

## Abnormal flowers and pattern formation in floral development

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**“From our acquaintance with this abnormal metamorphosis, we are enabled to unveil the secrets that normal metamorphosis conceals from us, and to see distinctly what, from the regular course of development, we can only infer.”**

– J. W. von Goethe (1790)

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### Introduction

The development of flowers is a mystery. Each flower starts as a small clump of undifferentiated cells, but develops into a complex structure in which different organs occupy precisely defined positions. In addition, each organ has its own characteristic cell types, organization and function. Since there is no cell migration in higher plant development and since flowers can develop normally without positional information being specified maternally, each cell in the developing floral primordium must somehow learn its position relative to other cells, and differentiate accordingly. Mutant plants in which cells do this improperly have recently become a focus of attention. In this review, we will briefly describe past work in morphological analysis of abnormal flowers and in the inheritance of floral abnormalities. While many instances of inherited alterations in flower development have been recognized and studied to answer genetic or evolutionary questions, there has, surprisingly, been little use of such material in experiments directed to understanding flower development. Recently such work has started, though, with several plant species, and in several laboratories. We will examine the past work and will describe recent work focussed on understanding the molecular mechanisms by which cells in developing flowers recognize their positions in space and in developmental time, and thus differentiate into appropriate organs in appropriate places.

### Monstrous flowers as curiosities

Monstrous flowers have been recognized as curiosities by botanists for more than two thousand years:

Theophrastus mentions double roses (flowers containing many more than the normal number of petals) in his *Enquiry into Plants*, written before 286 BC; double roses were also described by Pliny in the first century. Double peonies were known and selected by around 750 AD in China, and two Thang Dynasty authors described these flowers in the ninth century (Needham, 1986). Double flowers were described by the herbalists of the Renaissance, as well: Dodoens published such a description in 1568, and Gerard (1597) has illustrations of many double flowers beside their wild-type counterparts. Double flowers are thus the earliest recognized examples of floral abnormalities and of floral mutants. They have been of interest from the origins of botany to the present: Masters (1869) and Worsdell (1915–6) describe many of them, as do Reynolds & Tampion (1983) in a recent book that classifies the different developmental origins of the extra petals in different species with double flowers. Double flowers are at least as common in present-day gardens as they were in those of the herbalists: hybrid tea roses, carnations, double camellias, columbines and stocks, and so on, are all familiar examples (Reynolds & Tampion, 1983).

There are many additional, characteristic types of floral abnormalities, with homologous examples in many different species. One type of abnormal flower of considerable interest to developmental biologists is that said by early plant teratologists to exhibit ‘metamorphosis’ or ‘metamorph’, the appearance of a normal organ in the place where a homologous organ of a different type is typically found (Figs 1 and 2). The use of metamorphosis to refer to the replacement of one organ with a homologue was used by Goethe (1790), who in an essay on flower development called the appearance of one type of floral organ in a site normally occupied by an organ of a different type ‘abnormal

metamorphosis'. Goethe emphasized the homology of the organs in successive whorls of flowers, homology between organs having been suggested to him by cases of abnormal metamorphosis. Perhaps the earliest compendium of such abnormalities is that of Moquin-Tandon (1841), which summarizes a large number of reports of unusual flower forms published from the seventeenth to the early nineteenth centuries. The classic English work on floral abnormalities is by Masters (1869). Both Moquin-Tandon and Masters categorized the abnormalities according to the nature of the departure that they showed from the normal flower of the same species; neither considered seriously the distinction between inherited abnormalities and freaks. Moquin-Tandon (1841) traces the concept of metamorphosis to Jung in the seventeenth century; a critical review of the various definitions and interpretations of this concept is found in Sachs (1906).

In his 1894 classification of animal abnormalities, Bateson refers extensively to Masters' earlier work, and replaces Masters' 'metamorphosis' with another word, 'homoeosis'. Bateson defined homoeosis as 'the assumption by one member of a Meristic series, of the form or character proper to other members of the series...' and goes on to state "In the case of plants such Variation is very common and is one of the most familiar forms of abnormality." In flowers the meristic, or repetitious, series refers to the successive whorls of different organs that constitute the flower; "formation of sepaloid petals", as shown in the mutant flowers depicted in Fig. 1C,D and F and Fig. 2E and F, is one of Bateson's examples of homeotic floral variation. Sattler (1988) has recently reviewed some of the literature on homoeosis in plants, pointing out that some authors after Bateson have redefined the term to include phenomena not recognized as homoeosis or metamorphosis by the earlier authors, such as appearance of similar organs in different places in plants of different species.

