Review

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On the mechanisms of development in monocot and eudicot leaves

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Summary

Comparisons of concepts in monocot and eudicot leaf development are presented, with attention to the morphologies and mechanisms separating these angiosperm lineages. Monocot and eudicot leaves are distinguished by the differential elaborations of upper and lower leaf zones, the formation of sheathing/nonsheathing leaf bases and vasculature patterning. We propose that monocot and eudicot leaves undergo expansion of mediolateral domains at different times in ontogeny, directly impacting features such as venation and leaf bases. Furthermore, lineage-specific mechanisms in compound leaf development are discussed. Although models for the homologies of enigmatic tissues, such as ligules and stipules, are proposed, tests of these hypotheses are rare. Likewise, comparisons of stomatal development are limited to Arabidopsis and a few grasses. Future studies may investigate correlations in the ontogenies of parallel venation and linear stomatal files in monocots, and the reticulate patterning of veins and dispersed stoma in eudicots. Although many fundamental mechanisms of leaf development are shared in eudicots and monocots, variations in the timing, degree and duration of these ontogenetic events may contribute to key differences in morphology. We anticipate that the incorporation of an ever-expanding number of sequenced genomes will enrich our understanding of the developmental mechanisms generating eudicot and monocot leaves.

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I. Introduction

Monocotyledonous and eudicotyledonous plants are ultimately classified according to the morphological differences in the number and arrangement of their embryonic leaves. The monophyletic monocots diverged from their eudicot relatives quite early in angiosperm evolution (Hertweck *et al.*, 2015), although details of their evolutionary relationships are blurred by evolutionary convergences among the nearly 300 000 species within the two orders (Christenhusz & Byng, 2016). This review focuses on the comparative ontogeny of monocot and eudicot foliar leaves, which develop from the shoot apical meristem (SAM) and are dorsiventrally asymmetric from their inception (Roth, 1949; Hagemann, 1970; Caggiano *et al.*, 2017).

The two main differences defining most monocot and eudicot leaves are the patterning of the vasculature, which is typically parallel in monocots and reticulate in eudicots, and the presence of a sheathing leaf base in monocots that encircles the stem (Kaplan, 1973). We first describe the comparative ontogeny of leaf development in monocots and eudicots and present testable models for the differential elaboration of upper and lower leaf zones, the formation of sheathing leaf bases and the patterning of monocot and eudicot vasculature. Next, we investigate the interplay between adaxial–abaxial and mediolateral patterning in monocot and eudicot leaves, and compare and contrast the shared and disparate programs of leaf dissection during the development of compound leaf morphologies. Lastly, we discuss the homology of enigmatic structures, such as eudicot stipules and the ligules of monocot grasses, and present comparisons of stomatal development in monocot and eudicot leaves. We speculate that, although there are profound differences in very early and late stages of leaf development in monocot and eudicot leaves, our current understanding is greatly skewed by redundancies in genes and gene regulatory networks, and the paucity of model species examined to date. Future analyses of leaf evo-devo will be enriched by extending these molecular genetic comparisons to include more of the expanding array of angiosperm species with sequenced genomes.

II. Leaf zones in monocot and eudicot leaves

Monocot and eudicot lineages have evolved tremendously diverse leaf morphologies (Fig. 1), including variations in lobed and dissected lamina, petiole length and morphology, and the presence or absence of lateral stipules at the leaf base. Likewise, monocot leaves vary from the dorsiventrally flattened (i.e. bifacial) strap-like leaves of grasses, to simple and dissected leaves with broad lamina and petioles, to leaves with unifacial, cylindrical lamina. The German plant morphologist Eichler hypothesized that, in spite of this extreme variation in adult leaf morphology, all angiosperm leaves contain a basal, clasping, lower leaf zone (LL) that inserts into the stem at the node and a distal upper leaf zone (UL) that extends the lamina away from the stem (Eichler, 1861). Advocates for the analyses of leaf development throughout ontogeny as opposed to simple comparisons of mature stages, Troll, Knoll, Hagemann and Kaplan all proposed that the major morphological differences between monocot and eudicot leaves evolved via differential elaborations and/or suppression of these primordial LL and UL zones (Fig. 2) (Troll, 1939; Knoll, 1948; Hagemann, 1970; Kaplan, 1970, 1973).

Eichler theorized that the LL of all angiosperm leaf primordia inserts into the stem at the node and extends to encompass some portion of the circumference of the shoot apex, thereby forming the

Box 1 Glossary.

Abaxial – bottom (ventral) side of a plant organ or organism. The abaxial surface is opposite to the shoot apical meristem and the stem.
Adaxial – top (dorsal) side of a plant organ or organism. The adaxial leaf surface is adjacent to the shoot apical meristem and the stem.
Auricle – a triangular tissue adjacent to the ligule that forms a hinge-like structure at the boundary between the basal sheath and the distal blade of grass leaves.
Bifacial – having distinct adaxial–abaxial (top–bottom; dorsal–ventral) identities on both sides of the leaf throughout its proximodistal axis, and typically forming a flattened, asymmetrical leaf.
Dorsiventral – an axis of plant development pertaining to the dissimilar/asymmetric dorsal (adaxial/top) and ventral (abaxial/bottom) surfaces of an organ or organism.
Ligule – an outgrowth on the adaxial (top/dorsal) surface of grass leaves at the boundary between the distal blade and the proximal sheath.
Mediolateral – an axis of plant development, extending from the median plane to the margins (sides) of an organ or organism.
Ontogeny – the developmental history of an organ or an organism from fertilization to maturity.
Plastochron – a developmental time interval, defined as the time between the formation of successive leaf primordia during vegetative plant development.
Prepattern – a pre-existing, molecular genetic pattern that predicts the development of an embryonic or primordial structure *before* any evidence of morphogenesis is observed.
Proximodistal – an axis of plant development, extending from the basal/proximal region to the tip/distal region of an organ or organism.
Sheathing base – the basal portion of the leaf that wraps around the stem and adjoins the leaf node.
Shoot apical meristem (*SAM*) – an organogenic, stem cell reservoir that ultimately forms all the lateral organs of the plant shoot.
Stipule – a lateral, leaf-like outgrowth at the base of a leaf.
Unifacial – having a single dorsiventral (i.e. adaxial or abaxial) identity on nearly all sides of the leaf: a radialized or cylindrical leaf lacking dorsiventral asymmetry throughout most of its proximodistal axis, yet which typically forms a bifacial (two-sided, dorsiventrally asymmetric) leaf base where the leaf inserts into the stem.

leaf base (Eichler, 1861). It is from this leaf base that the stipules develop in some basal eudicot and core eudicot species, as lateral appendages of the LL (Fig. 2d,e). In most eudicots, the leaf base extends to include just a fraction of the perimeter of the nascent stem (Fig. 3a). By contrast, the LL typically expands mediolaterally to surround the circumference of the shoot apex in most monocots,

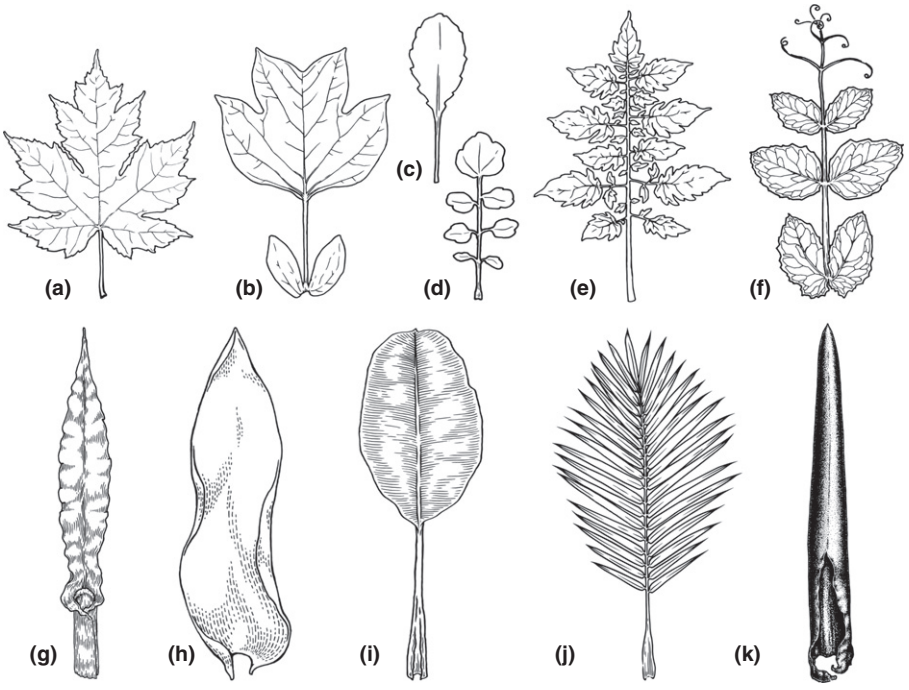

Fig. 1 Morphological diversity in a sampling of angiosperm leaves. (a, c–f) Core eudicot, (b) basal eudicot and (g–k) monocot leaf samples include those with (a–c, g–i, k) simple leaves, (a, b) extensive leaf lobing, (d–f, j) dissected leaves, (b, f) leaves with prominent stipules and (k) unifacial leaves. Samples are from (a) *Acer saccharum*; (b) *Liriodendron tulipifera*; (c) *Arabidopsis thaliana*; (d) *Cardamine hirsuta*; (e) *Solanum lycopersicum*; (f) *Pisum sativum*; (g) *Zea mays*; (h) *Tulipa gesneriana*; (i) *Musa acuminata*; (j) *Chrysalidocarpus lutescens*; and (k) *Sansevieria cylindrica*.

