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Review Paper

Genetic Control of Morphogenesis in *Arabidopsis*

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INTRODUCTION

The indeterminant pattern of growth characteristic of plants establishes a simple repetitive morphology [Walbot, 1985]. Although studies of plant anatomy, morphology, and physiology have contributed much to our understanding of plant morphogenesis, virtually nothing is known about the molecular mechanisms involved. In contrast to the important role of genetics in our understanding of the regulation of development in a number of animal and microbial systems, the genetic analysis of such processes in plants has not been a widely employed approach [Marx, 1983]. We believe that this difference in approach is related primarily to the vagaries of scientific research rather than to an intrinsic difficulty in applying genetic methods to the study of plant development. Whatever the case, the recent application of molecular methods to problems in plant biology has invigorated interest in the parallel use of genetics.

One plant particularly well suited for studying the molecular genetics of many aspects of plant development is *Arabidopsis thaliana*. The size and short generation of this small crucifer allows for the rapid isolation and characterization of mutants

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The genetic nomenclature used here follows the recommendations developed by an ad hoc committee at the Third International *Arabidopsis* Meeting, Michigan State University, April 1987. (1) Genotypes are italicized; (2) the genotype of the wild type is capitalized and the genotype of mutant alleles is lower case; (3) phenotypes are designated by the gene symbol that is not italicized but has the first letter capitalized; (4) where the gene symbol includes a number it is not separated by a hyphen; and (5) when appropriate, the allele is specified by a hyphenated number. Dominance relationships are not indicated in genotypic designations.

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[Estelle and Somerville, 1986]. One of the most important characteristics is the unusually small genome size, which may allow *Arabidopsis* genes to be cloned solely on the basis of their position on the genetic map by the technique of chromosome walking [Meyerowitz and Pruitt, 1985]. The potential utility of *Arabidopsis* as a tool for developmental genetics, although recognized for several decades [Redei, 1969], is only beginning to be exploited. For this reason the number of available mutants and the extent of their characterization lags behind several other plant genetic systems, including maize and tomato. Early studies of the mutagenic effects of chemicals and radiation on *Arabidopsis* (for a review see Redei, 1969) led to the isolation of hundreds of mutants that affect morphology [Burger, 1971; Koornneef *et al.*, 1982; Koornneef *et al.*, 1983; Kranz, 1978; McKelvie, 1962; Relichova, 1976]. In an early review McKelvie [1962] described 190 distinct morphological changes resulting from single nuclear mutations. Unfortunately, because the emphasis of these early studies was on mutagenesis, not development, the majority of such mutant lines were not characterized further and have not been maintained. However, mutant lines that have been maintained illustrate the potential of the system and may serve as a starting point for the study of a number of developmental processes.

In this brief review we have attempted to summarize the results of all previous studies describing mutants of *Arabidopsis* with altered morphology at one of three different levels: embryogenesis, organ formation (flower development), and cell development (trichomes). These mutants give clues regarding the way these developmental processes are regulated, and they provide direct access to the molecular genetics of these systems by marking relevent genes. Although much of the information is preliminary or has been incompletely developed, we believe that it is possible to recognize in the available information many promising opportunities. We have not included the classes of mutants of *Arabidopsis* that do not respond normally to environmental stimuli or to exogenous plant-growth regulators because these are described elsewhere [Finkelstein *et al.*, 1987]

EMBRYO-LETHAL MUTANTS

The formation of an embryo from a single cell involves a complex pattern of directed cell division and differentiation in which the body axis (hypocotyl), root and shoot apical meristems, and cotyledons are formed. During later stages the embryo becomes autotrophic, and metabolic changes occur to prepare for seed dormancy and germination. Embryo development in *Arabidopsis* follows a pattern similar to that of the extensively studied dicotyledonous plant *Capsella bursa-pastoris* [Marsden and Meinke, 1985] (Fig. 1). The zygote gives rise to an embryo proper supported by a six- to eight-cell stalk or suspensor. Embryonic morphogenesis occurs over a 7-day period, during which time the embryo becomes autotrophic, the suspensor degenerates, and the cotyledons change into storage organs that accumulate lipid, carbohydrate, and protein to be used during germination.

Meinke and coworkers have attempted to identify genes that control embryogenesis through the use of embryo-lethal mutants [reviewed by Meinke, 1986]. In two separate studies [Meinke and Sussex, 1979a, 1979b] over 30 embryo-lethal mutants were isolated. The approach, originally developed by Muller [1963], was to examine seed pods (siliques) of mutagenized plants for the presence of aborted seeds. The lethal mutations were then rescued as heterozygotes by advancing phenotypically

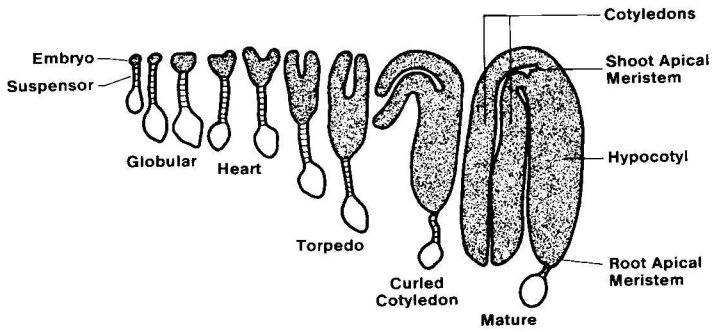

Fig. 1. Developmental stages during *Arabidopsis* embryogenesis. The names of each of the stages lie underneath. Parts of the early globular (far left) and mature (far right) embryo are indicated.

normal seeds from the same silique. Development of these mutants was found to be arrested in a variety of embryonic stages, ranging from preglobular to mature cotyledon. Arrest of embryo development was sometimes accompanied by abnormalities in embryo color or morphology. Although neither genetic complementation nor linkage analysis of the mutations causing embryo lethality were done, the variety of different phenotypes observed and the high frequency at which they were found suggests that most of the mutations occurred at different loci.

