Genetic and Molecular Bases of Rice Yield

Yongzhong Xing and Qifa Zhang

National Key Laboratory of Crop Genetic Improvement, National Center of Plant Gene Research (Wuhan), Huazhong Agricultural University, Wuhan 430070, China; email: qifazh@mail.hzau.edu.cn

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number of panicles, number of grains, grain weight, quantitative trait locus, agronomic trait, development

Abstract

Grain yield in rice is a complex trait multiplicatively determined by its three component traits: number of panicles, number of grains per panicle, and grain weight; all of which are typical quantitative traits. The developments in genome mapping, sequencing, and functional genomic research have provided powerful tools for investigating the genetic and molecular bases of these quantitative traits. Dissection of the genetic bases of the yield traits based on molecular marker linkage maps resolved hundreds of quantitative trait loci (QTLs) for these traits. Mutant analyses and map-based cloning of QTLs have identified a large number of genes required for the basic processes underlying the initiation and development of tillers and panicles, as well as genes controlling numbers and sizes of grains and panicles. Molecular characterization of these genes has greatly advanced the mechanistic understanding of the regulation of these rice yield traits. These findings have significant implications in crop genetic improvement.

Contents

INTRODUCTION

Rice (*Oryza sativa* L.) is one of the most important food crops worldwide. In the past half century, rice yield has benefitted from two major genetic improvements: improved harvest index and plant architecture through use of semidwarf genes, and production of hybrids that exploit heterosis. Consequently, rice yield has more than doubled in most parts of the world and even tripled in certain countries and regions in the last 50 years.

Rice has also been adopted as an important model system for plant science research because (*a*) rice has the smallest genome among crop plants; (*b*) rice shares substantial colinearity with members of the grass family, including all the important cereals (69); (*c*) high-precision genome sequences have been obtained (34); (*d*) efficient transformation technology is available; (*e*) germplasm resources and genetic stocks are abundant; (*f*) saturated molecular maker linkage maps have enabled mapping and identification of hundreds of genes and quantitative trait loci (QTLs); and (*g*) breeding programs are diverse and large in scale. There have also been coordinated efforts in rice functional genomics research, which have made tremendous progress (24, 38, 120). Among the most important outcomes is that large numbers of genes have been cloned and functionally characterized, many of which are directly related to yield traits. Such progress has greatly enhanced the understanding of the genetic and molecular controls and related biological processes underlying the formation of yield traits in rice.

The objective of this review is to provide an overview of such progress in the context of yield as a complex agronomic trait. We also discuss the implications of these findings for advancing the knowledge base and the significance in crop genetic improvement.

YIELD AND ITS COMPONENT TRAITS

As a complex agronomic trait, grain yield of a rice plant is multiplicatively determined by three component traits: number of panicles per plant, number of grains per panicle, and grain weight. Number of panicles is dependent on the ability of the plant to produce tillers (tillering ability), including primary, secondary, and tertiary tillers. Number of grains per panicle can also be attributed to two subcomponents: number of spikelets, which is mainly determined by the numbers of primary and secondary branches, and seed setting rate of the spikelets. Grain weight is largely determined by grain size, which is specified by its three dimensions (length, width, and thickness), and the degree of filling.

Rice varieties differ tremendously in the levels of grain yield, with immense variability in the combinations of component traits owing to the vast diversity of genetic constitutions. In addition, yield levels of rice varieties are also greatly influenced by the environmental conditions and the field management practices. There are also remarkable interactions

between genotypes and environments such that varieties are adapted to specific environmental conditions.

