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Leaf Morphogenesis in Flowering Plants

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INTRODUCTION

Leaves, or organs derived from leaves, are the most morphologically diverse structures in a plant (Gifford and Foster, 1989). They occur as prominent photosynthetic structures; as inconspicuous fragments of tissue in tubers, rhizomes, and the stems of many cacti; as nonphotosynthetic storage organs in bulbs; and, in carnivorous plants, as complex structures specialized for catching and digesting small animals. Given this natural diversity, it is not surprising that there is some disagreement about what actually constitutes a leaf (Sattler and Rutishauser, 1992; Rutishauser, 1994).

Most leaves have three more or less distinct parts: a leaf base, which may ensheathe the stem; a basal stalk, known as the petiole or rachis; and a distal portion, known as the blade or lamina, which is usually green and flat (Figure 1). Two major types of leaves, simple and compound, are distinguished by the shape of the blade. In a simple leaf, the blade is a unitary structure that may have a relatively smooth outline, or it may be moderately or very highly indented. In a compound leaf, the rachis produces a series of leaflets known as pinnae. Some leaves do not have all of these parts, and the form that each part takes in different species or within a single plant is highly variable.

Several other features are considered characteristic of leaves. Most leaves have dorsoventral asymmetry. That is, the side of the leaf toward the stem (the dorsal or adaxial surface) and the side oriented away from the stem (the ventral or abaxial surface) are morphologically and anatomically different. Second, leaves are determinate structures, meaning that they do not grow indefinitely. Finally, leaves can usually be distinguished from other leaflike structures by the presence of one or more axillary buds at the junction between the base of the leaf and the stem.

It is helpful to divide leaf development into several stages based on the time at which various features of the leaf become determined (Sylvester et al., 1996). During the first stage, the leaf primordium is initiated and acquires its identity as a leaf. During the second stage, the major parts of the leaf become determined and acquire their basic shape, and during the final phase, the histogenesis of the leaf is completed. This review focuses on the first two stages of leaf development and deals primarily with the development of simple leaves. Reviews of leaf development, some of which have been published recently, should be consulted for more detailed summaries of the literature in this field (Cusset, 1986; Tsukaya, 1995; Hall and Langdale, 1996; Smith, 1996; Sylvester et al., 1996).

This paper is dedicated to Ian Sussex, on the occasion of his retirement, in recognition of his many contributions to plant developmental biology.

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LEAF INITIATION

Role of the Shoot Apical Meristem

Leaves arise at regular intervals (termed plastochrons) and in regularly spaced and predictable positions around the periphery of the shoot apical meristem (Figure 2). They are produced by several external layers of cells and emerge from the surface of the meristem either as dorsally flattened bumps or as ridges. After a primordium emerges, it extends laterally by recruiting additional cells from the shoot meristem and may grow to encircle the entire shoot meristem.

Leaves almost always arise in association with a shoot meristem. Nevertheless, the function of the shoot meristem in leaf initiation has never been clearly established, and there is evidence that a shoot meristem is not actually required for leaf initiation. For example, in *Begonia* (Sattler and Maier, 1977) and watercress (Selker and Lyndon, 1996), leaves or leaflike structures can develop in the absence of a shoot meristem. Leaves arising in the absence of a shoot meristem are also observed frequently in tissue culture (Sattler and Maier, 1977). Moreover, in Arabidopsis, mutant alleles of the *PINHEAD* gene block the development of a fully functional shoot meristem but may permit the development of one or more leaf primordia (McConnell and Barton, 1995). Histological analysis of *pinhead* mutants that have produced a single leaf reveals no evidence of a shoot meristem, implying that if a meristem ever existed, it was completely consumed in leaf production.

These observations suggest that although leaf initiation is facilitated by the unique physiology or structure of the shoot meristem, the shoot meristem does not actively direct this process. That is, the meristem may represent a region (or type of tissue) in which leaves can spontaneously self-organize

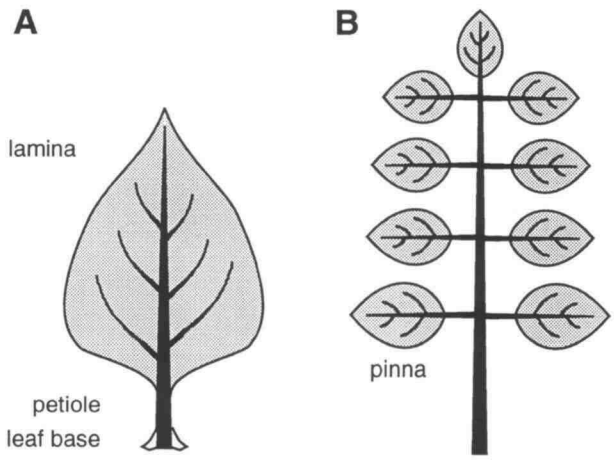

Figure 1. Parts of a Leaf.