To determine the extent of cellular differentiation in arrested embryos, the deposition of seed-storage proteins in wild-type and arrested embryos was analyzed biochemically [Heath *et al.*, 1986] and ultrastructurally [Meinke, 1986]. In every mutant examined, the level of seed storage protein at the time of embryonic arrest was the same as the level in a wild-type embryo at the same morphological stage. These observations demonstrated that developmental arrest occurred at the biochemical and cellular as well as at the morphological level, but they gave little information about the nature of the defect that caused embryo arrest.

As an approach to the study of development, embryo lethality has at least two major related drawbacks. First, the approach does not identify mutations that specifically affect development. A large number of genes required for cell maintenance and growth would be expected to cause embryo lethality. Thus, the number of possible mutations that cause embryo lethality may be so large as to preclude the identification of the subset that directly affects development. Second, because the mutations prevent the analysis of later developmental processes, it is impossible to determine which of the mutations affect processes specific to embryo development. In attempting to address the second problem, Meinke and collaborators examined the distribution of aborted seed within the siliques of various mutants [Meinke, 1982, 1985; Meinke and Baus, 1985]. Some mutants had a nonrandom distribution of aborted embryos within the silique, with most of the aborted embryos lying in the upper (stigma proximal) half. This phenomenon might be due to a decrease in fitness of the male gametophyte, indicating that the mutant phenotype was not limited to the embryo. However, the lack of an effect on the gametophyte cannot be taken as evidence that the mutation is specific for embryo development. A second genetic approach to studying lethal mutations, the use of temperature-conditional phenotypes, was also attempted [Meinke, 1985]. Previously isolated mutants were grown at 18°C and examined for the loss of embryo lethality. Although no temperature-sensitive mutants were identi-

fied, only a few mutants were screened, and the temperature at which these were isolated (24°C) was relatively close to the temperature at which they were screened (18°C). Thus, the use of conditional lethals may warrant further investigation.

Recently, Meinke and coworkers have successfully used tissue culture as an embryo-rescue technique that permits the evaluation of the effects of several mutations in all stages of the life cycle. Two of 17 mutants tested were rescued by this approach. Embryos of mutant 122G-E, which were arrested throughout embryogenesis, could be rescued on a medium enriched with vitamins, amino acids, and nucleosides [Baus *et al.*, 1986]. The mutant specifically requires biotin during development of the inflorescence and seeds but not during vegetative growth, suggesting that some genes involved in biotin metabolism are specific to different stages of development.

Tissue from a second mutant (12A-2A), which arrests growth in the linear cotyledon stages, could be induced to form shoots, but not roots, in the presence of the appropriate concentrations of the plant-growth regulators auxin and cytokinin [Baus *et al.*, 1986]. Thus, the defect in this mutant may interfere with initiation of root apical meristems. However the similarity in phenotype between this mutant and a tobacco mutant [Muller *et al.*, 1985] has led to speculation that the *Arabidopsis* mutant might be defective in some aspect of metabolism or action of the plant-growth regulator auxin [Meinke, 1986].

The isolation of these mutants is consistent with a previous suggestion [Nelson and Burr, 1973] that auxotrophic mutants may be embryo lethal because the plant becomes autonomous for the synthesis of most metabolites during embryo development. Indeed, it seems likely that any mutant that can be rescued by transfer to exogenous nutrients may not represent a developmental mutant per se.

MUTATIONS ALTERING FLORAL MORPHOGENESIS

The flower can be considered a specialized shoot having a determinate growth pattern, short internodes, and specialized organs and lacking lateral meristem development. Studies of comparative morphology and anatomy have indicated that all flower organs are structurally related to the leaf, which is consistent with the hypothesis that flowers evolved from lateral shoots and floral organs from modified leaves [Arber, 1937]. This concept of diversity through morphological variation on a standard body unit has elements in common with current concepts concerning *Drosophila* segmentation [Gehring and Hiromi, 1986]. However, the extent to which such parallels can be drawn must await the investigation of genes that control floral morphogenesis.

The simplicity of its inflorescence and flower structure makes *Arabidopsis* particularly attractive for molecular genetic analysis of floral morphogenesis. The inflorescence of *Arabidopsis* has been the subject of several anatomical [Miksche and Brown, 1965; Vaughn, 1955] and morphological [Muller, 1961] studies. The flowers have a perfect, bilaterally symmetric structure typical of the crucifers (Figs. 2, 3A) and are arranged on a stem in an indeterminate spiral or raceme. Floral organs are initiated in whorls from outside to inside, as follows: four sepals, four petals, two short and four long stamens, and a bicarpellate pistil or gynoecium [Vaughn, 1955], which may arise from four primordia [Sattler, 1973].