DISSECTION OF THE GENETIC BASES OF RICE YIELD TRAITS

The inheritance of quantitative traits classically involves multiple genes, each having a small effect that is sensitive to environmental changes. These traits are known in general as having low heritability and thus have earned the reputation of being difficult to investigate. However, the development of molecular marker, genome mapping, and QTL analysis technologies have greatly facilitated the investigation of genetic bases of quantitative traits. In rice, researchers have constructed high-density genetic linkage maps based on restriction fragment length polymorphism (RFLP) and simple sequence repeat (SSR) markers (50, 65, 66). Mapping populations specifically designed for dissecting the genetic bases of yield traits via QTL mapping have been constructed, producing large amounts of data leading to the identification of hundreds of QTLs for yield traits. While it is not our intent here to present a comprehensive account of all of the QTLs identified, we summarize some of the salient features of the QTLs for yield traits. For ease of comparison, we use mostly the results from populations developed from a cross between two *indica* rice varieties, Zhenshan 97 and Minghui 63, the parents of Shanyou 63, the mostly widely cultivated hybrid in China in the 1980s and 1990s. This includes an $F_{2:3}$ population, a VF_2 population, a recombinant inbred line (RIL) population, and an immortalized F_2 population. In the $F_{2:3}$ population, a linkage map was constructed using 240 F_2 individuals and the yield traits were measured using the F_3 families (117); the VF_2 population was obtained by ratooning the F_2 plants and transplanting the vegetative shoots (55); 240 RILs were derived by single-seed descent of the F_2, and a linkage map based on the RILs was again constructed (111); and three rounds of random crossing of the 240 RILs produced a population of 360 hybrids that were analogous to an F_2 population in its genetic composition, thus referred to as immortalized F_2 (IF_2) (26, 27). The yield data for each population were obtained from replicated field trials in two years. When comparable information is available, data from other populations are also included.

QTLs for the Yield Traits

The QTLs resolved in the four populations over 10 years (27, 55, 111, 117) are summarized in **Figure 1** and **Table 1**. For grain yield per plant, 10 QTLs were detected in $F_{2:3}$, 5 in VF_2, 4 in RIL, and 7 in IF_2 populations. These QTLs are distributed in 20 distinct locations and thus regarded distinct QTLs. Only 5 of the 20 distinct QTLs were recovered in two or more populations. Inspection of the QTLs detected in each of the populations showed that only 5 QTLs have relatively large effects if a cutoff is made by adopting a threshold individually explaining >10% of the variation of the trait (**Table 2**). Moreover, only 2 of the 10 QTLs in $F_{2:3}$ (117), 1 of the 5 in VF_2 (55), 2 of the 4 in RIL (111), and none of the 7 in IF_2 (27) were observed in both years of the field experiments (**Table 1**).

For number of tillers per plant, 4 QTLs were identified in $F_{2:3}$, 3 in VF_2, 6 in RIL, and 10 in IF_2, which were located in 21 distinct regions and thus regarded as distinct QTLs (**Figure 1**). Only 2 of the 21 QTLs could be detected in two populations, and the remaining 19 were each detected in only a sinlge population. Two QTLs could individually explain >10% of the variation of the trait (**Table 2**). Two of the QTLs were detected in both years in VF_2, whereas only one QTL was detected in both years in each of $F_{2:3}$, RIL and IF_2 populations (**Table 1**).

For number of spikelets (grains) per panicle, 8 QTLs were identified in $F_{2:3}$, 7 in VF_2, 5 in RIL and 7 in IF_2, which were situated in 17 distinct locations (**Figure 1**). Seven of the 17 distinct QTLs were detected in two or more populations. Six QTLs could individually explain >10% of the variation of the trait (**Table 2**). Four of the 8 QTLs in $F_{2:3}$, 1 of the 7 in VF_2,

