PCP
PLANT & CELL PHYSIOLOGY

Grass Meristems II: Inflorescence Architecture, Flower Development and Meristem Fate

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(Received October 12, 2012; Accepted January 22, 2013)

Plant development depends on the activity of various types of meristems that generate organs such as leaves and floral organs throughout the life cycle. Grass species produce complex inflorescences and unique flowers. The grass inflorescence is composed of different types of branches, including a specialized branch called a spikelet. The spikelet is a special unit of the inflorescence and forms one to several florets, depending on the species. In the floret, floral organs such as perianth organs, carpels and stamens are formed. In Arabidopsis, because the inflorescence meristem (IM) forms the floral meristems (FMs) directly on its flanks, the change of meristem fate is relatively simple. In contrast, in grasses, different types of meristem, such as the IM, the branch meristem (BM), the spikelet pair meristem (SPM) in some grasses, the spikelet meristem (SM) and the FM, are responsible for the elaboration of their complex inflorescences and flowers. Therefore, sequential changes of meristem fate are required, and a number of genes involved in the specification of the fate of each meristem have been identified. In this review, we focus on the following issues concerning the fate of the reproductive meristems in two grass species, maize (*Zea mays*) and rice (*Oryza sativa*): (i) meristem regulation during inflorescence development; (ii) specification and fate change of the BM and the SM; (iii) determinacy of the FM; and (iv) communication between the meristem and lateral organs.

Keywords: flower • inflorescence • maize • meristem • rice • spikelet.

Abbreviations: AM, axillary meristem; BM, branch meristem; FM, floral (flower) meristem; IM, inflorescence meristem; pBM, primary branch meristem; QTL, quantitative trait locus; SAM, shoot apical meristem; sBM, secondary branch meristem; SM, spikelet meristem; SPM, spikelet pair meristem; RAM, root apical meristem; TTP, trehalose-6-phosphate phosphatase.

Introduction

The plant body plan is governed by the activities of the shoot apical meristem (SAM) and the root apical meristem (RAM), which are formed during embryogenesis. All shoot parts of the plant, such as leaves, stems and flowers, develop from the SAM, whereas the root system is formed from the RAM. Post-embryonic development depends on the function of the meristems; therefore, regulation of meristem maintenance and fate is very important for plant growth.

In the vegetative phase, the SAM sequentially initiates leaf primordia. The axillary meristems (AMs) are formed in the leaf axils, and then develop the secondary shoots, or tillers. After transition from the vegetative to the reproductive phase, the vegetative SAM changes its fate, and converts into the inflorescence meristem (IM). In some species, the IM initiates the floral (flower) meristem (FM) directly, in the axil of a bract. For example, in Arabidopsis, transition of the meristem appears to be simple, as if the FM was directly formed from the IM because of suppression of bract growth. Unlike Arabidopsis, there are several intermediate types of meristem between the IM and the FM in the grasses, as described in the next section. Therefore, the transitions of meristem fate are complex, and involve a number of genes regulating this process. Grass inflorescences, such as the rice panicle and maize tassel, consist of a main axis, long branches and spikelets. These unique inflorescence units develop from specialized meristems: the branch meristem (BM) and the spikelet meristem (SM).

In the accompanying manuscript, Pautler et al. (2013) describe the genetic and hormonal regulation of the shoot meristem, as well as the transition from vegetative to inflorescence fate. In this review, we focus on the inflorescence, first considering the regulation of the initiation and determinacy of the BM. In the next section, we describe the genes involved in changes in meristem fate: transition from the BM to the SM and from the SM to the FM, respectively, and in determinacy of the SM. Then, we focus on the genes involved in both the regulation of determinacy of the FM and specification/development of floral organs. Finally, we briefly mention the communication between the meristem and lateral organ primordia. Although there are several excellent reviews on grass meristems (Bortiri and Hake 2007, Thompson and Hake 2009, Yoshida and Nagato 2011), the accumulation of the papers describing

Plant Cell Physiol. 54(3): 313–324 (2013) doi:10.1093/pcp/pct016, available online at www.pcp.oxfordjournals.org

Table 1 Genes appearing in this review

Rice	Maize	Arabidopsis
Meristem regulation during inflorescence development		
	ramosa1 (*ra1*)	
	ramosa2 (*ra2*)	
	ramosa3 (*ra3*)	
ABERRANT SPIKELET AND PANICLE1 (*ASP1*)	*ramosa enhancer locus2* (*rel2*)	*TOPLESS* (*TPL*)
	Tunicate1 (*Tu1*)/*zmm19*	
RFL/ABBERANT PANICLE ORGANIZATION2 (*APO2*)	*zfl1*, *zfl2*	*LEAFY* (*LFY*)
TAWAWA1 (*TAW1*)		
OsPINOID	*barren inflorescence2* (*bif2*)	*PINOID* (*PID*)
LAX PANICLE1 (*LAX1*)	*barren stalk1* (*ba1*)	
MONOCULUM1 (*MOC1*)/*SMALL PANICLE* (*SPA*)		*LATERAL SUPPRESSOR* (*LAS*)
LAX PANICLE2 (*LAX2*)		
GRAIN NUMBER 1a (*GN1a*)/*OsCKX2*		*CYTOKININ OXIDASE* (*CKX*)
DENSE AND ERECT PANICLE (*DEP1*)/*DENCE PANICLE1* (*DN1*)		
WEALTHY FARMER'S PANICLE (*WFP*)/*IDEAL PLANT ARCHITECTURE* (*IPA1*)/*SQUAMOSA PROMOTER BINDING PROTEIN-LIKE 14* (*OsSPL14*)		
	fasciated ear2 (*fea2*)	*CLAVATA2* (*CLV2*)
ABBERANT PANICLE ORGANIZATION1 (*APO1*)/*STRONG CULM2* (*SCM2*)		*UNUSUAL FLORAL ORGANS* (*UFO*)
Specification of the spikelet meristem identity		
FRIZZY PANICLE (*FZP*)	*branched silkless1* (*bd1*)	*PUCHI*
OsIDS1	*indeterminate spikelet1* (*ids1*)/*Tasselseed6* (*Ts6*)	*TARGET OF EAT1* (*TOE1*), *TOE2*, *TOE3*
supernumerary bract (*snb*)	*sister of indeterminate spikelet1* (*sid1*)	*TARGET OF EAT1* (*TOE1*), *TOE2*, *TOE3*
miR172a, *miR172b*	*tasselseed4* (*ts4*)	*miR172*
LEAFY HULL STERILE1 (*LHS1*)/*OsMADS1*		*SEP-like*
MOSAIC FLORAL ORGAN1 (*MFO1*)/*OsMADS6*	*bearded-ear* (*bde*)/*zag3*	*AGL6-like*
OsMADS3	*zmm2*, *zmm23*	*AGAMOUS* (*AG*)
OsMADS58	*zag1*	*AGAMOUS* (*AG*)
OsMADS13	*zag2*	*SEEDSTICK* (*STK*)
OsMADS17		*AGL6-like*
DROOPING LEAF (*DL*)	*ZmDL*	*CRABS CLAW* (*CRC*)
Communication between the meristem and lateral organs		
TOBGARI-BOUSHI1 (*TOB1*)	*zyb14*	*FILAMENTOUS FLOWER* (*FIL*), *YAB3*

meristem function and flower development has been very rapid. We have tried to summarize the related studies, including new findings. Genes mentioned in this review are listed in **Table 1**.

Inflorescence Development and Meristem Transitions in Grasses

Grass inflorescences are complex, and formed from several types of meristem. After transition from the vegetative phase to the reproductive phase, the SAM converts into the IM, which initiates the BMs and forms the main axis of the inflorescence (**Fig. 1A**). The BMs initiate the SMs, which then initiate the FMs. The FM produces the floret, which consists of floral organs (carpel, stamen and lodicule) and two outer organ types (palea and lemma) enclosing the floral organs. The spikelet is composed of one to several florets and two glumes that enclose them. Thus, the SM initiates the glume primordia in addition to the FM. Although this is a general scheme of the inflorescence and flower development in grasses, various modifications result in diverse structures of the inflorescence, depending on species.