A wide variety of mutants affecting floral morphology in *Arabidopsis* have been isolated and described [Arnold, 1965; Buggert and Robbelen, 1970; Burger, 1971;

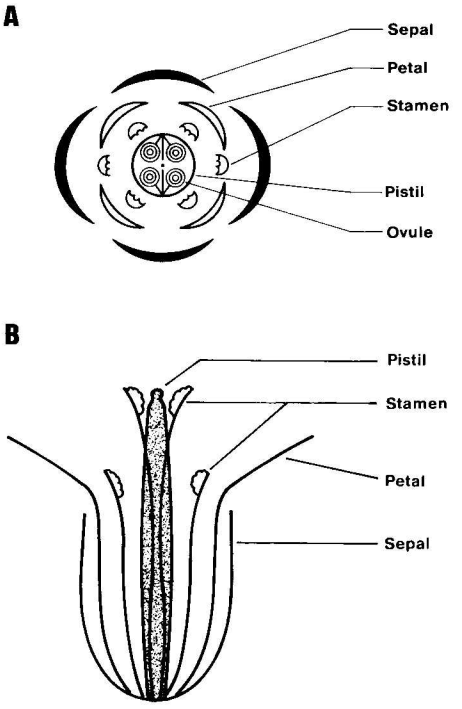

Fig. 2. Diagrammatic representations of a cross section **A:** and a longitudinal section **B:** of an *Arabidopsis* flower.

Goto *et al.*, 1987; Koornneef *et al.*, 1983; Kranz, 1978; McKelvie, 1962; Reinholz, 1966; Relichova, 1976; Robbelen, 1965; Usmanov, 1970; von Conrad, 1971; reviewed by Napp Zinn, 1985]. Table 1 lists many of the mutants still available but does not include the relatively uncharacterized mutants of the *Arabidopsis* Information Service Seed Bank [Kranz, 1978]. Most of these mutants have been isolated within the last 5 years. Although none of the mutants described in Table 1 has been fully characterized, the phenotypes suggest that the products of the mutated genes play key roles in floral development. Alterations in floral morphology observed among the mutants include changes in the number and position of organs (all mutants listed in Table 1); an increase in the length of the floral internodes (Lfy1); the appearance of lateral shoots in the floral bud (Ag1, Ap2, Lfy1, Flo4; see Fig. 4F); and the loss of determinant state of growth (Ag1). Further, phenotypic changes in most of these mutants are specific to the inflorescence (excepting pinformed and Flo5). Like many of the mutations affecting morphology of *Drosophila*, most of the mutations causing the phenotypes described in Table 1 are incompletely penetrant and may vary in expressivity, so that substantial variation may be observed even between flowers on the same plant.

The majority of mutants have one or more organ types replaced by another as a part of their phenotype and can therefore be considered homeotic mutants. For example, several mutants have carpels in place of either sepals (*Flo2*; Fig. 3B) or the entire perianth (*Flo3*, *Flo4*; Fig. 4E). In the *Flo9* mutant the stamens are often replaced by organs that have features characteristic of both anthers and petals (Fig.