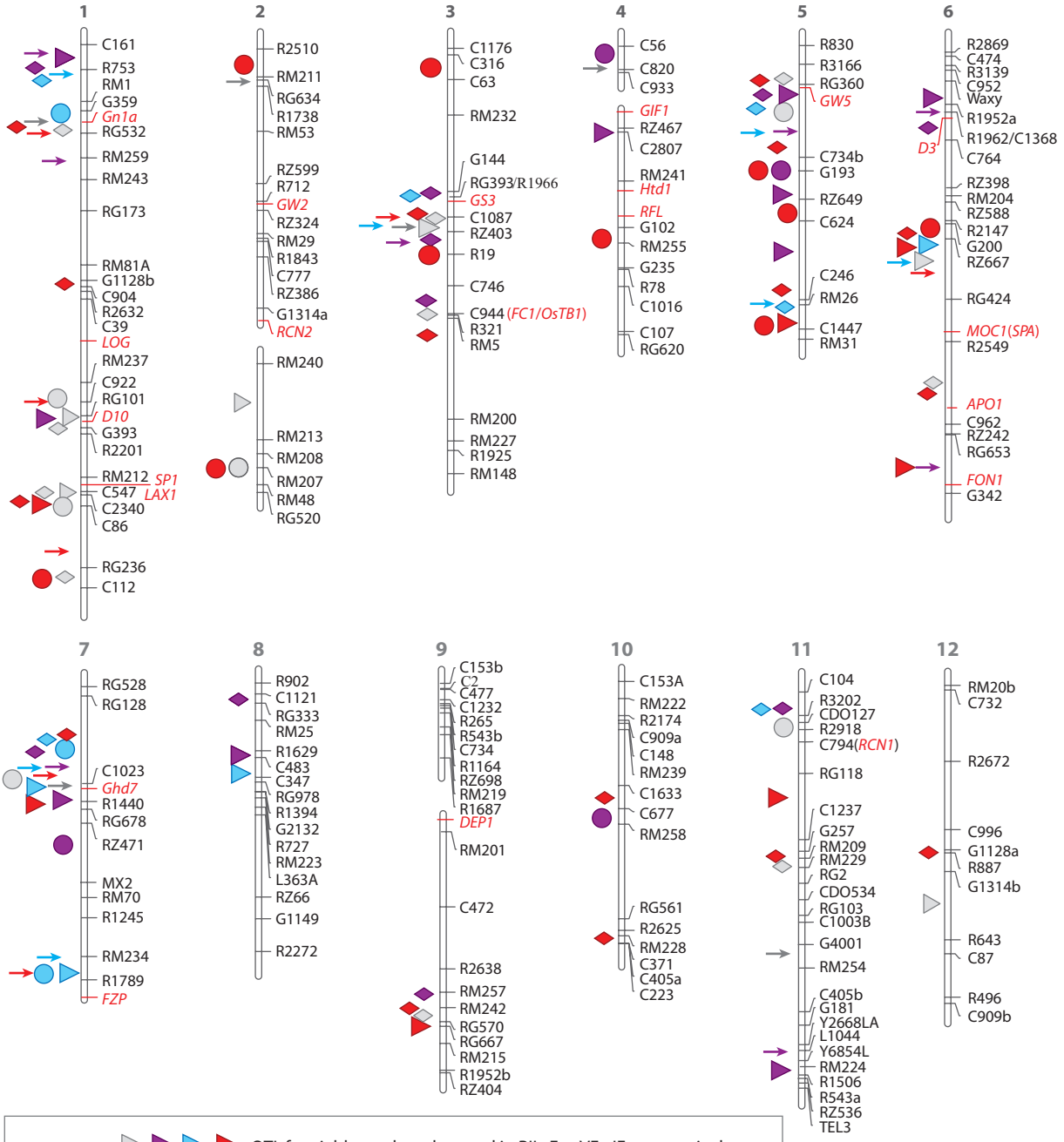

Table 1 Numbers of QTLs detected in two years in each population derived from the cross between Zhenshan 97 and Minghui 63

Population	Yield			Tillers/plant			Spikelets/panicle			Grain weight		
	Year 1	Year 2	Both	Year 1	Year 2	Both	Year 1	Year 2	Both	Year 1	Year 2	Both
$F_{2:3}$	5	6	1	3	2	1	5	7	4	7	9	6
VF_2	2	3	1	3	2	2	3	5	1	5	5	4
RIL	3	4	2	3	4	1	3	5	3	7	6	4
IF_2	3	4	0	6	5	1	4	6	3	12	13	9

Table 2 QTLs that individually explain >10% of the variation for the yield traits in the populations derived from the cross between Zhenshan 97 and Minghui 63

