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Developmental genetics of maize vegetative shoot architecture

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Abstract More than 1.1 billion tonnes of maize grain were harvested across 197 million hectares in 2019 (FAOSTAT 2020). The vast global productivity of maize is largely driven by denser planting practices, higher yield potential per area of land, and increased yield potential per plant. Shoot architecture, the three-dimensional structural arrangement of the above-ground plant body, is critical to maize grain yield and biomass. Structure of the shoot is integral to all aspects of modern agronomic practices. Here, the developmental genetics of the maize vegetative shoot is reviewed. Plant architecture is ultimately determined by meristem activity, developmental patterning, and growth. The following topics are discussed: shoot apical meristem, leaf architecture, axillary meristem and shoot branching, and intercalary meristem and stem activity. Where possible, classical and current studies in maize developmental genetics, as well as recent advances leveraged by "-omics" analyses, are highlighted within these sections.

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Introduction

An urgent challenge facing plant scientists and breeders is how to feed the world's growing population on limited arable land (Tilman et al. 2011). The cereal maize (*Zea mays* ssp. *mays*) is one of the most cultivated crops, making up over 38% of the world's cereal production (FAOSTAT 2020). Increased global production is attributed, in part, to adapting maize shoot architecture to dense planting (Duvick 2005). Shoot architecture is defined as the three-dimensional structural arrangement of the above-ground plant body (Sussex and Kerk 2001; Reinhardt and Kuhlemeier 2002) and is a major factor contributing to unit area grain yield (Russell 1991). Structure of the maize shoot is integral to all aspects of productivity and modern agronomic practices: row cropping, light interception, photosynthetic capacity, planting density, canopy shape, synchronized flowering, yield, and mechanized harvestability (Crosbie 1982; Troyer 1996; Tuberosa and Salvi 2009; Duvick 2005; Wang et al. 2018a). Maize stands out among the cereals as a crop with an impressive history of genetic investigation, owing largely to its amenability for forward, reverse, and modifier genetic screens (Candela and Hake 2008; Schnable and Freeling 2011; Nannas and Dawe 2015). Additionally, advances in new methodologies such as single cell technology (Efroni and Birnbaum 2016; Libault et al. 2017; Nelms and Walbot 2019; Satterlee et al. 2020), plant transformation (Lowe et al. 2016; Altpeter et al. 2016; Kaush et al. 2019), image processing (Clark et al. 2020; Guo et al. 2020), genomics (Kremling et al. 2018; Yang et al. 2019; Ricci et al. 2019; Franco et al. 2020; Soyk et al. 2020; Wang et al. 2020a; Hufford et al. 2021), metabolomics (Xu et al. 2019; Zhou et al. 2019), proteomics (Walley et al. 2016), phenomics (Pauli et al. 2016; Yang et al. 2017; Gage et al. 2019), genome editing (Kelliher et al. 2019; Gao et al. 2020; Liu et al. 2020; Atkins and Voytas 2020; Taagen et al. 2020), machine learning (Wang et al. 2020b; Washburn et al. 2019, 2020), and synthetic biology (Leydon et al. 2020) have deepened the toolboxes of plant scientists and breeders for more efficient and comprehensive strategies toward maize improvement.

Domestication of maize from teosinte reshaped shoot architecture

Maize was domesticated from the teosinte *Z. mays* ssp. *parviglumis* about 9000 years ago through a single domestication event in the Balsas region of southwest Mexico (Matsuoka et al. 2002; Piperno et al. 2009). Vegetative and reproductive morphologies of domesticated maize differ drastically compared with teosinte (Stitzer and Ross-Ibarra 2018). The domestication of maize resulted in a conspicuous increase in apical dominance of the shoot and morphological transformations of the pistillate inflorescence, or ear (Doebley and Stec 1991; Doebely et al. 1997). Maize has relatively few, if any, basal axillary branches, or tillers. A tiller originates from an actively growing axillary bud that nests in the axil between the attachment point of the leaf sheath and the stem node. Growth of the axillary bud is suppressed in maize shortly after it produces a whorl of protective leaves. About midway up the adult maize shoot, 2–3 aerial lateral branches (also called ear shanks or ear shoots) originate singly at each node in the sheath axil, each of which is terminated by a large ear. By comparison, the teosinte shoot has multiple, long tillers and lateral branches that originate basally and from aerial mid-shoot nodes, each of which terminates with a staminate inflorescence, or tassel. Teosinte tillers are prolific, bearing clusters of reduced ears at each node (Iltis 1983). Maize geneticists and breeders use "degree of prolificacy" to describe the potential for ear production (Liable and Dirks 1968; Hallauer 1974); prolificacy is reduced in modern maize and remains high in teosinte (Wills et al. 2013). Thus, domestication of maize from teosinte, with its strong selection for tiller suppression at the base of the shoot and elongation of lateral ear shoots in the middle of the plant, impacted several agronomically important aspects of plant architecture.

Maize domestication also conditioned more subtle effects on the shoot architecture through changes in leaf morphology. In maize and teosinte, leaves alternate at each node. Each leaf has a stem clasping proximal sheath and a distal blade that angles away from the shoot axis. Leaf sheath and blade are separated by a hinge-like auricle and epidermally derived ligule. In teosinte, each long lateral branch is akin to the maize shoot in that leaf attachment alternates at each node, with each leaf having sheath, auricle, ligule, and pronounced blade. In maize, the shorter internodes of the ear shank are wrapped entirely in a whorl of leaves of predominantly sheath tissue, though auricle, ligule, and blade are present, albeit highly reduced (Iltis 1983; Doebley 2004). Leaf traits, such as blade width and length, sheath length, size, and total estimated area, show significant quantitative variation between maize and teosinte, with each showing a proportional increase in maize (Milla and Matesanz 2017; Fu et al. 2019). Additionally, leaf blades along the teosinte main axis can be slightly more upright compared to blade angle for some maize inbred lines (Tian et al. 2019).

Shoot architecture as a readout of developmental patterning and growth

Plant development lies at the heart of shaping shoot architecture through the size, identity, timing, and duration of maturation schedules of active, pluripotent stem cell tissues called meristems. As plant stem cells, meristems give rise to daughter cells that form new organs as well as undergo self-renewal (Steeves and Sussex 1989). Meristem tissues of the maize shoot include the shoot apical meristem (SAM), axillary meristem, intercalary meristem, and meristems of the inflorescences. Meristems subsequently produce leaves, lateral branches, stem (discussed herein), and, upon floral transition, tassel and ears (discussed elsewhere).

Shoot apical meristem

The capacity for continuous postembryonic shoot development requires establishment and maintenance of shoot meristems (Steeves and Sussex 1989). In maize, shoot meristematic tissue develops during embryogenesis approximately a week after pollination (Randolph 1936; Nardmann and Werr 2009; Takacs et al. 2012). Subsequently, SAM initiates a leaf primordium singly at its flank in a distichous (alternate) phyllotaxy (Kiesselbach 1949; see Jackson 2009). Concomitant with cells becoming fated to a leaf primordium, cells from the meristem also are recruited to establish an axillary meristem in the axil of the initiating leaf and the internode section of stem. Collectively, these vegetative organs form a repeating unit of the shoot called the phytomer (Fig. 1a) (Galinat 1959). To describe the relatively uniform time interval between successive initiation of a primordium and its associated phytomer, plant biologists use the term plastochron. Plastochron index, abbreviated as "P" and a number, denotes the position of the primordia relative to an apical meristem actively producing primordia (Lamoreaux et al. 1978). For example, P2 is the second youngest primordium produced by the meristem, P1 is the most recently initiated primordium, and P0 is the incipient primordium at the meristem periphery (Fig. 1a, inset). Meristem size is dynamic in between plastochrons and during shoot maturation: the SAM is smallest in the embryo and increases as each successive primordium is initiated until floral transition (Abbe et al. 1951; Ledin 1954; Bassiri et al. 1992; Thompson et al. 2014). The geometry and size of the SAM is influenced by genetic modifier loci in various maize inbred backgrounds (Vollbrecht et al. 2000; Thompson et al. 2014, 2015; Leiboff et al. 2015). SAM traits such as size, height, and volume can be predicative of adult plant phenotypes (Thompson et al. 2015; Leiboff et al. 2015).

Histology of the SAM captures the beauty of its internal organization and cell division patterns. Longitudinal sections through the SAM expose cells that divide almost exclusively anticlinal (perpendicular to the surface) as clearly marked outer cell layer termed the L1 or tunica (Esau 1965). Beneath the L1/tunica is an inner mass of cells collectively termed L2 (subepidermal) or corpus, where cells division is in all orientations (Abbe et al. 1951; Ledin 1954). The central zone of the SAM houses initial cells akin to stem cells; cells of this zone are less dense in cytoplasm with infrequent divisions (Jackson 2009). The subjacent peripheral zone cups the central zone and is characterized by cells that are more concentrated in cytoplasm engaged in more frequent division, which relates to this zone's function in organogenesis. Underneath the girth of the peripheral zone is the centrally positioned rib zone, a region of more regular cell files that produce stem tissues.

Cell fate in the SAM is determined according to position, as demonstrated by numerous clonal analyses (Poethig 1987). Poethig et al. (1986) demonstrated that phytomers are clonally iterated, and others have shown that ~200 "founder" cells are recruited in the SAM for development of a phytomer (Steffensen 1968; Poethig 1984, 1987; McDaniel and Poethig 1988; Poethig and Szymkowiak 1995). Additionally, SAMs can be reprogrammed by responding to signals originating from lateral primordia (Irish and Karlen 1998; Je et al. 2016).

Leaves and the blade/sheath boundary

Maize leaves develop at the SAM flank. Leaf initiation is first detected by periclinal cell divisions in the L1 and L2 cell layers that correspond positionally to the emergent midrib along the medial axis of the incipient primordium (Sharman 1942; Esau 1965). Cell divisions spread bidirectionally and laterally around the entire circumference of the meristem flank to define an ensheathing, crescent-shaped ridge of founder cells known as the disc of insertion (Sharman 1942; Poethig 1984; Johnston et al. 2015). Additionally, lateral domains of the eventual distal blade and the proximal sheath are initiated during founder cell recruitment (Scanlon et al. 1996; Johnston et al. 2015). With initiation of each primordium, the sheathing leaf base surrounds the meristem and all younger leaves with characteristic overlapping lateral margins. Leaf margins trace back to overlapping domains of founder cells in the disc of insertion (Poethig and Szymkowiak 1995; Scanlon 2000). Delineation of the distinct founder cell population is a fundamental step in forming a boundary between the SAM and the developing leaf primordium (Johnston et al. 2015). Establishing and maintaining a boundary between the meristem and primordium is crucial to support stem cell homeostasis in the meristem and to ensure proper leaf morphology (Richardson and Hake 2019). From the disc of insertion, the primordium grows upward along the curvature of the meristem to

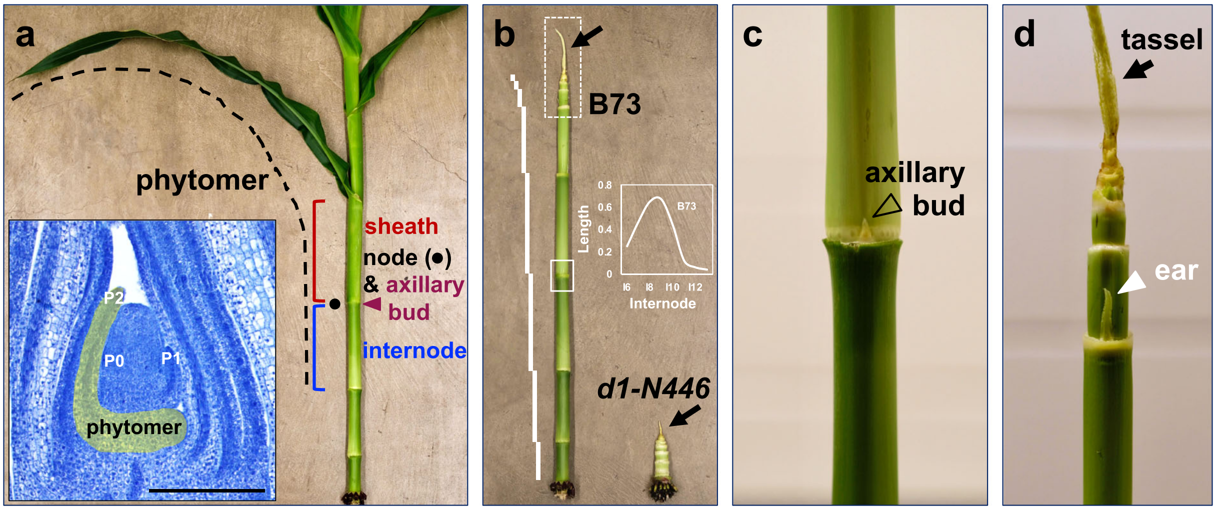

Fig. 1 The maize shoot. **a** Representation of the B73 phytomer consisting of internode, node with axillary bud, and leaf with sheath attached at the node and blade. Inset, longitudinal section of maize shoot apex depicting development of the phytomer pseudocolored in yellow. P0–P2, leaf primordia of plastochron stages. **b** Same plant shown in (**a**) but with all leaves removed. For comparison, a *d1-N446* mutant with leaves dissected to expose short internodes. White bars represent internode length plotted in graph for adult internodes 8–12 (inset), showing sigmoidal distribution of length across the shoot. Arrows denote tassels; solid outline box around an axillary bud shown in (**c**); dashed outline box around an ear shown in (**d**). **c** Axillary bud. **d** Tassel (arrow) and ear (arrowhead)

produce its distinctive hood shape (Sharman 1942). Uniform cell divisions throughout the primordium account for growth that is continuous from the circumference of the meristem flank with the base of the primordium (Poethig 1984; Sylvester et al. 1990; Poethig and Szymkowiak 1995).