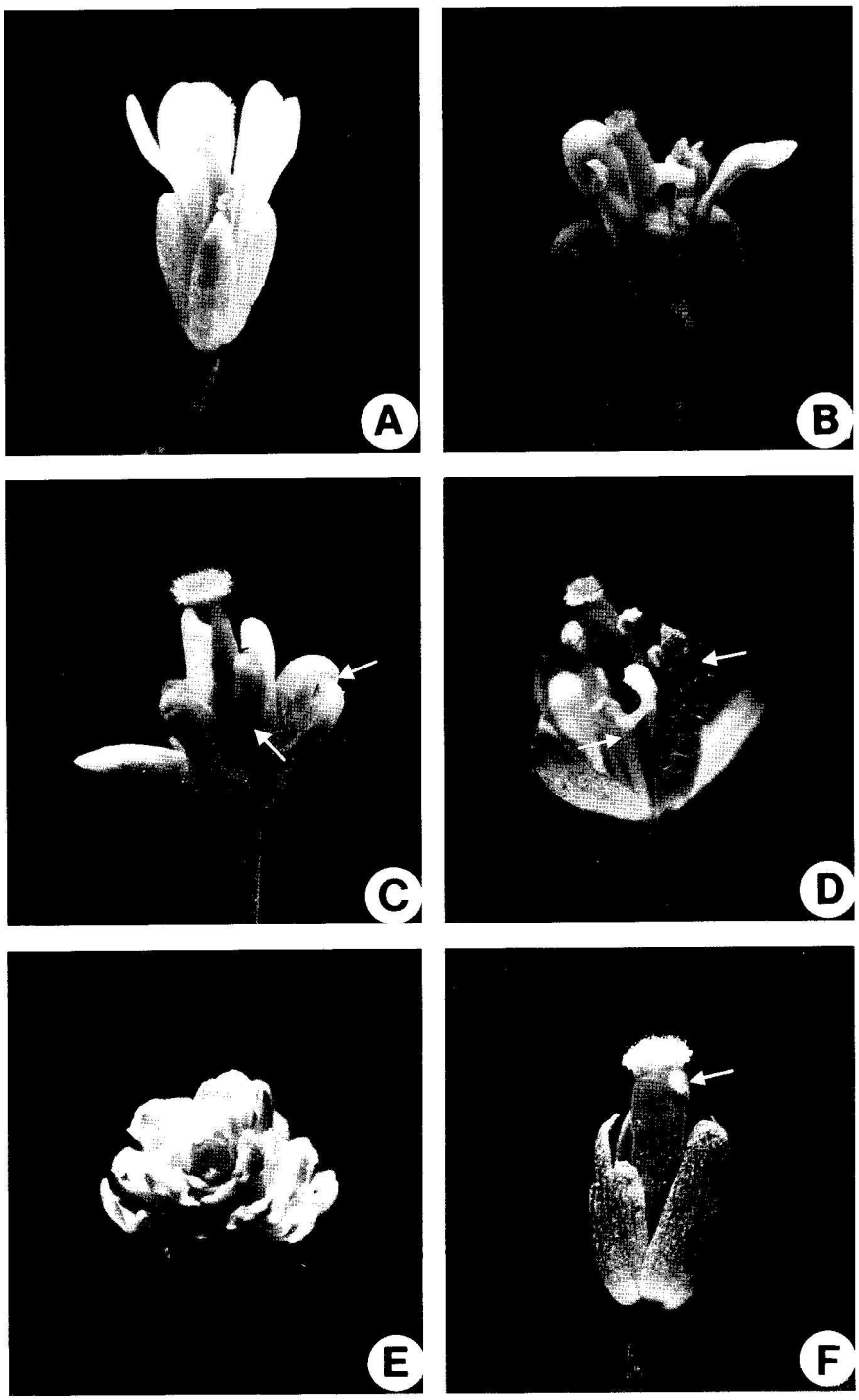

Fig. 3. Mutants of *Arabidopsis* with altered flower morphology. **A:** Wild type flower. **B:** Flo2 flower. **C:** Flo9 flower; arrows indicate structures that are intermediate between petals and stamen. **D:** Ap2 flower; the upper arrow indicates a leaflike flower organ with trichomes, and the lower arrow indicates a structure intermediate between a petal and a stamen. **E:** Ag1 flower. **F:** Pi1 flower; the arrow indicates a filamentous structure with carpel-like papillae at the top. All photographs are magnified ×11.

3C). Other mutants have more than one homeotic change induced by a single recessive mutation. The *ap2* mutation (Fig. 3D) causes sepals to be replaced by leaves and often causes petals to have characteristics of stamen. In some instances the phenotypes are not readily interpretable. For instance, in the *Pi1* mutant (Fig. 3F), normal stamens are absent and are replaced by a variety of filamentous structures that have features in common with the carpel. If we ignore such difficulties in interpretation for the sake of simplicity, it is possible to consider such phenotypes as discrete organ-for-organ replacements. Under this assumption, the homeotic changes in floral organs of various mutants of *Arabidopsis* can be integrated into a single scheme, as illustrated in Figure 5. The diagram includes the mutant phenotypes listed in Table 1 as well as the phenotype of the mutant 8/76 [Robbelen, 1965], where all floral organs were carpel-like. Figure 5 is intended as a device to assist in the organization of information on the available mutant phenotypes and not to imply genetic pathways for the regulation of development. However, one trend is immediately noticeable: the direction of homeotic changes is more frequently from stamens, petals, and sepals and toward carpels and leaves. Thus, carpel and leaf formation may represent genetic ground states for the fate of developing organ primordia.

Mutant phenotypes can be further characterized and the interaction of gene products determined by the construction of lines homozygous for two different mutations. Recently a *pi1 ap2* line was constructed (E. Meyerowitz, personal communication). This line has two whorls of leaves in place of sepals and petals. Thus, *pi1* appears to be epistatic to *ap2* with respect to the fate of petal primordia. The complex phenotypes of the *pi1* and *ap2* mutations and the properties of the double mutant can be most easily explained by a hypothesis that takes into account the interaction of several homeotic gene products in determining the fate of organ primordia. A simple model of this type is presented in Table 2. A loss of function mutation in gene *a* would result in the Ap2 phenotype if it is assumed that an off-on-off pattern produces an organ intermediate between stamen and petals. Similarly a loss-of-function mutation in gene *b* would result in a phenotype similar to the mutant *Pi1*. The double mutant would be expected to have two whorls of leaves, as observed. Although attractively simple, this hypothesis does not account for all the available data. The *pi1 ap2* double mutant should have carpels replacing stamens but it instead results primarily in the loss of stamens (E. Meyerowitz, personal communication). Other inconsistencies arising from the phenotypes of additional mutants suggest that the hypothesis presented in Table 2 is too simple. For example, the stamen-to-petal change observed in the *flo9* mutant cannot be explained by a single mutation in *a*, *b*, or *c*. However, the model does indicate that the apparent complexity of flower development need not involve more than a few genes to set the pattern of differentiation at the organ level.