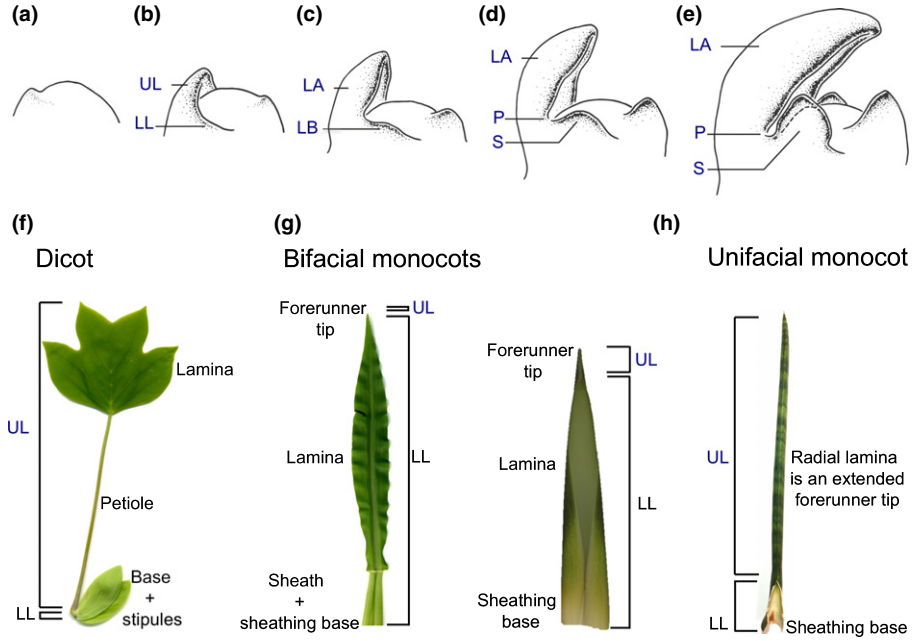

Fig. 2 The concept of leaf zonation. (a, b) Plant morphological models for leaf ontogeny hold that all leaf primordia contain a distal, upper leaf zone (UL) that projects away from the shoot apical meristem and a proximal, lower leaf zone (LL) that directly contacts the shoot apical meristem. (c) In this example from a stylized eudicot leaf, the UL gives rise to the distal leaf lamina (LA) or blade, and the LL forms the leaf base (LB). (d, e) As primordial development proceeds, the petiole (P) comprises the basal region of the UL, and the stipules (S) are derived from the leaf base in the LL. When extended to mature leaf morphologies, this classical model predicts that the petiole and lamina of most eudicot leaves (tulip poplar is used in this example) are derived from the UL, whereas just the leaf base and stipules comprise the LL (f). By contrast, most bifacial monocot leaves (g; examples from *Zea mays* and a juvenile leaf of *Sansevieria cylindrica*) contain a greatly truncated and radialized UL that forms the 'forerunner tip', whereas the lamina, sheath and leaf base are derived entirely from the LL. Lastly, in 'so-called' unifacial monocot leaves, such as this adult leaf from *Sansevieria cylindrica* (h), the radialized UL is greatly expanded and the bifacial LL is reduced to comprise the sheathing leaf base. Figures in (a–e) were redrawn from Kaplan (1997).

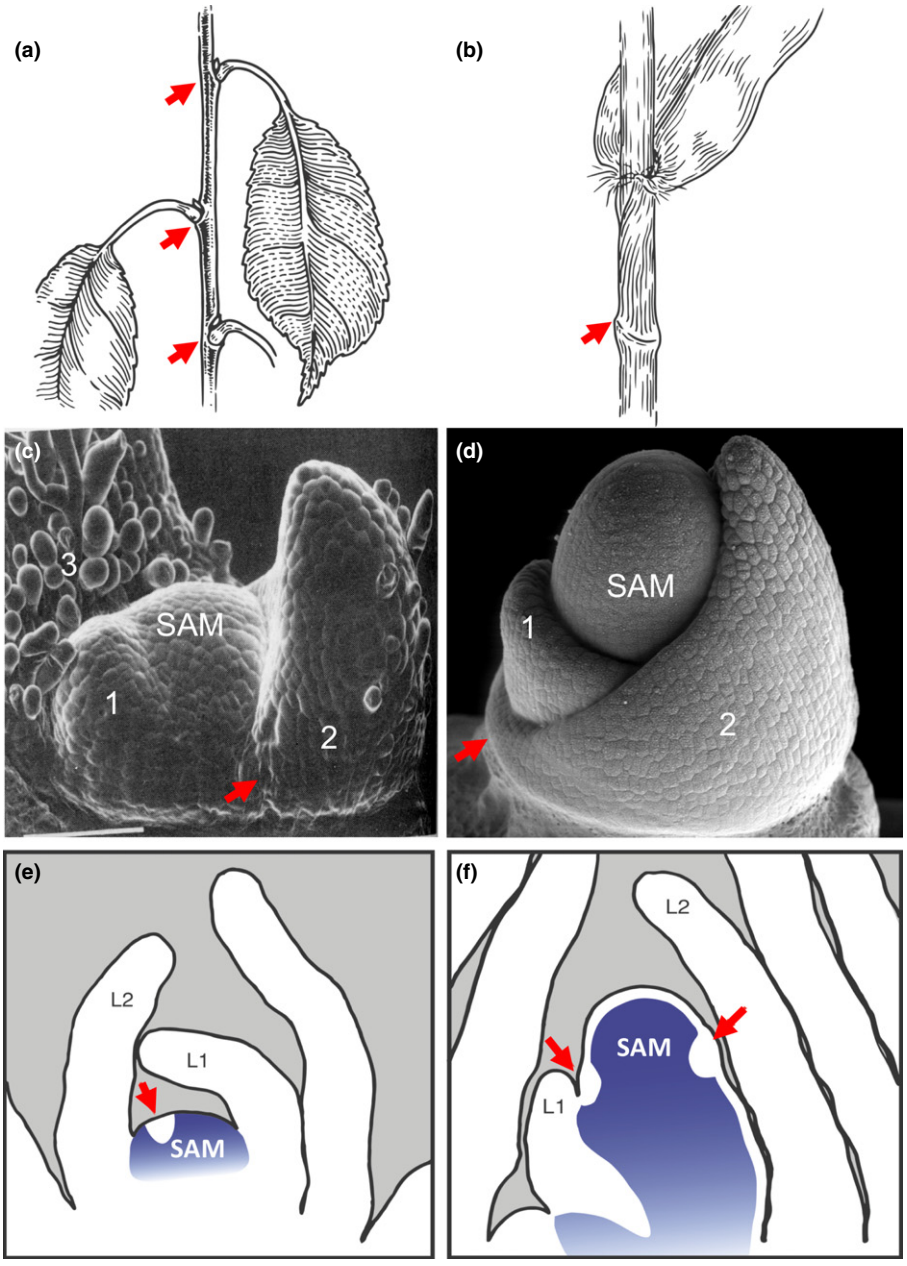

Fig. 3 Comparative ontogeny of the monocot and eudicot leaf base. In most eudicots, the leaf base (arrow) extends only partially around the circumference of the node (a), whereas monocots typically develop a sheathing leaf base that surrounds the stem (b). Scanning electron micrographs (SEMs) of (c) tobacco and (d) maize shoot apices reveal that, in monocots, the sheathing leaf base (arrow) is already established in young primordia. Cartoons of Class 1 *KNOX* gene expression (blue) in vegetative shoot apices reveal that, in Arabidopsis (e), a small patch of *KNOX* down-regulation in the shoot apical meristem (SAM) (arrow) corresponds to the founder cells of the newly initiating leaf primordium. In maize (f), however, this patch of *KNOX* down-regulation (arrows) extends completely around the circumference of the SAM. (c) Reprinted from Poethig & Sussex (1985).

forming the sheathing leaf base as a defining morphological feature of this group (Fig. 3) (Eichler, 1861; Troll, 1939; Hagemann, 1970; Kaplan, 1973). Furthermore, should the margins of the sheathing leaf base fail to separate (e.g. 'fuse'), tubular or cylindrical leaves will develop (Kaplan, 1973).

Extending this model, Kaplan and progenitors proposed that the LL of eudicot leaf primordia ultimately comprises just the leaf base and stipules of adult leaves, whereas the UL forms the distal lamina (Fig. 2d,e) (Troll, 1939, 1955; Knoll, 1948; Hagemann, 1970; Kaplan, 1973). This model stipulates that the petiole derives from the proximal region of the UL, which fails to undergo substantive, post-primordial mediolateral expansion. Thus, the eudicot petiole persists as a proximodistally elongated but narrow strip of the eudicot leaf that connects the leaf base to the distal lamina (Fig. 2).

By contrast, this model predicts that the strap-like, bifacial leaves of monocot grasses are almost wholly derived from the LL, as are bifacial monocot leaves that contain a leaf base, distal lamina and intervening petiole. In this way, just the tiny (*c.* 2 mm), unexpanded and radialized, forerunner tip (Knoll, 1948), comprising the distal end of bifacial monocot leaves, is derived from the primordial UL. Therefore, the monocot blade and petiole (if present) are not homologous to the eudicot lamina (Fig. 2). The phyllode theory likewise purports that the bifacial leaves of monocots are not equivalent to the lamina of eudicot leaves, but are instead composed of 'pseudolaminate', flattened petioles and leaf bases (Arber, 1918).

Intriguingly, monocot unifacial leaves lie at the opposite end of the spectrum of this model for leaf ontogeny, such that the radialized, forerunner tip comprising the monocot UL is greatly

elaborated in unifacial leaves and the LL is reduced to comprise just the sheathing leaf base (Fig. 2) (Knoll, 1948; Kaplan, 1973). Accordingly, monocot unifacial leaves develop radially symmetrical leaf blades that are homologous to the lamina of eudicot leaves, whereas only the much reduced, sheathing leaf base becomes bifacial. Originally presented as an evolutionary mechanism to explain parallel venation in monocot leaves, the phyllode theory instead proposes that unifacial monocot leaves are homologous to eudicot petioles that have lost the distal lamina (Arber, 1918, 1922). However, Troll and Kaplan rejected the phyllode model based on the lack of evidence for rudimentary/vestigial lamina during *any* stage in the ontogeny of monocot unifacial leaves (Troll, 1939; Kaplan, 1973).

Morphological support for these leaf zone models was provided by comparative analyses of the growth and development of the UL and LL in four monocot leaf types that vary in the proportions of the mature leaf that are unifacial vs bifacial (i.e. *Ornithogalum caudatum*, *Sansevieria trifasciata*, *Hosta lancifolia* and *Zantedeschia aethiopica*) (Kaplan, 1973). Remarkably, molecular genetic analyses of leaf mutants have generated very few tests of these models for the ontogeny and evolution of monocot and eudicot leaves (Tsiantis *et al.*, 1999; Nardmann *et al.*, 2004; Slewinski *et al.*, 2013, 2014). Where applicable, discussions of leaf mutant phenotypes and their relevance to these leaf zonation models for monocot and eudicot leaves will be provided later in this review in Section V.

III. Monocot and eudicot leaf initiation: differences in degree and timing, but not kind

Monocot and eudicot leaves initiate from cells within the peripheral zone (PZ) of the multicellular SAM. A plastochron (P) is defined as the time interval between successive leaf initiations from the SAM (Askenasy, 1880). The use of P number designations (wherein P1 represents the most recently initiated leaf primordium from the SAM, and P0 represents the leaf primordium that will initiate next) enable unambiguous descriptions of leaf development throughout ontogeny (Lamoreaux *et al.*, 1978; Sylvester *et al.*, 1990). Among the earliest-described molecular markers of leaf initiation are the polarized localization of members of the PINFORMED (PIN) family of auxin efflux transporters at the P0 stage, and the concomitant down-regulation of Class I *KNOTTED1-LIKE HOMEOBOX* (*KNOX*) gene expression (Smith *et al.*, 1992; Long *et al.*, 1996; Reinhardt *et al.*, 2000, 2003; Benkova *et al.*, 2003). As described below, these early events in leaf initiation are conserved in all monocot and eudicot species analyzed to date.

