The Plant Cell, Vol. 22: 1019–1032, April 2010, www.plantcell.org © 2010 American Society of Plant Biologists

REVIEW

Morphogenesis of Simple and Compound Leaves: A Critical Review

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The leaves of seed plants evolved from a primitive shoot system and are generated as determinate dorsiventral appendages at the flanks of radial indeterminate shoots. The remarkable variation of leaves has remained a constant source of fascination, and their developmental versatility has provided an advantageous platform to study genetic regulation of subtle, and sometimes transient, morphological changes. Here, we describe how eudicot plants recruited conserved shoot meristematic factors to regulate growth of the basic simple leaf blade and how subsets of these factors are subsequently re-employed to promote and maintain further organogenic potential. By comparing tractable genetic programs of species with different leaf types and evaluating the pros and cons of phylogenetic experimental procedures, we suggest that simple and compound leaves, and, by the same token, leaflets and serrations, are regulated by distinct ontogenetic programs. Finally, florigen, in its capacity as a general growth regulator, is presented as a new upper-tier systemic modulator in the patterning of compound leaves.

THE LEAVES OF FLOWERING PLANTS: THEIR ROLE, CONSTITUENTS, AND POSSIBLE ORIGIN

Leaves are the major photosynthetic organs of flowering plants and serve as their prime mediator with the environment above the soil surface. Leaves arise at the flank of the shoot apical meristem (SAM) and feature determinate growth. In eudicots, the primordium initially acquires a peg-like structure, whereas in grasses, it is broader and acquires a half-ring shape. Final leaf form is defined by dorsiventral (DV), mediolateral (ML), and proximodistal (PD) polarities. Leaves are composed of a terminal flat unit called a blade (also called a lamina) that is typically supported by a narrow petiole (see Figure 1A for prototype leaves). In monocots, the petiole is broad and extended in a characteristic sheathing base, and its precise distinction from the blade is debated. The eudicot blade generally features a reticulate vasculature with a central longitudinal midvein, whereas parallel vein systems characterize the monocot leaf. Although leaves vary dramatically in size and shape, they are traditionally divided into two major morphogenetic classes: simple and compound. Simple leaves have a single flat blade (synonymous here with the lamina), the margins of which are continuous and may be smooth (entire), lobed, or serrated. In typical compound leaves of eudicot plants, the blades are composed of several regularly spaced sessile or petiolated appendages called leaflets that are attached to a central rachis. Leaflets arise in two basic organizations called pinnate and palmate (Figure 1A), and in some plants, such as legumes, appendages of compound leaves may take the form of thread-like tendrils. A comprehensive glossary of leaf forms and the nomenclature of leaf elaborations can be found at http://en.wikipedia.org/wiki/Leaf_shape.

Available paleobotanical evidence suggests that the leaves of seed plants developed from a radial indeterminate branched-shoot system and were thus compound (Zimmermann, 1952), but the nature of the events leading to determinate laminar organs is debated (Sanders et al., 2009). By contrast, the first recorded angiosperm leaves were simple and entire, suggesting that compound leaves reappeared independently several times in different taxa, some of which may have evolved back to simple leaves (Taylor and Hickey, 1996; Doyle and Endress, 2000; Bharathan et al., 2002). The origin of leaves from branched indeterminate shoot systems and the interconversion of simple and compound forms (first in basal plants and then within flowering plants) have impacted the diversity of mechanisms regulating simple and compound leaves, as we discuss below.

As leaf morphology and ontogeny are variable, our description and discussion will focus on eudicot leaves, where most studies related to simple and compound architecture have been performed. We start with the genetic regulation of the basic morphogenetic events leading to the formation of leaves in flowering plants and then explore the different ways in which plants have rewired general meristematic programs to form complex leaf structures.

LEAF INITIATION AND THE ESTABLISHMENT OF LEAF AXES

The initial developmental program of all leaf types is fairly stereotypical. First, a group of cells at the flanks of the SAM, 50 to 100 cells based on clonal analysis (Figures 1B and 1C;

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www.plantcell.org/cgi/doi/10.1105/tpc.109.073601

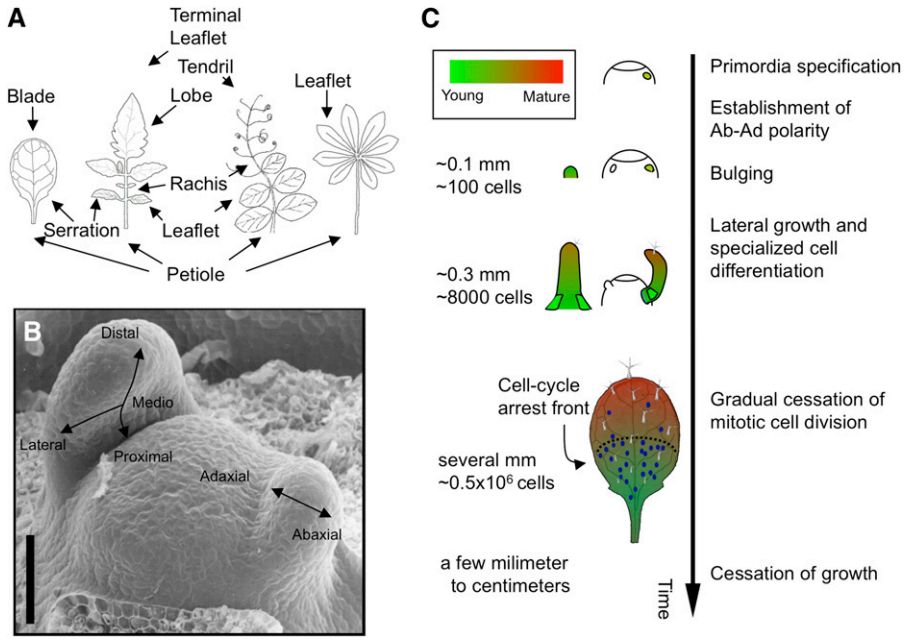

Figure 1. Morphology and Ontogeny of the Leaf.

(A) Different types of leaves and their parts. From left: the simple leaf of *Arabidopsis*, the pinnately compound tomato and pea, and the palmate leaf of *Lupinus perennis*. All leaves have a basal petiole and a distal domain made of continuous or separated laminae units.
(B) SAM of *Petunia*, showing developing leaf primordia. Arrows mark the three axes of leaf asymmetry. Bar = 50 μm.
(C) Stages in the ontogeny of a simple leaf. Color gradient indicates the maturation state of the leaf cells. Rough estimates of size and number of cells are typical of leaves of *Arabidopsis* and tobacco. Light-green (immature) polygons mark the region of slow maturation associated with the marginal blastozone.
(*Photograph in **[B]** courtesy of John Alvarez.*)

Poethig and Sussex, 1985a; Dolan and Poethig, 1998), is fated to form the leaf primordium in a species-specific pattern of phyllotaxis, marked by local auxin maxima (reviewed in Braybrook and Kuhlemeier, 2010). Concurrently, the peg-like primordium is partitioned into the dorsal (adaxial) and ventral (abaxial) domains (Figures 1B and 1C). The early establishment of this asymmetry is a prerequisite for the subsequent formation of a lamina (reviewed in Bowman et al., 2002), and it has been suggested that the juxtaposition of the abaxial and adaxial leaf domains in fact initiates lamina formation (Waites and Hudson, 1995).

