA in *Antirrhinum* and AG in *Arabidopsis*). Conserved positions are typed in bold face letters and homologous exchanges by light letters. Two consensus sequences comparing the six proteins (cons) and the four plant proteins only (cons^{plant}) are shown at the bottom. The asterisk indicates a point mutation (G–D) detected in the *deficiens*^{nicotianoides} allele. The recognition site for calmodulin-dependent protein kinases is underlined (Cohen, 1988).

SRF (Normal *et al.* 1988) and MCM1 (Passmore *et al.* 1989). The serum response factor (SRF) is essential for the serum-inducible transcriptional control of the *c-fos* proto-oncogene in mammals. In yeast the MCM1 protein is essential for the control of a- and alpha-cell-type-specific genes (Herskowitz, 1989). Thus it seems that these four proteins, in different organisms, participate in the determination of the fate of cells during differentiation. By analogy to the homeobox domain, common to several proteins controlling development processes in animals, the conserved domain of MCM1, AG, DEF A and SRF has been designated 'MADS-box' (Schwarz-Sommer *et al.* 1990).

Whether the DEF A protein can bind to DNA is not known. But structural analysis of the *deficiens*^{nicotianoides} allele, in which a single amino acid exchange causes the mutant phenotype (Fig. 1), has provided suggestive evidence that this domain is essential for the wild-type DEF A function. The observation that DEF A is a nuclear protein also points to its function as a transcription factor.

Homology between floral homeotic genes

Ten additional MADS-box genes were detected in *Antirrhinum*, when the conserved domain of DEF A was used as a molecular probe to screen a cDNA library. All are expressed in floral organs and some of them are expressed in vegetative tissues as well. Two of the cDNAs could be assigned to known floral homeotic genes, *squamosa* and *globosa* (SQUA and GLO in Fig. 4). While *squamosa* is involved in the establishment of flower primordia after evocation, (that is, it controls an early event in flower morphogenesis) the phenotype of *globosa* mutants suggests that it participates in the control of petal and stamen development, like the *deficiens* gene.

The analysis of the MADS-box homologues is not complete yet, but one can assume that more of them will turn out to represent floral or vegetative morphogenic genes. Families of MADS-box genes have been detected in *Arabidopsis*, yeast, mammals, frogs and flies as well. These findings may be significant for the question of evolution of regulatory mechanisms for differentiation processes in diverse organisms.

Homeotic genes are strongly expressed in two adjacent whorls: transcriptional control

Temporal and spatial expression patterns

deficiens is a flower-specific gene, because it is expressed only in the flower and is repressed, or not induced, in vegetative tissues (Sommer *et al.* 1990). *In situ* hybridization experiments have revealed that *deficiens* is expressed most strongly in petals and stamens, in the organs that are homeotically transformed in *deficiens* mutants (Fig. 5). Similarly, mutation in the *agamous* gene in *Arabidopsis* conditions petaloid stamens and sepaloid carpels and, accordingly, the wild-type gene is expressed at highly elevated levels in stamens and carpels (Yanofsky *et al.* 1990). But the expression of homeotic genes is not strictly tissue-specific. Northern blot analysis of dissected *Antirrhinum* organs, for example, revealed that *deficiens* is expressed weakly also in sepals and carpels.

deficiens gene expression is induced early in flower primordia and is virtually constant during flower development (Sommer *et al.* 1990). This indicates that DEF A function is not only required as an early developmental switch, but that it is also essential late in differentiation. The function of the DEF A protein in the control of late developmental events is not clear. As already mentioned, sepaloid development of organs in the second whorl of the *globifera* mutant can be changed to petal-like development by late somatic reversion of the *deficiens* gene. On the other hand, the morphological variability of third whorl organs in the

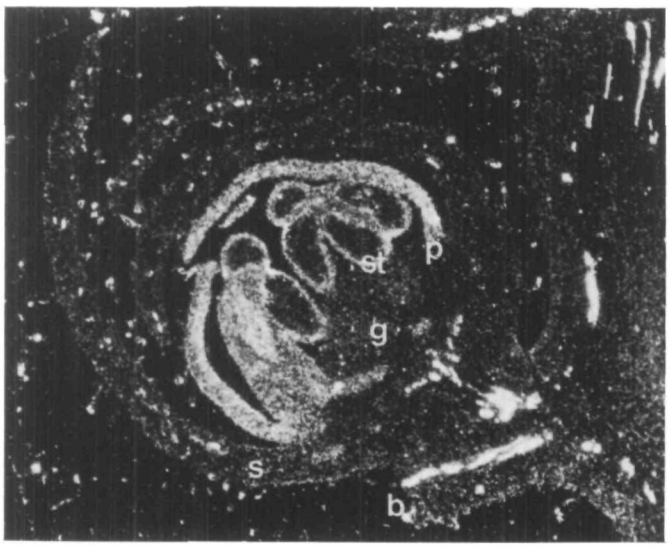

Fig. 5. *In situ* hybridization pattern of *deficiens* gene expression. A longitudinal section through a young bud was hybridized with a molecular probe of the *deficiens* gene. b=bract, s=sepal, p=petal, st=stamen, g=gynoecium.

deficiens morphoalleles points to a requirement of *deficiens* function late in stamen differentiation.