In maize, for example, several major differences occur. First, there are two types of inflorescence: the male inflorescence, or

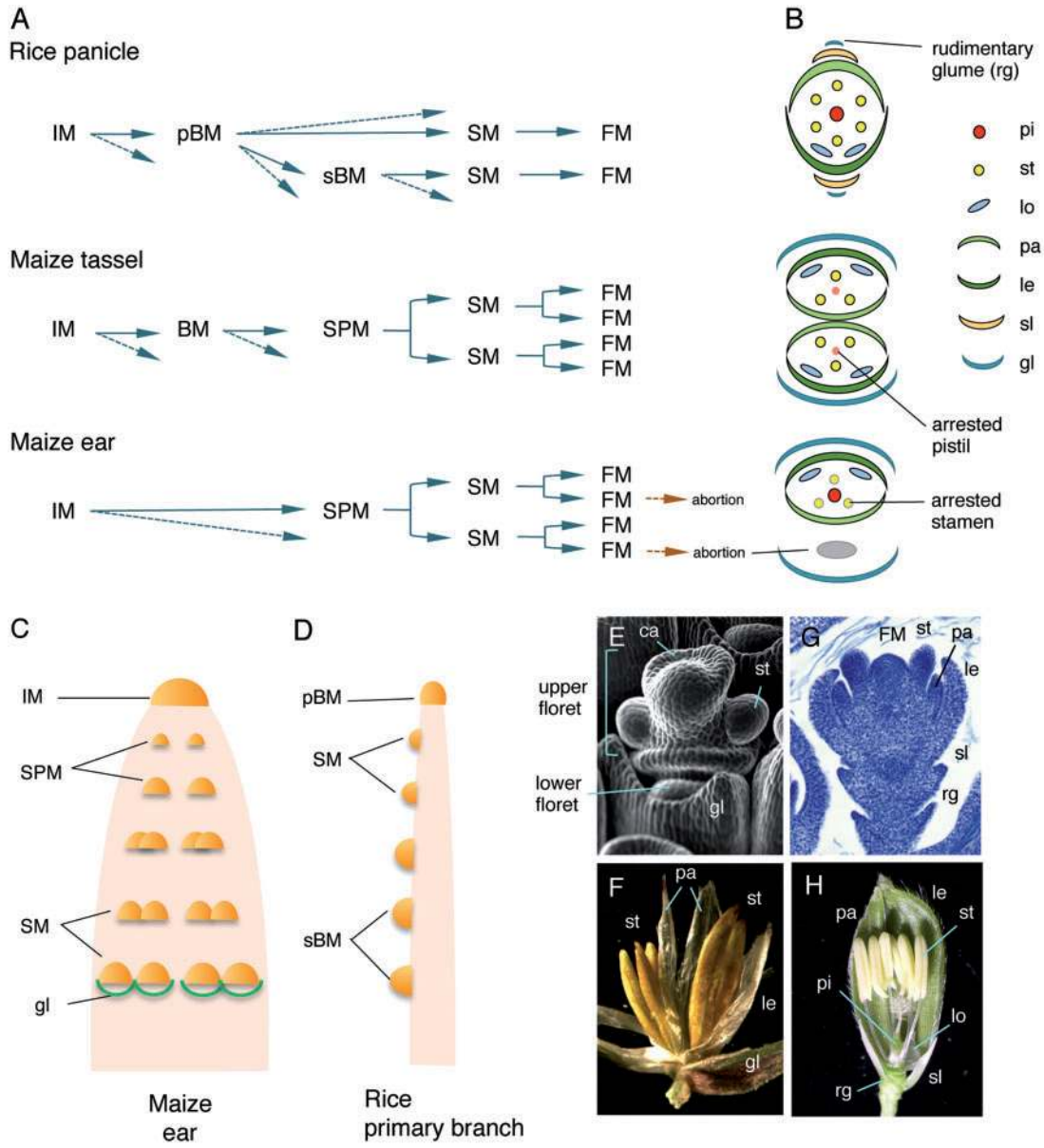

Fig. 1 Transition of the reproductive meristems, and the flower and inflorescence in rice and maize. (A) Transition of the meristems. A dashed arrow indicates multiple formation of the meristem. (B) Schematic representation of the spikelet of rice (upper) and maize (male, middle; female, lower). (C) Schematic representation of the ear in maize. (D) Schematic representation of the primary branch of a rice panicle. (E) Spikelet of a maize ear at the early developmental stage. (F) Mature maize male flower. (G) Cross-section of a rice flower at the initiation stage of the stamen. (H) Mature flower of rice. BM, branch meristem; ca, carpel; FM, flower meristem; gl, glume; IM, inflorescence meristem; le, lemma; lo, lodicule; pa, palea; pi, pistil; rg, rudimentary glume; sl, sterile lemma; SM, spikelet meristem; SPM, spikelet pair meristem; st, stamen.

tassel, which forms long branches; and the female inflorescence, or ear, which does not (**Fig. 1A**). Secondly, the IM initiates a novel kind of meristem, the spikelet pair meristems (SPMs), that are responsible for making paired spikelets typical of the Andropogoneae. SPMs are made in the tassel after the formation of several BMs with an indeterminate fate. The SPMs subsequently divide into two SMs (**Fig. 1A, C**). Next, the SM initiates two FMs (**Fig. 1A, E**). The two FMs normally develop florets in the tassel, whereas the lower FM aborts in the ear, such that only one floret is formed in the female spikelet (**Fig. 1B, E, F**) (for reviews, see McSteen et al. 2000, Bommert et al. 2005, Thompson and Hake 2009).

In rice, the SMs are initiated from the primary or secondary BMs, which are formed from the IM or primary BMs (pBMs), respectively (**Fig. 1A, D**). The SM initiates one fertile floret, two sterile lemmas and two glumes (**Fig. 1B, G, H**) (for reviews, see Bommert et al. 2005, Thompson and Hake 2009). The glumes are highly reduced, and are called rudimentary glumes. The sterile lemma, a tiny flap-like organ, is thought to be a reduced lemma of two sterile lateral florets, and develops as a lateral

organ of the SM between the fertile floret and the rudimentary glume (Yoshida et al. 2009). Because the SM initiates a single FM, the transition from the SM to FM is less clear in rice, compared with maize.

Meristem Regulation During Inflorescence Development

The *ramosa* pathway

Branch formation in the inflorescence is a key determinant of plant architecture. The branching pattern of the rice panicle is created by the formation of pBMs from the inflorescence meristem, and secondary BMs (sBM) from the pBMs (**Fig. 1A**). In maize, several long branches are formed from BMs in the tassel, but these are completely absent in the female inflorescence and allow efficient seed packing (**Fig. 1A, C**) (Sigmon and Vollbrecht 2010). BMs and SPMs are formed around the same time, and are virtually indistinguishable at initiation. Each SPM forms two SMs, and each SM forms two FMs upon which a determinate fate is imposed.

Three classical mutants of maize, *ramosa1* (*ra1*), *ra2* and *ra3*, are characterized by increased long branches in the tassel and ear; thus, these genes function to enforce the determinacy of SPMs to limit the production of long branches (**Fig. 2A–D**). *ra1* and *ra2* encode putative transcription factors of the C2H2 zinc finger and lateral organ boundary (LOB) domain-containing families, respectively (Vollbrecht et al. 2005, Bortiri et al. 2006). Interestingly, *ra3* encodes a trehalose-6-phosphate phosphatase (TPP), an enzyme that catalyzes the production of trehalose sugar (Satoh-Nagasawa et al. 2006). Genetic analysis has placed all three genes into a pathway, with *ra2* and *ra3* acting in parallel upstream of *ra1* (Satoh-Nagasawa et al. 2006). The three genes are expressed in overlapping, but distinct, domains, either within or directly subtending the SPMs and SMs that they regulate, pointing to non-cell-autonomous signals emanating from these domains.

Two possible mechanisms for a mobile signal regulating SPM determinacy include an RA3-dependent sugar signal, or an RA1-dependent protein or small molecule signal. TPP catalyzes the final step in the production of trehalose: removal of a phosphate group from the intermediate metabolite trehalose-6-phosphate (T6P) (Paul et al. 2008). Both trehalose and T6P have been proposed to act as sugar signals, due to their low abundances relative to primary metabolites. These molecules have been shown to regulate enzymes involved in central carbon metabolism, and may serve to couple sugar availability and plant growth (Paul et al. 2008). *ra3* is a member of a large TPP gene family, whose members possess unique and diverse expression patterns in Arabidopsis, indicating that some degree of subfunctionalization has occurred (Vandesteene et al. 2012). It is not feasible to measure changes in T6P and trehalose levels within these specific domains in situ. Nevertheless, key questions, such as whether the enzymatic activity of RA3 is required for function, can be addressed. An alternative hypothesis positions RA3 as a transcriptional regulator, as proposed for the glycolytic enzyme *HEXOKINASE1* in Arabidopsis (Cho et al. 2006, Satoh-Nagasawa et al. 2006). Transcriptome profiling by digital gene expression (DGE) signatures revealed a list of differentially expressed genes that could be responsible for mediating the *ra3* phenotype (Eveland et al. 2010). Candidate genes included those involved in primary carbon metabolism as well as those involved in hormone response pathways, such as ethylene response factor (ERF) family members. Overall, this study suggests an interesting link between sugar sensing, hormone signaling, and growth and development (Eveland et al. 2010, Eveland and Jackson 2012).A hint at the mechanism of action of RA1 comes from the identification of *ramosa enhancer locus2* (*rel2*) that encodes a TOPLESS (TPL)-like co-repressor (Gallavotti et al. 2010). RA1 and REL2 physically interact through the C-terminal EAR domains of RA1 (Gallavotti et al. 2010). Therefore, it is likely that this complex plays a role in repressing transcription of target genes. A rice mutant named *aberrant spikelet and panicle1* (*asp1*), which displays a range of vegetative and reproductive defects (**Fig. 2E, F**), encodes the rice ortholog of *REL2* (Yoshida et al. 2012). Several of the mutant phenotypes, as well as molecular knowledge of TPL function in Arabidopsis, strongly implicate defects in auxin signaling in the genesis of the *asp1* phenotype (Yoshida et al. 2012). Consistent with this idea, hormones have long been known to modify the number of long branches in the maize tassel upon exogenous application, including auxin and gibberellic acid (Nickerson 1959, McSteen 2006). Therefore, hormone biosynthesis and signaling components are potential downstream effectors of the *ramosa* pathway. Moving forward, it will be important to determine direct transcriptional targets of RA1 through methods such as chromatin immunoprecipitation-sequencing (ChIP-seq).