(A) Simple leaf.
(B) Compound leaf.

rather than a structural entity that makes leaf primordia; stated another way, one function of the shoot meristem may be to "make tissue that can make leaves" rather than to "make leaves."

Models for Leaf Initiation

Chemical models for organ initiation postulate the existence of an inducer and an inhibitor and usually model the interaction between these factors by one of several reaction–diffusion mechanisms (Meinhardt, 1984; see Nelson and Dengler, 1997, in this issue). These models are very good at generating spatial patterns that resemble the phyllotactic patterns seen in nature. However, because the existence and identity of these hypothetical signaling factors have not been demonstrated, it is not clear how to evaluate the accuracy or usefulness of these models. Nevertheless, it has long been known that leaf and shoot initiation can be induced in a variety of tissues by either a combination of auxin and cytokinin (Skoog and Miller, 1957) or cytokinin alone (Grayburn et al., 1982), and it is not unreasonable to suppose that one or both of these hormones may be important in leaf initiation. In this respect, it is interesting that the Arabidopsis cytokinin-resistant mutant *cyr1* produces few or no leaves (Deikman and Ulrich, 1995).

Two biophysical mechanisms for the regulation of leaf initiation have been proposed by Green. The first model (Green and Lang, 1981) assumes that the key event in leaf initiation is the production of a field of cells in which cellulose microfibrils are in a roughly circular arrangement. This circular array of cellulose microfibrils is believed to prevent cells from expanding laterally, thus forcing the primordium to expand out of the plane of the shoot meristem. In a meristem with preexisting leaves, the site of new leaf primordia is specified by the way in which these preexisting leaves modify the cellulose pattern within the shoot meristem (Green, 1985). More recently, Green and colleagues (Selker et al., 1992; Green et al., 1996) have advanced the idea first proposed by Schüepp (1938) that leaf primordia are formed by the spontaneous buckling of the outer layers of the shoot meristem. This is believed to occur because stresses in the shoot apex (largely generated by preexisting leaf primordia) prevent the excess tissue that is produced at the center of the apex from expanding laterally.

An attractive feature of these biophysical models is that they provide a mechanism both for the spatial positioning of leaf primordia and for leaf morphogenesis. This latter aspect is missing from chemical models, which do not address how a leaf is actually made. On the other hand, there is no evidence that the orientation of cellulose within the shoot apex or in a leaf primordium actually regulates the rate or orientation of cell expansion. Furthermore, whereas the second biophysical model predicts that the leaf primordium is initially subject to compression, most studies have shown that cells in incipient leaf primordia either are under tension or show no evidence of being under mechanical stress (Selker et al., 1992).

Early Events in Leaf Initiation

The earliest histological evidence of leaf initiation is a change in the orientation of cell division both in the epidermis and in internal layers of the shoot meristem (Lyndon, 1970; Tiwari and Green, 1991). At a molecular level, the first

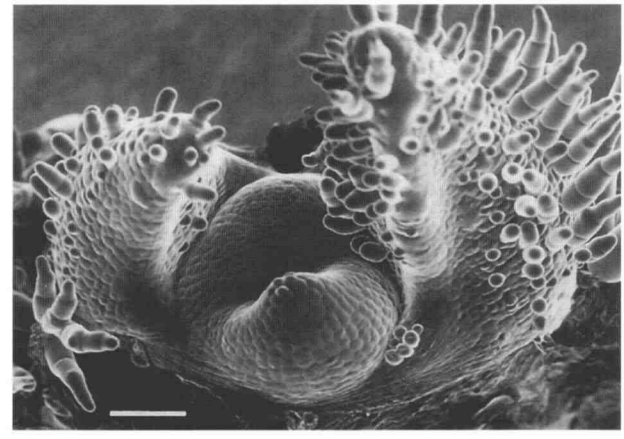

Figure 2. Scanning Electron Micrograph of a Tobacco Shoot Apex with Four Leaf Primordia.

Leaf primordia emerge from the shoot apical meristem as dorsally flattened bumps and develop a lamina three to four plastochrons later. A prominant feature of these young leaf primordia is their epidermal hairs, which develop on the leaf axis before the initiation of the lamina. Bar = 100 μm.

obvious sign of a leaf primordium—at least in maize and Arabidopsis—is a change in the expression pattern of the homeobox gene *KNOTTED1* (*KN1*) and the related Arabidopsis genes, *KNOTTED*-like from *Arabidopsis thaliana* (*KNAT1*) and *SHOOT MERISTEMLESS* (*STM*) (Jackson et al., 1994; Lincoln et al., 1994; Long et al., 1996). These genes (so-called class 1 *knox* genes; Kerstetter et al., 1994) are expressed throughout the shoot meristem but not in leaf primordia, and it has been hypothesized that downregulation of class 1 *knox* genes may be required for leaf initiation (Hake et al., 1995; see Clark, 1997; Kerstetter and Hake, 1997, in this issue).