There have been other encyclopedic catalogues of floral abnormalities since Moquin-Tandon and Masters, including those by Penzig (1890-4), arranged taxonomically, and Worsdell (1915-6), arranged by type of abnormality. Remarkably, not even the latter, compiled 15 years after the rediscovery of Mendel's experiments, differentiates between regularly inherited abnormalities and individual aberrant forms resulting from environmental treatments, insect infestations, and chance. A more recent review (Meyer, 1966) summarizes many of the findings of the earlier authors, and the categories of homeotic conversions of floral organs that have been observed in a wide variety of plant species.

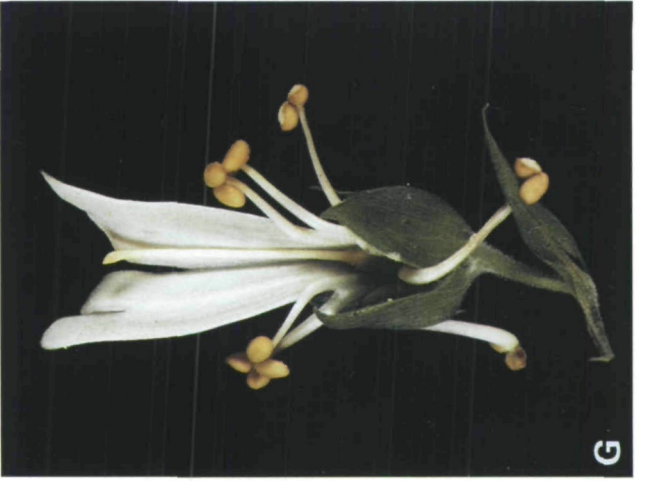
### Evolutionary Interpretation

One early consideration of abnormal flowers as something other than curiosities was by Linnaeus, who in 1744 described a mutant flower phenotype in toad flax (*Linaria vulgaris*), and briefly recognized that the occurrence of mutations casts doubt on the then-current view (earlier propounded by Linnaeus himself) of the

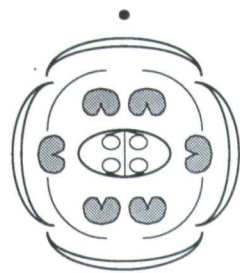
**Fig. 1.** Homeotic flower mutants of various plant species. (A-D) Phenotypes of flowers of the snapdragon, *Antirrhinum majus* (see Stubbe, 1966). (A) Wild-type morphology. The plant is homozygous for the anthocyanin mutation *nivea* which arose by insertion of the transposable element *Tam1* into the chalcone synthase gene. Pigment is absent in the corolla except where a reversion has occurred following excision of the transposable element during growth. Transposons *Tam1* and *Tam3* will be useful in tagging homeotic mutations in *Antirrhinum* (Coen & Carpenter, 1986). (B) The *cycloidea*<sup>radialis</sup> phenotype in which the five petals of the corolla tube all resemble the central petal of the lower lip. The corolla is now radially symmetrical whereas in the wild type it is zygomorphic. (C) The *deficiens*<sup>chlorantha</sup> phenotype. In this mutant line the petals are reduced in size and partially green. (D) A more extreme allele, *deficiens*<sup>globifera</sup>. In this case the sepals grow in place of petals. Also in place of the stamens, a tube is formed around the gynoecium with a stigmatic surface at the top and a ring of ovaries at the base. (*Antirrhinum* plants photographed at the Max-Planck-Institut für Züchtungsforschung, Köln, with thanks to Ekkehard Lönig.) (E) Flowers of the green rose, *Rosa* cv. 'Viridiflora', in which all flower organs resemble leaves. (Photographed at the Huntington Botanical Gardens, San Marino; California) (F) Phenotype of the green petals mutation of *Petunia hybrida* (see de Vlamming *et al.* 1984). (Photographed at the Department of Genetics, Vrije Universiteit, Amsterdam.) (G) The *heptandra* mutant phenotype of the common foxglove, *Digitalis purpurea* (see Saunders, 1911; Shull, 1912). The wild-type flower has a tubular corolla with five lobes, two in the upper lip and three in the lower lip. Four stamens are attached inside the corolla tube. In the *heptandra* mutant the three petals of the lower lip are often replaced by three stamens. The resulting flowers have seven stamens. (Photo: Keith Roberts)

immutable fixity of species (Moquin-Tandon, 1841; DeVries, 1903; Gustafsson, 1979). The type of abnormality recognized and named by Linnaeus is peloria, in which a plant of a species with flowers that are ordinarily bilaterally symmetric (such as snapdragon, *Antirrhinum*) has radially symmetric flowers. Peloric flowers are known in many families of flowering plants (DeVries, 1906). The *Antirrhinum cycloidea* mutation shown in Fig. 1B is an example.