The developmental origin of early leaf polarity was proposed by Sussex (1955) to be an instructive signal from the SAM because surgically separated leaf primordia of potato (*Solanum tuberosum*) produced unifacial radial organs. Sachs (1969), however, showed that wounded flat leaflet primordia of pea (*Pisum sativum*) can give rise to tendrils and suggested that wounding per se may induce the formation of radial centric organs. A more encompassing model by Hagemann and Gleissberg (1996), effectively a plant version of the prepattern paradigm (Stern and Tokunaga, 1967), suggests that the seemingly radial leaf primordium is intrinsically dorsiventral from the outset. In those authors' own words, "dorsiventrality is imposed on the leaf primordium from the beginning by its position within the longitudinal gradient of the shoot apex" (Hagemann and Gleissberg, 1996). Accordingly, it is impossible for a lateral leaf appendage to be primarily radial sensu shoot. The apparent symmetry of some unifacial monocot leaves (Kaplan, 1975) is indeed the result of secondary morphogenetic elaborations, and using the criteria of relative positioning of vascular elements, pea tendrils are not radially symmetric (Tattersall et al., 2005; J. Hofer and D. DeMason, personal communication). Likewise, the centric organs formed in response to surgical manipulation appear abaxialized and do not have a shoot-like vascular organization (Steeves and Sussex, 1989).

Once asymmetry has been established, the primordium undergoes elongation and partitioning into a proximal petiole, which is used only to connect the future blade with the stem and a distal generative rachis. The initial partitioning of the primordia is followed by concurrent initiation of blade expansion to form, in leaves such as those of tobacco (*Nicotiana tabacum*), a distal terminal lamina (Figures 1B and 1C; Poethig and Sussex, 1985b). Additional appendages such as leaflets subsequently may be formed along the generative rachis and the primary blade (Figure 1C).

LEAF MATURATION

Leaf maturation follows a conserved schedule and requires local and transient activities of different meristem types. The transition from a uniform peg-like primordium into an elaborated leaf involves a dynamic differential maturation process as evident

by morphological and anatomical hallmarks. The term maturation will be used herein to describe the normal, directional, and unperturbed differentiation process from primordium initiation to senescence. In its early stages, 12 to 48 h after initiation, the primordium is composed of histologically uniform dividing cells (Lyndon, 1998), and subsequent gradual changes in cell morphology are a hallmark of differentiation, otherwise referred to as gradual loss of developmental potentials. The maturation in many leaves progresses from the distal tip basally (Figure 1C) as indicated by the sequential appearance of morphogenetic markers: trichomes (Avery, 1933; Poethig and Sussex, 1985b), provascular strands (Aloni, 1987), enlarged epidermal cells (Hagemann and Gleissberg, 1996), modified cellular morphology, and differentiating guard cells (Bergmann and Sack, 2007). The gradual changes are also apparent in the extent of developmental potential in the developing leaves, determined, for example, by regeneration capacity of the primordium following injury (Sachs, 1969; Sena et al., 2009). These maturation processes are also reflected molecularly in temporally dynamic changes in >50% of the transcriptome of the growing blades (Schmid et al., 2005), thereby serving as a molecular signature that predicts the maturation state of leaf samples (Efroni et al., 2008).

Growth of the leaf involves oriented cell division throughout the blade, in what has been termed the plate meristem (Esau, 1977). Clonal analyses in simple cotton (*Gossypium hirsutum*) and tobacco leaves have revealed both shared and species-specific cell division patterns, although their relevance to the morphological variation between the species remained unclear (Poethig and Sussex, 1985a; Dolan and Poethig, 1998). Similar analysis of *Antirrhinum majus* petals suggested that it is the orientation of cell divisions, signaled by as yet unknown long-range messages, rather than their rate of division that determines differential growth (Rolland-Lagan et al., 2003). Yet, interestingly, application of EXPANSIN, a cell wall–loosening enzyme, causes local coordinated growth in tobacco leaves, suggesting at least some growth feedback can act locally in expanding leaves (Pien et al., 2001). When the blades of tobacco or *Arabidopsis thaliana* reach ~10% of their final size, mitotic divisions gradually cease in a basipetal wave (Poethig and Sussex, 1985b; Donnelly et al., 1999; Nath et al., 2003) and lamina growth proceeds via cell expansion that is frequently associated with endoreduplication cycles (Dale, 1988). Growth rates along the blade are modified with age, peaking in expanding primordia. Growth can last from days to weeks, and at many stages can pause or be induced, indicating that active growth arrest is as significant as growth promotion in regulation of leaf final size (Sachs, 2006).

How the final size of leaves, or in fact any organ, is determined is poorly understood (Day and Lawrence, 2000). Leaf size is reproducible under constant conditions but is also highly responsive to environmental signals, as shown for *Epipremnum aureum* and *Arabidopsis* (Figures 2A and 2B). Often, the overall number of cells in a given leaf correlates well with leaf size. Nevertheless, experimentally increasing, decreasing, or even halting mitotic cell activity results in normally sized leaves due to compensatory cell sizing. Two contrasting theories describe the relationships between cells and organs: the cellular theory asserts that the organ is the sum of its cells (reviewed in Tsukaya, 2003), whereas the organismal theory posits that cells act merely as tiles, responding to shape cues determined on an organismal level (Kaplan, 1992). At this point, conflicting evidence exists for both theories, and none provides a coherent model for shape and size determination. Regardless, and despite its morphological plasticity, leaf form is determined primarily by its genetic composition; some details of which will be described next.

MERISTEMATIC FACTORS REGULATING LEAF GROWTH AND FORM

The PHB-KAN Module

Lamina initiation is triggered by the establishment of leaf dorsiventrality, the genetics of which was first addressed in *Antirrhinum*, where mutations in *PHANTASTICA* abolished adaxial cell types and inhibited laminar growth (Table 1; Waites and Hudson, 1995). In *Arabidopsis*, the antagonistic function of two gene families, *PHABULOSA* (*PHB*)*-like* and *KANADI* (*KAN*), appears to underlie the gradual partitioning of the primordium into anatomically distinct abaxial and adaxial domains along the DV axis, respectively (Eshed et al., 2001; McConnell et al., 2001). The redundant *PHB-like* adaxial factors are expressed in the SAM and the adaxial domain of the leaf primordia (McConnell et al., 2001; Emery et al., 2003; Prigge et al., 2005), and when inactivated, only radial abaxialized cotyledons develop (Emery et al., 2003). Also, when *PHB* factors are excluded specifically from the leaf primordia via ectopic expression of *miR165/6*, miniature radial leaves are formed (Alvarez et al., 2006). Conversely, loss of *KAN* results in expansion of the adaxial domain, while its ectopic expression induces abaxialized radial leaves (Eshed et al., 2001, 2004). *PHBs* and *KANs* are the only known factors that are both required and, in a wild-type context, sufficient for specifying the two leaf domains (Chitwood et al., 2007; Bowman and Floyd, 2008). Their functions thus support the idea that polarity in the primordial stage is a prerequisite for lamina initiation and growth (Waites and Hudson, 1995), linking DV with ML elaboration. The polar expression of YABBY genes along the DV axis of the primordioum in *Arabidopsis*, *Antirrhinum*, and maize (*Zea mays*) and their requirement for lamina growth (Eshed et al., 2004; Golz et al., 2004; Juarez et al., 2004) further links the DV and ML axes. The molecular basis for PD primordial partitioning is poorly understood, and thus far, the distal activation of *STYLISH* genes (Kuusk et al., 2002), potent inducers of auxin biosynthesis genes (Sohlberg et al., 2006), serves as the earliest marker of this process. Importantly, it is not known when, and in what order, the three leaf axes are determined, and the significance of the sequence of these events for different leaf forms is not clear.