Genetic control of deficiens *gene expression*

Structural analysis of the *chlorantha* allele of *deficiens* (Fig. 1) indicated that *deficiens* is under genetic control. Due to a small rearrangement in the promoter region, expression of *deficiens* in *chlorantha* is reduced to 10–15% of that of wild type. This reduction of expression of the gene occurs in petals and stamens only. The deletion thus seems to affect the *cis*-acting binding site of a transcription factor that upregulates *deficiens* expression specifically in the two organs.

SRE (serum response element), the DNA-binding site of SRF, is functionally and structurally related to the binding sites of the MCM1 protein (Passmore *et al.* 1989). Interestingly, SRE-homologous sequences have been found upstream of the promoters of *deficiens* and *agamous*. This could indicate that some of the other MADS-box proteins, for example those encoded by the *squamosa* or *globosa* genes, could control the spatial and temporal expression of *deficiens*.

Post-translational processes specify homeotic gene functions

The simultaneous expression of homeotic genes in two different organs obviously contradicts their distinct functions in each organ. Consequently, one has to postulate that organ specificity of homeotic genes is the result of post-transcriptional and post-translational processes that determine their function in different organs.

Floral homeotic genes known so far are transcription factors and hence exert their function in the nucleus. Little is known about mechanisms that facilitate the transport to the nucleus of regulatory proteins subsequent to their synthesis in the cytoplasm. But any alteration that affects this transport process would influence the regulatory function of such proteins.

Post-translational events, such as cooperative interactions with other proteins or chemical modification, may also modulate the temporal and spatial pattern of homeotic gene function.

Interactions with other proteins

One striking feature of the DEF A protein is its small size and the lack of structural properties (for example an acidic domain) necessary for activation of transcription (Sommer *et al.* 1990). It is conceivable that this function is provided by other proteins which interact with DEF A and whose expression may be subject to temporal and spatial control. Cooperative types of interactions, for example, could modify the DNA-binding specificity of DEF A and modulate the stability of the complex. Experimental evidence for the existence and the nature of such accessory proteins is not available yet, but several morphological and genetic data, discussed in this report, hint at the existence of such modifying factors which may specify the pattern of *deficiens* gene functions.

Phosphorylation

DEF A is a phosphorylated nuclear protein (data not shown), but the functional significance of its phosphorylation for DNA-binding is not known. Preliminary results have shown that the protein occurs in different states of phosphorylation in wild-type flowers. The relative abundance of the different forms is altered in the *deficiens* morphoalleles *chlorantha* and *nicotianoides*. The results are very interesting for the following reasons. Firstly, in the DNA-binding domain of DEF A the recognition sequence of calmodulin-dependent protein kinases is found (Fig. 4), indicating that DEF A activity may be controlled by phosphorylation. Such calmodulin-dependent protein kinases could mediate environmental influences on development, *via* changes in Ca^{2+} concentration as a result of environmental and hormonal changes. Secondly, the alteration of the DEF A phosphorylation pattern in *deficiens* morphoalleles may indicate that DEF A is involved in the transcriptional control of a protein kinase. This protein kinase may phosphorylate not only DEF A, but other proteins as well.

Interestingly, the functions of SRF and MCM1 are also specified by interactions with other proteins and by phosphorylation. Hence one may speculate that the conserved structural homology of members is not the only common feature of MADS-box proteins. Possibly also the mechanisms which regulate and establish their cell-specific functions are similar.

Conclusions and perspectives

Some floral homeotic genes belong to a family of genes

encoding transcription factors, with a conserved domain called the MADS-box. MADS-box proteins in plants, mammals and yeast participate in the control of developmental processes and they reveal, besides conserved structural features, several other common properties. These are their constant expression in cells whose developmental fate they control and consequently, the requirement of post-translational modification (such as chemical modifications and interactions with accessory proteins) to specify their spatial and temporal function. Whether these similarities reflect evolutionary conservation of the control of differentiation remains an open question.

The regulatory function of the homeotic *deficiens* gene in floral organogenesis is diversified and specified by transcriptional and post-translational control. In this respect, the molecular basis of control in plant development may resemble that of animals. Genetic control of a homeotic gene, however, suggests that its analysis will not shed light on the mechanisms that generate the positional information that is subsequently interpreted by the homeotic genes.