Traits	Chromosome	Marker interval	Populations	Largest var%	Also detected by others
Yield	1	C547-C2340	RIL	10.0	(51)
	5	G193-RZ649	$F_{2:3}$	11.7	(14, 36, 108)
	5	C624-C246	$F_{2:3}$	10.2	
	6	R2147-RG424	VF_2, IF_2	18.5	(12)
	7	C1023-R1440	VF_2, IF_2	19.8	(108)
Tillers/plant	7	RZ471-MX2	$F_{2:3}$	11.3	(36)
	7	C1023-R1440	RIL	12.7	(43)
Spikelets/panicle	1	RM1-R753	$F_{2:3}$	15.5	(80)
	1	RG173-RG532	$F_{2:3}$	17.8	(58)
	3	C1087-RZ403	$F_{2:3}$, RIL	17.6	(68, 77)
	5	RG360-C734	VF_2	15.2	(67, 108)
	5	C624-C246	VF_2	31.7	(14, 124)
	7	C1023-R1440	VF_2, IF_2	16.3	(14, 121)
Grain weight	1	G359-RG532	RIL	10.2	(62, 123)
	3	C1087-RZ403	$F_{2:3}$,VF_2 RIL, IF_2	24.0	(11, 95)
	3	C944-C746	$F_{2:3}$	16.8	
	3	R1966-G144	$F_{2:3}$	22.5	
	5	RG360-C734	$F_{2:3}$, VF_2, RIL	24.1	(62, 108)
	7	C1023-RG128	$F_{2:3}$, VF_2	21.8	(80)

Var%, percentage of variation explained by the QTL.

3 of the 5 in RIL, and 3 of the 7 in IF_2 were detected in both years (**Table 1**).

For grain weight, 10 QTLs were identified in $F_{2:3}$, 6 inVF_2, 9 in RIL and 16 in IF_2, which are distributed in 24 distinct locations (**Figure 1**). Thirteen of the 24 distinct QTLs were detected in two or more populations. Six QTLs could individually explain >10% of the variation of the trait (**Table 2**). Six of the 10 in $F_{2:3}$, 4 of the 6 in VF_2, 4 of the 9 in RIL, and 9 of the 13 in IF_2 were detected in both years.

The above comparison reveals several features regarding the QTL analyses. First, the number of QTLs that can be detected in a single experiment or single population is usually small, typically—three to six for a typical yield

Figure 1

A genetic linkage map showing the positions of yield quantitative trait loci (QTLs) based on the data from populations derived from the cross between Zhenshan 97 and Minghui 63. The map is adapted from Xing et al. (111). The cloned genes related to the yield traits are indicated in red.

trait (**Table 1**). However, this does not indicate that the trait variation in that particular population is contributed only by a handful genes. Rather, the small number of QTLs reflects an artifact resulting from the very stringent thresholds used to control false positive effects (type I error) in the QTL analyses (52, 101, 118). The consequence of such stringent thresholds is that only loci conferring sufficiently large effects could be recovered as QTLs, leaving the majority of the genes causing smaller effects in that particular experiment undetected. Second, smaller numbers of QTLs were observed for yield, the most complex trait, than its component traits. One reason for such seemingly surprising results is that the data for yield as a trait usually are subjected to more experimental errors, as any experimental error in any of the component traits would accrue to amplify the experimental errors of the yield data. Such experimental errors reduce the statistical power of QTL detection. Moreover, the component traits of the yield are often negatively correlated. If such negative correlations involve the same or closely linked genes, the genetic effects of these genes or genic regions on yield would cancel each other, producing as a consequence that fewer QTLs would be detected for yield per se than for its component traits. Third, among the three yield component traits, the number of QTLs resolved for grain weight is the largest followed by number of spikelet per panicle, whereas the QTL number for tillers is the smallest. And QTLs for number of tillers are less repeatable compared with the other two component traits. This is because tillering as a developmental process is more susceptible to environmental conditions and management practice under field conditions. All these features commonly occur in QTL analyses of yield traits in the literature.

Interactions among genes, or epistasis, also have major contributions to the genetic bases of the yield traits. Analyses based on molecular marker linkage maps (27, 111, 117) showed that significant digenic interactions, including additive by additive, additive by dominance, and dominance by dominance, are frequent and widespread in the genome, contributing significantly to the genetic bases of the yield traits. More than 100 digenic interactions were usually detected for each of the traits in each of the populations, many of which were repeatedly detected in both years. The interactions involve large numbers of marker loci, most of which are not detectable on a single-locus basis. However, analytic tools still needs to be further developed to accommodate such epistatic effects in a single model to evaluate their relative importance against the QTLs.