A prediction of this hypothesis is that the constitutive expression of these genes in the shoot meristem will prevent leaf initiation. However, plants expressing *KN1* or *KNAT1* under the control of the constitutive cauliflower mosaic virus 35S promoter have no obvious defect in leaf initiation, although the leaves produced by these plants are morphologically abnormal (Sinha et al., 1993; Lincoln et al., 1994; Chuck et al., 1996). One possible explanation of this result is that *KN1* is not expressed in leaf primordia of *35S::KN1* plants. Although the distribution of *KN1* mRNA in the primary and axillary meristems of *35S::KN1* transgenic plants has not been described, it is interesting that leaf primordia in ectopic shoot meristems in Arabidopsis plants transformed with *35S::KNAT1* have no detectable *KNAT1* mRNA (Chuck et al., 1996). Whether this is due to the idiosyncrasies of the 35S promoter or, more interestingly, to the post-transcriptional degradation of *KNAT1* mRNA in leaf primordia is not known. In any case, the suppression of *KN1* gene expression is the best marker available for identifying cells in the early stages of leaf initiation in the shoot apical meristem.

Cell Lineages in the Leaf Primordium

Whatever the nature of the factors that initiate leaf production may be, it is clear that these factors operate on a group of cells. Periclinal chimeras (plants in which one of the cell layers in the shoot meristem is genetically different from other layers) demonstrate that a leaf primordium usually originates from at least three cell layers of the shoot meristem in both dicotyledons and monocotyledons (Figure 3; Stewart and Dermen, 1975, 1979). The number of cells recruited to form a leaf primordium in each of these layers can be determined directly, in the case of the epidermis, from time-lapse observations of the surface of the shoot during leaf initiation (Tiwari and Green, 1991) or indirectly, in the case of internal layers, from the size of genetically marked sectors produced just before leaf initiation (Poethig and Sussex, 1985b; Furner and Pumfrey, 1992; Irish and Sussex, 1992; Poethig and Szymkowiak, 1995).

These studies demonstrate that the size of a leaf primordium varies in different species, ranging from approximately five to 10 cells per layer in Arabidopsis (Furner and Pumfrey, 1992; Irish and Sussex, 1992) to somewhere between 50 and 100 cells per layer in tobacco (Poethig and Sussex, 1985b), maize (Poethig and Szymkowiak, 1995), and cotton (Dolan and Poethig, 1997a). They also show that these simple leaves are not derived from an apical cell or group of cells at the apex of the leaf primordium, as is the case in ferns (Bierhorst, 1977). Thus, different longitudinal sections of the leaf arise from different founder cells in the shoot meristem rather than from different cells in the apex of the leaf primordium. As shown in Figure 3B, this is particularly obvious in the lanceolate leaves of monocotyledons.

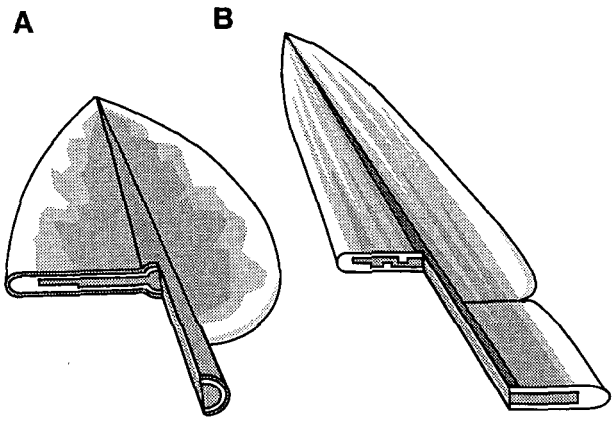

Figure 3. Distribution of Tissue Derived from Different Primary Cell Layers of the Shoot Apical Meristem in a Tobacco Leaf and a Maize Leaf.

(A) A leaf from a green (L1)–white (L2)–green (L3) periclinal tobacco chimera. In tobacco, the L1 lineage is confined to the epidermis. Most of the tissue at the margin of the leaf is derived from the L2 lineage because of periclinal divisions in this layer early in the expansion of the lamina.

(B) A leaf from a white (L1)–green (L2)–green (L3) periclinal maize chimera. In maize, the L1 layer produces all of the tissue at the margin of the leaf, whereas the L3 layer produces little, if any, of the tissue in the lamina (R.S. Poethig, unpublished observations).