PIN-like proteins enable polar auxin transport (PAT) (Galweiler *et al.*, 1998), and their activity can be blocked by several PAT inhibitors (Geldner *et al.*, 2001). Studies in both monocots and eudicots illustrate that PAT inhibitors prevent leaf initiation, which can be re-activated by the exogenous application of auxin to the SAM (Reinhardt *et al.*, 2000, 2003, 2005; Scanlon, 2003). In Arabidopsis, a convergence point of PIN1 protein localization in L1 (i.e. the outermost 'protodermal' layer) of the SAM creates an auxin maximum at P0, the site of the incipient leaf primordia. At this convergence point, PIN1 localization is reoriented to form a stripe of PIN1-expression domain (PED) (Scarpella *et al.*, 2006) in the interior of the SAM, which has been termed the provascular trace, marking the region in which the midvein of the developing leaf will later develop (Benkova *et al.*, 2003; Reinhardt *et al.*, 2003). Recent work in tomato has suggested that *LEAFLESS* (*LFS*), a homolog of the Arabidopsis *DORNRÖSCHEN-like* (*DRNL*) *AP2* transcription factor gene (Chandler *et al.*, 2007), may function to translate this P0 auxin maximum into the leaf initiation response (Capua & Eshed, 2017). Auxin induces *LFS*, and leaf initiation is prevented in *lfs* mutants; however, the intervening mechanisms whereby LFS induces leaf initiation are unclear. Although the expression patterns of several maize homologs of *DRNL* genes have been described during embryogenesis and leaf initiation (Zimmermann & Werr, 2007), there are currently no analyses of *DORNRÖSCHEN* or *DRNL* gene function reported in any monocot species.

Surprisingly, when the accumulation of PIN1 homologs was analyzed during leaf initiation in grasses, no L1-localized convergence point was observed in P0 of the SAM, although PIN1 does form the provascular trace in grass shoot apices (Carraro *et al.*, 2006; Gallavotti *et al.*, 2008; Lee *et al.*, 2009; O'Connor *et al.*, 2014). Working in Brachypodium, O'Connor *et al.* (2014) showed that a P0 auxin maximum associates with the L1-localized convergence of the PIN1 homolog SISTER OF PIN (SoPIN). Loss of SoPIN function blocks leaf organogenesis (O'Connor *et al.*, 2017), in keeping with a model whereby leaf initiation and development of the provascular trace in grasses are subfunctionalized by SoPIN and PIN1a, respectively. Phylogenetic analyses suggest that the grasses are *not* the outliers in their utilization of SoPIN during leaf initiation. By contrast, the *SoPIN* gene is found in all sampled angiosperms, but was lost in Arabidopsis and throughout the Brassicaceae (O'Connor *et al.*, 2014). Indeed, recent studies in tomato have reported that mutations in the *SoPIN* homolog *ENTIRE2* (*E-2*) render defects in phyllotaxy (leaf arrangement around a stem) and leaf patterning, although vascular development is unperturbed (Martinez *et al.*, 2016). This study concluded that *E-2* is required to maintain convergence of PAT in the L1 layer of the tomato SAM.

Studies in monocots and eudicots alike implicate PIN-mediated PAT as a prerequisite for the down-regulation of Class I *KNOX* gene expression during leaf initiation (Scanlon, 2003; Hay *et al.*, 2006). Extensive analyses of Class I KNOX function in a wide variety of angiosperm model species have illustrated that these gene products promote indeterminate/stem cell identity in meristematic tissues – properties that are repressed during the development of determinate lateral organs such as leaves (Smith *et al.*, 1992; Jackson *et al.*, 1994; Hareven *et al.*, 1996; Long *et al.*, 1996; reviewed in Tsuda & Hake, 2015). In most eudicots, *KNOX* down-regulation during leaf initiation occurs in a localized patch in the LL, spreading a short distance from the site at which the leaf primordium inserts into the SAM (Fig. 3e) (Long *et al.*, 1996). However, in monocots, *KNOX* down-regulation extends completely around the circumference of the shoot apex, to form a pre-primordial sheathing LL at its inception (Fig. 3f). Thus, a simple model is proposed for the ontogeny of the sheathing leaf base (Fig. 3d), which is a defining feature of most monocot leaves

(Eichler, 1861; Troll, 1939; Hagemann, 1970; Kaplan, 1973). Simply stated, monocot leaf bases surround the stem at maturity because *KNOX* down-regulation in the LL encircles the SAM PZ during leaf initiation (Johnston *et al.*, 2015).

IV. Reticulate and parallel venation: extending the model?

A complex molecular mechanism for the development of vascular bundles has been well described mainly in the eudicot Arabidopsis, and is the focus of many excellent, recent review articles (De Rybel *et al.*, 2016; Bhalerao & Fischer, 2017; Campbell & Turner, 2017; Ramachandran *et al.*, 2017; Tameshige *et al.*, 2017; Linh *et al.*, 2018). In lieu of retelling the molecular genetics of Arabidopsis vascular differentiation, this review instead focuses on models for the developmental patterning of reticulate vs parallel venation that distinguishes most monocot and eudicot leaves.

In eudicot leaves, several size orders of veins branch from the centralized, larger midvein, forming an anastomosed net-like structure whose outline mirrors the overall shape of the leaf (Fig. 4a) (Cheadle *et al.*, 1953; Kang & Dengler, 2002; Linh *et al.*, 2018). Lateral veins connect to the midvein to form closed-ended loops. Minor veins extend from the midvein, or from lateral veins, and will either form closed connections between the larger veins or will end freely as open veins. Grasses develop longitudinally arranged or striate lateral veins on either side of the larger midvein. Lateral veins often converge and anastomose at both ends of the monocot blade, where the leaf tapers near the tip and at the proximal, blade–sheath boundary (Fig. 4b). Minor, longitudinal intercalary veins are also found in between lateral veins, whereas other minor veins establish lateral interconnections between the longitudinally arranged bundles, to form a ladder-like pattern. In bifacial monocot leaves containing a broad lamina and a narrow petiole (e.g. *Musa acuminata* in Fig. 1i), lateral veins are arranged in a pinnate pattern seemingly connecting to the midvein. However, longitudinal venation is also found within the petiole and midrib regions of these leaf types, whereupon these striate veins then diverge laterally in the lamina to form pinnately arranged lateral veins (Troll, 1939; Cheadle *et al.*, 1953; Kaplan, 1973).

As described above, the major midvein in both monocots and eudicots is associated with the formation of the provascular trace, a PED that forms internal to the L1 convergence of PIN-like auxin transporters in the SAM during leaf initiation (Fig. 5a,b) (Benkova *et al.*, 2003; Reinhardt *et al.*, 2003; Carraro *et al.*, 2006; Gallavotti *et al.*, 2008; Bayer *et al.*, 2009). Subsequent analyses performed in Arabidopsis suggest that the convergence of PIN1-mediated auxin transport likewise controls the initiation of lateral veins (Scarpella *et al.*, 2006; Sawchuk *et al.*, 2013; Verna *et al.*, 2015). In this way, a single mechanism is proposed to pattern leaf initiation, midvein formation and lateral vein development in the eudicot leaf (reviewed in (Linh *et al.*, 2018).

The formation of lateral veins in Arabidopsis begins with an epidermal PIN1 convergence at the leaf primordial margin *c.* 2.5–3 d after germination, characterized by a broad PED that extends from the margin to the previously existing midvein (Fig. 5c) (Scarpella *et al.*, 2006; Sawchuk *et al.*, 2013). PEDs associated with lateral vein initiation in Arabidopsis begin exclusively at leaf margins and connect to the pre-existing midvein, which accounts for how the vasculature mirrors the overall shape of the Arabidopsis leaf. Unlike the initiation of the midvein and lateral veins, the development of the minor veins that form both the closed loops and open-ended veins in Arabidopsis leaves is not associated with an L1/epidermal PIN1 convergence point. Instead, initiation of these minor veins correlates with the formation of a PED that emanates from a pre-existing PED associated with either a lateral vein or the midvein. Should the minor vein PED extend to contact the PED of a major vein (i.e. a lateral vein or the midvein), a closed vein loop will form. Open minor veins develop when the minor vein PED emanates from a major vein and fails to extend to a second

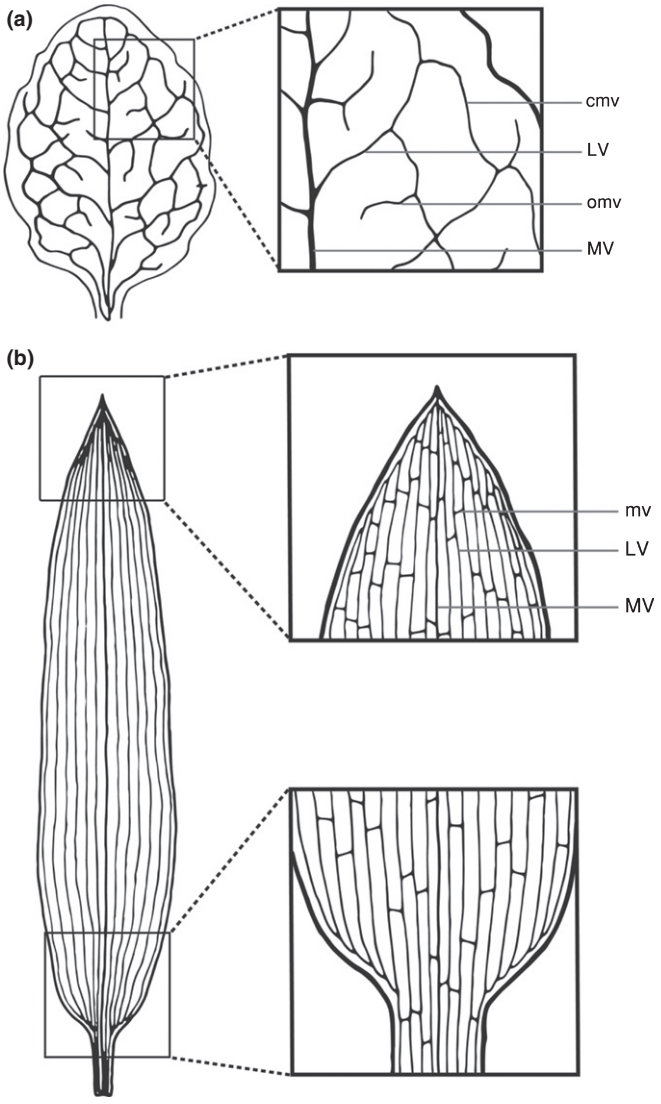

Fig. 4 Venation patterns in monocot and eudicot leaves. (a) Eudicot leaves have reticulately branched vasculature. Inset: lateral veins (LV) branch from the central midvein (MV). Closed minor veins (cmv) connect two major veins (LV to LV, or LV to MV) and open minor veins (omv) emanate from major veins but are open ended. (b) In monocot leaves, the LVs are arranged parallel to the MV and anastomose at the distal and basal ends of the leaf blade (insets). Minor veins (mv) develop in between the major veins to form short, transverse interconnections.

such PED (Fig. 5c–e) (Scarpella *et al.*, 2006; Sawchuk *et al.*, 2013; reviewed in Linh *et al.*, 2018).