Tunicate

The classical pod corn mutant of maize, *Tunicate1* (*Tu1*), has pleiotropic inflorescence phenotypes, but is most obviously characterized by elongated glumes that completely enclose the seeds (Han et al. 2012). This dominant mutant is caused by a chromosomal rearrangement, resulting in the MADS-box gene *ZMM19* gaining a novel expression pattern from a neighboring gene (Han et al. 2012. Wingen et al. 2012). Ectopic expression of ZMM19 in a *ramosa*-like domain confers indeterminacy to SPMs and results in the production of long branches. Therefore, when misexpressed in the *Tu1* mutant, ZMM19 plays a role in promoting growth and indeterminacy, in opposition to the *ramosa* genes (Han et al. 2012). The fact that a gene not normally expressed in the inflorescence can dictate such dramatic changes in inflorescence architecture is indicative of the modularity underlying developmental programs.

Genes regulating determinacy, maintenance and initiation of the BM

While the *ramosa* pathway defines the principal mechanism of branch meristem regulation in maize, several additional genetic

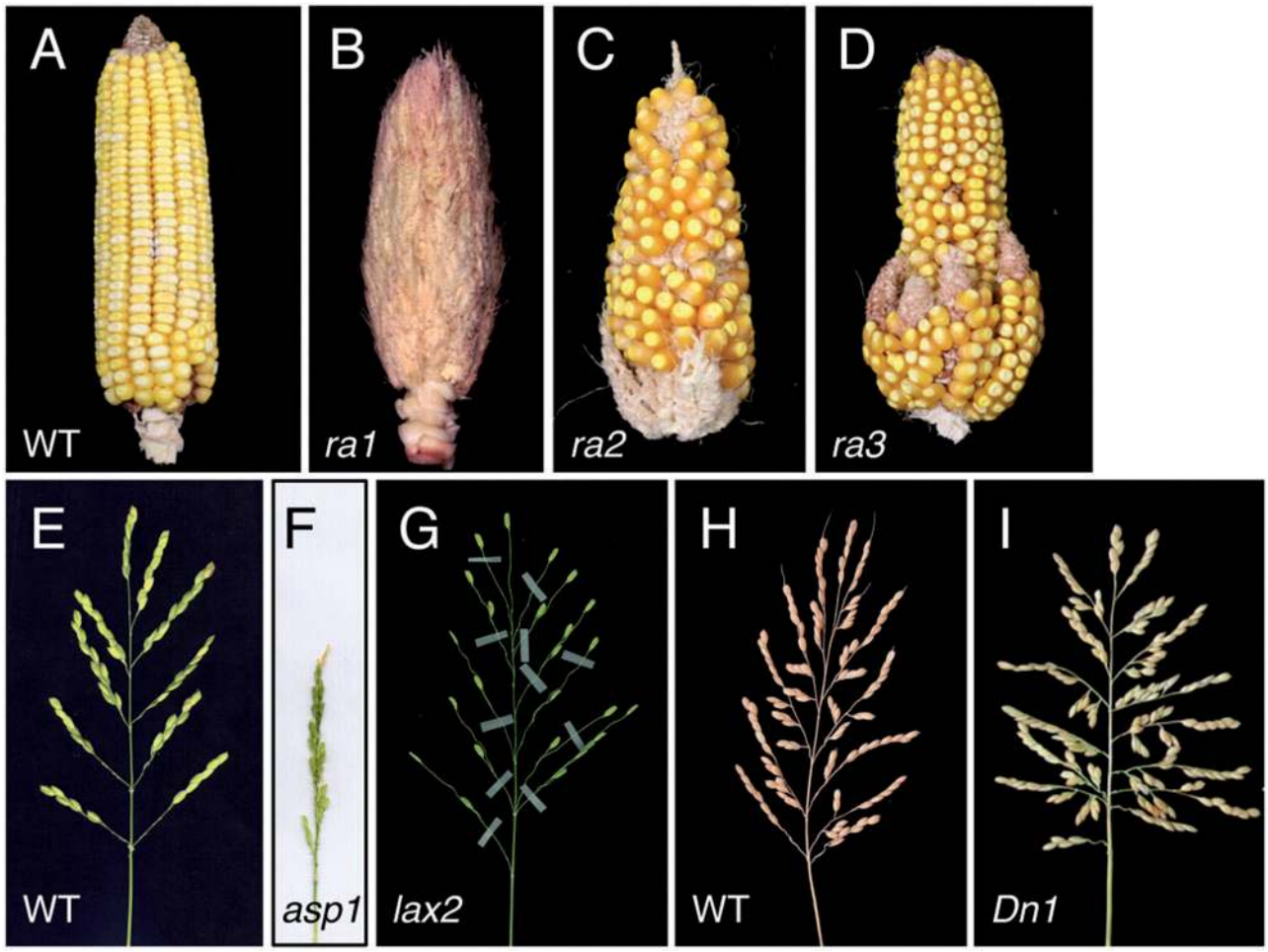

Fig. 2 Branching mutants of maize and rice. (A–D) Maize ears. (A) Wild type, (B) *ramosa1* (*ra1*), (C) *ra2* and (D) *ra3*. (E–I) Rice panicles. (E) Wild type (Taichung 65), (F) *aberrant spikelet and panicle1* (*asp1*), (G) *lax panicle2* (*lax2*), (H) wild type (Koshihikari) and (I) *Dense panicle1* (*Dn1*) on the Koshihikari background. Images were kindly provided by Drs. Akiko Yoshida (F), Yutaka Sato (G) and Fumio Taguchi-Shiobara (H, I).

factors can modulate the number of long branches produced in grass inflorescences. Constitutive overexpression of several TERMINAL FLOWER-related (TFL) genes in maize increases indeterminacy of axillary meristems in the inflorescence, consistent with TFL function in Arabidopsis (Bradley et al. 1997, Danilevskaya et al. 2010). Branch number is decreased in the mutant or transgenic knockdowns of the rice *LEAFY* homolog *RFL/ABBERANT PANICLE ORGANIZATION2* (*APO2*) (Rao et al. 2008, Ikeda-Kawakatsu et al. 2012) as well as double mutants of the duplicated maize orthologs *zfl1* and *zfl2* (Bomblies et al. 2003). These phenotypes imply close integration of flowering time regulation and inflorescence architecture; for example, there may be a competency period for production of branches, such that the number produced depends on the rate of progression through the transition. This idea is supported in recent studies of rice *TAWAWA1* (*TAW1*), which encodes a nuclear protein with an ALOG domain. In *taw1-D* gain-of-function alleles, IM activity is promoted and spikelet development is delayed, leading to prolonged branch formation. In contrast, loss of *TAW1* function leads to a reduction in IM indeterminacy, and small inflorescences (Yoshida et al. 2013).

Inflorescence architecture is also dependent on the initiation and maintenance of the AMs. There are a number of mutants in maize and rice that display a defect in forming BMs and SMs, leading to a reduced number of long branches, or even a completely barren inflorescence. *barren inflorescence2* (*bif2*), which encodes a maize ortholog of the Arabidopsis serine-threonine kinase *PINOID*, fails to initiate all axillary meristems of the inflorescence (McSteen et al. 2007). Mutants of the orthologous basic helix–loop–helix (bHLH) transcription factors BARREN STALK1 (BA1) and LAX PANICLE1 (LAX1), of maize and rice, respectively, fail to initiate AMs in both the vegetative and reproductive phases (Komatsu et al. 2003a, Gallavotti et al. 2004). Branching is also reduced in *monoculum1* (*moc1*)/*small panicle* (*spa*) and *lax2* mutants of rice (**Fig. 2E, G**), in addition to reduced tillering (Komatsu et al. 2003a, Li et al. 2003; Tabuchi et al. 2011). *MOC1/SPA* and *LAX2* encode a GRAS family transcription factor and a novel nuclear protein,

respectively. Combinations of *lax2* mutants with *lax1* or *moc1* mutants show enhanced sparse panicle phenotypes, suggesting synergistic genetic interaction between these genes (Tabuchi et al. 2011).

Quantitative trait loci (QTLs) and genome-wide association studies examining inflorescence architecture traits in maize have hinted at the contribution of other loci, such as *liguleless1*, which do not have strong loss-of-function branching phenotypes (Brown et al. 2011). It is important to pursue these types of powerful approaches in parallel with forward genetic studies, as genetic redundancy can limit the utility of traditional screens.