Validation of QTLs

Although thousands of QTLs have been reported in the literature (**http://www.gramene.org/plant_ontology/#to**), since QTLs are statistically determined, their biological significance needs to be validated individually by further experimentation. QTL validation can be summarized as three approaches. The first approach is development of QTL-based nearly isogenic lines (NILs) by crossing the two lines (usually the parents of the QTL mapping population) carrying the contrasting alleles of the QTL followed by successive backcrossing. Large numbers of QTLs have been validated using this approach in a number of plant species (some examples in rice are listed in **Table 3**). In a progeny population from a cross between the NILs, the trait controlled by the QTL would segregate in a simple Mendelian fashion, and the QTL can thus be precisely mapped and even cloned (see next section). However, the disadvantage of this approach is the large effort required to develop NILs for a single QTL. An alternative approach is construction of contiguous segmental substitution lines, in which a complete set of lines is developed by crossing and backcrossing the recipient line with a donor line. Each of the segmental substitution lines contains a small genomic fragment from the donor in the background of the recipient line, and collectively these substituted segments represent the entire donor genome. If the recipient and donor lines happen to be the parents of a QTL mapping population, analyses of the

Table 3 Major QTLs validated for rice yield traits in nearly isogenic genetic backgrounds

Traits	QTL	Chromosome	Intervals	Genetic effects			References
				A	D	Var%	
GL (mm)	*GS3*	3	GS09-MRG5881	1.47	1.06	95.6	18
GW (g)	*gw3.1*	3	JL8-RM3180			41.4	54
GW (g)	*gw8.1*	8	RM531-RM42	0.96		39.0	110
GW (g)	*gw9.1*	9	RM5661-RM215	0.98		42.5	109
GPP	*Gn1a*	1	3A28-3A20			44.0	4
GPP	*gpa7*	7	RM3325-3683	31.2	15.2	54.0	96
SPP	*SPP1*	1	YN27-YN34	22.05	0.55	63.4	63
SPP	*qSPP1*	1	MRG2746-RM490	16.7	4.8	68.1	121
SPP	*qSPP2*	2	MRG2762-MRG3515	12.3	3.7	73.0	121
SPP	*qSPP3*	3	RM135-RM49	21.8	8.0	68.8	121
SPP	*qSPP7*	7	RM3859-C39	59.8	28.3	74.2	112
SPP	*QSpp8*	8	RM544-RM310	28.3	22.6	83.0	122
GY	*qGY2-1*	2	RM279-RM5654			25.9	25

GL, grain length; GW, grain weight; GPP, grains per panicle; SPP, spikelets per panicle; GY, grain yield; A, additive effect; D, dominant effect; Var%, percent of variation explained by the QTL.

resulting substitution lines would allow not only the validation of the QTLs detected but also the discovery of loci with smaller effects that cannot be detected in ordinary QTL analyses. As a third approach, Tanksley & Nelson (94) outlined an advanced backcross QTL analysis method for simultaneous discovery and transfer of QTLs from unadapted germplasm into elite breeding lines. Using this approach, genomic segments from many exotic germplasms, including wild relatives of tomato, were introgressed into the backgrounds of elite cultivars. The resulting lines were not only immediately useful for breeding but also provided desirable materials for cloning and identification of the genes (20, 21). More recently, this method has been expanded into a strategy of molecular breeding by large-scale introgression of exotic germplasms into breeding lines in rice empowered by new genomic tools (59).

GENES CONTROLLING RICE YIELD TRAITS

Number of Tillers

Rice tillers are produced by shoot branching, which consists of two distinct steps. The first step is the formation of the axillary meristem on every leaf axil, which subsequently generates a few lateral leaves to form an axillary bud. The bud may continue outgrowth or become dormant until outgrowth is triggered. The second step is the outgrowth of axillary buds to form shoot branches called tillers. Normally, the first primary tiller of a rice plant would become visible when the main culm has the fourth leaf, and the second primary tiller would come out when the main culm has the fifth leaf (**Figure 2**). Similarly, a secondary tiller would extend out of the leaf sheath on the primary tiller when the primary tiller has the fourth leaf. Two consecutive tillers born on the same culm appear at 180°, and the secondary tillers are perpendicular to their respective primary tillers. If the environmental conditions permit, this process would continue to produce a large number of tillers, including primary, secondary, tertiary, and even high-ordered tillers.