As the primordium grows from the meristem periphery, the three axes that define the mature maize leaf—proximal-distal, medial-lateral, and adaxial-abaxial—become apparent (Fig. 2a, b). Immature leaf primordia can be separated into proximal-distal regions soon after primordium emergence, although regions of the sheath and blade are morphologically indistinguishable (Sylvester et al. 1990). The plastochron can be used to track early developmental events. For example, a primordium staged between P1 and P3 consists solely of cells fated to become distal blade (Poethig and Szymkowiak 1995). The sheath margins that originate from overlapping regions in the disc of insertion become fated as early as P3/P4 (Poethig and Szymkowiak 1995; Johnston et al. 2015). A pre-ligule inductive signal is thought to initiate from the medial plane of the primordium at the midrib and move laterally to each margin (Becraft and Freeling 1991). This signal sets up the formation of the pre-ligule band, the first morphological indicator of a boundary between the blade and sheath (Sylvester et al. 1990). The pre-ligule band becomes apparent around P6 due to higher rates of localized anticlinal cell division and a decrease in cell expansion (Sylvester et al. 1990; Moon et al. 2013). At about P7, epidermal cells at the base of the pre-ligule band subsequently undergo periclinal divisions to give rise to the emerging ligule (Fig. 2c) (Sylvester et al. 1990). Much less is known about auricle development; however, it has been suggested that the cells of the pre-ligule band and overlying cells, positioned immediately distal to the emergent ligule, differentiate to form auricle (Sylvester et al. 1996). In summary, leaf development is defined by three crucial stages that occur between P0 and P7: (1) formation of the crescent-shaped disc of insertion between the meristem and incipient primordium that will eventually produce a boundary between successive leaf whorls; (2) separation of sheath lateral margins at overlapping domains within the disc of insertion; and (3) delineation of the sheath, ligule, auricle, and blade along the proximal-distal axis.

At P8 and beyond, a developing maize leaf exhibits a gradient of differentiation until the leaf is fully mature: the distal tip consists of dividing cells that will produce specific cell types, whereas concomitantly cells at the proximal base are still acquiring identity of unique cell types (Marjeran et al. 2010; Wang et al. 2013).

Patterning along the adaxial-abaxial domains leads to differentiation of specific cell types on the dorsal and ventral sides of the leaf, respectively (Foster and Timmermans 2009). The adaxial epidermis is characterized by the ligule at the blade/sheath boundary and files of bulliform cells and macrohairs in the adult blade (Timmermans et al. 1998). Patterning of bulliform cell files, which may function in leaf rolling and blade angle, occurs in a region spanning 25–28 ml above the ligule during leaf expansion (Qiao et al. 2019). Adaxial-abaxial features are also expressed in internal tissues within polarized vascular bundles of xylem adaxially positioned to abaxial phloem and lignified sclerenchyma hypodermal cells that differentiate and connect to the adaxial epidermis at the midvein (Sharman 1942; Russell and Evert 1985). Along the medial-lateral axis of the blade, bundle sheath and mesophyll cells form rings around veins such that vascular bundles are separated in an ordered pattern of a bundle sheath cell, two mesophyll cells, and a bundle sheath cell (Hughes et al. 2019). Parallel to the midrib and leaf margins, densely arrayed lateral veins are separated from one another by two ranks of intermediate veins (Sharman 1942; Russell and Evert 1985; Hughes et al. 2019).

In the mature maize leaf, the proximal leaf sheath wraps around the stem and younger leaves to provide structural support (Kiesselbach 1949). The distal leaf blade projects away from the sheath at an angle, which maximizes light capture and gas exchange for photosynthesis. Sheath and blade tissues are joined at the ligule and auricle (Sharman 1942). The articulated auricle consists of a pair of triangular-shaped tissues that are widest at the leaf margin and relatively tapered at the midrib. The fringe-like ligule grows out from the epidermis at the base of the auricle (Fig. 2a) and may function in plant defense (Anderson et al. 2019; Kolkman et al. 2020). Interactions between the sheath, ligule, auricle, blade, and midrib significantly impact morphology of the shoot, primarily at the level of blade angle (Fig. 2b) (Sylvester et al. 1990; Becraft and Freeling 1991; Fowler and Freeling 1996; Harper and Freeling 1996; Strable et al. 2017; Tian et al. 2019). Because leaves are fundamental units of the canopy (Stewart et al. 2003), which is a key component of defining optimal plant density in row crops (Pepper et al. 1977; Lambert and Johnston 1978), regions of the leaf have undergone intense selection over the past century of maize breeding to alter its architecture (Tian et al. 2011; Tian et al. 2019). As a result, commercial maize hybrids exhibit a leaf architecture that is more upright in the upper nodes (Fig. 3) (Duvick 2005). Collectively, the constellation of traits that underlie leaf architecture has led to significant gains in grain yield in the last century of maize breeding (Pendleton et al. 1968; Duvick 2005; Tian et al. 2019).

Axillary meristems

Axillary meristems initiate in the axil of the leaf sheath at the stem node (Fig. 1b, c). Once formed, the meristem initiates a protective whorl of unexpanded leaves, producing an axillary bud. After the bud forms, its growth largely arrests when the bud enters a state of quiescence (Figs. 1a and 4a–c). In maize, an axillary bud can either remain dormant or grow based on its position along the shoot (Doebley et al. 1990). Axillary buds from the middle to upper nodes of the maize shoot grow in response to floral transition to produce a lateral branch that bears an ear. Buds of the basal nodes may remain dormant or grow into tillers. Tillers are akin to long branches that recapitulate phytomers of the main shoot. The capacity for a bud to remain dormant or become active is highly regulated by both internal and external stimuli (Whipple et al. 2011; Dong et al. 2019a). Maize concentrates resources to the primary shoot axis, resulting in the suppression of axillary bud growth. When overall apical dominance of the shoot is reduced, buds grow into tillers and compete with the shoot for resources (Fig. 4a) (Doebely et al. 1997).

Intercalary meristems

The maize stem is a cylindrical structure that provides mechanical support below the shoot apex (Peiffer et al. 2013). The stem is comprised of repeating units of node, where a leaf is attached, and internode, where stem growth occurs (Kiesselbach 1949). Shoot development is divided into juvenile and adult vegetative phases and a reproductive phase (Poethig 2003). During the juvenile phase in the first few weeks of shoot growth, juvenile internodes elongate very little, and as a result, node spacing is compact and the leaf whorl is tight. During transition to the adult phase and at the onset of the reproductive phase, adult internodes undergo substantial elongation, and spacing between nodes and leaves lengthens (Morrison et al. 1994; Poethig 2003). As a result, adult internodes constitute a significant proportion of overall plant height (Peiffer et al. 2014).

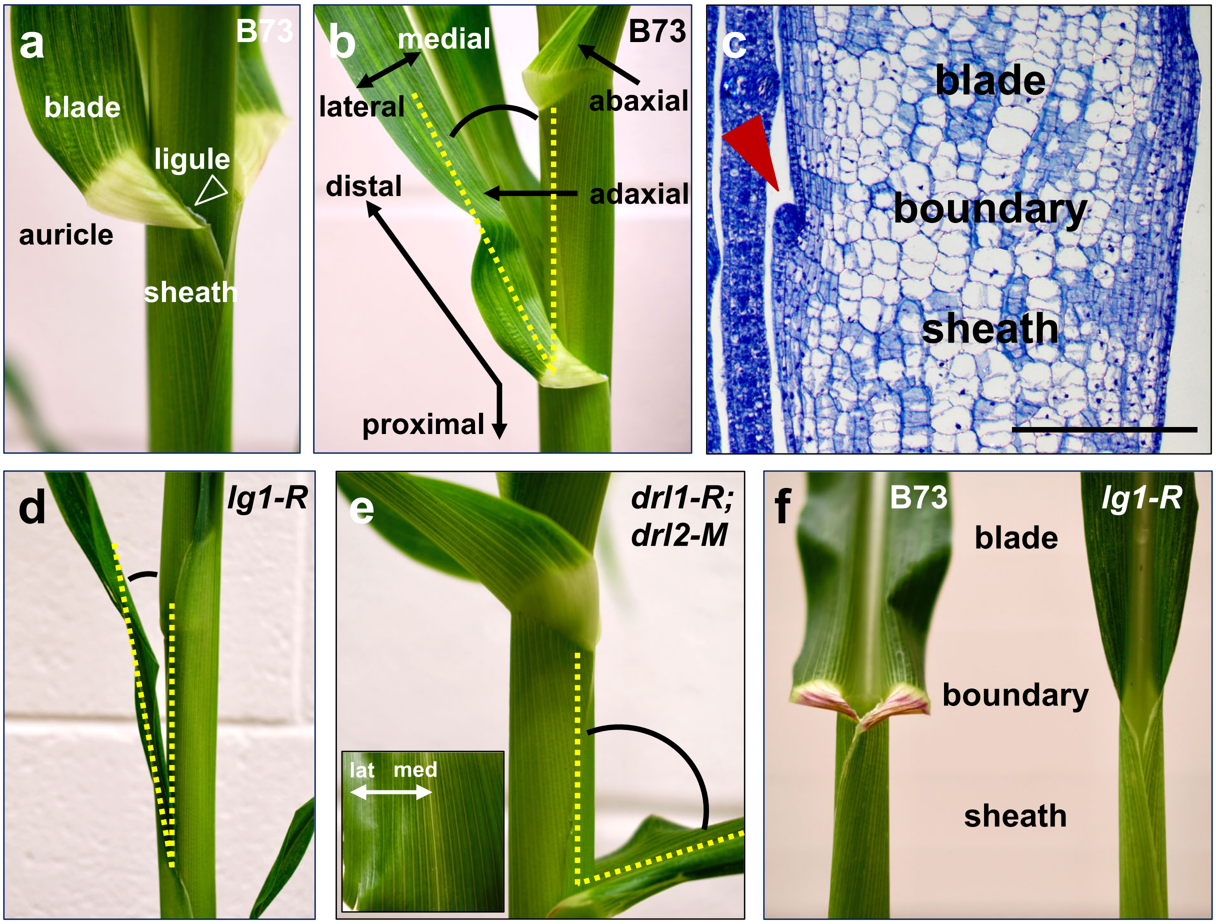

Fig. 2 Basic trait units that contribute to maize leaf architecture. **a** Blade/sheath boundary of a mature B73 leaf. **b** Side view of a mature B73 leaf denoting major axes and blade angle. **c** Longitudinal section through the P7 blade/sheath boundary with arrowhead denoting the adaxial ligule. Scale bar, 200 µm. **d** Side view of a mature *lg1-R* mutant leaf with blade angle labeled. Note the more upright blade with reduced angle. **e** Side view of a mature *drl1-R; drl2-M* double mutant leaf with blade angle labeled. Note the more horizontal blade with increased angle. Inset, adaxial view of *drl1-R; drl2-M* double mutant leaf blade showing reduced midrib. **f** Front view of B73 and *lg1-R* leaf showing deletion of the ligule and auricle and narrow blade in *lg1*. Images in **a**, **b**, and **d**–**f** are not to scale

Collectively, the variable individual growth rates and the differential elongation of each internode produce an overall sigmoidal pattern (Fig. 1b). This growth pattern importantly separates photosynthetic leaves along the shoot axis, improving efficient light capture and gas exchange, as well as elevating the tassel and ear to promote anemophily, i.e., wind pollination (Ku et al. 2015).