The KNOX-ARP Module

The Class I *KNOX* genes were the first homeodomain factors identified in plants (Vollbrecht et al., 1991; Freeling, 1992). The dominant gain-of-function *Knotted1* (*KN1*) mutation induces knot-like outgrowths on maize leaf blades. *KNOX* genes are normally expressed in apical meristems of all tested seed plants,

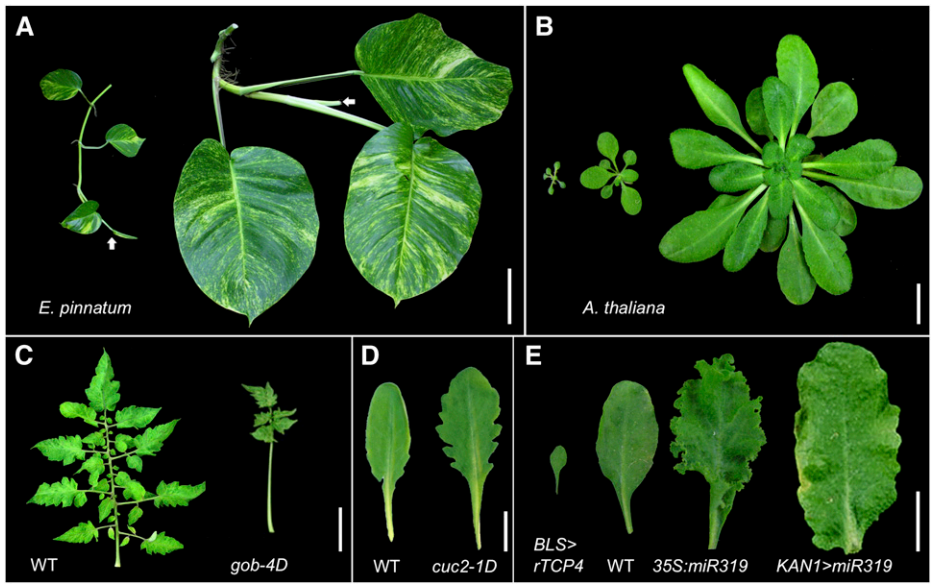

Figure 2. Environmental and Genetic Regulation of Leaf Size.

(A) Different branches of a single *Epipremnum pinnatum* plant. Left branch with small leaves was hanging, while right branch was wrapped around a supporting pole.

(B) Wild-type *Arabidopsis* plants grown under three environmental conditions natural for this species: agar plates (left), pots under long days (middle), and pots under short days (right) photographed together.

(C) and **(D)** The molecularly similar mutations *gob-4D* and *cuc2-1D* produce opposite effects on leaf size in tomato (Berger et al., 2009) **(C)** and *Arabidopsis* (Larue et al., 2009) **(D)**, respectively.

(E) Altering the levels of the CIN-TCP proteins can have dramatic effects on leaf shape and size (Efroni et al., 2008). Shown are leaves overexpressing a miR319-insensitive version of TCP4 (*BLS>rTCP4*) or overexpressing *miR319* that negatively regulate five CIN-TCPs (*35S:miR319* and *KAN1>miR319*). Note the large effect of the microRNA activated with the *KAN* promoter (right), which is transiently active during early stages of leaf primodium development.

Bars = 10 cm in **(A)**, 1 cm in **(B)**, **(D)**, and **(E)**, and 5 cm in **(C)**. (*Photographs in **[A]** and **[B]** are courtesy of I.E. **[C]** is reprinted from Berger et al. [2009]. **[D]** is courtesy of Clayton Larue, and **[E]** is reprinted from Efroni et al. [2008].*)

and some members of the family, such as *Arabidopsis STM*, protect the SAM from precocious termination (Long et al., 1996). While in some species *KNOX* expression is limited to the apical meristems, in others it is expressed in leaves as well (Hareven et al., 1996; Koltai and Bird, 2000; Bharathan et al., 2002). In all species, however, *KNOX* expression is excluded from the incipient leaf sites and all leaves examined are highly responsive to ectopic KNOX activity. *KNOX* genes exert some functions via interactions with BELL-like homeobox proteins (Cole et al., 2006). This interaction can be outcompeted by mini-KNOX proteins, such as the tomato (*Solanum lycopersicum*) TKD1/PTS, which lack a homeobox but can still interact with a range of BELL-like homeobox proteins (Ron, 2001; Kimura et al., 2008; Magnani and Hake, 2008). In addition, *KNOX* genes have been shown to modify hormonal balances by negatively regulating gibberellin (GA) biosynthesis genes (Sakamoto et al., 2001; Bolduc and Hake, 2009) or by promoting cytokinin biosynthesis (Jasinski et al., 2005; Yanai et al., 2005).

KNOX expression was initially studied in species in which it is excluded from the leaves (maize, *Arabidopsis*, and *Antirrhinum*), thus permitting the identification of genes that negatively regulate its expression in those organs. Prominent among these are mutations in genes encoding the orthologous *MYB* factors *ASYMMETRIC LEAVES1* (*AS1*) in *Arabidopsis*, *ROUGH SHEATH2* in maize, and *PHANTASTICA* in *Antirrhinum* (together known as ARP factors) that result in some *KNOX* genes being expressed in the leaf. But because in such cases *KNOX* expression is still excluded from early primordial sites, it was suggested that ARP factors act directly or indirectly to maintain KNOX genes in an off state (Timmermans et al., 1999). Notably, whereas *Antirrhinum PHAN* negatively regulates *STM* (Tsiantis et al., 1999), *Arabidopsis AS1* regulates other *KNOX* genes but not *STM* (Byrne et al., 2000; Ori et al., 2000). *ARP* genes are expressed in the leaf primordia, and in some species, are excluded from the SAM where *KNOX* genes are expressed. In other species, however, both *ARP* and *KNOX* are found in the same cells (Koltai and Bird, 2000; Kim et al., 2003), and *as1* mutants can suppress meristematic defects of *stm* (Schneeberger et al., 1998; Byrne et al., 2002). Thus, it is possible that both *KNOX* and *ARP* genes regulate meristematic functions in SAMs and in leaf primordia depending on the specific requirements of a given organ and a given species. Given that there is considerable divergence in noncoding regulatory regions as well as protein-coding sequences associated with these genes, there are ample opportunities to achieve such diverse roles and patterns of expression (Hay and Tsiantis, 2006).