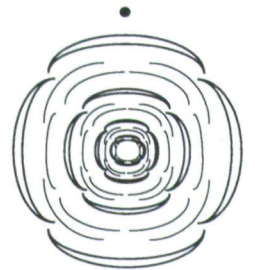
In the nineteenth and present centuries, the nature of floral monstrosities has been used to make inferences about the evolutionary homologies of different plant parts, and, by arbitrarily declaring certain mutations to be atavistic, about the structure of ancestral flowers. Darwin employed this type of reasoning cautiously (Darwin, 1876); his successors were not always cautious. For example, many instances of speculative use of teratological data are found in Worsdell (1915-6), a plant teratology work that is a successor to Masters' compendium, but which adds to the rigorous descriptions of Masters an imaginative evolutionary interpretation of many of the floral abnormalities. "In very many cases the so-called 'freaks' and 'monstrosities' represent reversions or harkings-back, in one form or another, to an ancestral condition..." The uncritical acceptance of



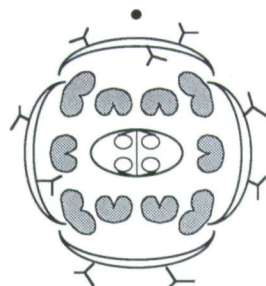




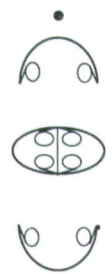
wild type



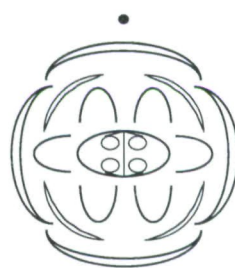
*agamous*



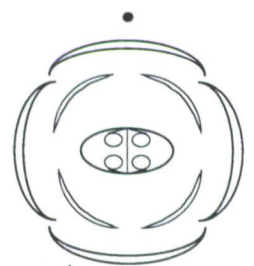
*apetala2-1*



*apetala2-2*



*apetala3*



*pistillata*

**Fig. 2.** Wild-type and homeotic mutants of *Arabidopsis thaliana* (see Bowman *et al.* 1989). In each case the phenotype is illustrated in the left panel while the right panel shows a floral diagram typifying the first flowers on plants raised at 25°C. (A) Wild-type inflorescence of the Landsberg *erecta* laboratory strain. Each flower has four sepals, four petals, six stamens (two lateral and four medial) and a bicarpellary ovary. (B) Inflorescence of an *agamous* plant (mutant S310) in which the stamens are replaced by petals and a new flower arises in place of the gynoecium in a serially repeated pattern. (C) Vertical view of an *apetala2-1* mutant plant. The sepals are leaflike and the petals have some of the properties of stamens. (D) The inflorescence of an extreme *apetala2* mutant, *ap2-2*. Here the two medial sepals are carpelloid and all other organs except the gynoecium are usually absent. (E) An *apetala3* mutant flower in which the petals are replaced by smaller, sepal-like organs and free carpels arise in place of stamens. (F) The *pistillata* phenotype (mutant S244). In this mutation, reduced sepals arise where petals normally occur and stamens are absent.

this point of view was in low repute well before Worsdell published it, as evidenced by Goebel's (1900) statement "We can only consider it as an error to look upon these kinds of malformations as *reversions*..." Nonetheless, there are many subsequent examples.

For instance, numerous uses of this sort of speculation are found in the long debate over the constitution of the pistils in mustard flowers. These pistils contain two chambers, separated by a septum. Since at least 1828 (Lindley, 1828) there has been a debate on the nature of the pistils: some believe them to be made of two carpels (the supposed evolutionary unit structure in the formation of gynoecia, recognizable in some but not all flower types), others believe them to be made of four (or even six). To pick a single example of the unacceptable use of evolutionary interpretation of abnormal flowers in this debate, we cite Saunders' (1923) work on abnormal fruits in the mustard *Matthiola*, which relates the structure of abnormal fruits with four rather than two valves. By assuming that this represents an ancestral form ("...there can be no doubt that in all these cases we are witnessing the reappearance of an ancestral character..."), she claims it as support for a multicarpellate ancestor, and thus as support for the plausibility of the four-carpel model. Arber (1931a,b) specifically criticizes this work, giving a detailed refutation of the type of arguments used by Saunders and calling specious all claims that morphological abnormalities can serve as genealogical data. Despite this, the debate continued for many years, with at least one author (Puri, 1941; 1945) arguing both sides. The assumption that abnormal flowers show ancestral forms is a continuing tradition (Guédès, 1966), and a continuing source of controversy, as detailed by Carlquist (1969), who briefly reviews some of the arguments in which teratological speculation has played a role, stating as a general conclusion that "Data from teratology is not useful in the study of the evolution of flowers."