Inflorescence architecture and yield

Meristematic activity in the inflorescence has a profound influence on grain yield. Characteristics such as the size and determinacy of the IM, BMs and SMs may drastically affect the number of spikelets, and eventually mature grains, per plant. Grain number per plant is a continuous trait that can be modified by a large number of genes controlling a range of developmental and physiological responses. Positional cloning has been successfully employed to identify several genes underlying yield QTLs in rice, and a few of these cases are highlighted below.

A major yield QTL on chromosome 1, *GRAIN NUMBER 1a* (*GN1a*), was fine-mapped to a single open reading frame encoding a cytokinin oxidase gene, OsCKX2 (Ashikari et al. 2005). High-yielding rice cultivars had less CKX2 activity, and therefore higher cytokinin levels, particularly in the IM of the panicle. This results in increased meristematic activity and a higher number of long branches and spikelets, leading to higher grain yield per panicle (Ashikari et al. 2005). A dominant-negative truncation of a phosphatidylethanolamine-binding protein (PEBP) underlies a yield QTL at the *DENSE AND ERECT PANICLE* (*DEP1*)/*DENSE PANICLE1* (*DN1*) locus (Huang et al. 2009, Taguchi-Shiobara et al. 2011). This mutation is responsible for creating high-yielding inflorescence architecture in many *japonica* rice varieties (**Fig. 2H, I**).

Another grain yield QTL called *WEALTHY FARMER'S PANICLE* (*WFP*)/*IDEAL PLANT ARCHITECTURE* (*IPA1*) corresponds to *Squamosa Promoter Binding Protein-Like 14* (*OsSPL14*) (Jiao et al. 2010, Miura et al. 2010). High branching accessions overexpress *OsSPL14* in the panicle in a domain associated with BMs (Miura et al. 2010). A single point mutation that relieves *OsSPL14* from its miR156-mediated repression is sufficient to decrease plant tiller number, while increasing panicle branching and grain number, thus creating the 'ideal plant' for agriculture (Jiao et al. 2010).

Many forward genetic screens uncover strong loss-of-function mutants with very severe phenotypes. While these 'monstrous' mutants are useful for uncovering the normal function of genes, they rarely provide useful substrate for breeding efforts, because they frequently display negative pleiotropic traits. For example, the increase in size of the IM in maize fasciated ear mutants is accompanied by a decrease in the length of the ear, as well as disorganized seed rows, which limit the number of seeds per ear. Reverse genetic resources, such as TILLING, can facilitate the discovery of hypomorphic alleles, which may have a beneficial effect on crop yield (Weil 2009). Bommert et al. (2013) isolated a weak allele of the fasciated ear mutant *fea2* and showed that this allele increases kernel row number and kernels per ear, without causing a fasciated IM or shorter ear. Furthermore, this study implicated natural variation in *fea2* expression in modifying meristem size in diverse inbred lines of maize (Bommert et al. 2013).

Dominant mutant alleles of the *aberrant panicle organization1* (*apo1*) gene of rice produce extra primary and secondary branches in the inflorescence, whereas loss-of-function alleles display the opposite phenotype (Ikeda et al. 2007, Ikeda-Kawakatsu et al. 2009). The contrasting effect on branching is explained by significantly different rates of cell division in each respective mutant (Ikeda-Kawakatsu et al. 2009). The beneficial panicle architecture of the gain-of-function mutant is associated with other negative traits, such as fewer panicles per plant. However, positional cloning of a QTL for culm strength, *STRONG CULM2* (*SCM2*), identified a dominant allele of *apo1* that conferred the improved panicle, without decreasing panicle number (Ookawa et al. 2010). The plant architecture QTLs described above reinforce the value of exploiting natural variation for yield improvement.

Specification of Spikelet Meristem Identity

Transition from the BM to the SM

The *branched silkless1* (*bd1*) and *FRIZZY PANICLE* (*FZP*) genes regulate the transition from the BM to the SM in maize and rice, respectively (Chuck et al. 2002, Komatsu et al. 2003b). In both *bd1* and *fzp* mutants, indeterminate branches are formed instead of spikelets (**Fig. 3**). Thus, it is likely that these two genes control the determinacy of the BM and establish the identity of the SM. *bd1* and *FZP* encode orthologous transcription factors in the AP2/ERF family. These two genes are expressed at the junction of the SM and the initiation site of the glume, but are not expressed in the meristem per se. This finding raises the possibility that the expression domain of these two genes might be important for the establishment of the SM identity.

In Arabidopsis, the *PUCHI* gene is the ortholog of *bd1* and *FZP*. A partial conversion from FM to IM is observed in the *puchi* mutant, in addition to other phenotypes (Karim et al. 2009). It seems likely that a fundamental function of these genes in controlling meristem transitions and/or determinacy is conserved in both grasses and Arabidopsis.

Identity of the SM and its determinacy

In maize, the transition from the SM to the FM is regulated by *indeterminate spikelet1* (*ids1*), and its close paralog, *sister of indeterminate spikelet1* (*sid1*) (**Fig. 3**) (Chuck et al. 1998, Chuck et al. 2008). In the *ids1* mutant, a few extra florets are

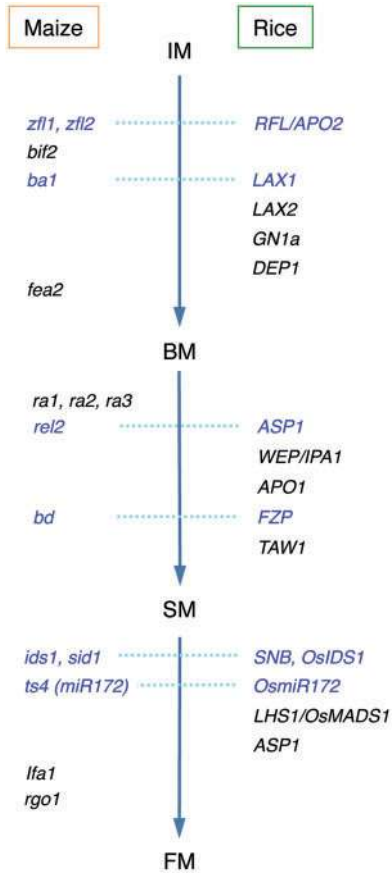

Fig. 3 Genes responsible for transition of meristem fate in maize and rice. Orthologs are indicated in blue. BM, branch meristem; FM, flower meristem; IM, inflorescence meristem; SM, spikelet meristem.

formed within a spikelet, in both the tassel and the ear, suggesting that the regulation of SM determinacy is compromised (Chuck et al. 1998). In the *ids1 sid1* double mutant, glumes (bracts) are continuously formed, implying that the transition of the SM to the FM is strongly inhibited (Chuck et al. 2008). Therefore, *ids1* and *sid1* redundantly act to control the transition from the SM to the FM, and the determinacy of the SM. The *ids1* and *sid1* genes also encode AP2/ERF transcription factors, but they belong to a class different from that of *bd1* (Chuck et al. 1998, Chuck et al. 2008).

In rice, *supernumerary bract* (*snb*) and *OsIDS1* have functions similar to those of *ids1* and *sid1* in maize (**Fig. 3**) (Lee et al. 2007, Lee and An 2012). The *snb* mutant displays repetitious production of rudimentary glumes (bracts) or extra lemma/palea, and, in rare cases, forms two florets in a spikelet (Lee et al. 2007). These phenotypes are enhanced in the *snb osids1* double mutant, suggesting that the transition from the SM to the FM is extremely delayed, and that SM determinacy is compromised (Lee and An 2012). *SNB* and *OsIDS1* encode homologs of *sid1* and *ids1* (Lee et al. 2007, Lee and An 2012), thus, the genetic mechanism controlling the transition from the SM to the FM appears to be conserved between maize and rice.

The classical maize mutants, *tasselseed4* (*ts4*) and *Tasselseed6* (*Ts6*), have been characterized as defective in sex determination (Chuck et al. 2007). These two mutants exhibit similar phenotypes: a failure in carpel abortion in the male flower and an increase in meristem branching. Gene isolation revealed that *ts4* encodes a member of the *miR172* microRNA family that restricts the function of AP2 domain transcription factors. *ids1* is more broadly expressed in the *ts4* mutant, compared with the wild type, and the branching and sex determination phenotypes of *ts4* are almost completely suppressed by *ids1* mutation. Therefore, *ids1* is likely to be a key target of miR172. The dominant *Ts6* mutant, on the other hand, has a mutation in the binding site of miR172 in the *ids1* gene. This mutation probably results in ectopic expression of *ids1* in the *TS6* mutant, because of the release from repression by miR172 (Chuck et al. 2007). In rice, overexpression of the *OsmiR172* genes largely phenocopies the *snb osids1* double mutant (Zhu et al. 2009, Lee and An 2012). These observations indicate that the function of *IDS1*-like genes and their proper regulation by *miR172* play important roles in specifying the fate of the SM in both maize and rice (**Fig. 3**).