Table 1. Genes Discussed in This Review

Gene/Family	Antagonist	Protein	*Arabidopsis/Cardamine*	Tomato	Pea	*Antirrhinum*
KNOX	*BELL-like homeobox*	Homeodomain transcription factor	*SHOOT MERISTEMLESS*	*TKN2*	*KNOX*	*STM*
CIN-TCP	*miR319*	bHLH-like transcription factor	*TCP4*	*LANCEOLATE*		*CINCINNATA*
NAM	*miR164*	Transcription factor	*CUP-SHAPED COTYLEDON*	*GOBLET*	*NAM*	*CUPULIFORMIS*
ARP		MYB transcription factor	*AS1*	*SIPHAN*	*CRISPA*	*PHANTASTICA*
LFY		Transcription factor	*LEAFY*	*FALSIFLORA*	*UNIFOLIATA*	*FLORICAULA*
UFO		F-box	*UNUSUAL FLORAL ORGANS*	*ANANNTHA*	*STAMINA PISTILLOIDA*	*FIMBRIATA*
Florigen precursor	*SP/TFL1*	Cofactor in signaling complexes	*FLOWERING LOCUS T*	*SINGLE FLOWER TRUSS*		

Global and Local Regulation of Leaf Form

Shortly after leaf initiation members of the class II TCP family are expressed throughout the primordium. Mutations in *CINCINNATA*, the first described *CIN-TCP*, resulted in excessive cell proliferation in *Antirrhinum* leaf margins, producing curled leaves (Nath et al., 2003). Leaf buckling and lobed margins are also conditioned by multiple losses of *CIN-TCP* genes in *Arabidopsis* and tomato (Palatnik et al., 2003; Ori et al., 2007; Efroni et al., 2008). However, because expression of a dozen cell cycle genes is reduced in young *Arabidopsis* leaves devoid of CIN-TCPs, regulation of mitotic activity by CIN-TCPs is probably indirect. Rather, based on expression profile analyses, it was suggested that the *CIN-TCPs* act at early stages to promote the maturation of leaf tissues (Efroni et al., 2008). In agreement with this, their precocious expression in *Arabidopsis* or tomato resulted in small and simple leaves with entire margins (Palatnik et al., 2003; Ori et al., 2007). Conversely, a transient delay in *CIN-TCP* expression induced giant leaves with otherwise normal morphology (Figure 2E; Efroni et al., 2008).

Members of the NAM/CUC family are initially expressed in the basal lateral boundaries of every initiating lateral plant primordium (Aida et al., 1997). In leaves, their expression is subsequently narrowed by *miR164* to focal points at the lamina margins adjacent to serrations and lobes (Nikovics et al., 2006; Blein et al., 2008; Berger et al., 2009). Progressive loss of *CUC* genes led to fusion of floral organs, leaves, and cotyledons and loss of serrations or lobes in the leaf lamina (Aida et al., 1997; Nikovics et al., 2006). Thus, similar to *STM*, loss of *CUC* activity results in precocious termination of meristematic tissues. *NAM/CUC* genes are implicated in regulation of growth as their increased expression can alter leaf size, but the direction of this change depends on the species in question: silent dominant mutations within the *miR164* binding site of *CUC2* genes resulted in smaller than wild-type leaves in tomato *gob-d* plants (Figure 2C; Berger et al., 2009) but larger than normal ones in *Arabidopsis cuc2-d* plants (Figure 2D; Larue et al., 2009). Such context-specific effects for growth regulators are similarly evident in the interactions between two growth-related quantitative trait loci, which produce opposing effects in two closely related *Arabidopsis* ecotypes (Kroymann and Mitchell-Olds, 2005). Furthermore, many genes have been implicated in modulating leaf morphology (a partial list along with references is provided in Supplemental Table 1 online), and given the status of leaves it is difficult to envision genes that do not contribute to leaf growth. In the next section, we will describe in detail a selected set of genes (Table 1) that are involved in meristem patterning and determination. Many of these genes play a limited role in shaping the simple leaf in the reference species *Arabidopsis*, but all have been implicated in patterning of compound leaves.

THE MAKING OF COMPOUND LEAVES

One of the most striking differences in leaf morphology is that found between simple and compound leaves. It has so happened that leaves of some of the most extensively studied plants, maize, *Antirrhinum*, tobacco, and *Arabidopsis*, are all simple, featuring a single undivided blade. In many plants, however, the organogenic potential of the primary leaf is extended to form a compound leaf, composed of several distinct lateral appendages, the leaflets. Leaflets, unlike leaves, do not host axillary buds, but like leaves they may give rise to secondary leaflets in a reiterative fashion.

Systematic and fossil analyses show that angiosperm compound leaves evolved from a simple form independently several times and often reverted back (Cronquist, 1988). Historically, discussions have centered on whether compound leaves are homologous or nonhomologous to shoots (reviewed in Champagne and Sinha, 2004). However, absence of knowledge of the underlying developmental mechanisms and the common evolutionary history nearly always enables claims of homology between two plant organs. With recent data on leaf development, we argue that it is more pragmatic to focus on the contrasting developmental and genetic programs for compound leaves in the genetically tractable systems of tomato, pea, and *Cardamine*. This comparison then serves as a reference point for comparing the mechanisms governing the formation of simple

and compound leaves in general and in the formation of distinct morphogenetic appendages in particular.

THE TOMATO COMPOUND LEAF: A REFERENCE SYSTEM

To dissect the organogenetic potential of compound leaves in tomato, Hareven et al. (1996) took advantage of the demonstrated meristematic activity of *KNOX* genes in conjunction with the modular organization, developmental versatility, and the wide range of genetic variants available in this species. The prototypical compound leaf of the cultivated tomato is composed of a petiole, a terminal leaflet, and four to six pairs of primary petiolate lateral leaflets that are generated alternately in basipetal mode along a central rachis (Figure 1A). Primary leaflets may form secondary leaflets, and small intercalary leafy appendages called folioles appear on later developing leaves between leaflets. Serrated lobes decorate the margins of all laminae.

Overexpression of the maize *KN1* in wild-type tomato leaves or in the bipinnate leaves of the *Petroselinum* mutant induces two to four additional reiterated rounds of ramification of the prototype blade, generating supercompound leaves bearing thousands of leaflets (Hareven et al., 1996; Figure 3A). Significantly, *KN1* misexpression eliminated foliole formation in the ramified leaves and did not increase the number of leaflets along the primary rachis. In the first of two critical experiments, overexpression of *KN1* in leaves of the *trifoliate* mutant resulted in each leaflet reiterating the ternate pattern, but again, no additional leaflets were formed along the bare petiole (Figure 3A). In the second experiment, the simple leaves of the *Lanceolate* (*La*) mutant responded to *KN1* as reported for tobacco and *Arabidopsis* by increased lobing only (Sinha et al., 1993; Lincoln et al., 1994), but again, no additional leaflets were formed (Figure 3B). Therefore, increased complexity of the prototypic compound architecture (i.e., number of appendages along the main rachis) is not correlated with its ramification by KNOX activity.