## Genetics

In contrast to the speculative use of floral abnormalities as a clue to ancestral characteristics, the use of genetic analysis to study both patterns of inheritance and the processes by which morphological evolution occurs is a viable and continuing tradition. Even before the publication of Mendel's work in 1866, it was known that pelorism in *Linaria* flowers is recessive to zygomorphy (Naudin, 1865); before the rediscovery of Mendel in 1900, Godron (1874) and Darwin (1876) studied the inheritance of floral monstrosities. Mutations that cause abnormal flowers were among those whose mode of inheritance was studied after the rediscovery of Mendel's paper as well. These striking and convenient characters were employed in crosses as tests of Mendelian patterns of segregation (Bateson *et al.* 1905; Baur, 1910; Keeble *et al.* 1910; Saunders, 1910; Bateson, 1913; White, 1914). Comprehensive bibliographies of these studies were published by Matsuura (1933) and Warner *et al.* (1934). A similar type of work, though for a different purpose, involved crosses between members of different species, as a way of using Mendelian ratios to infer the number and nature of the individual genetic differences between species with differing flower morphologies. One large research program of this type was started by Shull by 1906 (Shull, 1907), involving crosses of different species of Shepherd's Purse (*Capsella*). This work was summarized by Shull in 1929. He found that some strikingly different forms (such as a type with a 4-chambered ovary and the usual two-chambered type) differed in only a single gene that affected the character under study. A well-known example of work in this tradition is that cited by Beadle (1939) showing a close genetic relation between teosinte and maize, despite the different morphology of their female inflorescences and seeds, thus providing evidence that teosinte could be the direct ancestor of maize. Gottlieb (1984) has recently reviewed the many similar studies that were made in the first half of this century, and encourages a renaissance of this sort of work, since we still do not know how typical it is that marked morphological differences between species result from polymorphism at one or a few genes. All of these genetic studies do, however, show that changes in flower structure and organ differentiation can result from single-gene alterations, and that a wealth of material from old experiments is available for present analysis.

## Floral mutants in the study of development

Despite the long history of the knowledge of heritable abnormalities of flowers, and the long history of detailed studies of the development of normal flowers (Payer, 1857; Sattler, 1973), the study of abnormalities and the study of development have been separate traditions: there are only a handful of experiments in which mutant flowers have been used as a means of understanding the mechanisms by which flowers develop. Indeed, some recent hypotheses for the mechan-

isms by which floral organs appear in appropriate patterns can be seen to be incorrect from teratological evidence. Heslop-Harrison (1963) proposed a relay model for flower development, in which the organs of each whorl differentiate as a result of activation of an organ-specific gene complex. At the same time, this gene complex produces an organ-specific signal, which activates a second gene complex in the next inner whorl; this new gene complex then directs differentiation of the organs of the inner whorl, and produces another specific signal to activate differentiation in the next inner whorl. Green (1988) has proposed a related model, in which the shape changes in the floral primordium created by the development of individual floral organs in an outer whorl or whorls creates localized regions of stress closer to the center of the primordium. The position of these regions specifies the position of the inner organs, and the exact nature of the stress pattern, which depends on the type of organ primordium present in the outer whorls, specifies the type of inner organ that develops. In each of these models, the nature of the organs found in inner floral whorls is determined by the nature of the organs that are differentiating in the adjacent outer whorl or whorls. Thus, if mutant flowers were found in which stamens regularly develop in the positions normally occupied by petals, for example, both models would be shown to be incorrect. Such flowers have been recognized since at least 1821, and mutant plants that regularly produce such flowers have been described many times (Masters, 1869; Worsdell, 1915–6; Dahlgren, 1919; Shull, 1929; De Vlaming *et al.* 1984; Pruitt *et al.* 1987; Komaki *et al.* 1988; Bowman *et al.* 1989). In fact, it is clear from the literature on abnormal flowers that almost any type or number of organs can occupy any whorl, regardless of the character or number of organs in the adjacent whorls (Masters, 1869; Worsdell, 1915–6; Meyer, 1966; Lyndon, 1979a,b; Kinet *et al.* 1985; Bowman *et al.* 1989).