PATTERN FORMATION IN THE LEAF PRIMORDIUM

Simple versus Compound Leaves

Current concepts about the way in which leaves are specified are based almost entirely on microsurgical studies of compound leaves, such as those of ferns (Steeves, 1966; Steeves and Sussex, 1989), potato (Sussex, 1955b), or pea (Sachs, 1969). This conceptual framework is often applied to simple leaves, although there is relatively little evidence that this generalization is valid. In fact, recent analyses of the role of *KN1* in leaf development indicate that simple and compound leaves may develop by fundamentally different mechanisms. As noted above, class 1 *knox* genes are expressed

throughout the shoot except in regions that ultimately form leaf primordia (Jackson et al., 1994; Lincoln et al., 1994; Long et al., 1996). Misexpression of *KN1* or *KNAT1* in leaf primordia of maize (Smith et al., 1992; Schneeberger et al., 1995), Arabidopsis (Lincoln et al., 1994; Chuck et al., 1996), and tobacco (Sinha et al., 1993) produces irregularly expanded leaves.

By contrast, constitutive expression of *KN1* in transgenic tomato plants has a completely different effect on leaf morphology (Hareven et al., 1996). Instead of producing reduced leaves, tomato plants transformed with *35S::KN1* have highly ramified leaves with >1000 pinnae. This response is attributable to the compound nature of the wild-type tomato leaf because tomato mutations that produce simple leaves suppress this phenotype. In these simple leaf mutants, ectopic expression of *KN1* has the same effect on the morphology of the lamina as in maize, Arabidopsis, and tobacco. Because class 1 *knox* genes are believed to be important for initiating and maintaining the growth of the shoot apical meristem (Smith et al., 1992; Sinha et al., 1993; Long et al., 1996), this result has been interpreted to mean that compound leaves have a much greater capacity for indeterminate growth than do simple leaves.

The results of experiments on the development of the compound leaves of ferns are consistent with this conclusion (Steeves, 1966; Steeves and Sussex, 1989). In several fern species, leaf primordia that are isolated from the shoot apex by a series of cuts develop as shoots rather than leaves; similar results have been obtained by growing fern leaf primordia in culture. However, there is no conclusive evidence that the primordia of simple leaves can be transformed into shoots or any other type of indeterminate structure by these treatments (Steeves and Sussex, 1989).

Developmental Domains in a Leaf

During its initiation and early development, a leaf primordium divides into several more or less discrete domains along its dorsoventral, centrolateral, and proximodistal axes (Waites and Hudson, 1995; Harper and Freeling, 1996; Lu et al., 1996; Sylvester et al., 1996). The dorsoventral asymmetry of a leaf is apparent as soon as it emerges from the shoot meristem because the adaxial side of a leaf primordium is generally flatter than its abaxial surface. Later in development, the dorsoventral axis of the leaf is defined by the pattern of cellular differentiation. For example, many cell types (e.g., phloem, xylem, palisade mesophyll, trichomes, stomata, and ideoblasts) are located exclusively on the abaxial or adaxial surface of the leaf or are more abundant on one side than the other (see Nelson and Dengler, 1997, in this issue).

The demarcation of the leaf into central and lateral domains is marked by the differentiation of a distinctive band of cells along the lateral margins of the primordium shortly after it emerges from the shoot meristem. Hagemann and Gleissberg (1996) have termed this region of the leaf primordium the "blastozone" because the more usual term "marginal meristem" implies features (e.g., a prolonged or more rapid rate of cell division) that are not observed in all species. Cells in the blastozone expand laterally to form the lamina or several pinnae, whereas the central region of the primordium differentiates into the midrib or rachis.

The proximodistal domains of the leaf are defined by the way in which the blastozone develops (Harper and Freeling, 1996; Sylvester et al., 1996). Along this axis, a leaf can usually be divided into a distal region, which produces a relatively broad lamina, a proximal region (the petiole), in which the lamina is either absent or reduced in size, and a basal region, which extends around the stem to a greater or lesser extent.

Early Determination Events

Microsurgical studies of leaf development demonstrate that the determination of a leaf primordium occurs gradually and is completed after the leaf emerges from the shoot apex. Sussex (1955a, 1955b) transformed a potato leaf primordium into a determinate, radially symmetric structure by making a shallow cut between the primordium and the shoot apex when the primordium was less than ~50 μm in length. The leaves produced by this type of incision were circular in cross-section and had a radially symmetric vascular cylinder. In addition, they lacked a lamina and had no evidence of lateral cellular differentiation. This latter result is significant because it implies that the lateral differentiation of the leaf primordium may be regulated by the same mechanism that determines its dorsoventral polarity.