In further experiments with tomato, the expression of *KN1* or *TKN2* (tomato ortholog of *STM*) under different promoters (Hareven et al., 1996; Shani et al., 2009) or mutants carrying gain-of-function *KNOX* alleles such as *Mouse-ears* (*Me*; Chen et al., 1997; Parnis et al., 1997) resulted in novel patterns of ramification. A single reiteration of the basic compound pattern is found in leaflets lacking the BELL-like homeobox *BIPPINATA*. This gene behaves as a KNOX antagonist whose function can be outcompeted by the mini KNOX PTS (Kimura et al., 2008). Suppression of KNOX function via expression of a dominant-negative version led to small leaves with fewer leaflets (Shani et al., 2009). A similar situation was found with specific misexpressed forms of *KNOX* genes driven either by leaf-specific promoters (Shani et al., 2009) or in the tomato *Curl* mutant, carrying another gain-of-function allele of *TKN2* (Parnis et al., 1997). Thus, *KNOX* genes can modulate the level of ramification of a compound prototype, but, once again, the morphological form of this ramification is context dependent, even within the same species. This has led to the suggestion that to ramify the basic prototype, *KN1* exploits growth plasticity within a permissive developmental window and that the potential to reiterate a compound prototype is the best operational criterion for leaves being compound sensu tomato. As simple leaves, such as those of *Arabidopsis*, tobacco, the tomato *La* mutant, or sepals, do not turn compound in response to *KNOX*, it was inferred that the shapes of the two leaf types are regulated by two distinct developmental programs (Hareven et al., 1996). A reference point for future developmental genetic analyses and for scientific debates was thus established.

The expression pattern of *TKN2* in tomato is distinguishable from species with simple leaves in that it is expressed in developing leaves rather than only in the SAM proper, providing, for the first time, a potential molecular criterion for the distinction between simple and compound leaves (Hareven et al., 1996). To determine whether the correlation between *KNOX* expression and leaf forms in tomato is of general importance, Bharathan et al. (2002) conducted an extensive phylogenetic analysis in species with simple and compound leaves. Using antibodies able to detect any KNOX class I protein, a correlation between *KNOX* expression and leaf complexity in leaves of four *Lepidium* species was found, with one exception, likely due to secondary morphogenesis. The correlation held when additional taxa, including basal angiosperms, were included but with one additional conspicuous exception: compound leaves in legumes lacked detectable *KNOX* expression. This exception calls for a detailed description of the developmental mechanisms regulating compound leaves in pea and other species.

THE COMPOUND LEAVES OF PEA AND *CARDAMINE*

The legume pea provides an additional system in which compound leaves could be studied with the aid of an impressive battery of gene mutations (Figure 3C; Marx, 1987; Villani and DeMason, 1997; Gourlay et al., 2000). Unlike the basipetal mode of initiation and the homogeneous set of leaflets in tomato, common pea leaves bear three different types of appendages: serrated stipules, planar leaflets with smooth margins, and tendrils (Figure 1A), the latter two types developing, unlike in tomato, in an acropetal mode. The genetic regulation of the compound architecture is also different. In pea, the inactivation of *UNIFOLIATA* (*UNI*), an ortholog of *FLO*/*LFY*, conditions a simple leaf, primarily with one single terminal leaflet (Hofer et al., 1997). No single gene in pea is known to convert leaflets to the compound prototype, but such a modification is achieved by combining two recessive mutations: *afila*, which forms a ramified terminal blade comprised of tendrils, and *tendril-less*, which converts tendrils to planar leaflets (Figure 3C; Villani and DeMason, 1997). Similarly, inactivation of the FLO/LFY system in *Medicago truncatula* turned a ternate leaf into a simple one (Wang et al., 2008), and while this defect was rescued by introduction of a *LFY* transgene, no additional ramification was observed. Likewise, overexpression of a tomato *KNOX* in the clover leaf at best generated one or two ectopic leaflets but no regular duplication of the prototype (Champagne et al., 2007).

In both tomato and pea, the *FLO*/*LFY* orthologs are expressed in the SAM and leaf primordia (Hofer et al., 1997; Pnueli et al., 1998; Molinero-Rosales et al., 1999), but at least two *KNOX* genes are excluded from pea leaves (Hofer et al., 2001). Significantly,

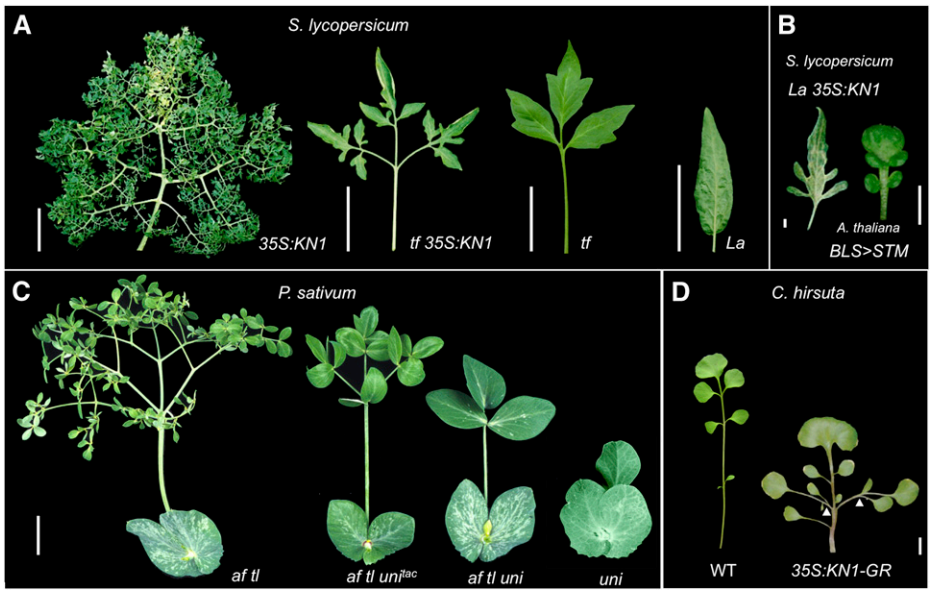

Figure 3. Genetic Regulation of Compound Leaf Patterning.

(A) *KN1*-induced reiterations of a compound pattern of tomato leaves (Hareven et al., 1996). Note the absence of additional primary leaflets, intercalary folioles, or marginal elaborations. *tf*, *trifoliate*.

(B) Similar marginal lobing response to elevated KNOX of the simple leaves of the tomato mutant *La* (left; Hareven et al., 1996) and *Arabidopsis* (right), where the KNOX gene *STM* is driven by the leaf-specific promoter *BLS*.

(C) A range of reiterations of the pea compound program conditioned by *uni*, *afilia* (*af*), and *tendrilless* (*tl*) (Hofer and Ellis, 1996).

(D) Compound *Cardamine* leaves respond to KNOX by initiation of additional leaflets (Hay and Tsiantis, 2006).

Bars = 10 cm in **(A)** and 1 cm in **(C)** and **(D)**. (***[A]*** *and tomato leaf of* ***[B]*** *are reprinted from Hareven et al. [1996], with permission from Elsevier. Arabidopsis leaf of* ***[B]*** *is reprinted from Shani et al. [2009]. Right image in* ***[C]*** *is reprinted from Gourlay et al. [2000]. Other genotypes in* ***[C]*** *are described in Hofer and Ellis [1996], and images were kindly provided by Julie Hofer.* ***[D]*** *is reprinted by permission from Macmillan Publishers Ltd.: Nature Genetics, Hay and Tsiantis [2006], copyright 2006.*)

UNI is also expressed in the incipient leaf primordia from which *KNOX* genes are excluded in all tested species, simple or compound (Gourlay et al., 2000; Bharathan et al., 2002).