There were earlier authors who understood the significance of floral abnormalities, but lacked the concepts and methods necessary to use such abnormalities for studying the mechanisms of development. Masters (1869) states quite clearly “The term metamorphosis [by which he meant what we now call homeosis], then, really implies an alteration in the organizing force, taking effect at a very early period in the life of the flower, at or before the period when the primitive aggregation of cells, of which it is at that time composed, becomes separated or ‘differentiated’ into the several parts of the flower. In other words, the ‘development’ of the flower pursues a different course from what is usual.” This was echoed by Goebel (1900), who attempted to separate evolutionary and developmental interpretations of abnormal flowers, stating “Our idea of metamorphosis is then primarily an ontogenetic one...”

In fact, Goethe (1790) proposed a theory for the differentiation of different organs in different floral whorls, based in part upon the frequent observations of petals differentiating in positions appropriate for sta-

mens or pistils. His theory was that the sap, as it rises higher in the plant, becomes more refined, and thus induces successive, different floral organs: “...the foliar organs are refined, the operation of the unadulterated saps becomes purer and stronger, and the transformation of the parts is rendered possible”. He considered the abnormal flowers to be “retrograde”, instances of the failure of this mechanism.

More recent plant developmental biologists have also recognized that inherited floral abnormalities may provide important keys to the nature of the gene products that regulate development in flowers (Wardlaw, 1965). Despite this recognition, the use of flower mutations in the study of development has not been a major area in plant developmental biology.

There do exist examples of experimental use of mutant flowers to understand floral development. Brieger (1935) studied the development of several mutant forms of *Primula sinensis* and *P. kewensis*. One mutation studied was *pistilloid* in *P. sinensis*, an X-ray-induced recessive mutation showing a carpelloid development of the primordia that would usually give rise to stamens. Brieger reported that wild-type flowers show a single ring-shaped primordium for both petals and stamens; this later divides into an outer zone that forms petal primordia, and an inner zone that forms the primordial stamens. In the mutant, the two ring zones are separate from the beginning, thus showing that the mutation acts long before the differentiation of any floral organs. Brieger proposes a model for organ specification in which organ-type-specifying ‘hormones’ reside in concentric rings in the flower primordium, and any primordium arising in the zone of a particular hormone differentiates into the organ specified by that hormone. The initial displacement of the stamen primordium toward the center of the flower thus explains its carpelloid development in the mutant. While such a model is unproven, it is noteworthy both because it is similar to a more recently published model for flower development (Holder, 1979), and because it avoids the problems of the models described earlier: it allows for cellular communication in establishment of what in animal embryology would be called embryonic fields, but does not require that differentiating organs communicate.

A more recent study of the development of double petunias (Natarella & Sink, 1971) leads to similar conclusions. In the development of these flowers, numerous extra primordia occur centripetal to the calyx. They differentiate into petals if they arise near the sepals, into stamens if they appear near the center of the flower, and into organs intermediate between petals and stamens if they arise between these positions. Again, a concentric zone model is suggested. That concentric fields of determined cells are not the sole determinant of fate choices in developing flowers is shown by the mutant *blind* of *Petunia hybrida*, which results in a corolla whose much-reduced limb bears supernumerary anthers. This mutation does not affect the time of initiation and location of petal primordia, but rather later stages of differentiation, with the upper

limb developing as anthers and the lower tube differentiating normally, as petals (Vallade *et al.* 1987).

A different sort of flower mutation has led Stebbins and his co-workers to emphasize a different set of processes in flower development. They have studied a number of morphological variants in barley; the best-studied floral one is *hooded* (Stebbins & Yagil, 1966). Flowers from plants bearing this single-gene semidominant mutation show a variable phenotype that in its most extreme form includes two extra rudimentary florets appearing at the apex of the lemma of the basal (normal) flower, with one growing from the other. The first of the extra flowers is upside-down relative both to the basal flower and to second (apical) extra floret. The earliest differences seen in the development of wild-type (awned) and hooded flowers are well after the differentiation of organ primordia has started; patterns of cell division and cell elongation vary. This fact led Stebbins (1986) to propose that plant mutations with morphological pattern phenotypes are generally due to disturbances in cell division patterns, and are mutations in genes whose primary products are either cytoskeletal proteins, or proteins associated with intercellular membranes.