Studies in maize also revealed a correlation between L1/epidermal PEDs and the formation of lateral veins, with important differences in developmental timing and PED polarity, which suggest a model linking the formation of parallel venation to the development of the monocot sheathing leaf base (Johnston *et al.*, 2014; reviewed in Linh *et al.*, 2018). As described above, leaf initiation in eudicots coincides with the formation of the provascular trace, a single PED that is associated with midvein development (Benkova *et al.*, 2003; Reinhardt *et al.*, 2003). However, in maize, PIN1a-associated provascular traces extending from L1 of the SAM are not only observed near the developing midrib region of newly initiated maize leaves (Fig. 5b). Instead, additional L1-derived PEDs are observed in lateral regions extending around the circumference of the emerging leaf primordium, in basipetal (tip-to-base) fashion from the edge of the pre-primordium towards the insertion point at the node (Johnston *et al.*, 2014). These data suggest a simple model wherein reiterative PIN1a-mediated auxin transport in the expanded lateral domains of the developing monocot leaf generates a series of additional provascular traces that will eventually give rise to a pattern of parallel lateral veins in the young primordium.

As in Arabidopsis, these monocot lateral vein PEDs also arise at the margins, but pre-primordially, that is, before the maize leaf primordium has fully emerged from the SAM. Notably, the midvein provascular trace is observed before the emergence of the primordium from the SAM in *both* Arabidopsis and maize. However, in maize, lateral vein provascular traces also arise as the leaf is initiating from the circumference of the SAM. This stands in contrast with Arabidopsis, in which lateral vein PEDs form in P3 leaf primordia, well after their emergence from the SAM (Scarpella *et al.*, 2006). In contrast with Arabidopsis, PEDs associated with lateral veins in maize do not connect to an existing larger midvein, but arise in parallel orientation to the midvein provascular trace (Fig. 5b).

We speculate that, because the maize lateral vein PEDs arise much earlier in developmental time than in Arabidopsis, that is, before an actual midvein is formed, the maize lateral vein PED cannot drain towards a pre-existing, larger vein. Simply stated, the model proposed by Johnston *et al.* (2014) predicts that the initiation of parallel lateral veins, together with the expanded recruitment of lateral leaf domains to form the typical monocot sheathing base, both occur concurrently during leaf initiation in maize. This model awaits further experimental support in maize and other monocot model systems. For example, monocot banana

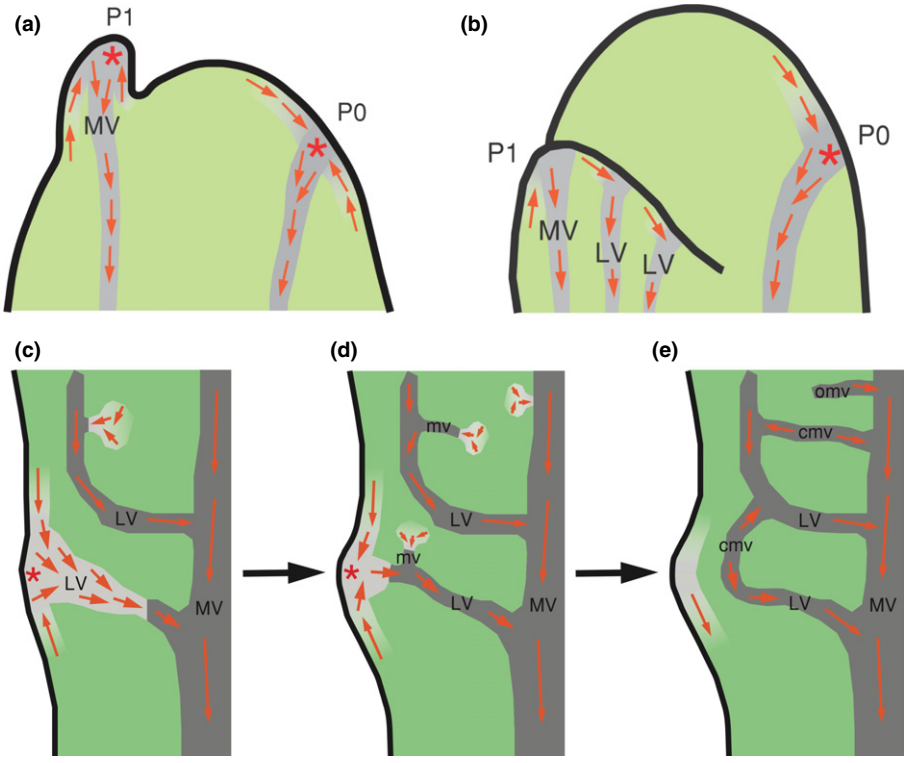

Fig. 5 Mechanisms of vascular patterning in Arabidopsis and maize leaf primordia. (a) Convergence of epidermally localized PIN1-mediated polar auxin transport (red arrows) at P0 marks the site of new leaf initiation in the Arabidopsis shoot apical meristem (SAM). Re-orientation of PIN1 directs auxin transport internally to form a broad band of PIN1 expression domain (PED), termed the provascular trace, which marks the site of midvein (MV) formation. In maize (b), epidermal localization of PIN1 is observed acropetal to P0, and also forms an internally localized provascular trace. In the initiating maize leaf (P1), several PEDs are formed lateral to the MV as the P1 leaf emerges from the entire circumference of the SAM. These lateral vein (LV) PEDs initiate at the epidermis of the emerging leaf primordium and transport auxin basipetally, towards the leaf insertion site at the node. (c–e) In Arabidopsis, LVs initiate from epidermally localized PIN1 convergence sites at the margins of the already emerged leaf primordium, and form PEDs that connect to the pre-existing midvein. Minor veins (mv) initiate from PEDs in contact with pre-existing LVs or the MV. When minor veins connect pre-existing LVs, or connect an LV to the MV, a closed minor vein (cmv) forms. When minor veins fail to connect at each end, an open minor vein (omv) is formed. Images are inspired by and redrawn from Linh *et al.* (2018).

leaves have a large midvein, connected to numerous lateral veins arranged pinnately (Fig. 1i). Although little is known about the mechanisms of vascular development in banana, this simple model would predict that lateral veins arise from epidermal PIN convergence points on the already initiated banana leaf primordium *after* the midvein is intact. Such lateral vein PEDs would then all canalize into the existing, larger midvein, as described by Scarpella *et al.* (2006), to generate pinnate venation. Amenable to transformation, banana leaves represent an excellent new model species to test these models for vascular patterning. Lastly, although it is currently unclear how intercalary and interconnecting minor veins form in monocots, mathematical modeling suggests that equivalent patterning mechanisms described for the formation of closed and open minor veins in Arabidopsis can be applied to monocot leaves (Runions *et al.*, 2005; Fujita & Mochizuki, 2006).

V. Flat laminar growth: patterning and coordination of adaxial–abaxial and mediolateral axes

Light energy is efficiently captured by flat surfaces. Current models predict that the pre-primordial juxtaposition of adaxial (top) and abaxial (bottom) domains organizes a new axis of expansive mediolateral growth from the SAM PZ, to give rise to the flattened lamina of bifacial leaves (Waites & Hudson, 1995; Caggiano *et al.*, 2017). In their landmark paper inspired by studies of the development of flattened wings in *Drosophila melanogaster* (Diaz-Benjumea & Cohen, 1993; Williams *et al.*, 1994), Waites and Hudson examined the abaxialized, radial, adult leaves of the Antirrhinum mutant *phantastica* (*PHAN*) and extended these animal models to describe the relationship between adaxial–abaxial and mediolateral patterning in plant leaves (Waites & Hudson, 1995). As described below, several decades of molecular genetic and microsurgical research have since been evaluated, and re-evaluated, in the light of the Waites–Hudson model.

The *PHAN* gene encodes a MYB domain protein (Waites *et al.*, 1998) that represses Class I *KNOX* gene expression in snapdragon leaves. Collectively, *PHAN* and its homologs from Arabidopsis, *ASYMMETRIC LEAVES1* (*AS1*), and maize, *ROUGH SHEATH2* (*RS2*), are referred to as *ARP* genes (Timmermans *et al.*, 1999; Tsiantis *et al.*, 1999; Byrne *et al.*, 2000; Ori *et al.*, 2000). Intriguingly, whereas ARP genes in maize, Arabidopsis and pea function in *KNOX* repression (Waites *et al.*, 1998; Timmermans *et al.*, 1999; Tsiantis *et al.*, 1999; Lodha *et al.*, 2013; DeMason & Chetty, 2014), tomato *PHAN* and *KNOX* genes are co-expressed in the same cells (Koltai & Bird, 2000; Kim *et al.*, 2003; DeMason & Chetty, 2014). Unlike the radialized, abaxial phenotypes observed in *phan* mutants, maize *rs2* mutants in the leaf exhibit narrow, half-leaf phenotypes with proximal–distal defects wherein ectopic sheath and ligule identity is expressed in the distal lamina (Schneeberger *et al.*, 1998). Tsiantis *et al.* (1999) invoked Kaplan's leaf zone model (1973) to explain these phenotypic discrepancies in the *arp* mutant phenotypes in maize and snapdragon. In this model, loss of *ARP* function causes ectopic expression of proximal leaf identity (i.e. the petiole in the eudicot Antirrhinum, and sheath and ligule tissue in maize) in the distal lamina. However, this interpretation fails to explain the mutant phenotype of the Arabidopsis *arp* mutant *as1*, which resembles the maize *rs2* mutant and does not include radial, abaxialized leaves (Byrne *et al.*, 2000; Ori *et al.*, 2000).