The varying complexity of homeotic changes resulting from a single mutation could be due to a hierarchy of decision making, with the most complex phenotypes resulting from a mutation in a gene that plays a role very early in floral development. One such complex phenotype is that of the mutant line GH110, which preliminary evidence suggests is due to a single recessive nuclear mutation designated *lfy1* (Haughn and McCourt, unpublished observations; Fig. 4A). Flowers of this mutant develop only leaves or sepal-like organs (Fig. 4A), except for the most apical organs, which often appear as open carpels. Unlike normal flowers, the internodes between organs of flowers of the *Lfy1* mutant often elongate to produce a determinate shoot

TABLE 1. Mutations That Cause Altered Flower Morphology in *Arabidopsis*

Name	Gene designation	Nature of lesion	Chromosome-map position	Phenotype	Origin	Mutants with similar phenotype
Agamous	*ag1*	Nuclear recessive	4-33.3	Nested perianths each with four sepals and approximately ten petals; indeterminate flower growth	1[a]	Multipetala [ref 36]
Apetala	*ap1*	Nuclear recessive	1-102.4	Lacks petals; bracts replace sepals; flowers initiated in sepal axils	1	Apetala, axillaris [ref 6,32,33]
Apetala	*ap2*	Nuclear recessive	4-58.7	Bracts replace sepals; petals replaced by structure with features of anthers and petals	1	—
Pistillata	*pi1*	Nuclear recessive	5-24.1	Sepals replace petals; stamens absent or with carpel-like features; carpels often misshapen	1	—
Clavata	*clv1*	Nuclear recessive	1-113.5	Gynoecium club shaped with 4 fused carpels	1	Clavata [ref 6]
	clv2	Nuclear recessive	1-88.4			
Leafy inflorescence	*lfy1*	Nuclear recessive	ND	Floral organs leaflike; carpels underdeveloped; internodes elongated; lateral flowers produced in all axils	2	—

Flower development	*flo3*	Recessive	ND	Perianth replaced by one whorl of carpel-like organs	2	—
Flower development	*flo4*	ND	ND	Like *flo3*; anthers usually absent	3	—
Flower development	*flo2*	Recessive	ND	Sepals replaced by carpels	4	Fasciata [ref 34]
Flower development	*flo9*	Nuclear recessive	ND	Anthers often petal-like	5	—
Pinformed	*pin*	Nuclear recessive	ND	Most floral stalks lack flowers, flowers have thin elongated pistils, multiple petals, no stamen, reduced sepals	6	*flo8* [ref 2]
Flower development	*flo5*	Recessive	ND	One or two extra organs per whorl; fasciated stem	2	—

[a]Numbers correspond to the researchers listed below:
[1]M. Koornneef, 1981.
[2]G. Haughn and P. McCourt, unpublished.
[3]S. Somerville, unpublished.
[4]J. Martinez-Zapater, unpublished.
[5]R. Rasooly, unpublished.
[6]Goto *et al.*, 1987.
ND = not determined.