Internode elongation is controlled by the intercalary meristem positioned immediately above the node at the base of each internode (Tsuda et al. 2017). Node and internode tissues are derived from a shared cell population from the shoot apex (Jorhi and Coe 1983). Intercalary meristems regulate internode elongation independent of the SAM; additionally, growth is not synchronous for all internodes (Morrison et al. 1994). Vascular tissue develops in association with internodes and, in transverse section, appears separate and dispersed throughout the stem (Tsuda et al. 2017).

Genes that shape shoot architecture of maize: an overview

Maize geneticists have been identifying, collecting, and maintaining mutants with altered shoot architecture for over 100 years (Fig. 5) (Neuffer et al. 1997). Hundreds of mutations affect nearly every organ and cell type in

Fig. 3 Canopy and leaf structure in field-grown maize. **a** Maize hybrid field showing the difference between lower (more sprawling) and upper (more upright) leaf canopies separated by dotted line. **b** Three commonly used maize inbred lines, B73, Mo17, and W22, denoting the position of the upper ear (arrow) and the upper three leaves (arrowhead). Note the shift in blade angle of the upper leaves in B73

maize; cloning numerous loci has provided a wealth of information about the functional and agronomic properties of maize. Some of the genes described in maize are unique to the grasses and therefore underscore the importance of maize as a model for discovering new developmental genes with no orthologs in eudicots. A current, albeit far from comprehensive, survey of the characterized loci that impact maize shoot architecture

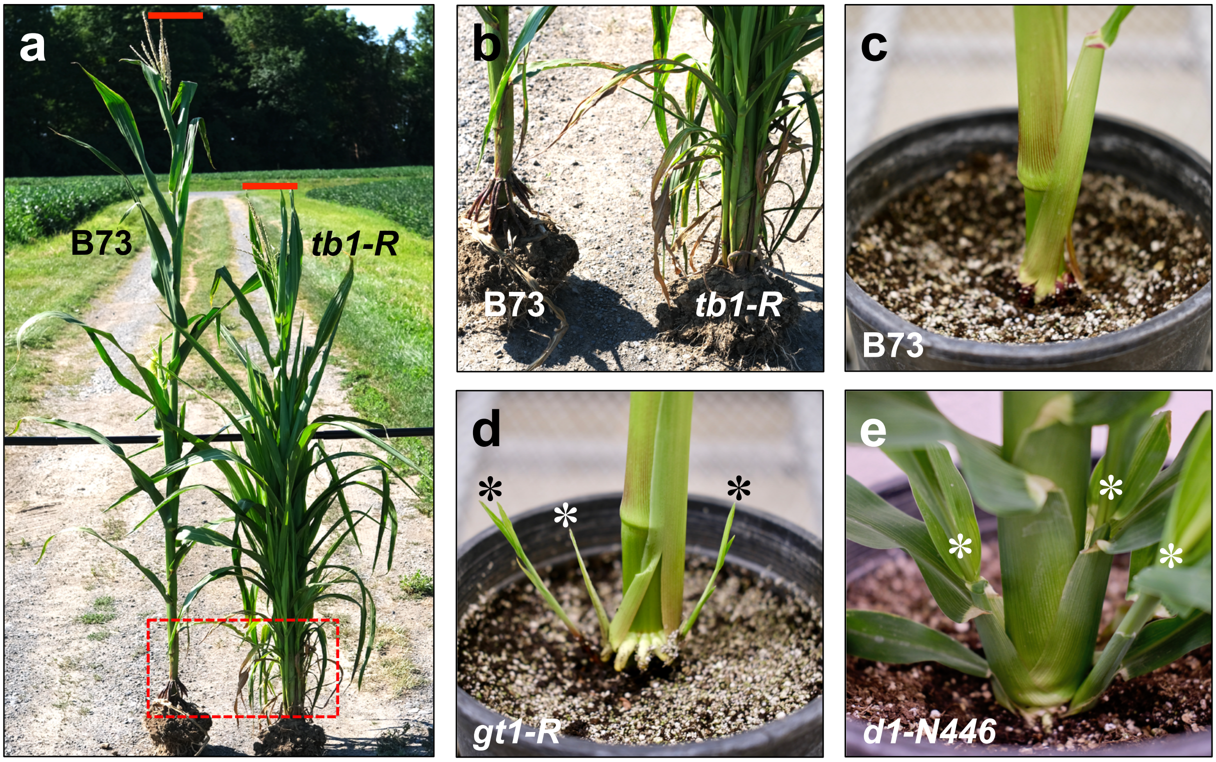

Fig. 4 Tiller growth in maize. **a** Field-grown B73 and *tb1-R* mutant (backcrossed in B73). Red bars denote plant height. Boxed region shown in (**b**). **b** High tiller number from outgrowth of basal axillary buds in *tb1-R*, whereas B73 axillary bud growth is suppressed. **c** B73 with suppressed axillary bud growth. **d** *gt1-R* mutant with active axillary bud growth (leaves have been removed). **e** *d1-N446* mutant with active axillary bud growth (leaves have been removed)

are summarized in Table S1 (in the supplementary material). Of the cloned genes, approximately 73% have significant associations with polymorphisms that explain natural variation in vegetative traits (Table S1). A common theme that emerges from mutant analyses is that many of the genes are pleiotropic (Table S1). Untangling the individual phenotypic components of pleiotropy is crucial if developmental genes are to be utilized to improve shoot architecture in maize. The following sections highlight several mutant and gene characterization studies with the primary focus on components of shoot architecture: shoot meristem, leaf, axillary bud, and stem.

Shoot apical meristem establishes and maintains shoot development

Shoot apical meristem maintenance

One of the best characterized plant transcription factors that regulates meristem activity is *KNOTTED1* (*KN1*), the founding member of the plant *KN1-LIKE HOMEOBOX* (*KNOX*) gene family (Hake et al. 1989; Vollbrecht et al. 1991). Some dominant, gain-of-function alleles of *KN1* produce “knots” by ectopically expressing *KNOX* genes along lateral veins in the blade (Fig. 5e, inset) (Freeling and Hake 1985; Sinha and Hake 1990). These knots are reprogrammed cells that continue to divide as if they were meristematic and eventually differentiate into sheath and auricle rather than blade. In contrast, strong loss-of-function *kn1* mutants show failure to initiate or maintain the SAM (Kerstetter et al. 1997). Loss-of-function alleles of *KN1* are sensitive to genetic modifier loci in various maize backgrounds (Vollbrecht et al. 2000). In permissive backgrounds, vegetative development is mostly normal, but development of the tassel and ear is severely compromised; in restrictive backgrounds, the SAM cannot be maintained, resulting in a limited (abortive) shoot (Vollbrecht et al. 2000). *KN1* transcripts accumulate throughout the SAM and developing stem, but not in the incipient primordium (P0), developing primordia (>P1), or the L1 layer of the meristem proper (Jackson et al. 1994). KN1 protein, however, traffics cell-to-cell via plasmodesmata and is found in the base of young leaf primordia and in the L1 (Lucas et al. 1995; Jackson 2002; Kim et al. 2002; Xu et al. 2011). By leveraging mRNA sequencing (RNA-seq) in *kn1* loss-of-function mutant immature ears and tassels and SAMs with chromatin immunoprecipitation paired sequencing (ChIP-seq) of normal developing ears, Bolduc et al. (2012b) found that KN1 binds to over 5000 loci in the maize genome and modulates the expression of around 650 genes. Many of KN1 target genes encode transcription factors and components of phytohormone pathways, which include auxin and gibberellin (Bolduc and Hake 2009; Bolduc et al. 2012b). Such candidate genes could be well-suited for improving maize shoot architecture.

Genetic analysis can be complicated by functional redundancy (Kafri et al. 2009). *KNOX* genes exhibit redundancy that is hierarchical: *KNOX* family members cannot substitute for *KN1* function, but *knox* mutants enhance *kn1* in a dose-dependent manner (Bolduc et al. 2014). A loss-of-function mutation in the *KNOX* gene *ROUGH SHEATH1* (*RS1*) does not condition a single mutant phenotype. However, in combinations with a *kn1* loss-of-function allele, *rs1* heterozygous or homozygous mutants interact synergistically, resulting in a limited shoot (Bolduc et al. 2014). *KN1* and *RS1* transcripts accumulate in overlapping domains in the shoot apex (Jackson et al. 1994), and KN1 and RS1 proteins bind and modulate the expression of similar genes (Bolduc et al. 2014). Furthermore, KN1 can heterodimerize with transcription factors that co-localize *in planta*, such as BELL1-like homoeobox12 (BLH12) and BLH14 (Tsuda et al. 2017). Neither *blh12* nor *blh14* single mutants display an obvious meristem phenotype; however, *blh12*; *blh14* double mutants have reduced SAM height and width, as well as suppressed axillary meristem maintenance (Tsuda et al. 2017), similar to *kn1* loss-of-function alleles (Kerstetter et al. 1997; Vollbrecht et al. 2000). Whether meristem phenotypes of *blh12; blh14* double mutants are sensitive to modifier loci in diverse genetic backgrounds remains to be explored.

Limited shoot phenotypes also arise from mutations in genes that participate in small RNA, phytohormone, metabolite, and nutrient pathways. Small RNAs (sRNAs) are crucial regulators of shoot development and have been linked to SAM establishment and maintenance (Klesen et al. 2020). Strong alleles of *RAGGED SEEDLING2*/*ARGONAUT7* and *RNA-DEPENDENT RNA POLYMERASE6* machinery components of sRNA production give rise to seedlings with limited shoots that terminate shortly after germination (Douglas et al. 2010; Petsch et al. 2015). Additional shoot abortive phenotypes can be teased out by generating double mutants between sRNA biosynthesis components, such as by