The phenotype of the *phantastisca* (*phan*) mutation in Antirrhinum provides striking evidence for this conclusion (Waites and Hudson, 1995). In plants that have a strong *phan* phenotype, leaves are completely radially symmetric, all of the cells in the leaf have abaxial identity, and there is no evidence of a lamina primordium. However, leaves with a weak *phan* phenotype have a rudimentary dorsoventral lamina with ectopic outgrowths of lamina tissue. These ectopic bits of lamina arise from the adaxial surface of the leaf and surround patches of abaxialized cells. This phenotype demonstrates that *PHAN* is required for the specification of adaxial identity both in the midrib and in the lamina and suggests that the differentiation of the lamina depends on the juxtaposition of cells with adaxial and abaxial identity (Waites and Hudson, 1995).

Microsurgical analysis of pea leaf development (Sachs, 1969) demonstrates that the central domain of a pea leaf loses its capacity to produce a marginal domain after it is ~30 μm in size. When primordia <30 μm in length are bisected in a saggital plane, each half regenerates a normal leaf. Primordia <30 μm in length are also capable of producing normal leaves when both margins of the primordium are removed. However, removal of the tip or margins of leaf primordia that are between 30 and 70 μm in length results in the loss of pinnae, tendrils, or stipules from the missing part

of the leaf. The character of the marginal structures produced by the leaf primordium is determined at a later stage of development; shallow cuts along the margins of primordia that are ~100 μm in length do not prevent the formation of pinnae but usually transform pinnae primordia into tendrils. In all of these experiments, regions of the leaf that had not been damaged developed normally, demonstrating that different parts of the leaf develop independently once they have become determined.

Although it is still unclear when the proximodistal domains of a leaf become determined, the genetic regulation of pattern formation along this axis has been extensively studied, particularly in maize (Freeling, 1992; Sylvester et al., 1996). In maize, the distal part (blade) and the proximal part (sheath) of the leaf differ in many aspects of their patterns of cellular differentiation (Sylvester et al., 1990). The boundary between the leaf blade and the leaf sheath is marked by a distinctive region known as the auricle, which forms a thin outgrowth (the ligule) on the adaxial side of the leaf.

At least 15 mutations that specifically affect the differentiation of one or all of these structures exist in maize (Freeling, 1992; Sylvester et al., 1996). The *liguleless1* (*lg1*) and *lg2* mutations block the formation of the ligule and auricle and eliminate the sharp boundary between the blade and sheath, suggesting that these genes play an important role in defining the region that gives rise to the auricle and ligule (Harper and Freeling, 1996). Mutations in 10 "ligule polarity" genes have the unusual effects of displacing the blade–sheath boundary and causing ligule-, auricle-, and sheath-like tissues to form in the lamina (Freeling, 1992; Fowler and Freeling, 1996). The three genes in this group that have been cloned (*KN1*, *ROUGH SHEATH1* [*RS1*], and *LG3*) are all related to the class 1 *knox* genes, and in every case the dominant phenotype of mutations in these genes results from their ectopic expression in the leaf blade (Smith et al., 1992; Schneeberger et al., 1995; Sylvester et al., 1996). Although the developmental basis for the phenotype of these dominant mutations is still unresolved (Freeling, 1992; Hake et al., 1995), this phenotype supports the conclusion that the blade and sheath regions of the leaf possess different, genetically regulated developmental identities.

LEAF EXPANSION

Initiation of the Lamina

After a leaf primordium emerges from the shoot apex, it expands laterally, both by recruiting cells from the shoot meristem and by virtue of the expansion of the lateral margins of the primordium (Hagemann and Gleissberg, 1996). In some species, the primordium gradually extends around the entire circumference of the shoot after initiation; in others, the lateral expansion of the primordium may be limited or largely restricted to the distal part of the lamina. Even compound leaves, which do not produce a lamina on the primary axis of the primordium, undergo some degree of lateral expansion. Histological studies and analyses of genetic mosaics demonstrate that this lateral expansion is initiated in a narrow marginal region of the leaf primordium, encompassing several files of cells in each of the three meristematic cell layers (Poethig and Sussex, 1985a, 1985b; Stewart and Dermen, 1975, 1979). The *lam-1* gene in *Nicotiana sylvestris* defines a key function in this process because mutations in this gene produce leaves that not only fail to produce a lamina but have no trace of marginal differentiation (McHale, 1992).

By contrast, in maize the lamina arises directly from the shoot meristem rather than from the lateral expansion of the midrib (Sharman, 1942; Poethig and Szymkowiak, 1995). The *narrow sheath1* (*ns1*) and *ns2* mutations play an important role in this process (Scanlon et al., 1996; Scanlon and Freeling, 1997). In combination, *ns1* and *ns2* produce leaves in which a basal, lateral domain of the leaf is missing. This region of the wild-type leaf is produced by a relatively small population of shoot meristem cells on the side of the meristem opposite the site of leaf initiation (Poethig and Szymkowiak, 1995), which fail to contribute to the expansion of the lamina in *ns1 ns2* plants (Scanlon and Freeling, 1997). Based on the phenotype of *ns1 ns2* plants, Scanlon et al. (1996) have proposed that the maize leaf possesses several discrete lateral domains, the development of each of which is regulated by a different set of genes.