As *ARP* genes are ubiquitously expressed in leaf primordia, the polarity defects seen in some *arp* mutants may be a result of interactions with their adaxially expressed binding partners comprising the LOB domain, such as AS2 transcription factors. Overexpression of *AS2* in Arabidopsis causes polarity defects (Ueno *et al.*, 2007), and the abaxializing *microRNAs 165/166* (*miR165/166*) are negatively regulated by AS2. In both maize and Arabidopsis, disruption of the *miRNA165/166* targeting site in the *CLASS III HOMEODOMAIN LEUCINE ZIPPER* (*HD-ZIPIII*) family of adaxial identity genes produces leaves with adaxial identity on abaxial surfaces, owing to ectopic expression of *HD-ZIPIII* genes throughout the primordium (McConnell *et al.*, 2001; Juarez *et al.*, 2004). In maize, mutations in the *AS2* ortholog *INDETERMINATE GAMETOPHYTE1* (*IG1*) can condition adaxial–abaxial phenotypes primarily in the flag leaf, which is the last leaf to develop from the SAM (Evans, 2007). The GARP domain KANADI (KAN) transcription factors confer abaxial identity in both monocots and eudicots, and act antagonistically to HD-ZIPIII function (Eshed *et al.*, 2001; Kerstetter *et al.*, 2001; Emery *et al.*, 2003; Izhaki & Bowman, 2007; Candela *et al.*, 2008; Zhang *et al.*, 2009). KAN1 inhibits *AS2* (Wu *et al.*, 2008) and physically interacts with the abaxializing factor AUXIN RESPONSE FACTOR3 (ARF3), whose transcription is, in turn, repressed by the adaxializing factors AS1/AS2 (Husbands *et al.*, 2015). Similarly, monocots and eudicots generate *TRANS-ACTING SMALL INTERFERING RNAs* (*ta-siARFs*) that target *ARF3* transcripts for degradation. In Arabidopsis, the ta-siARF pathway was first described as a mechanism for vegetative phase change, although studies in rice and maize have revealed adaxial–abaxial patterning defects in monocot ta-siARF mutants (Bohmert *et al.*, 1998; Timmermans *et al.*, 1998; Peragine *et al.*, 2004; Pekker *et al.*, 2005; Hunter *et al.*, 2006; Nagasaki *et al.*, 2007; Nogueira *et al.*, 2007; Itoh *et al.*, 2008; Douglas *et al.*, 2010). However, recent work in tomato and Arabidopsis has confirmed that a role for ta-siARF in leaf polarity is conserved in both monocots and eudicots (Yifhar *et al.*, 2012; Skopelitis *et al.*, 2017). Thus, many of the observed phenotypic differences of *ta-siARF* mutants are probably confounded by the lack of investigations in diverse angiosperms.

The UL of eudicots can be induced to become unifacial not only by mutation, but also through exogenous perturbations. Microsurgical incisions in L1 of the tomato SAM separating the meristem from the incipient primordium generate radial leaf phenotypes, suggesting that a signal from the SAM may establish adaxial leaf identity (Sussex, 1951; Reinhardt *et al.*, 2005; Kuhlemeier & Timmermans, 2016). However, more recent work in Arabidopsis has determined that microsurgical wounding depletes auxin from the SAM, which causes the down-regulation of *HD-ZIPIII* expression. This loss of *HD-ZIPIII* in the SAM subsequently activates the ectopic expression of abaxializing *KAN* genes in adaxial domains of the shoot apex (Caggiano *et al.*, 2017). Thus, in this model, wound-induced disruption of an adaxial–abaxial prepattern in the incipient, but as yet unelaborated, leaf primordium leads to

the formation of unifacial, abaxialized leaves. No SAM-derived signal is invoked (Caggiano *et al.*, 2017). This model remains to be tested in additional monocot and eudicot model species.

The Waites–Hudson model also predicts that some factor(s) at the marginal juxtaposition of adaxial–abaxial domains must be present to interpret this adaxial–abaxial prepatterning signal, and translate it into mediolateral outgrowth. *WUSCHEL-LIKE HOMEOBOX* (*WOX*) genes are candidate factors that interpret this adaxial–abaxial juxtaposition at the pre-primordial leaf margin into outgrowth of a leaf primordium. *NARROWSHEATH1* (*NS1*) and *NS2* are duplicate *WOX3* homeologs in maize that are required for mediolateral outgrowth of the sheath and lamina (Fig. 6a). Analyses of the *ns1;ns2* mutant phenotypes reveal a defect in mediolateral development from the midpoint of the blade that extends into the entire sheath (Scanlon *et al.*, 1996). According to the UL/LL model (Kaplan, 1973), this equates to a deletion of the lower portion of the LL (Nardmann *et al.*, 2004). By contrast, mutations in the *WOX3* ortholog *PRESSED FLOWER1* (*PRS1*) in Arabidopsis condition no leaf phenotypes, but delete the lateral stipules at the base of this eudicot leaf (Fig. 6a) (Matsumoto & Okada, 2001; Nardmann *et al.*, 2004; Shimizu *et al.*, 2009). This phenotype suggests that stipules are LL structures, in support of Kaplan's model, and likewise provides evidence that the lamina of maize and Arabidopsis are derived from non-homologous leaf zones (Fig. 2f,g). Only in double mutant combinations with *wox1* does *prs1* affect lamina development (Fig. 6a) (Vandenbussche *et al.*, 2009). These data suggest that *WOX1* is partially redundant with *WOX3* in the UL; there is currently no evidence that *WOX1* functions in the LL. In tobacco, *wox1* deletes all lateral outgrowth from the midvein without altering the leaf length or adaxial–abaxial identity (Fig. 6a) (McHale, 1993; Lin *et al.*, 2013). The interpretation of these data in light of the UL/LL model is complicated. Maize contains two additional, uncharacterized *WOX3* genes (*WOX3a* and *WOX3b*), and no homolog of *WOX1* (Nardmann *et al.*, 2007). Thus, if *WOX1* function in eudicots is restricted to the UL and monocot lamina are indeed comprised almost exclusively of LL (Kaplan, 1973), it is tempting to speculate that *WOX1* function is dispensable in monocots.

How marginally expressed *WOX* genes contribute to mediolateral outgrowth is an active area of investigation. In Arabidopsis, adaxially expressed *MONOPTEROS* activates *WOX1* and *WOX3* via auxin, whereas abaxially expressed *ARF3* represses these *WOX*s at the same *cis*-regulatory element (Guan *et al.*, 2017). Recent genetic analyses of the cell proliferation inhibitor genes *NGATHA* (*NGA*) and *CINCINNATA-class-TCP* (*CIN-TCP*) have found that *nga* and *cin-tcp* mutants exhibit upregulation of *PRS1* expression and vastly overgrown margins (Alvarez *et al.*, 2016). Therefore, these data place *WOX3* at the pre-primordial margin of the Waites–Hudson model, at the juxtaposition of adaxial and abaxial identities.

Another test of the Waites–Hudson model can be found in analyses of the abaxialized, unifacial monocot *Juncus prismatocarpus*, which expresses *ARF3* around the circumference of the PZ in the absence of detectable expression of the *HD-ZIPIII* gene *PHABULOSA* (Yamaguchi *et al.*, 2010). This lack of detectable pre-primordial adaxial–abaxial juxtaposition and the subsequent development of an abaxialized lamina are predicted outcomes of the Waites–Hudson model, although this study did not include an exhaustive survey of described adaxializing factors. Interestingly, despite the fact that *ARF3* is expressed throughout these abaxialized leaves, *PRS* expression is detected during the elaboration of the 'central-marginal domain' in *J. prismatocarpus* primordia (Fig. 6b) (Yamaguchi *et al.*, 2010). This seems to contradict observations in Arabidopsis in which *PRS1* is localized to the site of adaxial–abaxial juxtaposition, although it is currently unknown whether *MONOPTEROS* or auxin regulates *PRS* expression in *J. prismatocarpus*. Moreover, the unifacial leaves of *J. prismatocarpus* are not completely radialized, but form slightly flattened, ovoid-shaped lamina, and express *PRS* in the central-marginal domain (Fig. 6b). By contrast, leaves of the related species *J. wallichianus* are completely radialized, but *do not* express *PRS* in the central-marginal domain (Yamaguchi *et al.*, 2010). Thus, these data suggest that *PRS* has been recruited in some species to form slightly flattened unifacial leaves, despite the absence of adaxial–abaxial juxtaposition.

Furthermore, *DROOPING LEAF* (*DL*) orthologs are also implicated in the promotion of leaf flattening in *J. prismatocarpus* (Yamaguchi & Tsukaya, 2010; Yamaguchi *et al.*, 2010). *JpDL* and *JwDL* are orthologous to Arabidopsis *CRABS CLAW* (*CRC*), the rice *DL* gene and the maize homeologs *DROOPING LEAF1&2* (*DRL1&2*), which are members of the *YABBY* gene family (Yamaguchi *et al.*, 2004; Strable *et al.*, 2017). Contrary to their initial description as abaxializing factors (Siegfried *et al.*, 1999), *YABBY* genes function to promote growth and expansion (Eshed *et al.*, 2004), which may account for their labile functions in a variety of morphologically distinct structures (Bowman & Smyth, 1999; Yamaguchi *et al.*, 2004, 2010; Strable *et al.*, 2017). In eudicots, *CRC* expression is restricted to carpel and nectary primordia, and does not appear to function during leaf development (Bowman & Smyth, 1999; Orashakova *et al.*, 2009; Sarojam *et al.*, 2010). In addition to their function in developing flowers, rice *DL* and maize *DRL* genes also promote cell proliferation during leaf midrib development (Yamaguchi *et al.*, 2004; Strable *et al.*, 2017). *DL* expression in the genus *Juncus* correlates with morphological variation in unifacial leaves. Whereas the flattened, unifacial leaf primordia of *J. prismatocarpus* accumulate *DL* transcripts in the proliferating cells of the medial-central domain, radialized primordia of *J. wallichianus* do not express *DL* (Yamaguchi *et al.*, 2010). Thus, a likely shared function of *DL* orthologs is to promote cell proliferation in the medial-central domains of bifacial *and* unifacial flattened leaves.