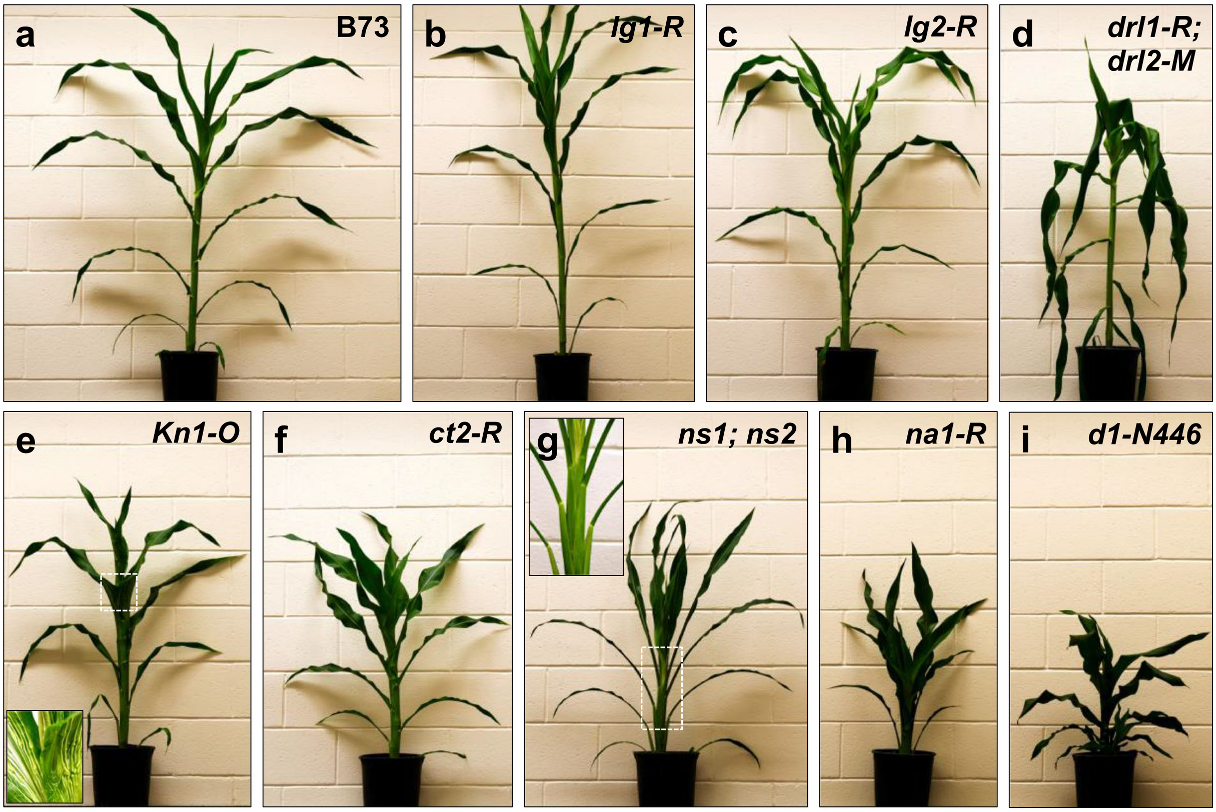

Fig. 5 Variation in shoot architecture caused by mutations in key developmental regulators. All mutants are backcrossed in B73, were greenhouse grown, and were imaged at the same stage of maturity. **a** B73. **b** *liguleless1-R* (*lg1-R*). **c** *liguleless2-R* (*lg2-R*). **d** *drooping leaf1-R; drooping leaf2-M* (*drl1-R; drl2-M*). **e** *Knotted1-O* (*Kn1-O*); inset, boxed region showing knots on *Kn1-O* leaves. **f** *compact plant2-R* (*ct2-R*). **g** *narrow sheath1; narrow sheath2* (*ns1; ns2*); inset, boxed region showing narrow sheaths and lower blade of *ns1; ns2* **h** *nana plant1-R* (*na1-R*) **i** *dwarf plant1-N446* (*d1-N446*)

stacking mutant alleles in *LEAFBLADELESS1/SUPPRESSOR-OF-GENE-SILENCING3* and *DICER-LIKE4* (Petsch et al. 2015). Plant hormones regulate development of specific regions of the SAM (Hepworth and Pautot 2015). The phytohormone cytokinin promotes proliferation of meristematic cells (Giulini et al. 2004; Kurakawa et al. 2007). The maize cytokinin biosynthesis gene *LONELY GUY7* (*ZmLOG7*) is expressed in the meristem tip, and *Zmlog7* mutants display a limited shoot (Knauer et al. 2019). Additionally, SAM maintenance is promoted by certain metabolites. *BLADEKILLER1* (*BLK1*) encodes a THIAMINE BIOSYNTHESIS2 (THI2) enzyme; *blk1/thi2* mutants display a progressive reduction in meristem height that leads to an abortive adult shoot (Woodward et al. 2010). *blk1/thi2* mutant shoots can be rescued with exogenous thiamine, highlighting the importance of thiamine in meristem maintenance (Woodward et al. 2010).

Shoot apical meristem size

A core pathway at the heart of stem cell homeostasis in plants is the *CLAVATA* (*CLV*)-*WUSCHEL* (*WUS*) feedback loop, which consists of peptide ligands, receptors, and transcription factors that operate in different regions of the meristem (Somssich et al. 2016). Briefly, stem cell fate is promoted by WUS, a homeodomain transcription factor. *WUS* transcripts accumulate in cells situated between the central and rib zones, but WUS protein moves into the central zone where activates the expression of *CLV3*, which encodes a secreted peptide. The central zone-derived CLV3 peptide ligand binds to the leucine-rich repeat receptor-like kinase (LRR-RLK) CLV1. *CLV1* is expressed beneath the central zone, whereas transcripts for the LRR-RL protein (LRR-RLP) encoding *CLV2* gene accumulate throughout the SAM. Both CLV1 and CLV2 are required to repress WUS expression. The CLV-WUS pathway is conserved in maize, as highlighted by genetic analyses that have mainly focused on meristems in inflorescences (Wu et al. 2018). Mutations in the *CLV2* in ortholog *FASCIATED EAR2* (*FEA2*) significantly enlarged the SAM (Taguchi-Shiobara et al. 2001; Je et al. 2018), as do mutations in a *CLV3* ortholog, maize *CLV3/EMBRYO SURROUNDING REGION-RELATED7* (*ZmCLE7*; Rodriguez-Leal et al. 2019). Interestingly, a mutant allele of the maize *CLV1* ortholog *THICK TASSEL DWARF1* (*TD1*; Bommert et al. 2005) reduces SAM size, which is consistent with the observation that *td1; kn1* double mutants cannot rescue the limited shoot of *kn1* loss-of-function mutants (Lunde and Hake 2009). Maize *WUS1* (*ZmWUS1*) and *ZmWUS2* are putative *WUS* co-orthologs (Nardmann and Werr 2006); however, laser microdissection, single-cell RNA sequencing, and RT-PCR analyses of the maize vegetative shoot apex did not detect cells that accumulated *ZmWUS1/2* transcripts, suggesting that the canonical *CLV-WUS* pathway appears to have been bypassed in the maize vegetative SAM (Knauer et al. 2019; Satterlee et al. 2020).

Enlargement of the SAM results from mutations in several key signaling components of stem cell homeostasis: the LRR-RLP encoding *FEA3* (Je et al. 2016), the *PERIANTHIA* (*PAN*) ortholog *FEA4* (Pautler et al. 2015), the α-subunit (Gα) of a heterotrimeric GTP binding protein encoded by *COMPACT PLANT2* (*CT2*; Bommert et al. 2013), the β-subunit (Gβ) of a heterotrimeric GTP binding protein (Wu et al. 2020), the maize ortholog of rice *FON2-LIKE CLE PROTEIN1* (*ZmFCP1*; Je et al. 2016; Knauer et al. 2019), the *CORYNE* ortholog *ZmCRN* (Je et al. 2018), and a cytokinin type A response regulator encoding gene *ABERRANT PHYLLOTAXY1* (*ABPH1*; Giulini et al. 2004). Additionally, a subset of genes that regulate meristem size also impact adult shoot architecture. Mutant *abph1* plants additionally display decussate phyllotaxy due to broadening of the SAM (Giulini et al. 2004). In certain genetic backgrounds, *fea4* mutants have reduced shoot stature (Pautler et al. 2015). FEA4 interacts with MALE STERILE CONVERTED ANTHER1 (MSCA1), a glutaredoxin protein (Yang et al. 2015). Mutant *mcsa1* plants exhibit reduced SAM size, whereas misexpression of *MCSA1* in the dominant *Abph2* mutant increases SAM size and results in decussate phyllotaxy (Yang et al. 2015), similar to *abph1* mutants. In addition to a larger SAM, *ct2* mutants display an overall reduced shoot stature, and leaf blades are relatively wider and more upright compared to non-mutants (Fig. 5f) (Bommert et al. 2013; Wu et al. 2018).

Besides core signaling pathways that operate within stem cells of the SAM, signaling feedback to the SAM from leaf primordia is crucial for balancing stem cell proliferation. The ZmFCP1 peptide is produced in lateral primordia and signals to its receptor FEA3, which is localized to cells that subtend meristem central and core regions (Je et al. 2016). Maize *YABBY* (*YAB*)/*CRABS CLAW* co-orthologous genes *DROOPING LEAF1* (*DRL1/YAB2*) and *DRL2/YAB7* are expressed in the incipient and initiating leaf primordia, but not in the SAM or subtending stem (Strable et al. 2017). However, meristem size is reduced in *drl1* and *drl2* single mutants and in a dose-dependent manner in *drl1; drl2* double mutants (Strable et al. 2017). These observations indicate the *DRL* genes function non-cell autonomously to regulate stem cell proliferation and suggest that activity levels and/or interactions among gene products from the loci are important to their function (Strable et al. 2017).

Micronutrients can impact SAM size. Mutations in an aquaporin encoding gene, *TASSEL-LESS1* (*TLS1*), confer a progressive reduction of SAM size that can lead to complete meristem consumption (Durbak et al. 2014). *TLS1* is necessary for boron transport during shoot development, as *tls1* mutants have low boron content. Furthermore, *tls1* meristem defects can be rescued by boron fortification, underlining the importance of the bioavailability of this micronutrient in soils (Durbak et al. 2014).

Shoot apical meristem determinacy

Maize shoots become determinate, or limited in growth, when the SAM differentiates into the tassel (Fig. 1b, d). Determinate meristems produce a limited number of organs before vegetative growth terminates and reproductive growth is acquired. Indeterminate activity of a meristem, on the other hand, results in prolonged growth due to sustained self-renewal of stem cells and continuous initiation of new organs. For a majority of maize lines, the SAM produces a predictable range of leaf primordia during vegetative growth prior to the reproductive phase. In temperate maize inbred lines, the number of leaves initiated before the SAM converts to a tassel is background dependent and can range from 13 to 22 leaves, in either short- or long-day photoperiods (Russell and Stuber 1983; Meng et al. 2011). By comparison, early-flowering Gaspé Flint maize produces 6–

8 leaves before flowering in either short- or long-day photoperiods, whereas photoperiod-sensitive tropical maize CML436 produces 26–30 leaves under long days and 18–20 leaves in short days (Meng et al. 2011).

Leaf number is a major component of shoot architecture in maize. Leaves along the shoot can be divided into two groups: those below the uppermost (primary) ear, where the canopy is relatively more sprawling, and those above the uppermost ear where the canopy is relatively more upright (Fig. 4a) (Vlăduțu et al. 1999; Flint-Garcia et al. 2005; Li et al. 2016). The proportion of lower and upper canopy leaves importantly enhances photosynthetic efficiency across pre- and post-anthesis stages of shoot development, respectively (Duncan 1971; Zhang et al. 2017). Maize is rich in natural variation for total leaf number (Russell and Stuber 1983; Flint-Garcia et al. 2005; Li et al. 2016). For example, in a set of 100 diverse inbred lines, total leaf number ranged from 7 to 19, leaf number below the primary ear ranged from 3 to 10, and leaf number above the primary ear ranged from 3 to 8 (Flint-Garcia et al. 2005). Most modern, temperate maize lines are photoperiod insensitive (Colasanti and Muszynski 2009), and developmental (i.e., endogenous) programs regulate vegetative to floral transition (Irish and Nelson 1991; Irish and Jegla 1997). Genetic studies on leaf number are largely deduced from variation in flowering time. This intricate that link between flowering time and total leaf number is due to the fact that floral transition converts the vegetative SAM into a tassel; thus, floral transition marks an end to leaf production.

Some genes regulate leaf initiation rate and total leaf number without significantly changing flowering time. Mutations in the *VIVIPAROUS8* (*VP8*) gene increase leaf initiation rate especially during early shoot growth, such that at any same stage of growth, *vp8* mutants display more leaves when compared with normal siblings (Evans and Poethig 1997). Interestingly, *vp8* have more leaves at maturity compared with normal sibling plants, and yet both genotypes flower simultaneously (Evans and Poethig 1997). *VP8* encodes a membrane-localized peptidase related to the *Arabidopsis* ALTERED MERISTEM PROGRAM1, which may be important for abscisic acid (ABA) homeostasis (Suzuki et al. 2008). Similarly, *terminal ear1* (*te1*) mutants initiate leaves more frequently compared with normal sibling plants, as well as alters phyllotaxy (Veit et al. 1998). *TE1* encodes an RNA binding protein (Veit et al. 1998), for which the target RNAs remain uncharacterized. The SQUAMOSA PROMOTER BINDING (SBP) transcription factor encoding *UNBRANCHED2* (*UB2*), *UB3*, and *TASSELSHEATH4* genes function redundantly in the central domain of the SAM to balance the rate of cell differentiation during lateral primordia initiation (Chuck et al. 2014). This balance is lost in *ub2*; *ub3*; *tsh4* triple mutants, where an abbreviated plastochron index increases total leaf number without altering flowering time (Chuck et al. 2014). Mutants with shifts in total leaf number and a subsequent shift in flowering time also greatly alter shoot architecture and are covered elsewhere (Colasanti and Muszynski 2009).