The activity of axillary buds is regulated by complex interactions of phytohormones, which are controlled by genetic, developmental, and environmental signals (53, 82). According to the well-known concept of apical dominance, auxin provided by the primary shoot apex inhibits the growth of axillary buds, whereas

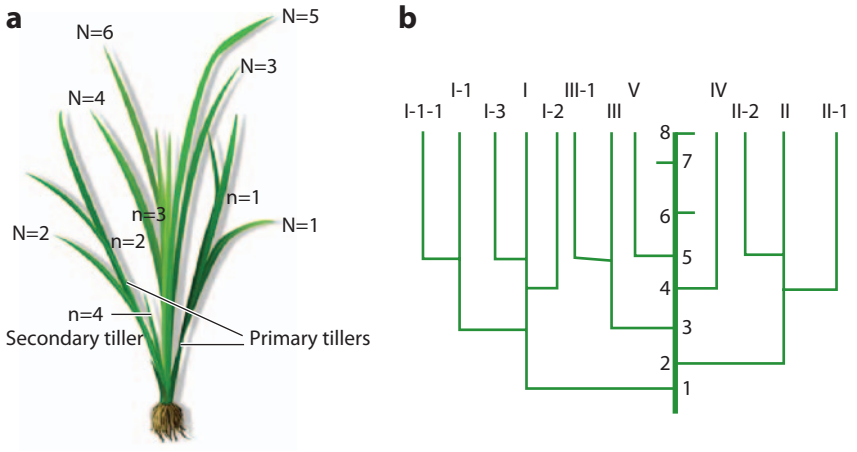

Figure 2

Synchronous growth of rice leaves and tillers. (*a*) Tillers and leaves when the plant is at the six-leaf stage. N represents the order of leaves in the main culm and n indicates tillers. (*b*) A model indicating tillers when the eighth leaf in the main culm is expanded. I, the first primary tiller; I-1, the first secondary tiller on the first primary tiller; I-1-1, the first tertiary tiller on the first secondary tiller of the first primary tiller. The same designations apply to II, III, etc.

cytokinin relieves the inhibition, resulting in the outgrowth of lateral branches (90).

Several mutants have been molecularly analyzed to understand the regulation of tillering in rice. The *moc1* mutant has a mono-culm phenotype producing no tillers owing to a defect in the formation of tiller buds (57). *MOC1* is the first gene isolated for rice tillering using a map-based cloning approach. The predicted protein is highly homologous to the tomato *LATERAL SUPPRESSOR* (79), whose loss-of-function mutation causes a branchless phenotype owing to a failure in axillary meristem initiation. Homology analysis demonstrated that *MOC1* is a member of the plant-specific GRAS family proteins. It has a nuclear localization, is expressed mainly in the axillary buds, and functions to initiate axillary buds and promote their outgrowth.

The molecular analysis of a series of bushy mutants in several plants suggested a novel graft-transmissible signal acting to inhibit bud growth (5, 53, 71). Three *Arabidopsis* mutants, *max2* to *max4* and pea *RMS1*, provided insights into the understanding of the signaling molecule. *MAX3* encoding carotenoid cleavage dioxygenase and *MAX4/RMS1* encoding polyene dioxygenase are predicted to function in the production of novel signaling molecules (6, 87), which are now identified as strigolactones (23, 98), acting downstream of auxin to regulate bud outgrowth (10). *MAX2* encodes an F-box protein identical to previously reported *ORE9* and is proposed to be required for the perception of the signal (88, 106).

A class of bushy mutants in rice also shows increased tiller numbers and reduced plant height. The axillary meristems of these mutants are normally established, but the activity of a tiller bud is not properly suppressed compared with the wild type, giving rise to a very large numbers of tillers, suggesting the critical roles of these genes in suppressing bud activity. This includes *D3* encoding an F-box leucine-rich-repeat (LRR) protein orthologous to *Arabidopsis* MAX2/ORE9 (35), *Htd1* encoding the rice ortholog of *Arabidopsis* MAX3 (125), *D10* encoding a carotenoid cleavage dioxygenase 8 orthologous to MAX4/RMS1/DAD1 (3), and *D27* encoding an iron-containing protein (61). All are involved in biosynthesis and signaling of strigolactones, indicating that monocots and

dicots share a conserved branching sinal pathway (104).