CRC and the *DL* orthologs appear to share an ancestral function in regulating carpel development in monocots and eudicots, whereas the involvement of *DL* orthologs in midrib development may have been acquired in the monocot lineage (Bowman & Smyth, 1999; Yamaguchi *et al.*, 2004; Sarojam *et al.*, 2010; Ohmori *et al.*, 2011; Strable *et al.*, 2017). These shared and diverged functions of *CRC/DL* orthologs in monocot and eudicot lineages are correlated with the distribution and sequence specificity of conserved noncoding sequences (CNSs). For example, a CNS is identified in intron 2 of *CRC/DL* homologs throughout the grasses; in rice, this CNS specifies the expression of *DL* in the leaf midrib region, but not in the carpel (Ohmori *et al.*, 2011; Strable *et al.*, 2017). Furthermore, *DL* expression differences between *J. prismatocarpus* and *J. wallichianus*

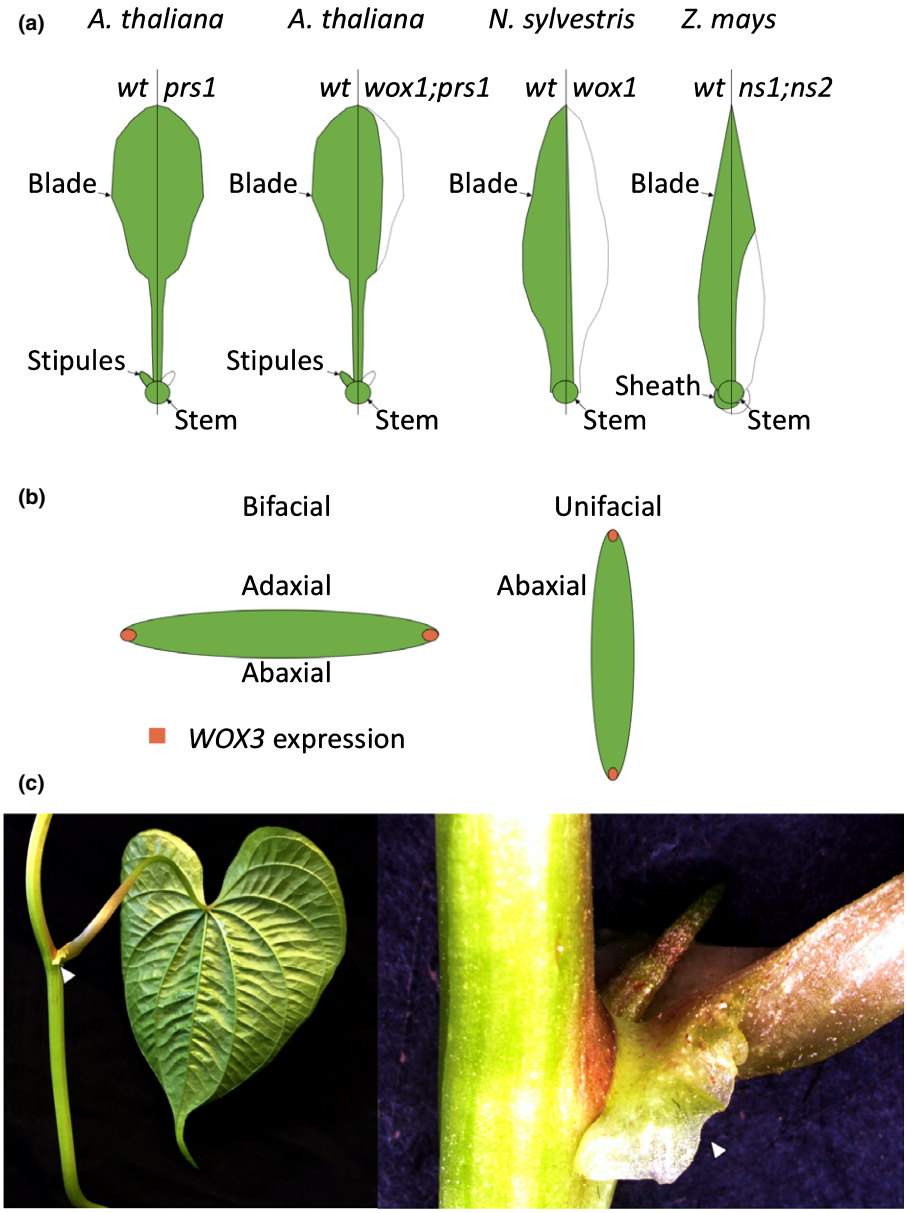

Fig. 6 WOX factors controlling laminar outgrowth. (a) Leaf cartoons showing mutant phenotypes (right side) and wild-type (wt) phenotypes (left side). In *Arabidopsis thaliana*, *prs1*(*wox3 ortholog*) mutants lack lower leaf zone stipule structure and have no effect on upper leaf zone laminar outgrowth. *wox1* and *prs1*(*wox3 ortholog*) double mutants have reduced upper leaf zone laminar outgrowth, whereas *wox1* mutants have no apparent phenotype. *Nicotiana sylvestris wox1* mutants are completely defective in laminar outgrowth without disrupting leaf length or adaxial–abaxial identity. *Zea mays ns1/ns2*(*wox3 orthologs*) mutants have narrow leaves and are most extreme in the sheath and lower blade where there are no margins. The upper part of the blade, however, is normal and includes the margins. (b) *WOX3* expression is found in the margins between the adaxial and abaxial juxtaposition, whereas in unifacial *Juncus prismatocarpus*, *WOX3* expression is found in the margins of medial-central areas of laminar outgrowth. (c) The monocot *Dioscorea bulbifera* lacks a sheathing base, but has stipule-like outgrowths (arrowheads).

are organ specific, and are probably a result of variation in *cis*-regulatory regions (Yamaguchi *et al.*, 2010). By contrast, *CRC* expression is limited to carpels and nectaries in Arabidopsis, where this CNS in intron 2 is not found (Lee *et al.*, 2005). Thus, it is tempting to speculate that the arrangement and sequence context of *cis*-regulatory regions diverged during the evolution of *CRC/DL* function in monocots and eudicots.

Several leaf flattening models have been tested, and re-evaluated, as developmentally distinct leaf forms have been investigated and with new molecular techniques. Some aspects to leaf polarity that were once thought to be distinctions between monocots and eudicots, such as the ta-siARF pathway, are probably a result of the limited number of tested organisms. Recent molecular reports challenge models establishing adaxial–abaxial polarity and suggest that SAM-dependent signals to promote leaf blade flattening are unlikely. However, the Waites–Hudson model of leaf flattening has provided a strong framework, tested by many gene functions, including the placement of *WOX1*/*WOX3* at the pre-primordial margin at the juxtaposition of adaxial and abaxial identities. In addition, the expression of *WOX3* and *DL* orthologs in the medial-central domains provides a potentially alternative mode of leaf flattening that seemingly contradicts the Waites–Hudson model. Evidently, expanding investigations to morphologically diverse leaves will provide an exciting opportunity to elucidate the mechanistic differences of leaf flattening in monocots and eudicots.

VI. Stipules and ligules: ontogeny of primordial elaborations

Within the LL of the grass leaf, the distal blade and proximal sheath are separated by taxon-specific structures that are defined differently by taxonomists, agronomists and geneticists (Kellogg, 2015).

The blade–sheath boundary in most grasses is separated by a collar that, in many species, is flanked by a ring- or wedge-shaped region of tissue, called a *leaf joint* in rice (Wu *et al.*, 2013), a *dewlap* in *Luziola* (Oryzeae) (Martínez-y-Pérez *et al.*, 2008) and sugarcane (*Saccharum* spp.) (Artschwager, 1951), and an *auricle* in maize (Sharman, 1942). Taxonomists reserve the term *auricle* to indicate the reduced prongs or outgrowths that extend from the wedge-shaped tissue at the leaf collar (Kellogg, 2015).

A prominent structure at the blade–sheath boundary of most grasses is the ligule, a flap of tissue that extends laterally from the adaxial surface. Ligule morphology varies across taxa, from membranous, to ciliate, to vascularized structures, and can range in size from reduced (a couple of millimeters) to extended (several centimeters) (Kellogg, 2015). Ligule development has been examined in maize using cell biological (Sharman, 1942; Sylvester *et al.*, 1990) and molecular genetic (Moreno *et al.*, 1997; Walsh *et al.*, 1998) (Muehlbauer *et al.*, 1999; Moon *et al.*, 2013) strategies. Recent transcriptomic analyses have suggested that ligule initiation from P5-P6 primordia is correlated with the accumulation of transcripts found at multiple organ boundaries throughout shoot development, including genes expressed during leaf and branch initiation (Johnston *et al.*, 2014). However, the ontogeny of the ligule remains an open question, and the homology of the ligule in eudicot leaves is even more enigmatic. Some have suggested that the ligule may arise as a lateral extension of the L1-derived margins of the grass leaf sheath (Philipson, 1935; Scanlon & Freeling, 1997). If the ligule is indeed an extension of a marginal domain in the lower part of the grass LL (i.e. sheath), many have suggested that ligules may comprise a grass stipule (Tyler, 1897; Glück, 1901; Majumdar, 1956; González & Rudall, 2001). Still others have proposed that there are no true homologs of stipules in the monocots (Lubbock, 1891). Unfortunately, there are currently no molecular genetic investigations of the homology of monocot ligules and/or eudicot stipules.

In Arabidopsis, *prs1* mutants lack stipules (Shimizu *et al.*, 2009), and maize *ns1;ns2* mutants lack mediolateral domains of blade and sheath. These data suggest that the lateral regions of the sheathing base of monocots may be homologous to the eudicot stipule. Tests of this proposed model are enabled in rare, monocot leaves that lack an extensive sheathing base. One such test case is *Dioscorea bulbifera,* an unusual monocot that *lacks* a sheathing leaf base, but develops stipule-like outgrowths from the site of leaf insertion at the node (Fig. 6c) (Isnard & Silk, 2009). Expanding these investigations to include molecular genetic analyses of *Dioscorea* and additional angiosperm taxa will provide robust tests of this model.

VII. Leaf architecture

Although the grass leaves described above have a very stereotyped morphology, angiosperms, in general, exhibit an extensive range of morphological diversity (Fig. 1). The evolutionary and environmental mechanisms driving leaf diversity have been reviewed recently (Chitwood & Sinha, 2016). Despite this diversity, leaves are placed into two major categories: simple and compound. Simple leaves terminally differentiate into a single unit. By contrast, compound leaves initiate as simple leaves that subdivide into leaflets by controlling marginal growth, and/or, uniquely to monocots, are dissected by tissue abscission or programmed cell death (PCD).

Simple leaves can be smooth or serrated, depending on the extent of elaborative, primordial growth at the leaf margin. Serrations in eudicot leaves are formed via the intermittent accumulation of *CUP-SHAPED COTYLEDON* (*CUC*) homologs, organ boundary genes that actively suppress growth at the margins (Fig. 7a) (Nikovics *et al.*, 2006). PIN1 promotes the accumulation of auxin at the site of the serrated outgrowth to inhibit CUC expression, which releases this CUC-induced suppression of marginal growth (Bilsborough *et al.*, 2011). In the case of *Cardamine hirsuta*, *REDUCED COMPLEXITY* (*RCO*) locally inhibits growth on the margins to enable leaflet outgrowth in areas in which *RCO* is not expressed; RCO is sufficient to form highly dissected leaves when overexpressed in the simple leaves of Arabidopsis (Fig. 7a) (Vlad *et al.*, 2014). Thus, intermittent suppression of marginal growth can be so extreme that the simple leaf becomes dissected to form compound leaves.

In most compound leaves, leaflets are formed wherein meristematic growth is reactivated in the margins of young leaf primordia, a process termed marginal blastozone fractionation (Fig. 7b) (Hagemann & Gleissberg, 1996). In monocots and eudicots alike, Class I *KNOX* expression is found in the SAM and is absent from initiating leaves (Fig. 3e,f). However, in many plants with compound leaves, *KNOX* expression is reactivated at the marginal blastozones (Hareven *et al.*, 1996; Janssen *et al.*, 1998; Bharathan *et al.*, 2002; Hay & Tsiantis, 2006). *KNOX* expression in leaves maintains indeterminacy, and overexpression of KNOX in tomato gives rise to super compound leaves with hundreds of leaflets (Hareven *et al.*, 1996). Thus, reactivation of meristematic growth in leaf primordia forms compound leaves.