In rice, *LEAFY HULL STERILE1* (*LHS1*)/*OsMADS1* is involved in SM identity, because loss-of-function mutants show reiterative formation of lemma/palea or extra floret formation in spikelets, in addition to defects in the identity of the lemma/palea (Jeon et al. 2000). *lhs1* phenotypes, including loss of determinacy of the SM, are enhanced by mutation in the *MOSAIC FLORAL ORGAN1* (*MFO1*)/*OsMADS6* gene (Ohmori et al. 2009, Li et al. 2010) (see below).

Whereas many of the mutations already described are in genes encoding specific transcription factors, more general transcriptional machinery is also important in control of meristem fate. Mutation in *ABERRANT SPIKELET AND PANICLE1* (*ASP1*) causes pleiotropic phenotypes in spikelet development and inflorescence architecture (Yoshida et al. 2012). These phenotypes are closely associated with various defects in meristem function, such as precocious transition of the BM to SM, partial failure in maintenance of the BM and SM, and disturbed initiation of the AM. In a rare case, a reversion from the SM to the BM is observed. *ASP1* encodes a transcriptional co-repressor, similar to Arabidopsis TPL. Therefore, it is likely that derepression of multiple genes in the *asp1* mutant causes the various defects in meristem fates.

In maize, the *INDETERMINATE FLORAL APEX1* (*IFA1*) and *REVERSED GERM ORIENTATION1* (*RGO1*) genes are also responsible for determinacy of the SM (Laudencia-Chingcuanco and Hake 2002, Kaplinsky and Freeling 2003). In *ifa1* and *rgo1* mutants, the SPM and the SM initiate extra SMs and FMs, respectively. Double mutants of *rgo1* and *ids1* show a more severe phenotype than either single mutant, and produce extra branches in the SPM (Kaplinsky and Freeling 2003). Reversion of the SM to SPM or BM fate is also observed in the double mutant of *ifa1* and *ids1*(Laudencia-Chingcuanco and Hake 2002). Thus, *IDS1*, *IFA1* and *RGO1* redundantly

regulate the fate of the reproductive meristems in maize; however the latter two genes have not yet been cloned.

Floral Meristem Determinacy

The vegetative SAM initiates leaf primordia, whereas the FM initiates floral organs. Apart from the type of lateral organs that these meristems produce, the essential difference between the SAM and the FM is determinacy. The SAM and IM are indeterminate, because the meristems continue to replace stem cells and repeatedly to initiate lateral organs and AMs. In contrast, the FM is determinate, because the stem cells are consumed by the formation of the final floral organs, such as the carpel and the ovule.

Function of C-Class MADS-box genes

In Arabidopsis, *AGAMOUS* (*AG*), which encodes a C-class MADS-box transcription factor, is a key gene responsible for meristem determinacy (for reviews, see Barton 2010, Sun and Ito 2010). *ag* mutants produce indeterminate flowers in which a set of floral organs (sepal–petal–petal) are repeatedly formed (Bowman et al. 1989, Yanofsky et al. 1990). *WUSCHEL* (*WUS*) expression persists in the FM of the *ag* mutant at a late stage of flower development, whereas it disappears after formation of the carpel in the wild type (Lenhard et al. 2001, Lohmann et al. 2001). Therefore, in the FM, *AG* regulates meristem determinacy by repressing *WUS*. *KNUCKLES* (*KNU*), which encodes a transcriptional repressor, has an important role to mediate the repression of *WUS* by *AG* (Sun et al. 2009). A recent study suggests the possibility that *AG* in turn directly represses *WUS* (Liu et al. 2011).

C-class MADS-box genes have increased in number during evolution of the grasses. Maize has at least three *AG* orthologs, and, among them, *zag1* has shown to be partially responsible for FM determinacy, as multiple carpels are formed in the *zag1* ear (Mena et al. 1996, Zanis 2007). Rice has two *AG* orthologs, *OsMADS3* and *OsMADS58*, and their functions are diversified (Yamaguchi et al. 2006). Whereas FM determinacy is partially compromised in an *osmads3* mutant, a severe loss of determinacy, similar to that of *zag1*, was observed in knockdown lines of *OsMADS58*. In these lines, a set of floral organs (lodicules, stamens and partial carpels) is repeatedly formed in the flower, and an FM-like structure remains, even in the mature flower (Yamaguchi et al. 2006). Although in a different genetic background, an *osmads58* mutation has little effect on floral phenotypes, but dramatically enhances the indeterminate phenotype of the *osmads3* single mutant, suggesting the importance of *OsMADS58* in FM determinacy in rice (Dreni et al. 2011). Taken together, the evidence suggests that C-class MADS-box genes play crucial roles in regulating the determinacy of the FM in both maize and rice.

OsMADS13, a MADS-box gene in the D-class lineage, is required for specification of the ovule (Lopez-Dee et al. 1999, Dreni et al. 2007, Yamaki et al. 2011). In the *osmads13* mutant, determinacy is partially lost, because *OSH1* expression is prolonged and multiple pistils are formed. Mutation of *osmads13* enhances the indeterminate phenotype observed in the *osmads3* or *osmads3 osmads58* double mutant (Dreni et al. 2011).

Additional genes responsible for FM determinacy

MADS-box genes in the *AGL6* subfamily, including rice *MOSAIC FLORAL ORGAN1* (*MFO1*)/*OsMADS6* and maize *bearded-ear* (*bde*)/*zag3*, are also responsible for FM determinacy (Ohmori et al. 2009, Thompson et al. 2009, Li et al. 2010). Mutation in *MFO1* causes production of extra carpels and spikelets in the center of the flower in rice (Ohmori et al. 2009, Li et al. 2010). *OsMADS17*, a close paralog of *MFO1*, seems to have a weaker function similar to that of *MFO1*, because RNA silencing of this gene enhances abnormal flower phenotypes in the *mfo1* mutant, but does not cause any obvious phenotype in the wild type (Ohmori et al. 2009). In the maize *bde* mutant, the upper FM forms extra floral organs, whereas the lower FM initiates additional FMs (Thompson et al. 2009). An evo–devo study has indicated that *AGL6*-like genes are expressed similarly in the FM in all grass species, whereas they are expressed differently in the floral organ primordia depending on the species (Reinheimer and Kellogg 2009). Thus, it is possible that the regulation of meristem determinacy is the ancestral function of *AGL6*-like genes in grasses.

Mutation of C-class MADS-box genes enhances the floral phenotype of *mfo1* and *bde* in both rice and maize (Thompson et al. 2009, Li et al. 2011). In maize, it has been demonstrated that BDE protein physically interacts with ZAG1 protein (Thompson et al. 2009). In rice, in contrast, it has been reported that C-class genes are regulated by *MFO1* (Li et al. 2010). However, this is inconsistent with the results of another group who found that the expression levels of two C-class genes are unchanged in the *mfo1* mutant, as compared with the wild type (Ohmori et al. 2009). More detailed analysis is required to resolve this discrepancy. Combination of mutations in *MFO1* and *OsMADS13* enhances defects in FM determinacy in each single mutant (Li et al. 2011). As described above, *LHS1* is also involved in FM determinacy, and *lhs1* mutations enhance the *mfo1* phenotype (Jeon et al. 2000, Ohmori et al. 2009, Li et al. 2010), suggesting that multiple MADS-box genes redundantly regulate determinacy of the FM in rice.

In rice, carpel identity is specified by a *YABBY* gene, *DROOPING LEAF* (*DL*), whereas this identity is regulated by *AG* orthologs in eudicots (Yamaguchi et al. 2004). Because spatial expression patterns of *DL* orthologs in maize and wheat resemble that of *DL* in rice, the function of *DL* orthologs is likely to be conserved in grasses (Ishikawa et al. 2009). In loss-of-function *dl* mutants, the carpels are homeotically transformed into variable numbers of stamens, and *OSH1* continues to be expressed after production of central stamens (Yamaguchi et al. 2004). Therefore, *DL* is also partially involved in the regulation of FM determinacy. Furthermore, the

indeterminate nature of *dl* is also promoted when combined with *mfo1* (Li et al. 2011).

ABERRANT PANICLE ORGANIZATION1 (*APO1*), which encodes an F-BOX protein similar to Arabidopsis *UFO*, has pleiotropic functions in both inflorescence and flower development (Ikeda et al. 2005, Ikeda et al. 2007). One function of *APO1* is the regulation of determinacy, as carpels are reiteratively formed in the *apo1* mutant. *APO1* promotes the expression of *OsMADS3*, and this may partially explain the loss of determinacy of the FM in *apo1* mutants. Defects in floral determinacy observed in each single mutant are strongly enhanced in *dl apo1* double mutants, in addition to homeotic change of stamens into lodicules (Ikeda et al. 2007).