Enhanced apical dominance has been regarded as an important consequence of the domestication of crop plants. The *teosinte branched1* (*tb1*) gene has been identified as a major contributor to this evolutionary change in maize, which acts to repress the growth of axillary organs and to enable the formation of the female inflorescences (16). Takeda et al. (92) studied the function of *OsTB1*, the rice homolog of the maize *TB1*, which encodes a transcription factor containing a basic helix-loop-helix (bHLH) type of DNA-binding motif. Transgenic rice plants overexpressing *OsTB1* show greatly reduced lateral branching without affecting the initiation of axillary buds, whereas a loss-of-function mutant of *OsTB1* exhibits enhanced lateral branching, indicating that *OsTB1* functions as a negative regulator for lateral branching in rice. Expression of *OsTB1* is significantly reduced in the *moc1* mutant (57), suggesting that *OsTB1* is downstream of *MOC1*. These examples indicate the conservation of mechanisms for controlling axillary bud activity between monocot and eudicot plants.

Although the examples described above represent tremendous progress in the study of tillering control, there are still important missing links. The most important gap is perhaps that variations of tiller numbers in the genetic populations for QTL analyses are usually quantitative rather than qualitative like the mono-culm or bushy mutants. Such quantitative variations may involve different mechanisms of regulation. Moreover, the quantitative difference in tiller number can provide a useful resource for rice breeding. Thus, cloning of QTLs for tillering would be necessary for understanding and utilizing this type of variation.

In addition, there have also been reports of environmental regulations of tillering. For example, tillering is strongly affected by planting density and fertilizer level. High plant density decreasing light quantity and changing light quality leads to the shade avoidance syndrome, which produces reduced branching, increased plant height, and decreased leaf-blade area (41, 84). These morphological changes are accompanied by a redistribution of auxin (85). Red light is absorbed by plants and far-red light is reflected. The red to far-red ratio is perceived by phytochrome. In *Arabidopsis*, phyB is believed to be the main photoreceptor of light, which is probably antagonized by phyA (13). Overexpression of *Arabidopsis phyA* in rice produced more tillers and higher grain yield than its wild type (22). Strigolactone biosynthesis is induced by low phosphorous resulting in fewer tillers (7), while with high nutrient levels strigolactone synthesis in rice roots is inhibited and thus more tillers are expected (98).

From an agronomic viewpoint, grain yield is usually contributed by the primary tillers and some early secondary tillers, whereas tertiary and late secondary tillers make little contribution, although they also consume nutrients, water, and photosynthates. In breeding programs, there is a trend to breed for fewer and larger panicles to achieve high yield (42). Thus, the ability to suppress axillary bud activity in a timely manner through genetic manipulation would be desirable for increasing rice yield.

Number of Grains Per Panicle

The number of grains per panicle is usually highly proportional to the spikelet number. To understand the making of the number of grains per panicle, it is essential to understand the basic biological processes of panicle development, as well as the differentiation of meristems into spikelets. From an agronomic perspective, the number of spikelets per panicle can be attributed to two components: the duration of panicle differentiation and the rate of spikelet differentiation (30). Thus, we first give a brief account of the current mechanistic understanding of the genes underlying the process of panicle development, then turn to genes and processes regulating the duration and rate of panicle differentiation.

Genes for the basic processes of panicle development. Transition from the vegetative phase to reproductive phase is marked by the