Each of the leaf appendages in pea has a distinct developmental status, as best illustrated by the stipules. These leaf appendages are insensitive to *UNI* or *TL* loss of function, but, amazingly, are converted to stipule-less perfect compound prototypes in *cochleata* mutants (Gourlay et al., 2000). In this respect, pea *cochleata* stipules behave like tomato leaflets expressing *KN1*. Notably, *crispa*, a mutation in the ARP homolog of pea, induces stipule-like organs along the main pea rachis (Tattersall et al., 2005). Indeed, in his evolutionary study, Cook (1923) suggested that lateral leaflets in walnut and hickory evolved from stipules.

Cardamine hirsuta represents a third model for the study of compound leaves, attractive because of its close relation to *Arabidopsis* (Canales et al., 2009). In *Cardamine*, leaflet initiation requires STM activity, but unlike in tomato, *KNOX* overexpression does not faithfully amplify the basic compound pattern. Instead, similar to clover, additional leaflets are formed along the central and lateral rachises, a phenomenon that is not observed in tomato (Figure 3D; Hay and Tsiantis, 2006). At this point, no mutations that cause complete reiteration of the basal compound pattern in *Cardamine* are known. *Cardamine* leaves with their different organogenic potential perhaps represent an intermediate form between simple and compound leaves sensu tomato.

In typical compound leaves, leaflet organization may be pinnate, with several pairs of leaflets aligned along the rachis, or palmate when all leaflets initiate from the distal end of the petiole. Tomato, pea, and to some extent *Cardamine* leaves may ramify to form bi- or tripinnate leaves. To our knowledge, however, there are no truly bi- or tripalmate leaves. And while there are many genetic ways to simplify compound leaves, comprehensive mutational surveys have yet to reveal one case in which the simple leaf of *Arabidopsis* is turned compound. Thus, comparison of ontogeny, genetic regulation of ramification, and sensitivity to the major meristematic factors suggests a diversity of mechanisms of compound leaves, and by the same token, different mechanisms may underlie the formation of simple leaves in different lineages.

KNOX AND LFY ORGANOGENESIS SYSTEMS

Compound leaves in flowering plants arose independently several times in simple leaf ancestors; thus, the deployment of (seemingly) different genetic programs for the same outcome in closely related species is both expected and intriguing (Cronquist, 1988; Bharathan et al., 2002). The formation of similar, albeit evolutionarily unrelated, structures may represent

deep homology among the genetic circuitries controlling generative processes (Shubin et al., 2009; Sobral et al., 2009). One such generative process may be encompassed by the meristematic program termed blastozone.

Hagemann and Gleissberg (1996) coined "marginal blastozones" (from the Greek: blastos = bud, germ; budding or germ zone) to replace "leaf marginal meristems" as cells at the margins of leaf primordia do not maintain typical cell-replenishing meristematic activity. The marginal blastozone is identified morphologically by a lack of mature cells and situated at the basal part of the blade primordium. Here, we use the term blastozone to encompass both the marginal leaf meristem and the reproductive meristem. We speculate that both the KNOX and LFY meristematic systems are equally adequate in regulating organogenesis in the blastozone, and their respective functions are deployed in the marginal leaf meristem of some species and in the reproductive blastozone of others.

For example, a *LFY* ortholog is likely to perform mostly sporophytic functions in nonvascular or early land plants (Maizel et al., 2005; Tanahashi et al., 2005), whereas *KNOX* genes play a critical role after the transition from haploid to diploid forms in *Chlamydomonas* (Lee et al., 2008). In flowering plants, *KNOX* genes maintain or enhance meristematic potential in apical and other meristems, whereas *LFY* is primarily reserved for the regulation of reproductive meristem fate (Weigel et al., 1992; Hake et al., 2004). *KNOX* genes positively regulate the formation of compound leaves in tomato (Hareven et al., 1996), while *UNI*/*LFY*, together with *Stamina pistilloida*, an ortholog of *FIM*/*UFO* (Hofer et al., 1997; Taylor et al., 2001; Chae et al., 2008; Souer et al., 2008), is a positive regulator of compound leaves in pea.

The compound sympodial inflorescence of tomato generates 4 to 10 flowers with perfect radial pedicels. In several tomato mutants, the inflorescence meristems duplicate reiteratively. Cauliflower-like curds with thousands of arrested inflorescence primordia are found in *anantha* (*an*), giant leafy inflorescences in *falsiflora* (*fa*) (Allen and Sussex, 1996), and hundreds of normal flowers in *compound inflorescence* (s). In all cases, the leaves are mildly or not at all affected. In tomato, *FA* encodes the ortholog of *FLO*/*LFY* (Molinero-Rosales et al., 1999), *AN* the putative ortholog of *FIM*/*UFO*, and *S* is the putative ortholog of *WOX9* (Lippman et al., 2008).

Loss of *FA*/*UNI*/*LFY* marginally suppresses organogenesis in tomato leaves and strongly suppresses leaf blastozone activity in pea, but maintains it in the reproductive blastozone in both species. *AN* (*FIM*/*UFO*) negatively regulates organogenesis in the reproductive blastozone of tomato but has no discernible role in leaves. By contrast, ectopic expression of *UFO* or *WOX9* induces deeply serrated leaf margins in *Arabidopsis* (Lee et al., 1997; Wu et al., 2005). *S* (*COMPOUND INFLORESCENCE*, *WOX9*) is a negative regulator of the reproductive blastozone in tomato, and we predict that overexpression in tomato of either *AN* or *S* will abolish leaf complexity. Therefore, just as tomato and pea use *KN1* and *LFY* differentially to regulate leaf marginal blastozones, so tomato deploys the *KN1* system in leaf and the *LFY* system in inflorescence blastozones. Thus, it appears that a battery of meristematic regulators is available for the multiple morphogenetic tasks of plant meristems. Each meristem employs a subset of these regulators, irrespective of the final morphological outcome. In chordates, highly divergent expression patterns are suggested to underlie similar body plans (Sobral et al., 2009). By the same token, Rao et al. (2008) reported that rice *LFY* is required for the ramification of the inflorescence meristem and for the suppression of leaf growth, reflecting once again an opportunistic use of conserved meristematic factors in species- and organ-specific contexts.

GENETIC PROGRAMS REGULATING LEAVES, LEAFLETS, AND SERRATIONS

The ontogeny of appendages in compound leaves traditionally has been viewed as secondary dissecting morphogenetic processes in an otherwise simple template (Kaplan, 2001). In part, this view correctly reflects the ontogeny of rare complex leaf forms in monocots where late folding or secondary cell death programs generate the dissected leaves, such as in palms or *Monstera deliciosa* (Gunawardena and Dengler, 2006). Irrespective of their complexity, all complex leaf forms, including tomato and pea, therefore were designated as dissected forms of simple leaves (Kaplan, 1984, 2001; Tsiantis and Hay, 2003; Champagne and Sinha, 2004; Blein et al., 2008). In an alternative view, appendages of compound leaves develop by distinct genetic programs, their ontogeny is different from that of the primary leaf blades, and simple and compound leaves therefore represent different genetic programs (Hareven et al., 1996).