Communication Between the Meristem and Lateral Organs

Signal from the meristem to lateral organs

Gene activity in the meristem is not restricted to the meristem itself, but also affects lateral organ development. Pioneering surgical experiments indicated that the leaf primordium develops into a radially symmetric abaxialized leaf, when an incision is made between the meristem center and the incipient leaf (P0) (Sussex 1951, Sussex 1954). This result suggests that a putative signal arising in the meristem specifies adaxial identity in the leaf primordia. Recent laser ablation studies have demonstrated the importance of the L1 layer of the meristem for the movement of the putative signaling molecule (Reinhardt et al. 2003, Reinhardt et al. 2005). This signaling seems to be transient, because the leaf develops normally without abaxialization when the incision is made between the meristem and an older leaf primordium (P2).

Molecular markers of adaxial and abaxial identity are expressed at early stages of stamen development in rice, with patterns similar to those observed in leaf development (Toriba et al. 2010). Subsequently, however, the expression patterns change, suggesting that rearrangement of adaxial–abaxial domains occurs. The former patterning probably depends on a signal from the meristem, whereas the latter patterning may result after release from the control of the meristem (Toriba et al. 2011). Therefore, the rearrangement of adaxial–abaxial polarity in the stamen might represent the transition from meristem-dependent development to organ-autonomous development. Although the molecular nature of this signal is still unknown, *MIR390* is a candidate for the signal from the SAM to the leaf primordia in Arabidopsis (Chitwood et al. 2009). Identification of the signal is critical for elucidation of the mechanism underlying communication between the meristem and lateral organs.

Signal from lateral organs to the meristem

Conversely, meristem activity is likely to be also affected by a signal from the lateral organs. Recent studies suggest that a class of *YABBY* (*YAB*) genes are involved in this process (Goldshmidt et al. 2008, Tanaka et al. 2012a).

In Arabidopsis, *FILAMENTOUS FLOWER* (*FIL*) and its related *YABBY* genes (*YAB2*, *YAB3* and *YAB5*) regulate leaf development, including establishment of adaxial–abaxial polarity and lamina expansion (Stahle et al. 2009, Sarojam et al. 2010). In addition, mutations in these genes result in defects associated with meristem function. For example, the expression domain of *CLAVATA3* and *WUS* is markedly expanded in the SAM of *fil* or *fil yab3* mutants, whereas the primary SAM fails to be maintained in triple and quadruple mutants of these *YABBY* genes.

In rice, mutation in the *TONGARI-BOUSHI1* (*TOB1*) gene results in pleiotropic defects in spikelet development, such as reduction in palea and lemma growth, formation of a seamless lemma/palea-like organ and production of two florets within a spikelet (Tanaka et al. 2012a, Tanaka et al. 2012b). In severe cases, the SM is arrested after formation of the sterile lemma. Formation of seamless organs and spikelets containing two florets is likely to be associated with a disorganized meristem. *TOB1* corresponds to *OsYABBY5* (Toriba et al. 2007), which belongs to the same subclass as FIL and YAB3.

Both rice *TOB1* and Arabidopsis *YABBY* genes are expressed in lateral organs, but not in the meristem (Goldshmidt et al. 2008, Tanaka et al. 2012a). The meristem defects observed in the above *YABBY* mutants are therefore likely to be caused by non-cell-autonomous action of the *YABBY* genes from the lateral organs. In Arabidopsis, mobility of YABBY protein or mRNA is not detected (Goldshmidt et al. 2008). Therefore, the FIL-clade *YABBY* genes are involved in communication between lateral organs and the meristem, possibly by producing a downstream signaling molecule that travels from the lateral organ to the meristem.

Perspective

In the past decade, much progress has been made towards understanding the molecular mechanisms underlying regulation of the fate, determinacy and maintenance of grass meristems. Many genes responsible for these activities have been isolated, and their functions have been revealed, together with genetic relationships between the genes. As discussed in the accompanying manuscript (Pautler et al. 2013), identification of positive regulators of stem cell maintenance, such as WUS, is also critical for understanding the determinacy of the FM. In Arabidopsis, determinacy is achieved by the repression of the positive regulator *WUS* by *AG*, after specification of the carpel. Although Class C genes such as rice *OsMADS58* and maize *zag1* are partially involved in this process, these genes do not specify the carpel in grasses, and meristems still persist after carpel specification by *DL*. Therefore, a complex mechanism might be required for repressing the putative WUS-like positive factor.

Furthermore, the genetic mechanism that regulates communication between the meristem and lateral organs is an

intriguing question in meristem biology. What genes are involved in the production and transduction of the signal connecting meristem maintenance and lateral organ development? Little is known about these important questions, even in Arabidopsis. We are expecting that increasing molecular genetic studies of maize and rice will contribute to understanding these issues.

Funding

This work was supported by the Ministry of Education, Science, Culture and Sport of Japan [Grants-in-Aid for Scientific Research for works on meristems (No. 23012011 to H.-Y.H.)]; the National Science Foundation Plant Genome Program of the USA [No. DBI-0604923 to D.J.]; National Institute of Food and Agriculture Award [No. 2010-04112 to D.J.]; the Japan Society for the Promotion of Science [a Research Fellowship for Young Scientists to W.T.]; NSERC (Canada) [a Post-Graduate Scholarship to M.P.].

Acknowledgment

We thank Drs. Y. Sato, F. Taguchi-Fumio and A. Yoshida for kindly providing us with images of rice mutants, and Dr. H. Yoshida for critical reading of the manuscript.

References

Ashikari, M., Sakakibara, H., Lin, S., Yamamoto, T., Takashi, T., Nishimura, A. et al. (2005) Cytokinin oxidase regulates rice grain production. *Science* 309: 741–745.

Barton, M.K. (2010) Twenty years on: the inner workings of the shoot apical meristem, a developmental dynamo. *Dev. Biol.* 341: 95–113.

Bomblies, K., Wang, R.L., Ambrose, B.A., Schmidt, R.J., Meeley, R.B. and Doebley, J. (2003) Duplicate *FLORICAULA/LEAFY* homologs *zfl1* and *zfl2* control inflorescence architecture and flower patterning in maize. *Development* 130: 2385–2395.

Bommert, P., Satoh-Nagasawa, N. and Jackson, D. (2013) Quantitative variation in maize kernel row number is controlled by the *FASCIATED EAR2* locus of maize. *Nat. Genet.* (in press).

Bommert, P., Satoh-Nagasawa, N., Jackson, D. and Hirano, H.-Y. (2005) Genetics and evolution of inflorescence and flower development in grasses. *Plant Cell Physiol.* 46: 69–78.

Bortiri, E., Chuck, G., Vollbrecht, E., Rocheford, T., Martienssen, R. and Hake, S. (2006) *ramosa2* encodes a LATERAL ORGAN BOUNDARY domain protein that determines the fate of stem cells in branch meristems of maize. *Plant Cell* 18: 574–585.

Bortiri, E. and Hake, S. (2007) Flowering and determinacy in maize. *J. Exp. Bot.* 58: 909–916.

Bowman, J.L., Smyth, D.R. and Meyerowitz, E.M. (1989) Genes directing flower development in *Arabidopsis*. *Plant Cell* 1: 37–52.

Bradley, D., Ratcliffe, O., Vincent, C., Carpenter, R. and Coen, E. (1997) Inflorescence commitment and architecture in *Arabidopsis*. *Science* 275: 80–83.

Brown, P.J., Upadyayula, N., Mahone, G.S., Tian, F., Bradbury, P.J., Myles, S. et al. (2011) Distinct genetic architectures for male and female inflorescence traits of maize. *PLoS Genet.* 7. e1002383.

Chitwood, D.H., Nogueira, F.T., Howell, M.D., Montgomery, T.A., Carrington, J.C. and Timmermans, M.C. (2009) Pattern formation via small RNA mobility. *Genes Dev.* 23: 549–554.

Cho, Y.H., Yoo, S.D. and Sheen, J. (2006) Regulatory functions of nuclear hexokinase1 complex in glucose signaling. *Cell* 127: 579–589.

Chuck, G., Meeley, R.B. and Hake, S. (1998) The control of maize spikelet meristem fate by the APETALA2-like gene *indeterminate spikelet1*. *Genes Dev.* 12: 1145–1154.

Chuck, G., Meeley, R. and Hake, S. (2008) Floral meristem initiation and meristem cell fate are regulated by the maize *AP2* genes *ids1* and *sid1*. *Development* 135: 3013–3019.

Chuck, G., Meeley, R., Irish, E., Sakai, H. and Hake, S. (2007) The maize *tasselseed4* microRNA controls sex determination and meristem cell fate by targeting *Tasselseed6/indeterminate spikelet1*. *Nat. Genet.* 39: 1517–1521.

Chuck, G., Muszynski, M., Kellogg, E., Hake, S. and Schmidt, R.J. (2002) The control of spikelet meristem identity by the *branched silkless1* gene in maize. *Science* 298: 1238–1241.

Danilevskaya, O.N., Meng, X. and Ananiev, E.V. (2010) Concerted modification of flowering time and inflorescence architecture by ectopic expression of TFL1-like genes in maize. *Plant Physiol.* 153: 238–251.

Dreni, L., Jacchia, S., Fornara, F., Fornari, M., Ouwerkerk, P.B.F., An, G. et al. (2007) The D-lineage MADS-box gene *OsMADS13* controls ovule identity in rice. *Plant J.* 52: 690–699.