Regulation of Leaf Shape

The expansion of a flattened structure such as the lamina, which occurs to varying extents in two dimensions, is a much more complex process than is the expansion of a cylindrical structure such as the petiole or the young leaf primordium. From a biophysical perspective, the extension of these cylindrical structures requires only that they be constrained from expanding laterally (Green and Lang, 1981). This could be accomplished by transversely aligned cellulose microfibrils, which are known to be important for maintaining the cylindrical shape of internodal cells in organisms such as *Nitella* (Green, 1980).

Some of the questions about leaf expansion that remain to be answered include the following: What keeps cells from expanding or dividing out of the plane of the lamina (as they do, for example, in the production of insect galls or knots)? What regulates the amount of lateral expansion within the plane of the lamina? How is the expansion of cells in different layers of the leaf coordinated so that the leaf remains flat rather than curling adaxially or abaxially? Conversely, what regulates the pattern of cell expansion that leads to the unrolling of leaf primordia in maize and other grasses?

Much of the diversity of leaf shape in nature arises from variation in the amount of expansion within the plane of the lamina. Allometric analyses of leaf expansion demonstrate that this may occur either during the initiation of the lamina or much later, as the lamina expands. For example, Harte

and Meinhard (1979a, 1979b) have shown for Antirrhinum that heteroblastic variation in leaf shape along the length of the shoot is due to variation in the growth of the lamina early in development, whereas mutations that affect the width of the lamina act either during the early phase of leaf development or later. In Arabidopsis (which has leaves similar in shape to those of Antirrhinum), the *angustifolia* and *rotundifolia3* mutations act after lamina initiation to control the polarity of leaf expansion (Tsuge et al., 1996). In cotton, the *Okra* mutation acts during lamina initiation to accelerate the growth of lobes relative to the growth of sinuses but does not affect the relative growth rates of these regions later in leaf development (Dolan and Poethig, 1991). By contrast, genes that affect leaf shape in *Tropaeolum majus* act after the initiation of the lamina to affect the rate of leaf expansion. Primordia of wild-type *Tropaeolum* leaves have well-defined lobes and sinuses, but at maturity the leaf is circular because the growth rate of the sinuses exceeds that of the lobes during leaf expansion (Whaley and Whaley, 1942). These examples demonstrate that the shape of the lamina arises from a variety of growth patterns, suggesting that the regulation of this process is quite complex.

Explanations for these and other patterns of leaf expansion have often been sought in the pattern of cell division. Detailed analyses of the rate, orientation, and duration of cell expansion and cell division in several species (Fuchs, 1975a, 1975b; Jeune, 1983; Poethig and Sussex, 1985a, 1985b) have revealed significant regional variation in all of these parameters during leaf expansion, some of which can be correlated with regional differences in the rate or orientation of growth. Despite its classical reputation as a meristematic region, the rate of cell division has actually been found to be significantly lower at the leaf margin than in adjacent internal tissue (reviewed in Cusset, 1986). The rate and orientation of cell division also vary along the proximodistal axis of the lamina, during the formation of lobes, and at different times in leaf expansion. For example, in both maize and tobacco, the rate of cell division is significantly higher and the orientation of cell division is less polarized near the base of the leaf than in other parts of the lamina (Poethig and Sussex, 1985a, 1985b; Poethig and Szymkowiak, 1995).

By contrast, evidence from comparative morphology (Cooke and Lu, 1992; Hagemann, 1992; Kaplan, 1992), cell lineage studies (Poethig and Sussex, 1985b), and experiments with γ-irradiated plants (in which leaf expansion occurs in the absence of cell division [Haber and Foard, 1963; Foard, 1971]), has led to the hypothesis that the expansion of the lamina is regulated by factors that operate non–cell autonomously to control growth, without regard to the orientation or rate of cell division (reviewed in Smith, 1996). Recent support for this hypothesis is provided by the phenotype of the *tangled* mutation in maize, which specifically blocks longitudinal anticlinal divisions during leaf expansion but has relatively little effect on the growth or final morphology of the leaf (Smith and Hake, 1996). Similarly, Hemerly et al. (1995) concluded that leaf morphogenesis is regulated independently of the rate of cell division because dominant negative mutations in cdc2 kinase, a cell-cycle regulator, decrease the growth rate and size of tobacco leaves but have relatively little effect on leaf shape. Although these latter two studies did not address the issue of whether regional variation in the orientation or rate of cell division still exists in these mutants, they are consistent with a large body of evidence suggesting that patterns of cell division do not play a primary role in regulating the morphogenesis of multicellular structures in plants (see Jacobs, 1997, in this issue).