Natural variation of the SAM morphology

Exploring natural diversity of SAM morphology and understanding its underlying genetic architecture requires phenotypic analysis at microscopic scale. A handful of studies in maize have utilized biparental recombinant inbred line (RIL) and maize diversity panels to quantify SAM width, height, arch length, midpoint width, height of P1, plastochron internode length, and cell number of the L1 layer along the length of meristem arc (Thompson et al. 2014, 2015) or to model the SAM as a paraboloid (Leiboff et al. 2015). Interestingly, candidate genes in quantitative trait locus (QTL) regions that associate with SAM size and morphology do not represent genes that have been characterized previously to regulate SAM activity, suggesting that genes crucial to SAM maintenance do not necessarily contribute to natural variation of SAM morphology (Thompson et al. 2014, 2015; Leiboff et al. 2015). Curiously, SAM size and flowering time are correlative, such that maize lines with large SAMs associate with earlier flowering (Thompson et al. 2015; Leiboff et al. 2015). As expected, maize lines with larger SAMs also correlate with fewer nodes and reduced plant height (Leiboff et al. 2015). The height of the SAM relates to stem diameter, whereas SAM radius is associated with the height of the primary ear along the plant axis (Leiboff et al. 2015). These SAM "microphenotypes" were recently utilized in genomic prediction models as a way to optimize trait selection in breeding programs (Yu et al. 2020). Furthermore, laser microdissection coupled with transcriptomic analysis of SAM functional domains uncovered gene regulatory circuits that also regulate adult shoot traits (Knauer et al. 2019).

Leaf architecture is governed by multiple interacting traits

Blade/sheath boundary and blade angle

Maize leaves play a significant role in defining overall shoot architecture. The blade/sheath boundary, articulated by auricle and ligule tissues, impacts blade angle and is a crucial feature of the maize leaf. Dominant, gain-of-function mutations in *KNOX* genes, such as in the transcriptional regulators *KN1*, *RS1*, *GNARLEY1* (*GN1*), *LIGULELESS3* (*LG3*), and *LG4*, have highlighted a key role for these genes in specifying proximal cues during the establishment of the blade/sheath boundary (reviewed in Bolduc et al. 2012a). Leaves of neomorphic *Knox* mutants produce ectopic blade/sheath boundaries due to the mis-regulation of *KNOX* genes during leaf development (Fig. 5e). Cells in the blade that mis-express *KNOX* genes take on a sheath-like identity; the juxtaposition of blade and ectopic sheath-like cells produces an ectopic boundary, where ligule-/auricle-like tissues subsequently develop (Smith et al. 1992; Jackson et al. 1994; Freeling and Hake 1985; Becraft and Freeling 1994; Schneeberger et al. 1995; Fowler et al. 1996; Muehlbauer et al. 1999; Foster et al. 1999). A dominant, gain-of-function allele of the *WAVY AURICLE IN BLADE1* (*WAB1*) gene similarly displaces auricle-like tissue distally into the blade due to *WAB1* mis-expression (Hay and Hake 2004; Lewis et al. 2014). Interestingly, ligule tissue does not develop with the ectopic auricle of *Wab1* mutants, and *kn1* loss-of-function mutants have no effect on *Wab1*, suggesting *WAB1* functions independent of the *KNOX* pathway (Hay and Hake 2004). *WAB1* encodes a TCP (TEOSINTE BRANCHED1, CYCLOIDEA, PCF) transcription factor that is not normally expressed during leaf development (Lewis et al. 2014). The *HAIRY SHEATH FRAYED1* (*HSF1*) gene encodes a maize *HISTIDINE KINASE1* receptor for cytokinin (Muszynski et al. 2020). Semi-dominant *Hsf1* mutants initiate proximal sheath-like prongs along the distal blade margin due to cytokinin hypersignaling, suggesting that ectopic activation of cytokinin response is sufficient for activating at least some leaf patterning programs (Bertrand-Garcia and Freeling 1991; Muszynski et al. 2020). Deciphering the downstream genetic networks for *WAB1* and *HSF1* will be important next steps in understanding how they regulate leaf patterning.

Once the blade/sheath boundary is established, the ligule and auricle region is elaborated (Fig. 2a, b). A recessive, loss-of-function class of *liguleless* (*lg*) mutants has illuminated the importance of specifying the pre-ligule band (~P6) and proper ligule outgrowth (~P7/P8) (Sylvester et al. 1990; Moreno et al. 1997; Walsh et al. 1998). Mutant *lg1* plants were originally identified by their acutely upright leaves (Figs. 2d and 5b) (Emerson 1912a). Ligule and auricle tissues are not established in *lg1* mutants, and yet despite lacking these tissues, the blade and sheath are separated by a clear boundary (Fig. 2d, f) (Becraft et al. 1990; Sylvester et al. 1990; Becraft and Freeling 1991; Moreno et al. 1997; Li et al. 2017). Genetic mosaic analysis indicates *LG1* functions cell autonomously in an early step of ligule and auricle initiation (Becraft and Freeling 1994). *LG1* encodes a grass-specific SBP transcription factor, and *LG1* transcripts and LG1 protein accumulate in the pre-ligule band and throughout the initiating ligule (Moreno et al. 1997; Moon et al. 2013; Johnston et al. 2014; Lewis et al. 2014). *LG1* is mis-expressed in dominant, gain-of-function *Wab1* mutants in domains of the leaf that also ectopically express *WAB1* (Lewis et al. 2014). However, in *wab1* loss-of-function mutants, expression of *LG1* at the pre-ligule band is unaltered, suggesting that although WAB1 is sufficient to regulate *LG1* expression, it is not required (Lewis et al. 2014). This observation leaves open the possibility that *LG1* is regulated by a different TCP transcription factor. Additional insight into *LG1* regulation was provided through fine mapping and cloning a QTL for leaf angle, *qLA2*. Cloning *qLA2* uncovered a maize gene orthologous to rice *INCREASED LEAF INCLINATION1* (*ZmILI1*; Ren et al. 2020). ZmILI1 is a helix-loop-helix (HLH) protein that has affinity for the *LG1* promoter (Ren et al. 2020). Because HLH proteins lack a basic domain required for DNA binding (Ruzinova and Benezra 2003; Zhang et al. 2009), but form heterodimers with bHLH proteins, it will be important to understand the mechanism for which ZmILI1 associates with target promoters. One such mechanism could be through heterodimerization with maize *ILI1 BINDING bHLH1-1* (*ZmIBH1-1*), a gene that underlies the *qLA2-1* leaf angle QTL and encodes a bHLH protein with a basic DNA binding domain (Cao et al. 2020). However, whether ZmILI1 and ZmIBH1-1 physically interact to regulate *LG1* expression currently remains to be reported.

The recessive, loss-of-function *lg2* mutant has upright leaves due to incomplete patterning of the auricle and ligule (Fig. 5c). In contrast with *lg1* mutants, a reduced ligule and auricle develop at the margin but not at the midrib in *lg2* mutants, and the blade/sheath boundary is shifted along the proximal/distal axis (Harper and Freeling 1996). Although the expression pattern of *LG2* has not been reported by RNA *in situ* hybridization, *LG2* is expressed broadly in the SAM and young primordia (i.e., ≤P4), earlier than *LG1* expression, according to transcriptomic analyses of laser microdissected sections of the maize shoot apex (Knauer et al. 2019; Leiboff et al. 2020). Genetic mosaic analysis suggests that *LG2* functions non-cell autonomously (Harper and Freeling 1996). *LG2* encodes a grass-specific TGA-class bZIP transcription factor (Walsh et al. 1998). Genetic interaction analysis between *lg1* and *lg2* mutants indicates the two genes work in the same pathway, with *LG2* potentially functioning earlier than *LG1* to establish the position of the blade/sheath boundary (Harper and Freeling 1996). In support, *LG2* is expressed early and is bound by KN1, whereas *LG1* is expressed late and is not a KN1 target (Bolduc et al. 2012b). The *Arabidopsis* TGA-class bZIP protein PAN can interact with organ boundary proteins BLADE-ON-PETIOLE1 (BOP1) and BOP2 in yeast and *in planta* (Hepworth et al. 2005; Xu et al. 2010). It is tempting to speculate that LG2 may similarly form heterodimers with other transcriptional regulators to establish and/or pattern the blade/sheath boundary.

Connecting developmental events, such as patterning with growth, along the proximodistal, mediolateral, and adaxial-abaxial axes of the leaf is critical to understanding leaf architecture. The semi-dominant *Liguleless narrow* (*Lgn*) mutant links development of the blade/sheath boundary with medial/lateral growth (Moon et al. 2013). Leaf width is significantly reduced in *Lgn* mutant leaves, and the ligule develops only at the midrib along the medial plane of an otherwise poorly patterned blade/sheath boundary (Moon et al. 2013). *LGN* encodes a serine/threonine kinase, in which the kinase activity is severely compromised in *Lgn* mutants (Moon et al. 2013). Expression levels of *LG1* and *LG2* are low in *Lgn* mutants; moreover, genetic interactions between *lg1* and *lg2* with *Lgn* are synergistic (Moon et al. 2013). Similar to the early expression pattern of the KN1-target *LG2*, *LGN* transcripts accumulate early and ubiquitously in the leaf, and *LGN* is bound by KN1 (Bolduc et al. 2012b; Moon et al. 2013). Thus, a LGN-mediated phosphorylation cascade may regulate a ligule signal from the medial midrib laterally to the leaf margin. Because TGA-class bZIP activity can be regulated post-translationally by phosphorylation (Schütze et al. 2008), it will be important to know if LG2 and LGN physically interact and if LGN can phosphorylate LG2. *Lgn* is sensitive to genetic modifier loci, as noted by its variable expressivity in various genetic backgrounds and response to temperature (Buescher et al. 2014). Cloning a large effect modifier QTL for *Lgn*, *Sympathy for the ligule* (*Sol*), uncovered a homolog of *Arabidopsis ENHANCED DISEASE RESISTANCE4* (*EDR4*), suggesting *Lgn* may trigger a MAP kinase signaling cascade, whereas *Sol/EDR4* may suppress this cascade (Anderson et al. 2019).

Cloning of the semi-dominant *Semidwarf3* (*Sdw3*) mutant revealed the importance of the phytohormone ethylene in regulating growth at the blade/sheath boundary (Li et al. 2020a). *SDW3* encodes a maize ethylene biosynthesis enzyme, ZmACS7, and the *Sdw3* mutant overproduces ethylene (Li et al. 2020a). Mutant *Sdw3* plants have a wide leaf angle due to growth-related expansion of the auricle at the margin (Li et al. 2020a). The phytohormone brassinosteroid plays a key role in regulating blade/sheath boundary development in maize. RNA interference (RNAi) downregulation of maize *BRASSINOSTEROID INSENSITIVE1* (*ZmBRI1*) homologs gave rise to leaves with highly reduced ligule and auricle tissues (Kir et al. 2015). Developing ligule and auricle cells accumulate a fluorescent protein marker of brassinosteroid activity, and *Zmbri1*-RNAi lines have reduced accumulation of this fluorescent protein marker (Kir et al. 2015). Mutations in the brassinosteroid biosynthesis genes *BRASSINOSTEROID DEPENDENT1* (*BRD1*; Makarevitch et al. 2012) and *NANA PLANT1* (*NA1*; Hartwig et al. 2011) disrupt margin-to-margin expansion of the ligule and result in acute involution of auricle tissue (Makarevitch et al. 2012; J. Strable, unpublished data). Furthermore, *na1* mutants have markedly upright leaves (Fig. 5h). Laser microdissection of the developing ligule and auricle region coupled with RNA sequencing of these tissues found that *BRD1* and a maize homolog of *Dwarf11* were both upregulated in preligule tissue, further implicating brassinosteroid biosynthesis in patterning the blade/sheath boundary (Johnston et al. 2014). Interestingly, the rice HOMEOBOX1 (OSH1) transcription factor dampens the brassinosteroid pathway by upregulating the expression of *CYTOCHROME P450* (*CYP*) brassinosteroid catabolic genes. Furthermore, *cyp*-RNAi lines have patterning defects of the blade/sheath boundary (Tsuda et al. 2014). In maize, *CYP* genes are direct targets of KN1 and are upregulated in *Kn1* dominant, gain-of-function mutants (Bolduc et al. 2012b). These observations suggest that KNOX proteins regulate *CYP* genes to maintain low levels of brassinosteroid in some cells of the developing leaf, which may be necessary to establish the blade/sheath boundary in the cereals (Tsuda et al. 2014).