Dreni, L., Pilatone, A., Yun, D., Erreni, S., Pajoro, A., Caporali, E. et al. (2011) Functional analysis of all AGAMOUS subfamily members in rice reveals their roles in reproductive organ identity determination and meristem determinacy. *Plant Cell* 23: 2850–2863.

Eveland, A.L. and Jackson, D.P. (2012) Sugars, signalling, and plant development. *J. Exp. Bot.* 63: 3367–3377.

Eveland, A.L., Satoh-Nagasawa, N., Goldshmidt, A., Meyer, S., Beatty, M., Sakai, H. et al. (2010) Digital gene expression signatures for maize development. *Plant Physiol.* 154: 1024–1039.

Gallavotti, A., Long, J.A., Stanfield, S., Yang, X., Jackson, D., Vollbrecht, E. et al. (2010) The control of axillary meristem fate in the maize ramosa pathway. *Development* 137: 2849–2856.

Gallavotti, A., Zhao, Q., Kyozuka, J., Meeley, R.B., Ritter, M.K., Doebley, J.F. et al. (2004) The role of *barren stalk1* in the architecture of maize. *Nature* 432: 630–635.

Goldshmidt, A., Alvarez, J.P., Bowman, J.L. and Eshed, Y. (2008) Signals derived from *YABBY* gene activities in organ primordia regulate growth and partitioning of *Arabidopsis* shoot apical meristems. *Plant Cell* 20: 1217–1230.

Han, J.J., Jackson, D. and Martienssen, R. (2012) Pod corn is caused by rearrangement at the *Tunicate1* locus. *Plant Cell* 24: 2733–2744.

Huang, X., Qian, Q., Liu, Z., Sun, H., He, S., Luo, D. et al. (2009) Natural variation at the *DEP1* locus enhances grain yield in rice. *Nat. Genet.* 41: 494–497.

Ikeda, K., Ito, M., Nagasawa, N., Kyozuka, J. and Nagato, Y. (2007) Rice *ABERRANT PANICLE ORGANIZATION 1*, encoding an F-box protein, regulates meristem fate. *Plant J.* 51: 1030–1040.

Ikeda, K., Nagasawa, N. and Nagato, Y. (2005) *Aberrant panicle organization 1* temporally regulates meristem identity in rice. *Dev. Biol.* 282: 349–360.

Ikeda-Kawakatsu, K., Maekawa, M., Izawa, T., Itoh, J.-I. and Nagato, Y. (2012) *ABERRANT PANICLE ORGANIZATION 2/RFL*, the rice

ortholog of Arabidopsis *LEAFY*, suppresses the transition from inflorescence meristem to floral meristem through interaction with *APO1*. *Plant J.* 69: 168–180.

Ikeda-Kawakatsu, K., Yasuno, N., Oikawa, T., Iida, S., Nagato, Y., Maekawa, M. et al. (2009) Expression level of *ABERRANT PANICLE ORGANIZATION1* determines rice inflorescence form through control of cell proliferation in the meristem. *Plant Physiol.* 150: 736–747.

Ishikawa, M., Ohmori, Y., Tanaka, W., Hirabayashi, C., Murai, K., Ogihara, Y. et al. (2009) The spatial expression patterns of *DROOPING LEAF* orthologs suggest a conserved function in grasses. *Genes Genet. Syst.* 84: 137–146.

Jeon, J.S., Jang, S., Lee, S., Nam, J., Kim, C., Lee, S.H. et al. (2000) *leafy hull sterile1* is a homeotic mutation in a rice MADS box gene affecting rice flower development. *Plant Cell* 12: 871–884.

Jiao, Y., Wang, Y., Xue, D., Wang, J., Yan, M., Liu, G. et al. (2010) Regulation of *OsSPL14* by OsmiR156 defines ideal plant architecture in rice. *Nat. Genet.* 42: 541–544.

Kaplinsky, N.J. and Freeling, M. (2003) Combinatorial control of meristem identity in maize inflorescences. *Development* 130: 1149–1158.

Karim, M.R., Hirota, A., Kwiatkowska, D., Tasaka, M. and Aida, M. (2009) A role for *Arabidopsis PUCHI* in floral meristem identity and bract suppression. *Plant Cell* 21: 1360–1372.

Komatsu, M., Chujo, A., Nagato, Y., Shimamoto, K. and Kyozuka, J. (2003b) *FRIZZY PANICLE* is required to prevent the formation of axillary meristems and to establish floral meristem identity in rice spikelets. *Development* 130: 3841–3850.

Komatsu, K., Maekawa, M., Ujiie, S., Satake, Y., Furutani, I., Okamoto, H. et al. (2003a) *LAX* and *SPA*: major regulators of shoot branching in rice. *Proc. Natl Acad. Sci. USA* 100: 11765–11770.

Laudencia-Chingcuanco, D. and Hake, S. (2002) The *indeterminate floral apex1* gene regulates meristem determinacy and identity in the maize inflorescence. *Development* 129: 2629–2638.

Lee, D.Y. and An, G. (2012) Two AP2 family genes, *SUPERNUMERARY BRACT* (*SNB*) and *OsINDETERMINATE SPIKELET 1* (*OsIDS1*), synergistically control inflorescence architecture and floral meristem establishment in rice. *Plant J.* 69: 445–461.

Lee, D.Y., Lee, J., Moon, S., Park, S.Y. and An, G. (2007) The rice heterochronic gene *SUPERNUMERARY BRACT* regulates the transition from spikelet meristem to floral meristem. *Plant J.* 49: 64–78.

Lenhard, M., Bohnert, A., Jurgens, G. and Laux, T. (2001) Termination of stem cell maintenance in Arabidopsis floral meristems by interactions between *WUSCHEL* and *AGAMOUS*. *Cell* 105: 805–814.

Li, H., Liang, W., Hu, Y., Zhu, L., Yin, C., Xu, J. et al. (2011) Rice *MADS6* interacts with the floral homeotic genes *SUPERWOMAN1*, *MADS3*, *MADS58*, *MADS13*, and *DROOPING LEAF* in specifying floral organ identities and meristem fate. *Plant Cell* 23: 2536–2552.

Li, H., Liang, W., Jia, R., Yin, C., Zong, J., Kong, H. et al. (2010) The *AGL6*-like gene *OsMADS6* regulates floral organ and meristem identities in rice. *Cell Res.* 20: 299–313.

Li, X., Qian, Q., Fu, Z., Wang, Y., Xiong, G., Zeng, D. et al. (2003) Control of tillering in rice. *Nature* 422: 618–621.

Liu, X., Kim, Y.J., Muller, R., Yumul, R.E., Liu, C., Pan, Y. et al. (2011) *AGAMOUS* terminates floral stem cell maintenance in *Arabidopsis* by directly repressing *WUSCHEL* through recruitment of Polycomb Group proteins. *Plant Cell* 23: 3654–3670.

Lohmann, J.U., Hong, R.L., Hobe, M., Busch, M.A., Parcy, F., Simon, R. et al. (2001) A molecular link between stem cell regulation and floral patterning in *Arabidopsis*. *Cell* 105: 793–803.

Lopez-Dee, Z.P., Wittich, P., Enrico Pe, M., Rigola, D., Del Buono, I., Gorla, M.S. et al. (1999) *OsMADS13*, a novel rice MADS-box gene expressed during ovule development. *Dev. Genet.* 25: 237–244.

McSteen, P. (2006) Branching out: the ramosa pathway and the evolution of grass inflorescence morphology. *Plant Cell* 18: 518–522.

McSteen, P., Laudencia-Chingcuanco, D. and Colasanti, J. (2000) A floret by any other name: control of meristem identity in maize. *Trends Plant Sci.* 5: 61–66.

McSteen, P., Malcomber, S., Skirpan, A., Lunde, C., Wu, X., Kellogg, E. et al. (2007) *barren inflorescence2* encodes a co-ortholog of the PINOID serine/threonine kinase and is required for organogenesis during inflorescence and vegetative development in maize. *Plant Physiol.* 144: 1000–1011.

Mena, M., Ambrose, B.A., Meeley, R.B., Briggs, S.P., Yanofsky, M.F. and Schmidt, R.J. (1996) Diversification of C-function activity in maize flower development. *Science* 274: 1537–1540.

Miura, K., Ikeda, M., Matsubara, A., Song, X.J., Ito, M., Asano, K. et al. (2010) *OsSPL14* promotes panicle branching and higher grain productivity in rice. *Nat. Genet.* 42: 545–549.

Nickerson, N.H. (1959) Sustained treatment with gibberellic acid of five different kinds of maize. *Ann. MO Bot. Gard.* 46: 19–37.

Ohmori, S., Kimizu, M., Sugita, M., Miyao, A., Hirochika, H., Uchida, E. et al. (2009) *MOSAIC FLORAL ORGANS1*, an *AGL6*-like MADS box gene, regulates floral organ identity and meristem fate in rice. *Plant Cell* 21: 3008–3025.