In monocots, although examples of marginal blastozone fractionation can be found in Araceae and Dioscoreaceae (Gunawardena & Dengler, 2006), palms display a different form of compound leaf development. In palms, differential growth at the margins forms plications, in which margins are folded into an accordion-like structure (Fig. 7c) (Dengler *et al.*, 1982). *KNOX* expression is found in some palm plications, suggesting a blastozone fractionation-like mechanism (Jouannic *et al.*, 2007). However, *KNOX* expression is not universal in the compound leaves of all palms (Nowak *et al.*, 2007) and, similarly, is not universal in eudicots. In the case of pea and *Medicago truncatula*, instead of *KNOX* reactivation, orthologs of *LEAFY* (*LFY*) are expressed in marginal blastozones (Hofer *et al.*, 1997, 2001; Gourlay *et al.*, 2000; Champagne *et al.*, 2007; Wang *et al.*, 2008). Although *KNOX* expression is observed in some palm plications, the mechanism of KNOX-independent palm plications remains to be determined.

During the transition from cell division to cell expansion, palm leaf primordia undergo a secondary phase of leaflet separation, wherein cell numbers are reduced at the ridges of plications (Kaplan *et al.*, 1982). As a result, mechanical forces eventually separate the leaflets as they expand, to form pinnately compound leaves. This cell reduction is not achieved by PCD or necrosis, but probably by

an abscission-like degradation of the middle lamella (Nowak *et al.*, 2007). Furthermore, PCD is implicated in the dissection of *Monstera* leaves, wherein PCD causes perforations early during leaf expansion, and these perforations are enlarged as the leaf grows (Fig. 7d) (Gunawardena *et al.*, 2005). Moreover, the lace plant *Aponogeton madagascariensis* eliminates tissue between veins, to form a striking, lattice-like, skeletal network (Fig. 7d) (Gunawardena *et al.*, 2004). The use of PCD and/or abscission like processes to shape leaf development in these species appears to be the result of specialization, rather than an inherent feature of monocot leaf development. Overall, a diverse array of species-specific mechanisms of compound leaf development have been identified in monocot and eudicot lineages, which may reflect the multiple, independent origins of compound leaf morphology during angiosperm evolution (Bharathan *et al.*, 2002).

VIII. Stomatal development: shared and diverged mechanisms for making epidermal pores

The epidermis of monocot and eudicot shoots is perforated with stomata, minute adjustable pores flanked by specialized guard cells (GCs) that overlie subepidermal airspaces. Stomata are essential for plant development; mutations blocking stomatal formation are lethal (Ohashi-Ito & Bergmann, 2006; MacAlister *et al.*, 2007; Pillitteri *et al.*, 2007; Raissig *et al.*, 2016). Striking differences in stomatal patterning are noted in comparisons of monocot and eudicot leaves. Namely, stomata appear to be randomly distributed across the epidermis of eudicot leaves, whereas monocot stomata are arranged in evenly distributed, parallel rows (Han & Torii, 2016; Simmons & Bergmann, 2016; Hepworth *et al.*, 2018). Genetic analyses in Arabidopsis have provided detailed, molecular genetic descriptions of the mechanisms of stomatal development (Han & Torii, 2016; Simmons & Bergmann, 2016). Comparative studies are shedding light on the shared and divergent mechanisms of angiosperm stomatal development, including new insights into stomatal patterning in monocots (Chater *et al.*, 2011, 2013; Hepworth *et al.*, 2018).

Stomatal morphology and distribution vary across plant lineages (Peterson *et al.*, 2010; Vatén & Bergmann, 2012; Chater *et al.*, 2017). The predictable patterns of cell division and cell fate acquisition that define stomatal patterning have been described for developing monocot and eudicot leaves. One common theme is that early asymmetric cell divisions in the stomatal cell lineage generate a stomatal precursor cell and a daughter cell; the former will ultimately differentiate into a pair of GCs and the latter will differentiate as an epidermal pavement cell. A generalized model for stomatal development in eudicots, summarized below, derives largely from observations in Arabidopsis (Fig. 8a; reviewed in Simmons & Bergmann, 2016). Stomatal patterning occurs across a proximal–distal gradient, with more terminally differentiated cells found at distal portions of the Arabidopsis leaf. New stomata can arise adjacent to cells neighboring existing stomata, although stomata are typically separated by an epidermal pavement cell (Zhao & Sack, 1999). At first glance, mature leaf stomata appear to be randomly distributed across the adaxial and abaxial surfaces of Arabidopsis leaves, although exquisite molecular genetic analyses have revealed that this pattern is decidedly nonrandom (Han & Torii, 2016; Simmons & Bergmann, 2016).

In the initial phase of stomatal development, protodermal stem cells become meristemoid mother cells (MMCs) that divide asymmetrically to produce a smaller, triangular meristemoid and a larger stomatal lineage ground cell (SLGC). An SLGC can have one of two fates: (1) differentiation into a pavement cell; or (2) a second asymmetric cell division to form a satellite meristemoid. Meristemoids can be maintained in self-renewing, amplifying divisions that preserve the developmental plasticity of these initial cells. Typically, after two rounds of asymmetric cell division, the meristemoid transitions to a guard mother cell (GMC). In the final stage of stomatal differentiation, the GMC undergoes a single symmetric cell division to produce the GC pairs that define the stomatal aperture. Thus, in broad-leaved eudicots, a stomatal cell lineage ultimately gives rise to a pair of kidney-shaped GCs that are irregularly dispersed across the leaf epidermis (Fig. 8a).

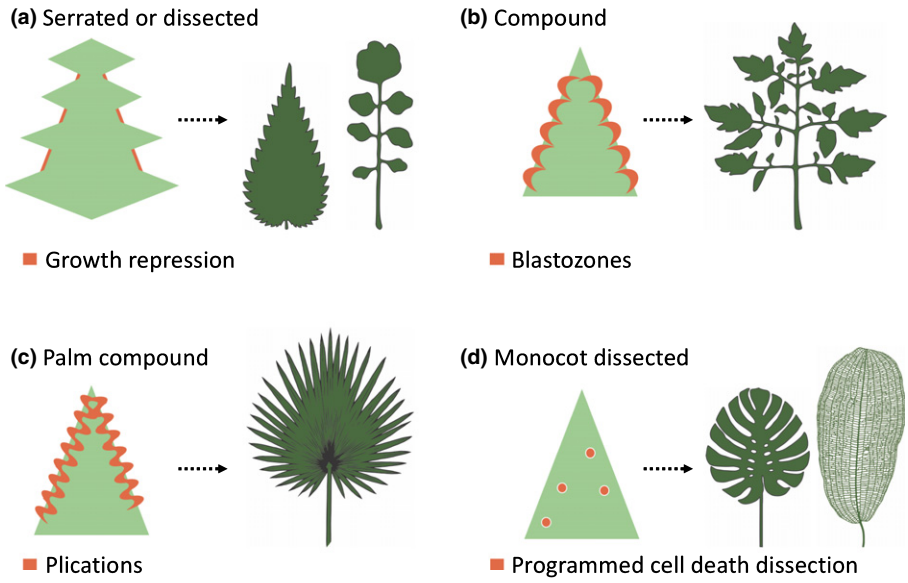

Fig. 7 Factors shaping leaf architecture. (a) Differential marginal growth can be controlled by intermittent expression of growth inhibitors *CUC2* and *RCO*. (b) *KNOX* or *LFY* expression is observed in emerging leaflets along the marginal blastozones. (c) Plication formation initiates palm leaflets. (d) Programmed cell death shapes leaves both marginally and internally in monocots.