Nevertheless, it is difficult to ignore the fact that patterns of cell division are highly regulated in particular regions of the leaf (e.g., at the leaf margin) and at particular times in development. Meyerowitz (1996, 1997) has proposed that this paradox could be resolved if there was short-range communication between cells that allowed them to adjust to differences in the activity of particular cells in a group. Variation in the orientation and rate of cell division in some cells would be compensated by an appropriate change in the cell division pattern of other nearby cells. This hypothesis implies that rather than being regulated independently of cell division, morphogenesis actually depends on highly regulated, albeit variable, patterns of cell division.

One tissue in which the orientation of cell division is highly regulated is the epidermis. Periclinal chimeras demonstrate that cells in this tissue layer divide almost exclusively in an anticlinal plane, except at the leaf margin of the linear leaves of monocotyledons (Figure 3; Stewart and Dermen, 1975, 1979; Poethig and Szymkowiak, 1995). Cell division in internal layers of the leaf also tends to be in an anticlinal plane, except at the margin of the lamina. This aspect of leaf development raises the question of whether one or another tissue layer of the leaf regulates leaf expansion. Periclinal and mericlinal chimeras in which the L1, L2, and L3 layers of the shoot are derived from species with different leaf shapes routinely demonstrate that the L2 layer of shoot meristem plays a major role in the determination of leaf shape, although the epidermis may have some effect on leaf shape as well (Szymkowiak and Sussex, 1996). The role of these tissue layers in leaf shape has also been investigated by taking advantage of mutations that affect leaf shape (Dolan and Poethig, 1991, 1997b). In cotton, the presence of the *Okra* mutation in any layer of the lamina has an effect on leaf expansion (Dolan and Poethig, 1997b). The magnitude of this effect is different for different layers, but it is remarkable that even when *Okra* tissue is present in only one layer of the lamina (abaxial or adaxial), it has a significant effect on leaf expansion. This result suggests that the epidermis may play a larger role in leaf expansion than had previously been suspected.

HETEROBLASTY AND REGULATION OF LEAF IDENTITY

Plants produce several different types of leaves and leaflike organs during their development (Allsopp, 1967; Poethig,

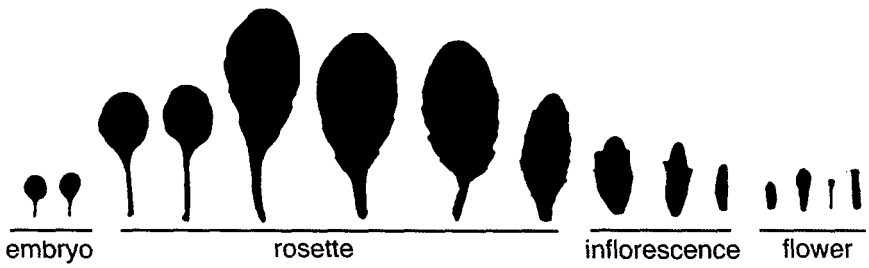

Figure 4. Sucessive Leaves and Leaflike Organs in an Arabidopsis Plant (ecotype Wassilewskjia).

Organs produced at different nodes on the shoot—and therefore at different times in development—are different. Cotyledons are similar to each other, as are the first two leaves, presumably because members of these pairs of organs are initiated at essentially the same time and at the same nodal position. Organs are shown life size, except for floral organs, which are shown at magnification ×1.6.

1990). During seed development, the embryo produces one or two cotyledons—small, relatively simple leaflike organs that accumulate large amounts of nutrients that are used by the germinating seedling. The first few leaves produced by the shoot meristem after germination (juvenile leaves) are usually also smaller, simpler, and anatomically and biochemically different from leaves produced later in development (adult leaves); leaves produced during the development of the inflorescence (bracts) also have a distinctive morphology and pattern of cellular differentiation (Figure 4).

Some of these differences are regular features of shoot development and are components of a genetically regulated program of shoot maturation, whereas other features are more plastic and are controlled by the physiological status of the plant and a variety of environmental factors (Allsopp, 1967). In maize, for example, epicuticular wax, epidermal hairs, epidermal cell shape, and cuticle thickness are developmentally regulated traits that change in concert at the same time in shoot development (Lawson and Poethig, 1995). Many other aspects of leaf anatomy (e.g., epidermal and bundle sheath cell size, interveinal distance, and the ratio of mesophyll area to vascular bundle area) change in a more complex fashion from leaf to leaf along the shoot. These features are likely to be regulated by many endogenous and environmental factors (Bongard-Pierce et al., 1996).