Following the phylogenetic survey and the difficulties of linking KNOX function consistently with leaf complexity, it was suggested that formation of compound leaves depends instead primarily on expression patterns of the *ARP* genes (Kim et al., 2003; Champagne and Sinha, 2004). Downregulation of tomato *ARP* results in leaves ranging from radial to palmate in correlation with the expression domains in the silenced lines. Moreover, the patterning of blades (simple, pinnate, or palmate) is correlated with the distribution of ARP expression domains in a wide range of species (Kim et al., 2003; Champagne and Sinha, 2004). However, *CRISPA*, the *ARP* ortholog in pea, is required for proper polarity of leaflet laminae but has only marginal effects on leaf complexity (Tattersall et al., 2005), whereas *Cardamine ARP* is required to maintain an elongated rachis (Hay and Tsiantis, 2006). In both species, however, ARP mutations condition *KNOX* expression in leaves, as expected. Notably, overexpression of *ARP* in *Arabidopsis* can only alter lamina dimension but does not induce lobes, serrations, or ectopic leaflets (Theodoris et al., 2003), as could be expected from a gene directing organ initiation (Kim et al., 2003). Thus, as with all other genes discussed, *ARP*s do not appear to be dedicated to a single common task in patterning compound leaves. And although the molecular networks (or modules) show some conservation, their phenotypic output, like that of KNOX, is species specific. For a detailed discussion of phylogenetic analysis of gene expression patterns, the reader may consult a recent review by Tomescu (2009).

Blein et al. (2008) objected to the conserved role of *ARP*s, the ARP/KNOX module, or *LFY* in determining leaves and their appendages and suggested instead a major patterning role for the *NAM*/*CUC* genes. In compound leaves of five species from different phylogenetic positions, including legumes, the *NAM*/*CUC* genes were found to be expressed at the boundaries of

leaves, leaflets, or serrations. It was therefore concluded that the conserved *NAM/CUC* genes are required to form leaves and leaflets and to dissect leaf blades into leaflets and leaflet margins into serrations and, consequently, that leaflets and serrations are formed by the same program as leaves (Blein et al., 2008).

NAM/CUC genes are expressed at boundaries of leaf and floral organ primordia, suggesting that expression-based homology in this case is inadequate (see Gaunt, 1997). Moreover, unlike with *KNOX*, overexpression of *NAM* hardly affected the number of leaflets in the compound tomato leaf, although lamina expansion was suppressed by both genes (Berger et al., 2009). Even without NAM/CUC3 functions, most compound leaves remained compound, although featuring a reduced number of leaflets (Blein et al., 2008). Implied relationships between programs controlling leaflets and serrations are also questionable; serrations are largely eliminated in ramified tomato leaves overexpressing *KN1* or in leaves bearing the dominant *TKN2/Me* (Figure 3A). Likewise, in a mutant form of the California Black walnut, serrations became pronounced while most leaflets of the compound leaves were lost (Babcock, 1914). Finally, Wang et al., (2008) showed that loss of *UNI* in *Medicago* results in transformation of the ternate clover leaf to a simple form. We noticed that the remaining terminal leaflet in such plants is still extensively serrated, a striking genetic uncoupling of leaflet formation and marginal serrations.

According to the prepattern paradigm (Hagemann and Gleissberg, 1996), dorsiventrality of the leaf derives directly from the existing radial prepattern of the SAM. Leaves are thus different from shoots. If we extend this rationale, leaflets cannot be identified with leaves because they emerge from the existing dorsiventral, not radial, prepattern of the leaf primordium and the same argument applied to the leaflets/serrations identity. Furthermore, clonal analysis in conjunction with careful examination of auxin maxima in *Cardamine* (Barkoulas et al., 2008) showed that leaflets are assembled by only one to four founder cells in contrast with the 50 to 100 initial founding cells in leaf primordia (Poethig and Sussex, 1985a), suggesting that leaves and leaflets have unique ontogeny.

If leaves and leaflets are different, the terminal leaflet, in representing the primary blade of the compound leaf, would be expected to be endowed with a distinct developmental status. Thus far, all mutants that have been found to inhibit leaflet initiation do not affect initiation of the terminal leaflet, be it precocious maturation conditioned by tomato *La* or inhibition of polar auxin transport in developing shoots of pea, tomato, or *Cardamine* (Gould et al., 1991; Avasarala et al., 1996; Barkoulas et al., 2008; see also the section below). In our view, therefore, despite the use of common genetic components in the development of leaflets, lobes, and serrations, leaves are different from leaflets, leaflets from lobes, and lobes from serrations, each representing a distinct morphogenetic entity.

THE GROWTH HORMONE FLORIGEN REGULATES LEAFLET PATTERNING IN COMPOUND LEAVES

Plant hormones contribute to the final shape of leaves as indeed they do for the whole plant. GAs reduce marginal elaborations of tomato leaves (Hay et al., 2002; Jasinski et al., 2008), whereas inhibitors of GA biosynthesis reduce leaflet number in pea and transform tendrils into laminate leaflets. However, DeMason (2005) found that GA could not convert pea leaflets to tendrils unless added with auxin. Direct evidence for the involvement of auxin in patterning compound leaves has been obtained in tomato and pea. In pea, inhibition of polar auxin transport via 1-*N*-naphtylphthalamic acid (NPA) favored leaflets over tendrils and determined the number of leaflets formed (Gould et al., 1991; DeMason and Chawla, 2004). As with pea, low NPA levels reduced leaflet formation in tomato (Avasarala et al., 1996).

More recently, Reinhardt et al. (2000) showed that at high NPA levels, tomato shoots do not produce leaves at all. However, these effects can be partially reversed by local auxin application, as with the *pin1* mutants of *Cardamine* (Barkoulas et al., 2008). However, as auxin maxima characterize differential meristematic activity at the SAM or along the rachis, it is likely that the role of auxin in compound leaf morphogenesis depends on a prior state of competence (Canales et al., 2009).

In numerous plant species with compound leaves, a sequential reduction of leaflets accompanies the transition to flowering of the SAM (Figure 4A). Recently, florigen has emerged as a new surprising regulatory tier of leaf patterning that is epistatic to the functions of *KNOX* or auxin maxima. Florigen is a systemic hormone that induces flowering, a protein in nature and the product of *FT* orthologs in flowering plants (Zeevaart, 1976, 2008). *SINGLE FLOWER TRUSS* (*SFT*), a shoot architecture gene that encodes the tomato ortholog of *FT*, was shown to generate universal flowering induction signals (Lifschitz et al., 2006) that were associated with severe growth retardation. As flowering in tomato is synonymous with termination of the SAM (Pnueli et al., 1998; Lifschitz and Eshed, 2006), it was surmised that flowering induction and attenuated growth are two facets of the same developmental mechanism targeted by florigen (Lifschitz et al., 2006).