One can expect rapid progress in the study of homeotic genes, their interactions with each other and the regulation of their expression. This will help to elucidate basic mechanisms controlling flower development, but many questions will still remain open.

Nothing is known, for example, about genes (so-called targets) whose expression and function is regulated by homeotic genes. The same is true for genes that control expression of homeotic genes. To achieve progress in this field, molecular studies must be complemented by genetic analyses. Such genes, for instance, could be identified, and perhaps isolated, when a search for modifiers and suppressors of homeotic mutations is carried out by genetic methods.

Similarities in the process of flower development among different plant species has often been emphasized. The homology between homeotic genes controlling organogenesis in *Antirrhinum* and *Arabidopsis* may support the current view that studying one plant species will allow the development of others to be understood as well. This may be true for the very basic molecular mechanisms that govern, by transcriptional control, developmental processes. But distinctive differences may also exist, such as the number and behaviour of genes involved in a particular process, or the absence of certain types of homeotic transformation in some plant species. Therefore, it is possible that the complexity of processes directing flower development is different in different plant species.

References

Bowman, J. L., Smyth, D. R. and Meyerowitz, E. M. (1989). Genes directing flower development in *Arabidopsis*. *The Plant Cell* **1**, 37–52.

Carpenter, R. and Coen, E. S. (1990). Floral homeotic mutations produced by transposon-mutagenesis in *Antirrhinum majus*. *Genes Dev.* **4**, 1483–1493.

Cohen, P. (1988). Protein phosphorylation and hormone action. *Proc. R. Soc. Lond B.* **234**, 115–144.

Green, P. (1989). Shoot morphogenesis, vegetative through floral, from a biophysical perspective. In *Plant Reproduction: From Floral Induction to Pollination* (ed. Lord, E. and Bernier, G.). The American Society of Plant Physiologists Symposium Series, Vol. I, pp. 58–75.

Haughn, G. W. and Sommerville, C. (1988). Genetic control of morphogenesis in *Arabidopsis*. *Dev. Genet.* **9**, 73–89.

Herskowitz, I. (1989). A regulatory hierarchy for cell specialization in yeast. *Nature* **342**, 749–757.

Heslop-Harrison, J. (1963). Sex expression in flowering plants. *Brookhaven Symp. Biol.* **16**, 109–125.

Hill, J. P. and Lord, E. M. (1989). Floral development in *Arabidopsis thaliana*: a comparison of the wild type and the homeotic pistillata mutant. *Can. J. Bot* **67**, 2922–2936.

Holder, N. (1979). Positional information and pattern formation in plant morphogenesis and a mechanism for the involvement of plant hormones. *J. theor. Biol.* **77**, 195–212.

Klemm, M. (1927). Vergleichende morphologische und entwicklungsgeschichtliche Untersuchung einer Reihe multipler Allelomorphe bei *Antirrhinum majus*. *Bot. Archiv* **20**, 423–474.

Meyer, V. (1966). Flower abnormalities. *The Botanical Review* **32**, 165–195.

Meyerowitz, E. M., Smyth, D. R. and Bowman, J. L. (1989). Abnormal flowers and pattern formation in floral development. *Development* **106**, 209–217.

Norman, C., Runswick, M., Pollock, R. and Treisman, R. (1988). Isolation and properties of cDNA clones encoding SRF, a transcription factor that binds to the *c-fos* serum response element. *Cell* **55**, 989–1003.

Passmore, S., Elble, R. and Tye, B.-K. (1989). A protein involved in yeast binds a transcriptional enhancer conserved in eukaryotes. *Genes Dev.* **3**, 921–935.

Sattler, W. (1988). Homeosis in plants *Am. J Bot* **75**, 1606–1617.

Schwarz-Sommer, Zs, Huijser, P., Nacken, W., Saedler, H. and Sommer, H. (1990). Genetic control of flower development by homeotic genes in *Antirrhinum majus*. *Science*, in press.

Sommer, H., Beltran, J.-P., Huijser, P , Pape, H., Lönnig, W.-E., Saedler, H. and Schwarz-Sommer, Zs. (1990). *Deficiens*, a homeotic gene involved in the control of flower morphogenesis in *Antirrhinum majus*: the protein shows homology to transcription factors. *EMBO J.* **9**, 605–613.

Stubbe, H. (1966). *Genetik und Zytologie von* Antirrhinum majus *L. sect.* Antirrhinum. VEB Gustav Fischer Cerlag, Jena.

Walbot, V. (1985). On the life strategies of plants and animals. *Trends Genet.* **1**, 165–169.

Yanofsky, M. F., Hong, M., Bowman, J. L., Drews, G. N., Feldmann, K. A. and Meyerowitz, E. M. (1990). *Agamous*, an *Arabidopsis* homeotic gene whose product resembles transcription factors. *Nature* **346**, 35–39.