Aquatic plants, which produce different types of leaves in submerged and aerial environments, provide an extraordinary example of the developmental plasticity of the leaf (Goliber and Feldman, 1990; Bruni et al., 1995). In these species, the primordia of aerial and submerged leaves are initially identical and can be induced to develop as either an aerial or a submerged leaf by a variety of factors. In general, the development of any particular trait can be changed until just before a leaf type–specific difference in the trait appears. Thus, major features of leaf shape (e.g., lobe number) are determined earlier in development than are traits such as epidermal cell shape, stomatal density, and the differentiation of mesophyll cells. In fact, in *Hippuris vulgaris*, leaf primordia do not become completely determined until they are one-half of their final size (Goliber and Feldman, 1990). Similar results have been obtained by Battey and Lyndon (1988) with *Impatiens balsamina*, in which switching shoots from floral inductive to noninductive conditions leads to the production of leaves that have petal-like features. We have found that the basal region of an adult maize leaf can be induced to revert to a juvenile pattern of development as late as six plastochrons after the leaf is initiated (H. Passas and R.S. Poethig, unpublished results).

Goethe (1790) proposed that all leaflike organs, including the parts of a flower, arise from transformations of a single type of organ—a leaf. Recent and striking evidence for this hypothesis is provided by the phenotype of Arabidopsis flowers that contain loss-of-function mutations in three genes that regulate the A, B, and C functions required for floral organ identity. In these triple mutant plants, all four types of floral organs develop as leaves (Bowman et al., 1991). Evidence that cotyledons are also fundamentally leaves is provided by three mutations in Arabidopsis, *leafy cotyledon1* (*lec1*), *lec2*, and *fusca3*, that transform cotyledons into leaves (Keith et al., 1994; Meinke et al., 1994; West et al., 1994).

These results raise an obvious set of questions: What is the default state of a juvenile leaf, an adult leaf, or a bract? Does one of these conditions represent a fundamental program of leaf morphogenesis, or are these organs derived from a more basic developmental program? The leaflike organs in the $A^-B^-C^-$ floral mutants and the *lec* mutants do not provide an answer to this question because the characteristics of the transformed organs in these mutants differ: leaflike organs in *lec* mutants have the trichome distribution pattern of juvenile leaves, whereas the leafy organs in flowers resemble bracts (Telfer et al., 1997; A. Telfer and R.S. Poethig, unpublished observations).

Insight into the genetic regulation of juvenile and adult leaf identity in maize has come from the phenotypic and molecular analysis of *GLOSSY15* (*GL15*). Loss-of-function mutations in this gene partially transform the epidermis of juvenile leaves into an adult epidermis (Evans et al., 1994; Moose and Sisco, 1994), demonstrating that *GL15* functions both

to promote juvenile epidermal identity and to repress an adult program of epidermal differentiation. This is reminiscent of the function of the *APETALA2* and *AGAMOUS* floral homeotic genes in Arabidopsis, which are required both to promote the identity of the outer and inner two whorls of floral organs, respectively, and to repress the pattern of differentiation typical of the adjacent pair of whorls (Coen and Meyerowitz, 1991; Weigel and Meyerowitz, 1994). In this regard, it is interesting that *GL15* contains a region that is closely related to a sequence in *APETALA2,* termed the *AP2* domain (Moose and Sisco, 1996). Although it is tempting to conclude from the phenotype of *gl15* mutations that the adult pattern of epidermal differentiation is the ground state in maize, this conclusion is premature. It may well be that juvenile and adult leaf identities are regulated in the same way that floral organ identity is determined, namely, by juvenile and adult identity genes that mutually repress each other's expression and that act by modifying a more basal program of leaf morphogenesis (Weigel and Meyerowitz, 1994).

PROBLEMS AND PROSPECTS

The experimental phase of plant developmental biology began in the 1940s with an attack on pattern formation in the shoot meristem and the mechanism of leaf morphogenesis (reviewed in Wardlaw, 1968). Although progress in defining the mechanism of leaf morphogenesis has been slow, the recent increase in interest in leaf development is already yielding significant new insights. A major problem in this field is the fact that leaf development has been studied in many different species; therefore, it is difficult to use the results from previous studies as a foundation for research on more genetically and molecularly tractable systems. Furthermore, analysis of one of the most important sources of variation in leaf morphology—the expansion of the lamina—is complicated by the fact that this morphogenetic process likely depends on a large number of factors, many of which function in general processes of cell physiology. Nevertheless, recent technical advances and the development of sophisticated genetic systems will provide opportunities to examine leaf morphogenesis in new ways and will make it possible finally to answer the question: How are leaves made?

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