The maize *DRL* genes regulate leaf development along all three axes (Strable et al. 2017). The syntenic paralogous *DRL1* and *DRL2* genes are co-orthologous to *Arabidopsis CRABS CLAW* and rice *DROOPING LEAF* genes (Bowman and Smyth 1999; Yamaguchi et al. 2004; Strable et al. 2017). The moderate droopy leaves of *drl1* single mutants are enhanced in *drl1; drl2* double mutants (Figs. 2e and 5d). Not only is the midrib markedly reduced by mutations in the *DRL* genes, auricle tissue is distally expanded at the presumptive midrib (Fig. 2e, inset), and hypodermal sclerenchyma cells are underdeveloped (Strable et al. 2017). The blade angle traits in *drl* mutants, along with narrower blades and elongated internodes, were noted previously (Strable et al. 2017) as reminiscent of rice brassinosteroid mutants (Yamamuro et al. 2000; Zhang et al. 2009; Sun et al. 2015), which either have elevated brassinosteroid levels or enhanced brassinosteroid signaling. In summary, the interaction of midrib, auricle, and supportive sclerenchyma cells are key factors that control blade angle, and all are under the control of the *DRL* genes (Strable et al. 2017).

Leaf morphometrics: length, width, and size

Leaf morphology and size greatly impact photosynthetic capacity. Leaves are the primary photosynthetic organ of the maize shoot and play crucial roles in shoot growth and grain filling (Duncan 1971; Stewart et al. 2003; Lee and Tollenaar 2007; Ma et al. 2014; Zhao et al. 2015; Zhang et al. 2017). Genetic analysis of leaf development has identified several key regulators of blade length, blade width, and sheath length. Many of the genes encode transcription factors that also regulate hormone biosynthesis, signaling, or response pathways. Dominant *Kn1-N* mutants have wider and shorter leaf blades and shorter sheaths. This is due in part to KN1 directly activating the gibberellin catabolic gene *GA2 OXIDASE1* (*GA2ox1*; Bolduc and Hake 2009). Mutations in a gibberellin biosynthesis gene *DWARF PLANT3* (*D3*) result plants with short stature and wide blades (Winkler and Helentjaris 1995), and *d3* mutants have low levels of bioactive gibberellin (Phinney and Spray 1982; Fujioka et al. 1988; Winkler and Helentjaris 1995). Transgenic maize that overexpress a gibberellin anabolic-related gene, *GA20ox5* homolog, has increased blade length along with increased plant height (Nelissen et al. 2015). Analysis of KN1 target genes by ChIP-seq found that KN1 preferentially binds adjacent to genes in the gibberellin pathway, including *GA2ox1* and *GA20ox5*, among other phytohormone pathways (Bolduc et al. 2012b). In *Rs1* and *Gn1* mutants, sheath length is similarly reduced, and blade length tends to be unchanged (Becraft and Freeling 1994; Foster et al. 1999). *GA20ox5* is mis-expressed in recessive *kn1* mutants, and *GA2ox1* is mis-expressed in dominant *Kn1*, *Rs1*, *Gn1*, and *Lg3* mutant leaves, where enrichment of KNOX binding overlaps KN1 target loci (Bolduc and Hake 2009; Bolduc et al. 2014). These observations suggest that *KNOX* genes have redundant functions in regulating hormonal response. In support, loss-of-function mutations in *RS2*, a negative regulator of *KNOX* genes during leaf development, produce short stature mutant plants with wide blades (Schneeberger et al. 1998). Mutant *rs2* plants ectopically accumulate KNOX proteins in developing leaves (Schneeberger et al. 1998; Tsiantis et al. 1999; Timmermans et al. 1999); however, the gibberellin levels or responsiveness have not been reported in *rs2* mutants. Collectively, these studies suggest that KNOX-mediated control of gibberellin levels at the base of newly initiating leaves is important in coordinating cell expansion and elongation during leaf development.

Studies of other phytohormones and growth regulators have expanded the framework for understanding the regulation of maize leaf morphology and size. In addition to critical roles in patterning the blade/sheath boundary, brassinosteroid also plays an important role in determining leaf size. Mutations in the rice *DWARF1* homolog *NA2*, a brassinosteroid biosynthesis gene, result in reduced brassinosteroid metabolites and short stature plants with shorter and narrower leaf blades (Best et al. 2016). Dampening brassinosteroid signaling in *Zmbri1*-RNAi knockdown lines also conditions reduced blade and sheath lengths largely through decreasing the number and rate of dividing cells (Kir et al. 2015). Mis-regulation of cytokinin signaling through the semi-dominant *Hsf1* mutant decreases blade and sheath length, as well as blade width (Muszynski et al. 2020). Similarly, the recessive *abph1* mutant, a negative regulator of cytokinin signaling, results in narrower blades (Jackson and Hake 1999; Giulini et al. 2004). Treating non-mutant seedlings with exogenous cytokinin phenocopies the effects of *Hsf1* on leaf growth (Muszynski et al. 2020). The ethylene pathway functions largely to restrict growth (Dubois et al. 2018). Increased stability of the ZmACS7 biosynthesis enzyme in the semi-dominant *Sdw3* mutant leads to increased ethylene production in sheath tissues and a subsequent reduction in sheath length (Li et al. 2020a). Exogenous application of the ethylene precursor ACC also reduces sheath length (Li et al. 2020a), as well as leaf blade length (J. Strable, unpublished data).

Further insight into the regulation of maize leaf morphology and size is provided by genetic analyses of transcription factors that pattern the blade/sheath boundary. In *lg1* and *drl1* single mutants, leaf blades are narrower and shorter, and sheaths are longer (Foster et al. 2004; Strable et al. 2017). These traits are enhanced in *drl1; drl2* double mutants (Strable et al. 2017). Furthermore, a genome wide association study (GWAS) identified rare polymorphisms with large phenotypic effects for leaf width within QTL intervals for DRL1/YAB2 and DRL2/YAB7 (Strable et al. 2017). The duplicate *NARROW SHEATH* genes (*NS1* and *NS2*) encode WUS-HOMEOBOX transcription factors and are required to recruit founder cells from the SAM into marginal domains of the primordium, mediating mediolateral growth of the sheath and blade (Scanlon et al. 1996; Nardmann et al. 2004). The *NS* gene products accumulate in a lateral domain of the primordium (Nardmann et al. 2004; Conklin et al. 2020). Double mutant *ns1; ns2* plants have narrow sheaths and proximal blades that lack morphological markers of margin identity (Fig. 5g) (Scanlon et al. 1996). Blade width is reduced in a dose-dependent manner by the *Wab1* mutation (Hay and Hake 2004). The narrow leaves observed in *Wab1* versus those found in *ns1; ns2* double mutants differ in telling ways: *Wab1* distal blade width is predominantly reduced, whereas the proximal blade and sheath are narrow in *ns1; ns2* (Scanlon et al. 1996; Hay and Hake 2004), suggesting that *WAB1* promotes growth in the blade lateral domain. Indeed, genetic interactions between *Wab1* and *ns1*; *ns2* mutants indicate *WAB1* regulates growth of a leaf domain distinct from the *NS* domain.

Coordination between cell division, expansion, and differentiation is crucial to determining mature leaf morphology and size. The AP2-EREB171 transcription factor DWARF & IRREGULAR LEAF1 (DWIL1) importantly regulates cell size, shape, and arrangement in the leaf that correlates with the shorter and wider blades in *dwil1* mutants (Jiang et al. 2012). Maize *SCARECROW1* (*ZmSCR1*) co-orthologs encode GRAS transcription factors, and *Zmscr1*; *Zmscrh* double mutants display significant reductions in blade length and width, along with overall stunted shoot growth (Hughes et al. 2019). The narrow blades of *Zmscr1*; *Zmscrh* double mutants have defects in mesophyll cell divisions that increase vein density, indicating these genes function to regulate cell-type patterning during leaf development (Hughes et al. 2019). In transgenic studies, constitutive (i.e., driven by the *UBIQUITIN* [*UBI*] promoter) or localized ectopic expression (i.e., driven by *ZmGA2ox* promoter) of the maize *PLASTOCHRON1* (*PLA1*) gene gives rise to longer leaves due to an increase in the duration of leaf elongation, which is also correlated with patterns of auxin accumulation (Sun et al. 2017). These results indicate that *PLA1* promotes leaf growth by increasing the duration of cell division and that auxin may play a key role in determining the period of growth (Sun et al. 2017). Similarly, constitutive expression of the maize *ARGOS1* (*AUXIN REGULATED GENE INVOLVED IN ORGAN SIZE1*; *ZAR1*) gene under the *UBI* promoter increased leaf size in field-grown transgenic plants largely by increasing cell number but not cell size (Guo et al. 2013). ARGOS proteins also negatively regulate ethylene signaling (Shi et al. 2015), suggesting that crosstalk between hormone pathways could play a role in regulating both cellular dynamics and leaf size.

Cell division and elongation defects that culminate in narrower and shorter leaf blades and shorter sheaths characterize *asceapen1* (*asc1*; Brooks et al. 2009), *narrow odd dwarf* (*nod*; Rosa et al. 2017), and *cellulose synthase-like D1* (*csld1*; Hunter et al. 2012) mutants. Natural variation in the *CSLD1* gene, which is important for cell wall development in maize, was found to underlie a QTL for leaf width (Li et al. 2018). *NOD* encodes the CELL NUMBER REGULATOR13/MID-COMPLEMENTING ACTIVITY membrane-localized protein that is enriched in dividing leaf tissues (Rosa et al. 2017), and ASC1 is a D4-CYCLIN (Brooks et al. 2009), emphasizing the importance of coordinating cell division during leaf morphogenesis. However, misregulation of cell division and cell shape is not always correlated with altered leaf shape. For example, leaves of *tangled-1* mutant plants are reduced proportionally in size largely due to much slower growth rate (Smith et al. 1996).

Natural variation of leaf morphology

Maize is rich in natural variation for leaf traits, such as blade width and length, sheath length, blade angle of middle and upper canopies, auricle area and length, and total leaf area (Flint-Garcia et al. 2005; Kong et al. 2017; Cui et al. 2017; Dzievit et al. 2018). As a result, numerous QTL mapping analyses have harnessed variation in leaf morphology to identify underlying genetic factors (Mickelson et al. 2002; Ding et al. 2015; Li et al. 2015; Ku et al. 2010, 2012a, 2012b; Tian et al. 2011; Wei et al. 2016; Pan et al. 2017; Cui et al. 2017; Dzievit et al. 2018; Li et al. 2018). The maize nested association mapping (NAM) population, in which 25 diverse founders were crossed to a common parent to generate 5000 RILs (Yu et al. 2008; Gage et al. 2020), was utilized to dissect the genetic architecture of leaf width, length, and angle (Tian et al. 2011). By conducting a GWAS, Tian et al. (2011) found that these three classes of leaf traits are governed by myriad QTL with small effects that display little epistasis, environmental interaction, or pleiotropy. Linkage mapping in bi- and quad-parental populations has also been leveraged to identify leaf trait QTL (Mickelson et al. 2002; Ding et al. 2015; Li et al. 2015; Ku et al. 2010, 2012a, 2012b; Wei et al. 2016; Cui et al. 2017; Dzievit et al. 2018; Li et al. 2018). Recent dissection of leaf angle in three historically important US maize heterotic groups isolated 12 QTL that, when merged into a meta-analysis, identified 58 genomic regions that harbored 33 candidate genes (Dzievit et al. 2018). This analysis identified several cloned genes with significant roles in leaf architecture, as well as paralogs of these genes. These include *LG1* (Moreno et al. 1997) and its paralog *SISTER OF LG1/SBP28*, *LG2* (Walsh et al. 1998), and its paralog *LG2 RELATED SEQUENCE1/bZIP90*, and *DRL1/YAB2* and its paralog *DRL2/YAB7* (Strable et al. 2017). Genetic associations at *LG1* and *LG2* (Tian et al. 2011) and *DRL1* and *DRL2* (Strable et al. 2017) were found to underlie significant QTL for blade angle and width traits. However, despite the large number of QTL that have been identified and mapped, a majority of the underlying causative loci remain to be cloned and characterized.