The hooded mutation of barley might be interpreted as a disturbance in the relative timing of developmental events, with a prolonged period of cell division occurring prior to the start of organ differentiation. Another example of the effect of changes in developmental timing on flower development is found in maize (Poethig, 1988). The phenotype of each of three non-allelic, semidominant mutations, *Teopod1*, *Teopod2*, and *Teopod3* includes certain floral organs, particularly in the tassel, becoming larger and more leaflike than in wild type. An analysis of these phenotypes indicates that each mutation prolongs vegetative development, such that the succeeding reproductive developmental program is disturbed. It seems that the floral effects are secondary consequences of an earlier primary effect of each mutation.

The potential involvement of known plant hormones in flower development is revealed by the work of Sawhney & Greyson (1973a,b) on the *stamenless-2* gene of tomatoes. Plants homozygous for this recessive gene have flowers in which the stamens are carpelloid, at times including marginal external ovules. The developing primordia of these organs appear identical to developing stamen primordia in wild-type flowers in their very early development; only after reaching a length of about 100  $\mu\text{m}$  can the differences in the organs be detected. Further studies showed that application of gibberellic acid (at  $10^{-3}\text{ M}$ ) to mutant floral primordia caused the development of the flowers to be almost wild type. This suggests that the mutation may act by interfering with hormone action. However, more recent work (Sawhney, 1983; Sawhney & Polowick, 1986) shows that the *stamenless-2* mutation is temperature-sensitive, and that gibberellin treatment mimics the effects of low temperature in the development of the mutant flower. Thus, it is not clear if the effect of

gibberellin on these plants is specific, or if a variety of different stresses might all have the same effect.

Whether hormones are involved in other morphogenetic mutants of flowers is not known. That mutants in *Arabidopsis* similar to *stamenless-2* in tomato are not influenced by exogenous application of gibberellins or other hormones (Bowman *et al.* 1989) indicates that hormones are not necessarily deficient in all abnormal flowers.

The possibility of involvement of another set of known substances in pattern formation in flower development is demonstrated by recent work on polyamine mutants of tobacco (Malmberg, 1980; Malmberg & McIndoo, 1983, 1984; Malmberg *et al.* 1985; Malmberg & Rose, 1987). These authors selected for tissue culture cells of tobacco that had mutations in the polyamine biosynthetic pathway, and regenerated plants from the mutant cells. Many of the plants were dwarfed, and all had some floral abnormalities, ranging from plants that did not flower, to male or female sterility and homeotic conversions of organs. One phenotype that arose in several independent regenerants was staminoid ovules; other observed abnormalities included petaloid anthers, stigmatic anthers, leaflike sepals, and partial flowers developing within the ovary. Only two of the regenerated lines showed enough fertility in either sex to be analyzed genetically; in both cases the floral abnormality and polyamine lesion segregated as nuclear dominants in the small  $F_1$  generations obtained, and cosegregated in the small numbers of progeny obtained from backcrosses of the  $F_1$  plants to wild type.

While there was a remarkable correlation between floral abnormalities and polyamine biosynthetic lesions in these experiments with tobacco, studies on polyamine levels in four different floral mutants of *Petunia* (Gerats *et al.* 1988) showed no disturbance in polyamine levels in three of the mutants (including green petals, Fig. 1F), and a disturbance in the fourth that could as easily be an effect of the abnormal morphology as a cause. Some normal-flower lines of *Petunia* were also shown to have different polyamine levels. Therefore, not all floral phenotypes result from polyamine lesions, and not all polyamine variation is associated with alterations in floral morphology.

Thus, despite a wealth of available mutations in which floral pattern formation is regularly and profoundly disrupted, there has been little use of abnormal flowers in understanding floral morphogenesis. What work has been done in the past has not answered questions of developmental mechanism. Nonetheless, it has been valuable in providing tests of some theories for mechanisms of flower development, in providing facts on which new general theories may be based, and in indicating the possible involvement of known substances in flower development. Furthermore, the existence of single-gene mutations with profound effects on organ patterns in developing flowers demonstrates that genes exist whose products play a key role in specifying the number and position of organ primordia, and in directing the cells of those primordia to differentiate to appropriate cell types.