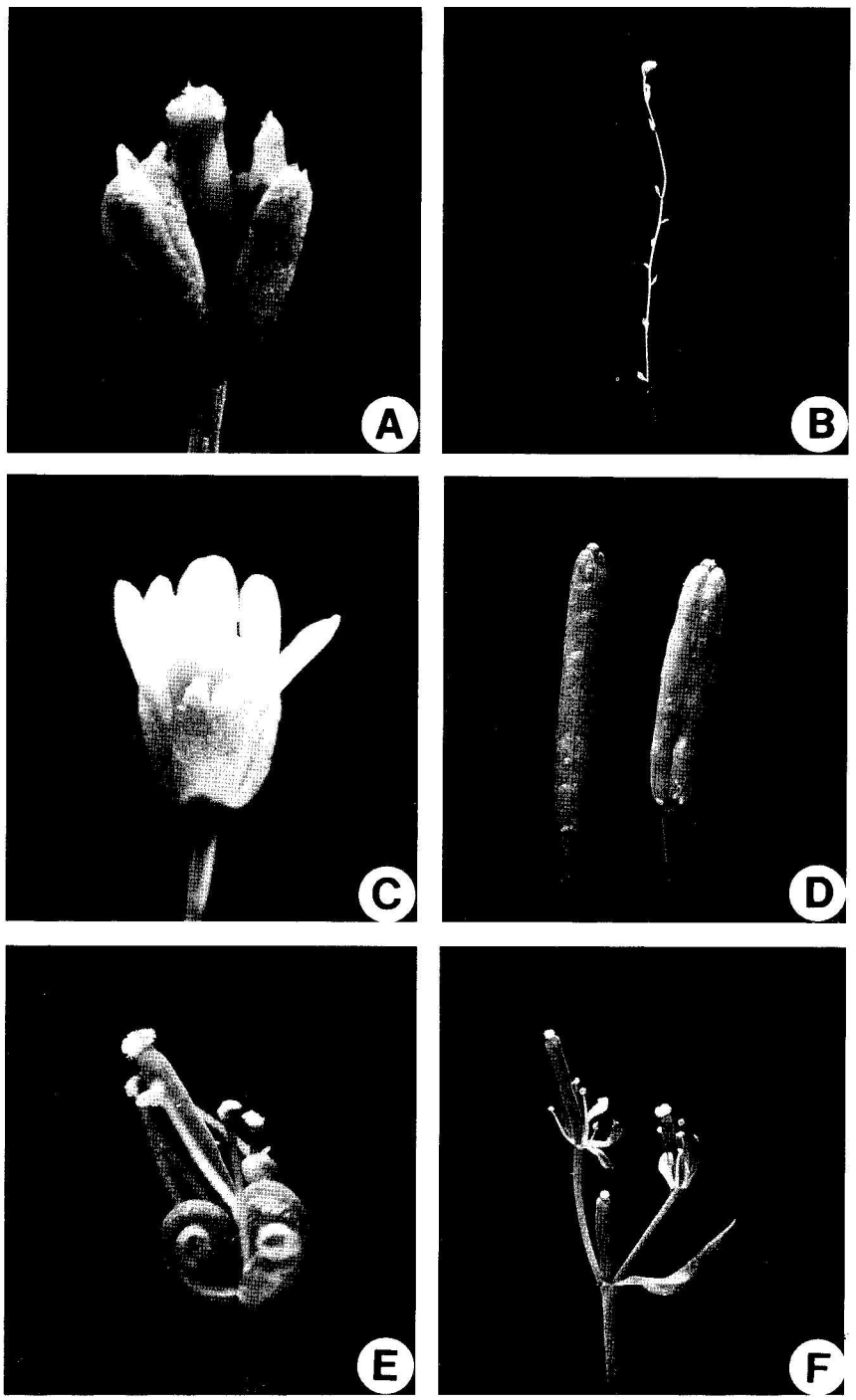

Fig. 4. Mutants of *Arabidopsis* with altered flower morphology. **A:** Lfy1 flower; magnification, ×11. **B:** Lfy1 flower following elongation of the stem, ×1.2. **C:** Flo5 flower, ×11. **D:** Siliques of wild type (left) and Clv2 (right), ×2. **E:** Flo3 flower, ×11. **F:** Ap1 flower showing secondary flowers initiated in the sepal axils, ×6.

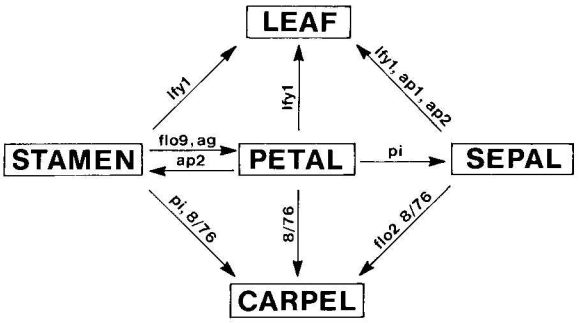

Fig. 5. A scheme indicating the homeotic transformations of flower organs caused by mutations of *Arabidopsis*. The arrow shows the direction of a homeotic transformation where an organ type observed in the wild-type flower (arrow points away) is replaced by a different organ type in the mutant flower (arrow points toward). Positioned above each arrow are the names of mutations that cause the homeotic transformation indicated by that arrow.

TABLE 2. A Model for Gene Regulation of Floral Organ Identity*

Organ	*a*	*b*	*c*
Carpel	Off	Off	On
Stamen	Off	On	On
Petal	On	On	Off
Sepal	On	Off	Off
Leaf	Off	Off	Off

*The letters *a, b, c* designate genes; the model proposes that these three genes together establish organ identity by being either expressed (On) or not expressed (Off) in the different organs shown.

(Fig. 4B). The phenotype of the mutant lends support to the hypothesis that a flower represents a modified shoot [Arber, 1937] and suggests that the product of the *LFY1* locus acts early in the process that determines the fate of lateral meristems.

In addition to the identity of flower organs, information concerning the number of whorls and number of organs in a whorl must be specified. Four mutants of *Arabidopsis* affect these characters. The *ag* mutation (Fig. 3E) causes an increase in the number of whorls per flower. Flowers of plants carrying the *ag* mutation consist of a number of nested perianths (sepals plus petals), each of which has one whorl of four sepals and three whorls of approximately ten petals. No stamens or carpels are produced. The number of petals in each perianth suggests a homeotic transformation of stamens to petals (6 stamens + 4 petals = 10). The sepals in the next whorl may represent a carpel-to-sepal transformation that starts another round of perianth development. With this interpretation, the AG1 locus can be considered to specify information needed to produce reproductive structures (the equivalent of gene *c* in Table 2). It is interesting that the only mutant to lack carpels is also the only mutant to have flowers with an indeterminant pattern of growth. Possibly, the determinant nature of the floral meristem is a consequence of carpel formation.

One mutation (*flo5*; Fig. 4c) exhibits an increase in the number of organs in a whorl. Each whorl in a flower typically has five sepals, five petals, eight stamens, and a pistil with five carpels (four fused around a fifth). Thus, the basic number of

primordia per whorl is increased from four to five. The *flo5* mutation also causes an increase in the number of leaves per whorl in the rosette, suggesting that both floral and vegetative meristems share a common control over the initiation of organ primordia.

Mutations at two loci, *clv1* and *clv2* (Fig. 4D), produce a pistil very similar to that of the *Flo5* mutant (four carpels surrounding what appears to be a degenerate fifth carpel) but no other characteristics of the Flo5 phenotype. These mutants may assist in resolving controversy concerning the number of primordia required for gynoecial organogenesis [Sattler, 1973].