A biparental maize-teosinte population that generated 866 RILs (Shannon 2012; Shannon et al. 2019) has proven to be a powerful resource for uncovering novel genetic variants for leaf architecture (Fu et al. 2019; Tian et al. 2019). This population has great potential (1) for allele discovery, such as beneficial genetic variants that were lost during domestication, and (2) as a source for alleles to improve maize shoot architecture (Tian et al. 2019). Uncovering genetic variation in wild ancestors not only provides insights into domestication, it also paves the way for future maize improvement. Tian et al. (2019) demonstrated the power of utilizing the maize-teosinte RILs to discover leaf architecture QTL and improve yield potential under dense planting. A screen for leaf angle variants important for high density planting uncovered two large effect QTL for leaf blade angle: *Upright Plant Architecure1* (*UPA1*) and *UPA2* (Tian et al. 2019). Cloning *UPA2* revealed a regulatory sequence 9.5 kb upstream of the maize gene *RELATED TO ABI3/VP1* (*RAV*)*-LIKE1* (*ZmRAVL1*), which encodes a B3-domain transcriptional activator (Tian et al. 2019). The *UPA2* regulatory sequence carries a DRL1 binding motif (Tian et al. 2019). This DRL1 binding motif is complete in most teosinte lines, whereas in all maize lines, two nucleotides are missing, which weakens DRL1 binding affinity (Tian et al. 2019). As a result, near isogenic lines (NILs) that retain the teosinte allele displayed slightly more upright leaves. Interestingly, the *ZmRAVL1* promoter carries a LG1 binding motif important for LG1-mediated activation of *ZmRAVL1* expression (Tian et al. 2019). DRL1 and LG1 proteins physically interact *in vitro* and *in vivo*, and DRL1 attenuates the activity of LG1 (Tian et al. 2019). Thus, in modern maize, *ZmRAVL1* levels are higher due to the reduced binding of DRL1 to *UPA2* that subsequently weakens its repressive interaction with LG1 (Tian et al. 2019).

In parallel, Tian et al. (2019) also cloned the large effect *UPA1* leaf angle QTL. *UPA1* QTL was found to be controlled by the brassinosteroid biosynthesis gene *BRD1*, and NILs with the teosinte *UPA1/BRD1* allele have a wider blade angle, due to an increase in auricle size and a reduction in abaxial hypodermal sclerenchyma (Tian et al. 2019). The *BRD1* promoter contains ZmRAVL1 binding motifs, which provides a mechanism for regulating brassinosteroid levels during development of the blade/sheath boundary (Tian et al. 2019). Therefore, in modern maize, DRL1-LG1-mediated activation of *ZmRAVL1* upregulates *BRD1/UPA1* and increases brassinosteroid levels at the developing ligule/auricle region (Tian et al. 2019). Understanding the mechanism by which DRL1 and LG1 proteins interact *in planta* to regulate *ZmRAVL1* expression will be an important area of future research.

Maize-teosinte RILs, additionally, have been leveraged to dissect genome-wide expression QTL to detect shifts in gene expression that may associate with domestication (Wang et al. 2018b). Additionally, maize-teosinte RIL populations with diverse maize common parents have been used to hunt for QTL that explain natural variation in leaf traits (Fu et al. 2019; Liu et al. 2019a). Fu et al. (2019) noted that large effect QTL underlie leaf length, whereas conversely numerous small effect loci control blade width and sheath length. This study implicated several cloned genes known to regulate these processes, such as *DRL1* and *DRL2* (blade width and sheath length, respectively; Strable et al. 2017), *microRNA396a* (sheath length; Nelissen et al. 2015), *DWIL1* (blade width; Jiang et al. 2012), and *LGN1* (leaf length; Moon et al. 2013). Recently, a teosinte NAM (TeoNAM) population was developed, in which 5 diverse teosinte inbred lines were crossed with a common maize parent to generate 1257 RILs, to facilitate genetic mapping of domestication and agronomically useful QTL (Chen et al. 2019; Chen et al. 2020). TeoNAM carries with it the potential for identifying beneficial alleles from teosinte that could be then introgressed into maize breeding lines and programs.

Axillary branching is regulated by external and internal cues

Axillary meristem initiation and maintenance

An axillary bud is initiated by an axillary meristem located at the node within the leaf axil. These lateral buds will either enter dormancy or remain active and grow into branches. Auxin and downstream transcription factors serve as key regulators of axillary meristem initiation. *SPARSE INFLORESENCE1* (*SPI1*) encodes a maize YUCCA-like flavin monooxygenase involved in local auxin biosynthesis (Gallavotti et al. 2008). The serine/threonine kinase BARREN INFLORESENCE2 (BIF2) is co-orthologous to *Arabidopsis* PINOID (McSteen et al. 2007) and functions in polar auxin transport (Skirpan et al. 2009). Transcripts for both *SPI1* and *BIF2* accumulate in axillary meristems, and *spi1; bif2* double mutants produce fewer vegetative phytomers compared with either single mutant (McSteen et al. 2007; Gallavotti et al. 2008). Mutations in *SPI1* and *BIF2* reduce tiller number in highly branched *teosinte branched1* (*tb1*; Doebely et al. 1997) mutants, indicating that these genes importantly regulate axillary meristem initiation (McSteen et al. 2007; Gallavotti et al. 2008). *BARREN STALK1* (*BA1*) encodes a bHLH transcription factor orthologous to rice LAX PANICLE1 (LAX1) (Gallavotti et al. 2004). Additionally, BA1 is regulated post-translationally by BIF2 phosphorylation (Skirpan et al. 2008). *ba1* mutants fail to initiate all vegetative and reproductive axillary meristems, and, as expected, *ba1; tb1* double mutants display complete tiller suppression (Ritter et al. 2002). *BA1* levels are under the control of BARREN STALK FASTIGIATE1 (BAF1), a transcriptional regulator with an AT-hook DNA binding motif (Gallavotti et al. 2011). Mutant *baf1* plants fail to initiate axillary buds that are fated to become lateral ear shoots; as a result, *baf1* mutants are earless (Gallavotti et al. 2011). *BA1* and *BAF1* are co-expressed in boundary regions flanking the axillary meristem stem cell niche, indicating expression of these genes represents one of the first committed steps of axillary meristem initiation (Gallavotti et al. 2004; Gallavotti et al. 2011). The *BA2* gene is required for axillary meristem initiation, much like *BA1*. *ba2* mutants fail to initiate vegetative and reproductive axillary meristems, and *ba2; tb1* double mutants display complete tiller suppression (Yao et al. 2019). BA2 encodes a nuclear localized protein orthologous to rice *LAX2* (Yao et al. 2019). BA1 and BA2 physically interact to form a protein complex, which likely functions downstream of auxin signaling (Yao et al. 2019). Whether *BA2* is regulated transcriptionally by BAF1 or whether BIF2 phosphorylates BA2 remains to be explored.

Once formed, maintenance of axillary meristems is equally critical for shoot branching. Genetic analysis of maize *BLH* genes has shed some light on this process. Vegetative shoots of *blh12; blh14* double mutants lack tillers and ears (Tsuda et al. 2017). However, cells in the axils of *blh12; blh14* double mutant leaves are histologically dense staining, and these cells accumulate KN1 protein (Tsuda et al. 2017). These hallmark features of meristematic cells indicate that axillary meristem initiation is unaffected by mutations in *BLH12* and *BLH14* at early plastochron stages. However, around P7, histological staining of cells weakens and KN1 accumulation lessens to the extent of eventual undetectable levels, suggesting that axillary meristem maintenance fails in the absence of *BLH12* and *BLH14* (Tsuda et al. 2017).

Axillary bud potential

Shoot branching of modern maize is largely at the level of lateral ears and not so much by tillers. This is due to growth arrest of axillary buds and bud dormancy, not a lack of axillary meristem initiation, at basal nodes. Key transcription factors expressed within basal buds regulate their dormancy (Dong et al. 2019a). *TB1* encodes a TCP transcription factor that acts as a growth repressor to promote apical dominance in modern maize (Doebely et al. 1997). Mutant *tb1* plants resemble teosinte in that the mutants are highly tillered and overproduce aerial lateral branches from mid-shoot nodes (Fig. 4a, b) (Doebley et al. 1995). Modern maize contains a *hopscotch* retrotransposon inserted ~58 kb upstream of the *TB1* promoter that predates maize domestication (Clark et al. 2006; Studer et al. 2011). This *hopscotch* element has transcriptional enhancer activity (Studer et al. 2011; Ricci et al. 2019), which conditions overexpression of *TB1* in domesticated maize (Doebely et al. 1997). Mutations in *GRASSY TILLERS1* (*GT1*), a class I HOMEODOMAIN LEUCINE ZIPPER transcription factor encoding gene, increases tiller growth (Fig. 4d) (Whipple et al. 2011). TB1 binds the *GT1* promoter to activate its expression (Whipple et al. 2011; Dong et al. 2019b). TB1 also binds to the domestication QTL *prolificacy1.1* (*prol1.1*; Wills et al. 2013) located 7 kb upstream of *GT1* to repress secondary branching in the ear (Dong et al. 2019b). Additionally, the *TASSELS REPLACE UPPER EARS1* (*TRU1*) domestication locus participates in a TB1 module to regulate shoot architecture. *tru1* mutants have long lateral ear shanks from mid-shoot nodes that terminate with a tassel (Sheridan 1988). *TRU1* encodes a BTB/POZ ankyrin repeat protein that accumulates in axillary buds, similar to TB1 (Dong et al. 2017). *TRU1* is a direct target of TB1 and is overexpressed in maize relative to teosinte in a tissue-specific manner (Dong et al. 2017). The *TB1* module highlights how overexpression was selected to produce a suite of domestication and agronomic traits (Dong et al. 2019a)

External factors, such as light, and internal signals, such as photohormones, metabolites, sugar status, and light, are key regulators of axillary bud activity. Suppression of bud growth is a component of the shade avoidance response and PHYTOCHROME (PHY)-mediated perception of red to far-red light ratios (R:FR; Dubois and Brutnell 2009). The TB1-GT1 regulatory module is sensitive to R:FR ratios, where a decrease in R:FR is perceived as shade (Whipple et al. 2011). Furthermore, FR light reduces tillering in teosinte and sorghum, which are two grass species where phytochrome signaling activates the shade avoidance response (Whipple et al. 2011). In sorghum, the red-light receptor PHYB signals to repress *TB1* expression in axillary buds, whereas high R:FR ratio suppresses PHYB activity and promotes shade avoidance (Kebrom et al. 2006). In maize buds, TB1 regulates metabolite levels involved in sugar sensing, as well as numerous phytohormone pathways that do not include auxin (Dong et al. 2019b). Levels of the phytohormone jasmonic acid are significantly reduced in *tb1* and *gt1* mutant buds; furthermore, TB1 directly targets the jasmonic acid biosynthesis gene *OXOPHYTODIENOATE REDUCTASE8* (*OPR8*), which is downregulated in *tb1* and *gt1* mutant buds (Dong et al. 2019b). *opr7; opr8* double mutants have many more nodes with de-repressed buds, and lateral ear shanks are longer relative to non-mutant plants (Yan et al. 2012), further indicating a role for jasmonic acid in suppressing axillary buds and branch growth.