### New approaches

The past few years have seen the development of methods for eukaryotic gene analysis at the molecular level, and the means for the molecular cloning of genes about which no more is known than their genetic map position. Recent use of these methods in the cloning of genes whose mutant phenotypes are disruptions of developmental pattern has led to rapid progress in unraveling the mechanisms by which genes direct pattern formation in the developing embryos of *Drosophila* (French *et al.* 1988). These experiments make it clear that the next stage in understanding flower development is to find the products of the genes known to be of critical importance in flower development, and to determine their sites of action, and activities. Current work is thus turning to those plants with extensive existing series of mutations that have profound effects on flower morphology, and from which it is possible to clone genes when no more is known about the genes than their phenotype.

Two methods, each developed initially in micro-organisms, are available for molecular cloning of plant genes when no biochemical information on their products is available. They are transposon tagging and chromosome walking. The former requires mutations in the gene of interest to have been caused by insertion of an active mobile element for which DNA hybridization probes are available. The element, and the adjoining gene, are then recovered from a library of genomic fragments of the mutant line. In the latter method, the DNA of a genetically mapped morphological mutation is recovered by isolation of successive overlapping DNA segments, starting at a nearby cloned DNA fragment. This method is only practicable (at present) in species with small genomes and little dispersed repetitive DNA, whose genetic maps have a large number of closely spaced DNA markers to use as starting points.

The snapdragon, *Antirrhinum majus*, is amenable to the first approach (Martin *et al.* 1985). Three different active transposons are known in this species, Tam1, Tam2 and Tam3 (Coen & Carpenter, 1986). Each was identified through its unstable insertion into genes controlling flower pigment production. When the element leaves the mutant gene, a phenotype approaching wild type may at times be restored. If this happens often during the growth of the corolla, a variegated flower results (Fig. 1A). The DNA sequence of Tam1 and Tam2 shows them to be related, and the elements appear to interact *in vivo* (Hehl *et al.* 1987; Hudson *et al.* 1987). On the other hand, Tam3, which is 1000 times more active at 15°C than at 25°C (Carpenter *et al.* 1987), has a unique DNA sequence and transposition properties (Sommer *et al.* 1985).

Many genes that affect flower structure are known in *Antirrhinum* (Stubbe, 1966). Among these are the multiallelic series at the *cycloidea* locus, the most extreme allele *cycloidea<sup>radialis</sup>* causing the corolla to become peloric (radially symmetrical) (Fig. 1B). Another multiallelic series of mutations, at the *deficiens*

locus, results in changes to the corolla and androecium (Fig. 1C,D). In the extreme mutant *deficiens<sup>globifera</sup>*, the petals become sepaloid and carpelloid organs replace the stamens, with stigmatic tissue and ovules present. If one were to clone these genes and study their products, it might be possible to gain insight into the mechanisms that produce the normal lateral asymmetry of the corolla, and also to learn about the early developmental decisions that control the determination of petal and stamen primordia.

A successful strategy to clone these genes might involve activating Tam elements by growing plants in nonrepressed genetic backgrounds (Hudson *et al.* 1987) or at low temperatures (Tam 3) and screening succeeding generations for new mutations at these loci. These could then be examined for new sites of Tam insertion by DNA probes, and the adjacent plant DNA cloned. It may not be necessary to produce new, Tam-induced alleles at these loci at all if the unstable mutants already known (Stubbe, 1966) have arisen through past Tam insertions (Coen & Carpenter, 1986). Continued instability might well be the consequence of regular somatic loss of the element, as in the case of genes producing variegated pigment patterns.

Many maize genes have already been cloned by transposon tagging with *Ac* and other endogenous transposable elements (Fedoroff *et al.* 1984; O'Reilly *et al.* 1985; Paz-Ares *et al.* 1986; Wienand *et al.* 1986; Cone *et al.* 1986; Schmidt *et al.* 1987); and there do exist mutations affecting development of the maize inflorescences (Coe & Poethig, 1982; Poethig, 1988). For the most part, these seem to have primary effects on general plant growth, however, and only secondary effects on flower development. Examples are the *Teopod* mutations, with general effects on juvenility discussed earlier, and the andromonoecious dwarf mutations, which cause reduced plant height and leaf length while at the same time allowing development of the normally suppressed stamens in ears. There are also maize mutations that affect the degree of branching in tassels, and others that allow development of the ordinarily suppressed pistils in tassel florets, but these mutants do not show the types of profound alterations of cell fate and developmental pattern that would indicate that their products act directly in specifying the early events of flower development.