MUTATIONS AFFECTING PLANT TRICHOMES

The use of genetics to study the growth and development of individual cell types in multicellular organisms is hampered by the fact that the cells are inaccessible and indispensable. The epidermal trichome has neither of these disadvantages and may represent an excellent genetic system for studying plant-cell development. The trichomes of *Arabidopsis*, like those of most Brassicaceae [Rollins and Banerjee, 1975], are single-stalked dendritic epidermal cells (Fig. 6A–C) with a thickened cell wall and papillate cuticle. The trichomes are present on the surfaces of leaves, stems, and sepals and are evenly spaced over each separate organ (Fig. 6C). The majority of trichomes on the leaves have three branches projecting from the top of a stalk. Unbranched trichomes are found along the perimeter of leaves and represent the predominant class on the floral stem. The base of the trichome cell is encircled by a single layer of support cells distinguished from other epidermal cells by their rectangular shape (Fig. 6A). Although the function of the Brassicaceae trichome cell is unknown, it has been suggested that they serve as organs that absorb water from the surface of the leaf, reflect light, or discourage insect predation [Uphof, 1962].

The trichome cell is easily visible, and mutants with altered trichome morphology have been isolated and characterized to serve as genetic markers [Koornneef *et al.*, 1983]. These are listed in Table 3, along with two novel mutants that have been isolated recently. In all but one instance (*ttg1*), phenotypic changes are specific to the trichomes. Elimination of the trichomes by one of two mutations, *gl1* and *ttg1*, does not result in any deleterious effects to the growth and development of the plant. Scanning electron micrographs of the leaf surface of the *gl1* mutant (Fig. 6D) failed to show any indication of rudimentary trichomes or of the characteristic support cells on the leaf surface. A few trichomes are produced at the leaf margin. The presence of these cells could be due to leakiness in expression of the phenotype or may represent a separate population that is unaffected by the mutations. It is likely that *gl1* and *ttg1* represent the only loci associated with loss of trichomes, because a number of independently isolated glaborous mutants have mutations that fail to complement one of these two loci [Koornneef *et al.*, 1982] (G. Haughn, unpublished results). The elimination of a specific cell type with few obvious pleiotropic effects and the small number of loci responsible suggest that products of the *GL1* and *TTG1* loci are involved in determination of trichomes from epidermal cells. In this respect these loci are good candidates for genes that specifically control development.

Loss of trichomes is the only known effect of *gl1* mutations. By contrast, *ttg* mutants show a number of pleiotropic effects [Koornneef, 1981], including loss of pigmentation (also associated with the transparent testa phenotype) and loss of seed