Inactivation or overexpression of *SFT* stimulates or represses, respectively, foliole formation along the rachis, but the typical compound architecture is maintained, suggesting a stage-dependent meristematic function (Shalit et al., 2009). This situation is dramatically changed in *35S:SFT* plants in which the *SELF PRUNING* (*SP*) gene, a florigen antagonist of the same family, has been inactivated. Leaves of *sp 35S:SFT* are reduced to only one pair of distal leaflets or even turned into completely simple leaves, whereas alone, *sp* only slightly reduces serration. Moreover, the differential effects of an overdose of *SFT* in *SP* or *sp* backgrounds are graft transmissible, suggesting that florigen targets local SFT/SP ratios (Shalit et al., 2009). This was best illustrated by manipulating SFT/SP ratios in sensitized *trifoliate* mutant leaves (Figures 4B and 4C). The formation of simple leaves or the attenuation of other plant meristems by the florigen system is achieved without the disruptive morphogenetic effects that are frequently associated with *AS1*, *NAM/CUC*, *KNOX*, and *LFY/FLO* in either leaves or flowers. When either *SFT* or *SP* is mutated (Pnueli et al., 1998; Lifschitz et al., 2006), it is the timing or the potential of the developmental events that is changed.

In the regulatory hierarchy of the morphogenetic gradient in compound leaves, florigen is evidently epistatic to the role suggested for auxin (Reinhardt et al., 2000). Remarkably, florigen

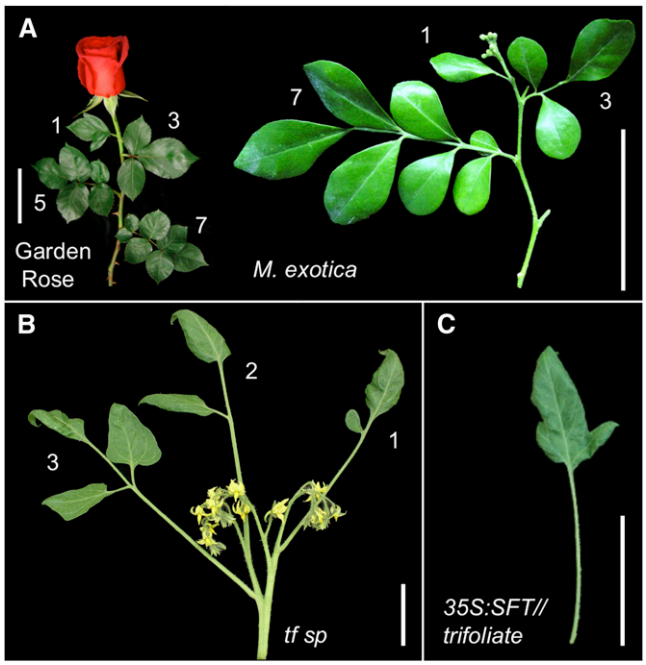

Figure 4. Regulation of Leaflet Initiation by Local and Systemic Florigen Levels.

(A) and **(B)** Selected examples of gradual leaflet loss correlated with transition to flowering in the domesticated rose (**[A]**, left; Shalit et al., 2009) or *Murraya exotica* (**[A]**, right). In tomato, such loss is evident in the sensitive *trifoliate self pruning* (*tf sp*) background **(B)**.

(C) Simple leaves induced by grafting a tomato *trifoliate* receptor with florigen-producing donor (*35S:SFT//trifoliate*; Shalit et al., 2009). Bars = 5 cm.

(*Images are reprinted from Shalit et al. [2009], except for the M. exotica image that was taken by I.E.*)

is also epistatic to the formidable organogenesis activity of *KNOX* genes: florigen completely suppresses induction of super compound leaves by *Me* (a gain-of-function allele of *TKN2*) in tomato. A balanced florigen function is therefore a prerequisite for generating the competence and the right organogenesis window in which *KNOX*, auxin, and other meristematic genes can pattern the blastozones of tomato leaves.

CONCLUDING REMARKS

Eudicot leaves are initiated as peg-like primordia from the flanks of the SAM, while in grasses the primodium already has an ML expansion. By an as yet unknown signaling mechanism, the primordium is endowed with PD, DV, and ML developmental potentials. Along its PD axis, the primordium is divided into a proximal nongenerative zone fated to form a petiole, whereas the distal zone has bilateral generative potential, designated here as a marginal leaf meristem/blastozone. When the primordium gives rise to one continuous lamina, the result is a simple leaf. If, however, bilateral meristematic potential persists, the generative rachis is expanded stepwise such that lateral meristematic foci generate leaflets causing the formation of a compound leaf.

The development of a planar bilateral lamina requires the function of the PHB and KAN families that function together with the YABBY and other meristematic factors to establish and maintain adaxial and abaxial fates and juxtaposition of the dorsal and ventral margins of the blastozone (Bowman et al., 2002; Chitwood et al., 2007). Numerous genes regulate the growth and expansion of the bilateral lamina in a continuous process called maturation, among which genetic interactions between the early acting CUC and CIN-TCPs direct elaboration/serration of the lamina (Ori et al., 2007; Blein et al., 2008; Efroni et al., 2008; Berger et al., 2009). While regulators of the maturation process are used to shape the lamina, rewired circuits involving the same general meristematic functions (Table 1) also regulate the initiation and ramification of lateral appendages in compound leaves. Yet, these modified regulatory modules, as illustrated by the interchangeable blastozone functions of *KNOX* and *LFY*, are species, and even organ, specific. This is likely the result of dormant developmental potentials embedded in leaves of flowering plants, a consequence of multiple independent interconversions of simple and complex forms during their evolutionary history.

To state unequivocally that any one particular gene system is responsible for the evolution and ontogeny of leaves in general, or of simple and compound leaves in particular, is premature, primarily because the scope of the phylogenetic data does not allow for satisfactory explanations of the emerging genetic exceptions (Tomescu, 2009). Indeed, if the meristematic elements that were used to define homology between a leaf and its leaflets (i.e., *KNOX* [Bharathan et al., 2002], *ARP* [Kim et al., 2003], and *NAM* [Blein et al., 2008]) are used to compare pea and tomato, we would have to conclude that leaves of these species are not homologous.

The formation of simple or compound leaves was the major focus of this review. Our view that the ontogeny and morphology of simple and compound leaves are regulated by distinct genetic programs with shared potentials in different contexts is based on extensive genetic evidence that, when looked at collectively, uncouples lateral leaflet and terminal blade formation, as well as leaflet formation and serrations. However, because not all meristematic gene systems are known and their basic cellular targets are mostly elusive, more extensive deep homology (Shubin et al., 2009) studies may link these morphogenetic structures in unexpected ways.

Supplemental Data

The following material is available in the online version of this article.

Supplemental Table 1. A Partial List of Genes Involved in *Arabidopsis* Leaf Development/Growth.

Supplemental References.

ACKNOWLEDGMENTS

This review is dedicated to the memory of Zvi Sachs, an eminent and inspiring scientist. We thank Darleen DeMason, Julie Hofer, Miltos Tsiantis, John Bowman, and the three reviewers for helpful comments, suggestions, and clarifications. We thank Zach Lippman for an excellent

editorial and linguistic work and John Alvarez, Clayton Larue, and Julie Hofer for the images in Figures 1B, 2D, and 3C, respectively. This work was supported by grants from the Israel Science Foundation (No. 863-06) and MINERVA to Y.E. and Israel Science Foundation Grants 381-03 and 1006-07 to E.L.

Received December 23, 2009; April 9, 2010; accepted April 15, 2010; published April 30, 2010.

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