In contrast to *Antirrhinum* and maize, the *Arabidopsis* genome is not known to contain active transposons that can be used in gene tagging. The prospects for gene tagging in *Arabidopsis* are thus limited to the introduction of heterologous transposons, such as *Ac* from maize (Van Sluys *et al.* 1987), and to insertional mutagenesis by T-DNA from the Ti or Ri plasmids of *Agrobacterium* (Feldmann & Marks, 1987; Feldmann *et al.* 1989). *Arabidopsis* does have the small genome (70 000 kilobase pairs, Leutwiler *et al.* 1984) and low level of interspersed repeat sequences (Pruitt & Meyerowitz, 1986) which aid chromosome walking. Further, a restriction fragment length polymorphism (RFLP) genetic map exists with enough DNA fragments mapped so that more than 50 % of the genome is



within less than 1.9 centimorgans (on average about 250 kilobase pairs) of the mapped DNA fragments (Chang *et al.* 1988). In addition, complementation of mutations by introduction of wild-type genes *via Agrobacterium*-mediated transformation is possible (Meyerowitz, 1987). Thus, available materials provide starting points for chromosome walks, and a means of knowing when the desired gene has been cloned; as well as the possibility for insertional mutagenesis.

A number of *Arabidopsis* mutations whose phenotype is homeotic transformation of the organs in different whorls, loss or gain of organs in different whorls, or abnormal differentiation of organs have been described (McKelvie, 1962; Conrad, 1971; Koornneef *et al.* 1983; Meyerowitz & Pruitt, 1984; Pruitt *et al.* 1987; Meyerowitz, 1987; Haughn & Somerville, 1988; Komaki *et al.* 1988; Bowman *et al.* 1989). The phenotypes of several mutants homozygous for these recessive, single-gene mutations are depicted in Fig. 2. The range of phenotypes is similar to that described for many other species, including double flowers (*agamous*), staminoid petals and leaflike sepals (*apetala2-1*), carpelloid sepals with absence of organs in the petal or stamen whorls (*apetala2-2*), sepaloid petals and carpelloid stamens (*apetala3-1*), sepaloid petals with no differentiation of organs in the stamen whorl (*pistillata*) and extra organs in various whorls (several different *clavata* loci).

The study of these mutations has gone beyond a description of the mutant phenotypes in mature flowers: the development of four genotypes of mutant flowers have been studied with the scanning electron microscope, and the phenotype of most of the possible double mutants have been described (Bowman *et al.* 1989), allowing conclusions on the interactions and roles of the wild-type products of these genes. One clear conclusion is that different single mutations and double mutant combinations can allow the regular differentiation of almost any organ in almost any whorl, and that the differentiation of any organ type is independent of the organ type developing in adjacent whorls. In addition, each of the homeotic flower mutations so far described in *Arabidopsis* affects organ identity or development in two or more adjacent whorls. These observations are consistent with those models for flower development in which genes establish separate identities for concentric rings of cells prior to any overt cellular differentiation in the flower primordium. Furthermore, the time of action of the products of the *apetala2-1* and *apetala3-1* genes has been determined using temperature-sensitive alleles and temperature-shift experiments (Bowman *et al.* 1989), and the times are different, implying that sequential action of several genes is necessary to fully specify the fate of the cells in each ring. The *apetala2* product is necessary only in the earliest stages of flower development, before the appearance of any organ primordia, for petals to develop normally. The temperature-sensitive period of *apetala3-1* is later, at a time when the affected organ primordia are present, but before any differentiation of the cells in the primordia is visible. This shows that the cells of the primordia are not irreversibly determined

until after the number and position of the primordia has been established.

Scanning electron microscope views of these mutants also show that single organs can develop as mosaics of cell types usually found in different floral organs, such that abnormal organs are formed largely from normal cell types in ectopic sites. This emphasizes the point that these sorts of mutations result from cells losing positional information, and not from being unable to differentiate normally. Understanding the role of these genes in the developing flower primordium awaits their molecular cloning, and an analysis of their products.

It is likely that new methods and new mutations will allow similar approaches to the molecular cloning of genes that direct flower development in many species in addition to those already mentioned. Whatever species and whatever means are used to clone these genes, the extension of findings to a wide range of further species with different flower forms will be potentially simple and rapid. Genes with important roles are likely to be found universally, and their isolation by DNA cross-hybridization straightforward. The result may eventually be an understanding both of the molecular mechanisms of flower development, and of the differences in developmental programs that give rise to the varied forms of flowers in different species.

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