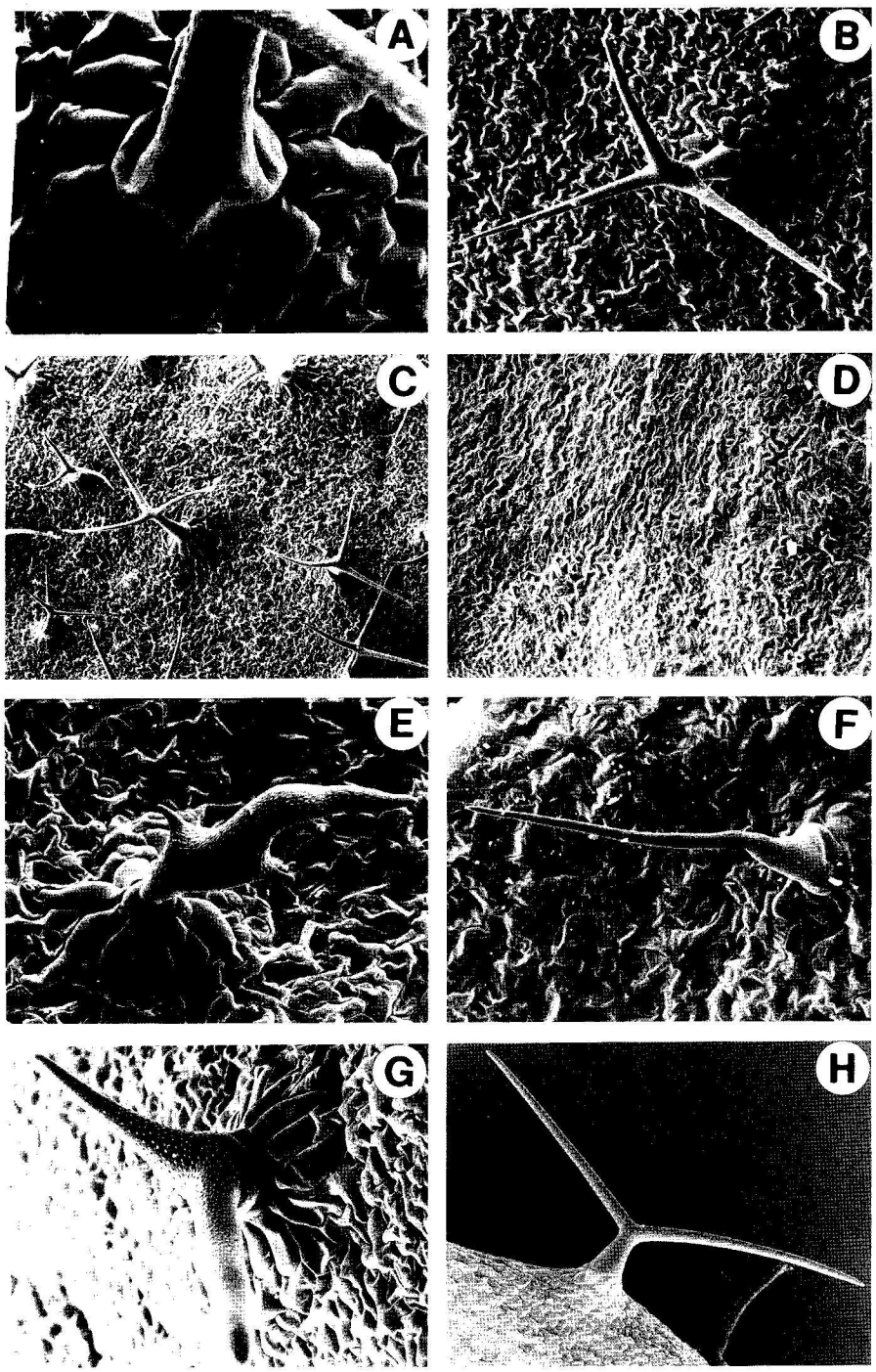

Fig. 6. Wild-type and mutant trichome morphologies in *Arabidopsis*. **A:** The base of a wild-type trichome showing the surrounding support cells, ×500. **B:** Wild-type trichome, ×130. **C:** Surface of a wild-type leaf, ×54. **D:** Surface of a Gl1 leaf, ×54. **E:** Dis1 trichome, ×240. **F:** Gl3 trichome, ×200. **G:** Skt1 trichome, ×200. **H:** Udt1 trichome, ×100.

TABLE 3. Mutations That Cause Altered Trichome Morphology

Name	Gene designation	Nature of lesion	Chromosome-map position	Phenotype	Reference
Glaborous	*gl1*	Nuclear recessive	3–39.6	Trichomes absent	[a]
Glaborous	*gl2*	Nuclear recessive	1–122.1	Trichomes undeveloped on first 2 leaves; reduced in number on the rest	[a]
Glaborous	*gl3*	Nuclear recessive	5–54.9	Trichomes relatively unbranched and reduced in number	[a]
Distorted	*dis1*	Nuclear recessive	1–18.5	Trichomes small and malformed	[b]
Distorted	*dis2*	Nuclear recessive	1–46.4	Trichomes small and malformed	[b]
Transparent testa, glabra	*ttg1*	Nuclear recessive	5–32.3	Like *tt3*, lacks trichomes except at leaf edge; altered seed-coat structure	[c]
Stalkless	*slt1*	ND[e]	ND	Trichomes lack stalk and have only two branches	[d]
Under-developed	*udt1*	ND	ND	Trichomes have two branches; surface papillae are slow to develop	[d]

[a]Goto *et al.*, 1987.
[b]Walbot, 1985.
[c]von Conrad, 1971.
[d]G. Haughn, unpublished observations.
[e]ND = not determined.

mucillage (also associated with the *Gl2* phenotype). This connection between pigmentation, mucillage, and trichomes is puzzling. Koornneef [1981] has speculated that a specific metabolite might be required for the formation of trichomes, anthocyanins, and mucillage or that *TTG* is a regultory gene controlling all three traits. In support of the latter point are the observations that accumulation of anthocyanins can be induced in *Arabidopsis* by stress and that both seed mucillage and trichomes may be involved in prevention of water loss. Thus, it is conceivable that all three characters share a common regulatory locus.

A second class of mutants shows altered trichome morphology (Table 3). Recessive mutations at loci *dis1* and *dis2* [Feenstra, 1978] result in a distorted trichome morphology (Fig. 6E). Other mutants have trichomes with reduced branching and stalk size (*Gl3, Slt1,* and *Udt1*; Fig. 6F, G, and H, respectively). These mutants should prove to be valuable in the study of the mechanisms by which the trichome establishes its unique three-dimensional structure. A current hypothesis about the control of cell morphogenesis maintains that cell growth is directed by the interaction of cytoskeletal proteins and cell-wall microfibril biosynthesis [Lloyd and Seagull, 1985]. It is conceivable that the loci affecting trichome morphology are cytoskeletal components. For example, *DIS1* and *DIS2* could encode species of tubulins that are synthesized only in the trichome. Regardless of their function, such loci are interesting in the context of cell development.

It is obvious from the few examples presented that mutations that specifically affect trichomes can be isolated. Few cell types are so easily accessible and dispensable. The trichome therefore represents a unique opportunity to use genetics to study cellular differentiation in higher plants.

CONCLUSIONS

The regulation of plant morphogenesis is a fascinating but still poorly understood process. Like mutant analysis in the dissection of developmental processes in organisms from other kingdoms, molecular genetics is likely to be a useful or even an essential approach to understanding plant development. Although only preliminary analyses have been carried out on the developmental mutants of *Arabidopsis*, we believe that the available information indicates the existence of a wide range of potentially useful mutants. Furthermore, the phenotypes of many of these mutants can be interpreted with reference to both classical anatomical hypotheses and models similar to those developed to assist in understanding homoetic mutations in *Drosophila*. Many of the mutations described here that specifically alter flower and trichome morphology give clues about the way those structures develop and seem likely to affect genes that regulate morphogenesis. A vast amount of work remains to be done to understand the function of the genes in which these mutations lie. We are confident, however, that through the application of molecular genetic techniques to the analysis of the *Arabidopsis* mutants [Meyerowitz and Pruitt, 1985], significant progress in this area will be forthcoming.

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