Ookawa, T., Hobo, T., Yano, M., Murata, K., Ando, T., Miura, H. et al. (2010) New approach for rice improvement using a pleiotropic QTL gene for lodging resistance and yield. *Nat. Commun.* 1: 132.

Paul, M.J., Primavesi, L.F., Jhurreea, D. and Zhang, Y.H. (2008) Trehalose metabolism and signaling. *Annu. Rev. Plant Biol.* 59: 417–441.

Pautler, M., Tanaka, W., Hirano, H.-Y. and Jackson, D. (2013) Grass meristem I. Shoot apical meristem maintenance, axillary meristem determinacy and the floral transition. *Plant Cell Physiol.* 54: 302–312.

Rao, N.N., Prasad, K., Kumar, P.R. and Vijayraghavan, U. (2008) Distinct regulatory role for *RFL*, the rice *LFY* homolog, in determining flowering time and plant architecture. *Proc. Natl Acad. Sci. USA* 105: 3646–3651.

Reinhardt, D., Frenz, M., Mandel, T. and Kuhlemeier, C. (2003) Microsurgical and laser ablation analysis of interactions between the zones and layers of the tomato shoot apical meristem. *Development* 130: 4073–4083.

Reinhardt, D., Frenz, M., Mandel, T. and Kuhlemeier, C. (2005) Microsurgical and laser ablation analysis of leaf positioning and dorsoventral patterning in tomato. *Development* 132: 15–26.

Reinheimer, R. and Kellogg, E.A. (2009) Evolution of *AGL6*-like MADS box genes in grasses (Poaceae): ovule expression is ancient and palea expression is new. *Plant Cell* 21: 2591–2605.

Sarojam, R., Sappl, P.G., Goldshmidt, A., Efroni, I., Floyd, S.K., Eshed, Y. et al. (2010) Differentiating Arabidopsis shoots from leaves by combined YABBY activities. *Plant Cell* 22: 2113–2130.

Satoh-Nagasawa, N., Nagasawa, N., Malcomber, S., Sakai, H. and Jackson, D. (2006) A trehalose metabolic enzyme controls inflorescence architecture in maize. *Nature* 441: 227–230.

Sigmon, B. and Vollbrecht, E. (2010) Evidence of selection at the *ramosa1* locus during maize domestication. *Mol. Ecol.* 19: 1296–1311.

Stahle, M.I., Kuehlich, J., Staron, L., von Arnim, A.G. and Golz, J.F. (2009) YABBYs and the transcriptional corepressors LEUNIG and

LEUNIG_HOMOLOG maintain leaf polarity and meristem activity in *Arabidopsis*. *Plant Cell* 21: 3105–3118.

Sun, B. and Ito, T. (2010) Floral stem cells: from dynamic balance towards termination. *Biochem. Soc. Trans.* 38: 613–616.

Sun, B., Xu, Y., Ng, K.-H. and Ito, T. (2009) A timing mechanism for stem cell maintenance and differentiation in the *Arabidopsis* floral meristem. *Genes Dev.* 23: 1791–1804.

Sussex, I.M. (1951) Experiments on the cause of dorsiventrality in leaves. *Nature* 167: 651–652.

Sussex, I.M. (1954) Morphogenesis in *Solanum tuberosum* L.: experimental investigation of leaf dorsiventrality and orientation in the juvenile shoot. *Phytomorphology* 5: 286–300.

Tabuchi, H., Zhang, Y., Hattori, S., Omae, M., Shimizu-Sato, S., Oikawa, T. et al. (2011) *LAX PANICLE2* of rice encodes a novel nuclear protein and regulates the formation of axillary meristems. *Plant Cell* 23: 3276–3287.

Taguchi-Shiobara, F., Kawagoe, Y., Kato, H., Onodera, H., Tagiri, A., Hara, N. et al. (2011) A loss-of-function mutation of rice *DENSE PANICLE 1* causes semi-dwarfness and slightly increased number of spikelets. *Breed. Sci* 61: 17–25.

Tanaka, W., Toriba, T., Ohomori, Y. and Hirano, H.-Y. (2012b) Formation of two florets within a single spikelet in the rice *tongari-boushi1* mutant. *Plant Signal. Behav.* 7: 793–795.

Tanaka, W., Toriba, T., Ohmori, Y., Yoshida, A., Kawai, A., Mayama-Tsuchida, T. et al. (2012a) The *YABBY* gene *TONGARI-BOUSHI1* is involved in lateral organ development and maintenance of meristem organization in the rice spikelet. *Plant Cell* 24: 80–95.

Thompson, B.E., Bartling, L., Whipple, C., Hall, D.H., Sakai, H., Schmidt, R. et al. (2009) *bearded-ear* encodes a MADS box transcription factor critical for maize floral development. *Plant Cell* 21: 2578–2590.

Thompson, B.E. and Hake, S. (2009) Translational biology: from Arabidopsis flowers to grass inflorescence architecture. *Plant Physiol* 149: 38–45.

Toriba, T., Harada, K., Takamura, A., Nakamura, H., Ichikawa, A., Suzaki, T. et al. (2007) Molecular characterization the YABBY gene family in Oryza sativa and expression analysis of *OsYABBY1*. *Mol. Genet. Genomics* 277: 457–468.

Toriba, T., Ohmori, Y. and Hirano, H.-Y. (2011) Common and distinct mechanisms underlying the establishment of adaxial and abaxial polarity in stamen and leaf development. *Plant Signal. Behav.* 6: 430–433.

Toriba, T., Suzaki, T., Yamaguchi, T., Ohmori, Y., Tsukaya, H. and Hirano, H.-Y. (2010) Distinct regulation of adaxial–abaxial polarity in anther patterning in rice. *Plant Cell* 22: 1452–1462.

Vandesteene, L., Lopez-Galvis, L., Vanneste, K., Feil, R., Maere, S., Lammens, W. et al. (2012) Expansive evolution of the TREHALOSE-6-PHOSPHATE PHOSPHATASE gene family in *Arabidopsis*. *Plant Physiol* 160: 884–896.

Vollbrecht, E., Springer, P.S., Goh, L., Buckler, E.S. and Martienssen, R. (2005) Architecture of floral branch systems in maize and related grasses. *Nature* 436: 1119–1126.

Wingen, L.U., Münster, T., Faigl, W., Deleu, W., Sommer, H., Saedler, H. et al. (2012) Molecular genetic basis of pod corn (*Tunicate* maize). *Proc. Natl Acad. Sci. USA* 109: 7115–7120.

Weil, C.F. (2009) TILLING in grass species. *Plant Physiol.* 149: 158–164.

Yamaguchi, T., Lee, Y.D., Miyao, A., Hirochika, H., An, G. and Hirano, H.-Y. (2006) Functional diversification of the two C-class genes, *OSMADS3* and *OSMADS58*, in *Oryza sativa*. *Plant Cell* 18: 15–28.

Yamaguchi, T., Nagasawa, N., Kawasaki, S., Matsuoka, M., Nagato, Y. and Hirano, H.-Y. (2004) The YABBY gene *DROOPING LEAF* regulates carpel specification and midrib development in *Oryza sativa*. *Plant Cell* 16: 500–509.

Yamaki, S., Nagato, Y., Kurata, N. and Nonomura, K.-I. (2011) Ovule is a lateral organ finally differentiated from the terminating floral meristem in rice. *Dev. Biol.* 351: 208–216.

Yanofsky, M.F., Ma, H., Bowman, J.L., Drews, G.N., Feldmann, K.A. and Meyerowitz, E.M. (1990) The protein encoded by the *Arabidopsis* homeotic gene *agamous* resembles transcription factors. *Nature* 346: 35–39.

Yoshida, A., Ohmori, Y., Kitano, H., Taguchi-Shiobara, F. and Hirano, H.-Y. (2012) *ABERRANT SPIKELET AND PANICLE1*, encoding a TOPLESS-related transcriptional co-repressor, is involved in the regulation of meristem fate in rice. *Plant J.* 70: 327–339.

Yoshida, A., Sasao, M., Yasuno, N., Takagi, K., Daimon, Y., Chen, R. et al. (2013) *TAWAWA1*, a novel regulator of rice inflorescence architecture, functions through the suppression of meristem phase transition. *Proc. Natl. Acad Sci. USA* 110: 767–772.

Yoshida, A., Suzaki, T., Tanaka, W. and Hirano, H.-Y. (2009) The homeotic gene *LONG STERILE LEMMA* (*G1*) specifies sterile lemma identity in the rice spikelet. *Proc. Natl Acad. Sci. USA* 106: 20103–20108.

Yoshida, H. and Nagato, Y. (2011) Flower development in rice. *J. Exp. Bot.* 62: 4719–4730.

Zanis, M.J. (2007) Grass spikelet genetics and duplicate gene comparisons. *Int. J. Plant Sci.* 168: 93–110.

Zhu, Q.-H., Upadhyaya, N.M., Gubler, F. and Helliwell, C.A. (2009) Over-expression of miR172 causes loss of spikelet determinacy and floral organ abnormalities in rice (*Oryza sativa*). *BMC Plant Biol.* 9: 149.