Bud activity can be regulated independently of *TB1*. A QTL that increases tiller number in sweet maize was identified as *TILLER NUMBER1* (*TIN1*; Zhang et al. 2019a). *TIN1* encodes a C2H2 ZINC FINGER transcriptional regulator, where a splice site variant in sweet maize causes intron retention that enhances *TIN1* expression and bud growth (Zhang et al. 2019a). TIN1 physically interacts with TOPLESS (TPL) co-repressor proteins and represses *GT1* expression (Zhang et al. 2019a). Mutations in the TPL member *RAMOSA ENHANCER LOCUS2* (*REL2*) result in earless shoots in some maize backgrounds due to the loss of lateral ear buds (Liu et al. 2019b). Interestingly, *rel2; tb1* double mutants only slightly impact tiller number compared with *tb1* single mutants, suggesting that basal axillary buds retain growth potential independent of *REL2* (Liu et al. 2019b). The phytohormone strigolactone controls lateral branching through *TB1* homologs in other plant species (Domagalska and Leyser 2011). In maize, however, strigolactone and *TB1* pathways work independently, as mutations in the maize strigolactone biosynthesis gene *CAROTENOID CLEAVAGE DIOXYGENASE8* (*CCD8*) have only a modest effect on axillary bud growth and do not impact *TB1* expression (Guan et al. 2012).

Genetic and hormonal regulation of intercalary meristem and stem traits

Regulators of cell size and number have key roles in internode elongation, a few of which have been referred to previously in this review (e.g., *NOD*, *ASC1*, *DWIL1*). Phytohormone pathways also play an integral role in the control of plant height. In fact, reduction of plant height in wheat drove the Green Revolution, and subsequent characterization of the genes responsible for this trait placed them in the gibberellin pathway (Hedden 2003). Short stature maize mutants have long been recognized by maize geneticists (Emerson 1912b; Emerson and Emerson 1922). Some of the short stature mutants that piqued Emerson's curiosity were later found to have genetic lesions in the gibberellin pathway. For example, recessive mutants *d1*, *d3*, *d5*, and *anther ear1* (*an1*) all respond to exogenous gibberellin (Figs. 1b and 5i) (Fujioka et al. 1988), and all four genes encode gibberellin biosynthesis enzymes (Benson et al. 1995; Winkler and Helentjaris 1995; Chen et al. 2014; Fu et al. 2016). Dominant *D8* and *D9* mutants are insensitive to exogenous gibberellin (Winkler and Freeling 1994; Peng et al. 1999) and encode DELLA proteins involved in gibberellin signaling (Cassani et al. 2009; Lawitt et al. 2010). Additionally, *D1* was found to underlie a major QTL for plant height (Teng et al. 2013), and GWAS for plant height traits using the maize NAM RILs found several significant associations with hormone genes that regulate internode elongation, including *D1* and *BRD1* (Peiffer et al. 2014). The genetic architecture of maize height is highly polygenic (Peiffer et al. 2014), and many candidate loci remain uncharacterized.

Several examples of hormonal regulation of internode elongation have been mentioned in previous sections of this review. These include brassinosteroid- (e.g., *BRD1*, *NA1*, *NA2*, *BRI1*), cytokinin- (e.g., *HSF1*, *ABPH1*), ethylene- (e.g., *SDW3*), strigolactone- (e.g., *CCD8*), abscisic acid- (e.g., *VP8*), and auxin (e.g., *BIF2*, *SPI1*)-related genes, all of which control plant height across all nodes of the shoot. Similarly, mutations in *BREVIS PLANT1* (*BV1*) significantly reduce plant height by decreasing stem elongation across all nodes (Avilla et al. 2016). *BV1* encodes an inositol phosphate phosphatase involved in auxin signaling (Avilla et al. 2016). Curiously, in *bracytic2* (*br2*) mutants, only lower internodes are reduced, while upper internode length remains relatively normal (Multani et al. 2003; Pilu et al. 2007; Xing et al. 2015; Li et al. 2020b). *BR2* encodes an MDR class of P-glycoproteins necessary for polar auxin transport (Multani et al. 2003), whereas others have suggested that BR2 is more closely related to an ATP binding cassette type B protein that exports auxin from intercalary meristems of lower nodes (Knöller et al. 2010). Given the lower versus upper node differences in *br2* mutants, it would be interesting to know whether BR2 levels are regulated by a program related to the juvenile phase.

In addition to regulating plant height, auxin transport also plays a critical role in promoting negative gravitropism. The *prostrate stem1* mutant fails to maintain an upright shoot, which becomes recumbent as an adult (Dong et al. 2013). Genetic complementation tests between *prostrate stem1* and the maize mutant *lazy plant1* (*la1*; Jenkins and Gerhardt 1931; van Overbeek 1936) indicated the two are allelic (Dong et al. 2013). *ZmLA1* is functionally orthologous to rice and *Arabidopsis LAZY* genes. Interestingly, the ZmLA1 protein localizes to both the plasma membrane and the nucleus where it interacts with auxin transport and singling proteins, respectively (Dong et al. 2013; Howard et al. 2014). In future work, it will be important to know the mechanism by which ZmLA1 regulates both auxin transport and signaling proteins.

Transcriptional regulators of internode development have also been characterized. Double mutant *drl1; drl2* plants have longer adult internodes compared with non-mutant sibling plants (Strable et al. 2017). Additionally, a GWAS identified rare polymorphisms with large phenotypic effects for average internode length within QTL intervals for DRL1/YAB2 and DRL2/YAB7 (Strable et al. 2017). Through a mechanism that may involve partnering with KN1, BLH12 and BLH14 proteins prevent precocious differentiation of the internode by maintaining intercalary meristems, as well as balance anastomosis of provascular bundles in early stem development (Tsuda et al. 2017). Intercalary meristem activity is also governed by GROWTH REGULATING FACTOR

(GRF)-INTERACTING FACTOR1 (GIF1; Zhang et al. 2018), a transcriptional regulator that forms complexes with GRF transcription factors (Kim and Tsukaya 2015). Mutant *gif1* plants have short, asymmetric (kinked) internodes and show decreased expression of gibberellin-related genes (Zhang et al. 2018). In rice, *GRF1* activity in the intercalary meristem is induced by gibberellin (van der Knaap et al. 2000), suggesting that hormonal regulation of *GIF1* and *GRF* binding partners may be similar in maize. *GRF1* levels are regulated by microRNA396 (miR396; Rodriguez et al. 2010); maize plants that overexpress a miR396-resistant *GRF1* transgene have short internodes and wide leaves (Nelissen et al. 2015), phenotypes that are reminiscent of mutants with altered gibberellin levels. GRF1, along with twelve other GRFs, physically interacts with GIF1, which is enriched in meristematic tissues, suggesting that the spatiotemporal context and modularity of GRF-GIF1 complexes are likely a key aspect of their function during stem development (Nelissen et al. 2015; Zhang et al. 2018).

Conclusions and outlook

Plant developmental genetics provide a framework for translating information about gene function into breeding programs to improve agronomic traits. Maize has a long history as a genetic and breeding model and is an indispensable global crop. Positional cloning of genes has revealed the importance of structural variation (e.g., *Kn1*, Veit et al. 1990; Ramirez et al. 2009; *Abph2*, Yang et al. 2015) and transposable elements (e.g., *Corngrass1*, Chuck et al. 2007; *Kn1*, Hake et al. 1989; Ramirez et al. 2009; *tru1*, Dong et al. 2017; *drl1*, Strable et al. 2017; *Sdw3*, Li et al. 2020a) in generating novel shoot phenotypes. Genome mining of diversity panels and RILs has uncovered natural variants of cloned genes, which have given rise to a plethora of quantitative shoot phenotypes (*qPH3.1/D1*, Teng et al. 2013; *qPH1/BR2*, Xing et al. 2015; *DRL2*, Strable et al. 2017; *qLW10/ZmCSLD1*, Li et al. 2018; *UPA1/BRD1*, Tian et al. 2019; *qLA2-1/ZmIBH1*, Cao et al. 2020; *qLA2/ZmILI1*, Ren et al. 2020). A major step forward will be to functionally characterize candidate genes that underlie QTL more efficiently, such as demonstrated with quantitative trait gene sequencing (Zhang et al. 2019b). Given the tremendous diversity found in maize (Tenaillon et al. 2001; Romay et al. 2013), more high-quality inbred line genome assemblies are needed to fully catalog genetic variation, such as those captured by the (Hufford et al. 2021). High-quality genome assemblies are also needed for teosintes, landraces, and tropical maize lines, as illustrated by the assembly of a tropical maize landrace genome, which uncovered thousands of structural variations when compared with either the genomes of B73 or Mo17, both temperate maize inbred lines (Yang et al. 2019). The previously mentioned implementation of *UPA1* and *UPA2* regions in maize to increase yield under dense planting nicely demonstrates the power of utilizing teosinte-maize populations to find rare and beneficial genetic variants and incorporating pre-domesticate alleles into elite maize inbred lines (Tian et al. 2019). Recent advances including the construction of a practical haplotype graph in maize and a pangenome representative of haplotype diversity and structural diversity should prove to be valuable resources for modern breeding programs by improving the effectiveness of deciphering the genetic basis of phenotypic variation (Franco et al. 2020).

Current sequence databases catalog a wealth of genetic variation; however, a vast number of genes in maize are yet to be functionally characterized, providing scant resolution to the traits they may regulate. Plant transformation has been a historical roadblock for in maize functional genetics, where high cost, low efficiency, and a limited number of amenable transformable genetic backgrounds have hampered progress. Recent advances in transformation technology have circumvented two of these obstacles by increasing transformation efficiency and making the process independent of genotype (Lowe et al. 2016, 2018). Dovetailing with advances in transformation, CRISPR/Cas9 genome engineering has allowed for the generation of multiple alleles of a single gene, simultaneous targeting multiple genes of a functionally redundant gene family, or broadly targeting many independent genes. Genetic redundancy has challenged maize geneticists for decades; however, multiplex gene knockout with CRISPR/Cas9 is a method to overcome functional redundancy by generating higher-order mutants in a single experiment (Wu et al. 2018). Therefore, scalability of maize CRISPR/Cas9 targets has recently become less of an obstacle. In an astounding tour de force, high-throughput usage of multiplex CRISPR/Cas9 achieved editing of >740 candidate genes in maize that generated >410 alleles in ~190 genes with discernable phenotypes (Liu et al. 2020). Considering the recent identification of 160 loci with adaptive agronomic potential, as well as >1800 loci associated with trait improvement in 350 elite inbred lines (Wang et al. 2020a), multiplex CRISPR/Cas9 provides an efficient platform to discover and characterize loci in QTL regions and provides opportunities to accelerate precision maize breeding.

Many of the developmental mutants mentioned in the previous sections display pleiotropy, which can have a negative effect on yield. Pleiotropy that is often uncovered in developmental studies can arise from functional null mutations due to lesions in coding sequences and/or the inherent nature of phytomer, where genes that regulate phytomer development often have pleiotropic effects. Genetic variation in *cis*-regulatory elements (CRE) located in promoters and other regulatory regions are often less pleiotropic because transcript levels and/or spatiotemporal accumulation patterns are shifted versus completely removed (Wittkopp and Kalay 2012). Thousands of CREs in the maize genome function over long ranges (>20 kb) to regulate gene activity (Ricci et al. 2019). Untangling the individual phenotypic components of pleiotropy is one way to utilize developmental genes to improve yield. Mutagenesis of CREs through CRISPR/Cas9 has thus far proven to be an effective method of modulating expression levels and/or patterns, as well as generating quantitative phenotypes in crops by inducing dosage variation within a genetic circuit that regulates meristem size (Rodriguez-Leal et al. 2017, 2019). In recent efforts to regulate gene expression in maize, promoter sequences have been inserted, albeit at low efficiency, at endogenous loci using defined CRISPR/Cas9 target sites and homology directed repair (Svitashev et al. 2015; Shi et al. 2016). CRISPR/Cas9 could be used to overcome pleiotropic effects if the abundance of binding partners or downstream targets within a network could be modulated in a developmental or tissue-specific context. Equally promising is the implementation of CRISPR/Cas9 to generate complex trait loci to enable trait stacking, as demonstrated in multi-trait maize hybrids (Gao et al. 2020). Chromosomal regions engineered with a site-specific insertion landing pad are utilized to accept transgene(s) through recombinase-mediated cassette exchange, thus providing an effective method for overcoming linkage issues (Gao et al. 2020). Collectively, approaches that harness CRISPR/Cas9 technologies, along with rapid advances in transformation, genomics, and machine learning, demonstrate the transformative utility ahead for functional genetics and applied breeding programs.

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