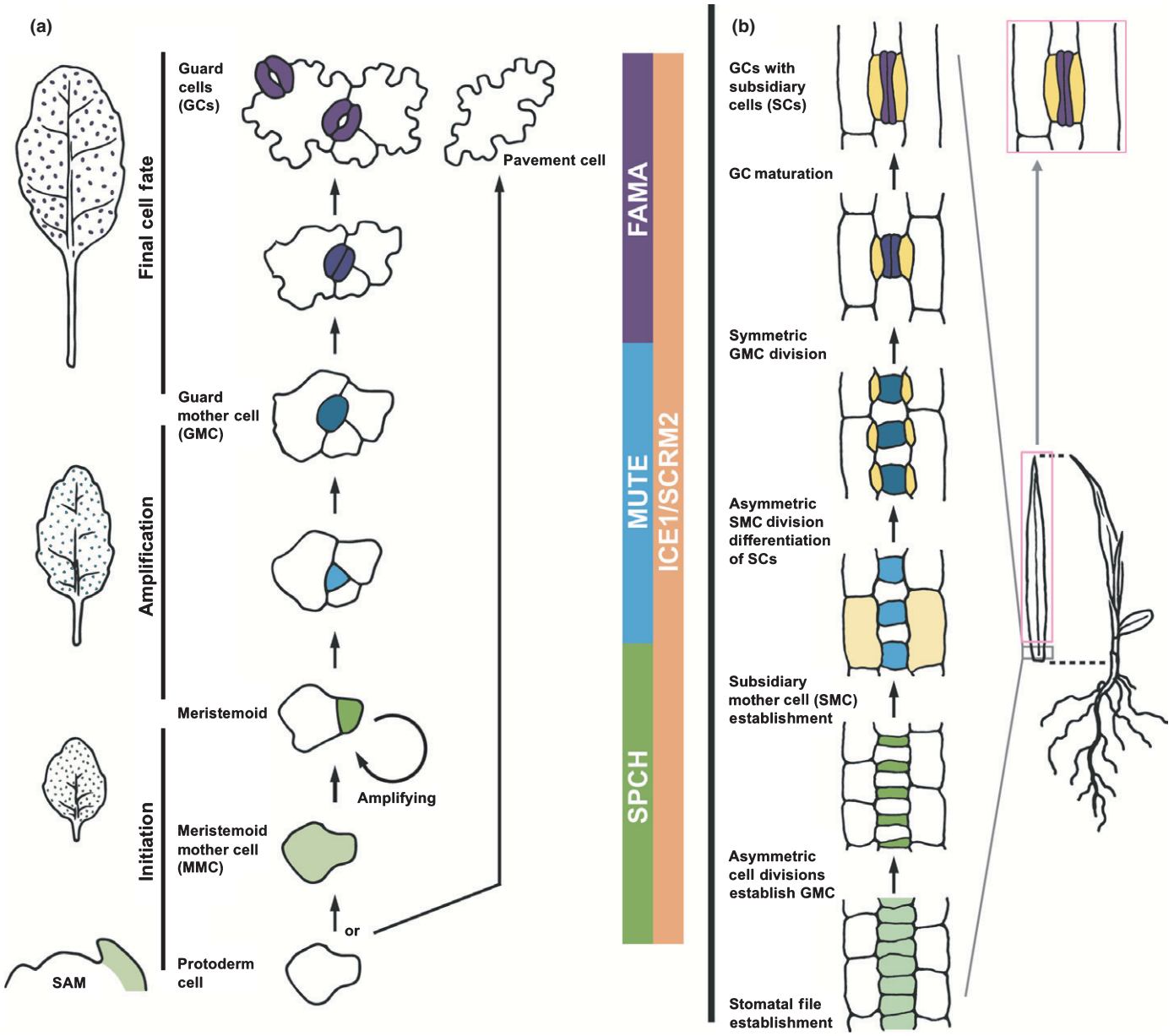

Fig. 8 Overview of stomatal development in the context of leaf maturation in Arabidopsis and in grasses. (a) The eudicot (Arabidopsis) leaf epidermis is derived from protodermal cells in the L1 layer of the shoot apical meristem (SAM) and is specified as leaf primordia are initiated from the SAM. Meristemoid mother cells (MMCs) are established at the initiation phase of stomatal development in the young leaf. MMCs accumulate SPCH protein and those that form a complex with ICE1/SCRM2 divide asymmetrically to form meristemoids. In the amplification phase, MMCs and meristemoids divide asymmetrically to promote the maturation of the epidermis and appropriate stomatal patterning. MUTE protein accumulates and forms a complex with ICE1/SCRM2 in cells fated to become guard mother cells (GMCs), committing entry into the stomatal cell lineage by terminating asymmetric cell divisions. In the final stage of stomatal differentiation, the GMC undergoes a single symmetric cell division to produce a pair of guard cells (GCs) that define a stoma aperture. This requires the FAMA protein, which forms a complex with ICE1/SCRM2. (b) In grasses (monocots), stomatal precursor cells arise in the proximal base of the growing leaf blade in cell files with higher rates of cell division relative to flanking cell files. All cells in the stomatal lineage divide asymmetrically in the same orientation (i.e. parallel to the marginal axis of the leaf) to produce a smaller GMC and a larger interstomatal sister cell. Cells in the flanking files become polarized in response to cues from the GMC and divide asymmetrically to become subsidiary mother cells (SMCs), each of which then differentiates into a subsidiary cell (SC) positioned adjacent to the GMC. The GMC then undergoes a terminal symmetric division to produce daughter cells that differentiate into GCs. The end result is a four-cell complex of two SCs and two GCs.

Studies of stomatal development in monocots have focused on grasses, where recent advances in genomic technologies have facilitated the functional characterization of Arabidopsis gene homologs (Liu *et al.*, 2009; Raissig *et al.*, 2016, 2017; Hughes *et al.*, 2017; Yin *et al.*, 2017). A generalized model for stomatal development in monocots, summarized below, derives from observations in rice, maize, barley and *Brachypodium distachyon* (Liu *et al.*, 2009; Facette & Smith, 2012; Raissig *et al.*, 2016, 2017;

Hepworth *et al.*, 2018). In the proximal base of the growing leaf blade, stomatal precursor cells arise in cell files with higher rates of cell division relative to flanking cell files. Importantly, this proliferation phase is much more abbreviated compared with that in Arabidopsis, and another major distinction is that grasses lack a self-renewing meristemoid phase. Every cell in the precursor cell file undergoes one asymmetric division in the same orientation (i.e. parallel to the marginal axis of the leaf) to produce a smaller GMC and a larger inter-stomatal sister cell. Notably, in grasses, cells in the flanking files become polarized in response to cues from the GMC and divide asymmetrically to become subsidiary mother cells (SMCs), each of which then differentiates into a subsidiary cell (SC) positioned adjacent to the GMC. The GMC then undergoes a terminal symmetric division to produce daughter cells that differentiate into GCs. Thus, in monocots, grass stomatal cell lineages produce a pair of dumbbell-shaped GCs flanked by two SCs arranged in collateral rows between parallel veins (Fig. 8b) (Zeiger *et al.*, 1987).

The sequential steps that specify stomatal differentiation are orchestrated by the transient expression of three genes that encode basic helix-loop-helix (bHLH) transcription factors: *SPEECHLESS* (*SPCH*), *MUTE* and *FAMA* (Han & Torii, 2016; Simmons & Bergmann, 2016). Within each gene's specific developmental window in the stomatal lineage, SPCH, MUTE and FAMA form obligate heterodimer complexes with the bHLH proteins, SCREAM/INDUCER OF CBF EXPRESSION1 (SCRM/ICE1) and SCRM2, which are broadly expressed, but enriched in stomatal lineage cells (Kanaoka *et al.*, 2008). Briefly, protodermal stem cells enter the stomatal cell lineage by expressing *SPCH* (MacAlister *et al.*, 2007). Cells that accumulate SPCH/ICE1/SCRM2 protein complexes will become MMCs that divide asymmetrically to produce an SLGC. Later, as *SPCH* expression dampens, the meristemoid transitions to a GMC, and *MUTE* is upregulated in GMCs, committing entry into the stomatal lineage by terminating asymmetric divisions (Pillitteri *et al.*, 2007; Kanaoka *et al.*, 2008; Davies & Bergmann, 2014; Adrian *et al.*, 2015). In the final stage of stomatal differentiation, FAMA directs a single symmetric cell division in GMCs to produce the GC pairs that define a stoma aperture (Fig. 8a) (Ohashi-Ito & Bergmann, 2006; Kanaoka *et al.*, 2008).

Highly orchestrated cell–cell communication properly orients the division of SLGCs, to prevent stomata from developing immediately adjacent to one another. Ultimately, spatiotemporal regulation of SPCH stability early on in the stomatal lineage controls stomatal density (MacAlister *et al.*, 2007). EPIDERMAL PATTERNING FACTOR (EPF) peptide ligands bind to family members of the ERECTA receptor kinase and the receptor-like protein TOO MANY MOUTHS to inhibit SPCH (Nadeau & Sack, 2002; Shpak *et al.*, 2005; Hara *et al.*, 2007; Hunt & Gray, 2009; Kondo *et al.*, 2009; Sugano *et al.*, 2010). Thus, extracellular signaling peptides and a suite of plasma membrane-associated proteins control the appropriate spacing of stomata by regulating SPCH.

Forward and reverse genetic studies in rice and *Brachypodium* have revealed that expansion of the *SPCH*, *MUTE*, *FAMA*, *ICE1*/*SCRM* and *SCRM2* homologs during the evolution of the grasses produced an 'alternatively wired' genetic program for stomatal patterning (Liu *et al.*, 2009; Raissig *et al.*, 2016, 2017; Chen *et al.*, 2017). Functional analyses of *SPCH* paralogs in *Brachypodium* (*BdSPCH1* and *BdSPCH2*) indicate partially redundant roles in establishing stomatal fate (Raissig *et al.*, 2016; Chen *et al.*, 2017). BdSPCH2 is sufficient to induce stomatal fate, suggesting that it functions as a GMC master regulator (Raissig *et al.*, 2016). Although the Arabidopsis *ICE1* and *SCRM2* genes are broadly expressed in the protoderm and are functionally redundant in the Arabidopsis stomatal cell lineage, *BdICE1* and *BdSCRM2* paralogs have evolved to perform diverse functions in brachypodium (Raissig *et al.*, 2016). BdICE1 establishes stomatal fate during initial asymmetric cell divisions, whereas BdSCRM2 is required for differentiation of the stomatal complex in GMCs before the formation of SMCs (Raissig *et al.*, 2016). Unlike in Arabidopsis, where MUTE is required solely for GMC identity by preventing asymmetric divisions of the meristemoid, the mobile BdMUTE is trafficked from the GMC to the adjacent SMCs, where it establishes SMC identity and promotes cell division in brachypodium (Raissig *et al.*, 2017).

As a model organism for understanding stomatal development in eudicots, research in Arabidopsis has elucidated the molecular mechanisms of stomatal patterning, and has provided a platform for comparative analyses in other species. This work underscores the power inherent in studies on the evolution of development (evo-devo), as demonstrated by the identification of grass stomatal cell lineages (SCs) that are absent in Arabidopsis. Comparative studies have revealed that the core stomatal developmental genes are retained across Arabidopsis and grass lineages, despite the fact that stomatal patterning is altogether distinct in monocots and eudicots. In grasses, core stomatal patterning genes are restricted to cell files at the base of the leaf in which cells are devoid of self-renewing divisions, whereas, in Arabidopsis, these self-renewing divisions allow stomata to develop at later stages of leaf maturation. The end result is that Arabidopsis leaves are dotted with stomata defined by GCs in all orientations with respect to the leaf proximal–distal axis, and grass leaves develop files of GCs in uniform orientation along the proximal–distal axis. Clearly, evolutionary tinkering of the stomatal pathway, probably through subfunctionalization, has led to alternatively wired developmental programs and outcomes in monocots and eudicots.

IX. Conclusion

Several distinguishing characteristics that contrast monocot and eudicot leaf development may stem from early patterning events, including the formation of a sheathing leaf base and parallel lateral veins during initiation of monocot leaves from the SAM. Classical studies in plant morphology predict that bifacial leaves in monocots and eudicots are derived from distinct, primordial leaf zones, whereas the radialized lamina of unifacial monocot leaves are homologous to the eudicot lamina. Genetic analyses of monocot and eudicot leaf development are often subject to ambiguity, owing to differing levels of functional redundancy within a relatively small sampling of model species. In spite of these challenges, numerous studies of monocot and eudicot leaves provide robust support for

the model of Waites & Hudson (1995), which describes the interplay between adaxial–abaxial patterning and mediolateral outgrowth of the lamina. Comparative analyses suggest that adaxial–abaxial patterning is prepatterned in the shoot apices of both eudicots and monocots, before the outgrowth of leaf primordia. However, subsequent mediolateral laminar outgrowth may occur during the P1 stage in grasses, but in slightly older leaf primordia in Arabidopsis. Unlike the conserved events during the initiation of leaf primordia from the SAM, the development of compound/dissected leaves involves a number of lineage- and species-specific mechanisms, which may reflect the multiple, independent origins of compound leaf morphology in angiosperm evolution. Questions concerning the possible homology of monocot sheathing leaf base and eudicot stipules, and the homology of grass ligules, remain open for investigation. Likewise, the parallel arrangement of monocot vasculature and stomatal cell files, which are reticulately arranged and evenly distributed/interspaced in eudicots, raises interesting questions as to whether vascular patterning may influence stomatal patterning in both these angiosperm lineages. Future analyses of leaf evo-devo will be enriched by interdisciplinary approaches that combine phenomics, functional genomics and computational modeling in comparative studies of the expanding array of angiosperm species with sequenced genomes.

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Author contributions

P.A.C., J.S. and M.J.S. wrote the manuscript. S.L. created the artwork in Figs 1–5 & 8, and provided comments on the manuscript. P.A.C. and J.S. contributed equally to this work.

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