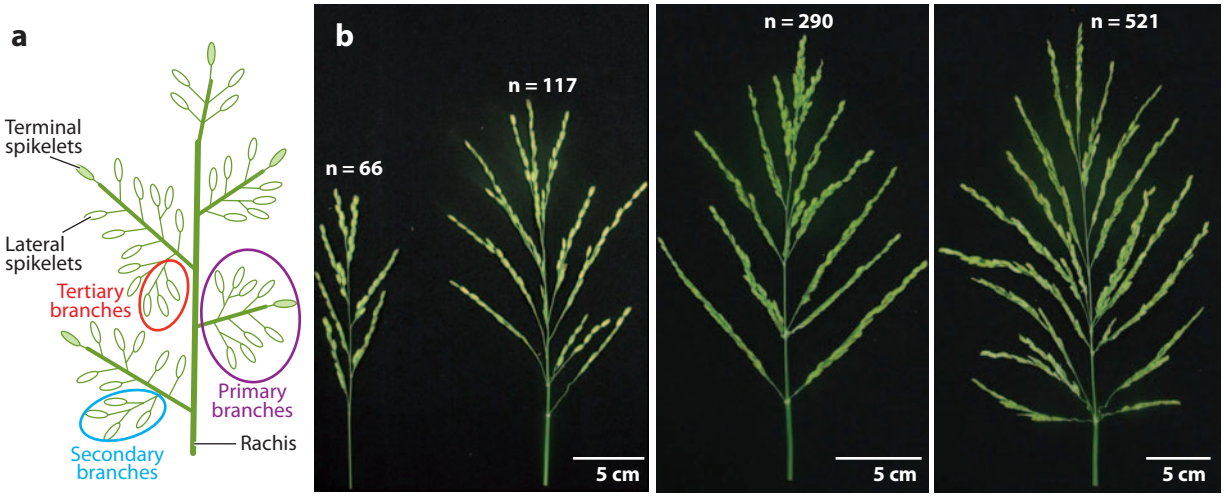

Figure 3

(*a*) Sketch of a rice panicle. A mature rice panicle consists of the main stem, often referred to as the rachis, a number of primary branches, one or more secondary branches on the primary branch, and occasionally tertiary branches on the secondary branch. There are lateral spikelets and terminal spikelets on the branches. The primary node is where the first primary branch formed, and the vestige is where the terminal primary branch formed. (*b*) Rice panicles of various sizes with 66, 117, 290, and 521 spikelets.

transformation of the shoot apical meristem into an inflorescence (or panicle) meristem. During panicle development, the panicle meristem produces a number of lateral meristems, each of which becomes a primary branch. The panicle meristem eventually loses its activity by forming the terminal primary branch marked by a vestige at the base. Each of the primary branches also produces a certain number of second-order lateral meristems. Some of the earlier second-order lateral meristems differentiate into secondary branches, each of which bears a certain number of spikelets, while the later ones directly form spikelets. Tertiary branches may similarly form on the secondary branches. Several panicles of different sizes are illustrated in **Figure 3**. In genotypes with a large number of spikelets per panicle, secondary branches often contribute more than half of the total spikelets (30, 114).

Analyses of a series of mutants have uncovered a number of genes involved in the regulation of panicle development. In the *lax panicle1* mutants, initiation and/or maintenance of rachis branches, lateral spikelets, and terminal spikelets are severely restricted, indicating that the *LAX1* gene is required for the initiation/maintenance of axillary meristems in the rice panicle (46). Molecular cloning of the gene identified that *LAX1* encodes a bHLH transcription factor, which is involved in the formation of all types of axillary meristems throughout the ontogeny of a rice plant (44). Reduction of the tiller number was also observed in the mutant of *lax1-2*, a strong allele of *LAX1*, suggesting that *LAX1* function is required for the generation of axillary meristems of both tillers and panicles (73). Interestingly, the accumulation of LAX PANICLE1 protein in axillary meristem formation in rice is subjected to a two-step regulation such that in *lax1* mutants, the proliferation of meristematic cells is initiated but fails to progress into the formation of axillary meristem. There is a close relationship between axillary meristem initiation and leaf development such that plastochron stage 4 of leaf development is crucial for axillary meristem formation. *LAX1* expression starts in the axils of leaves at this stage, and LAX1 protein is trafficked to the axillary meristem, which occurs only around this stage.

SMALL PANICLE (*SPA*) is another main regulator of axillary meristem formation (44). Similar to *lax* mutants, panicle branches and spikelets of *spa* mutants are severely reduced, but the mutations do not seem to affect tillering. Analysis of the nucleotide sequence of the coding region of *MOC1* in the *spa* mutant revealed that *spa* is a new allele of *MOC1* (*moc1-3*) (73). The panicle of the *lax1moc1-3* double mutant becomes a wirelike structure devoid of all branches, and initiation of tillers and vegetative branch shoots is almost completely suppressed.

In a mutant called *frizzy panicle* (*fzp*), the formation of florets is replaced by sequential rounds of branching, such that several rudimentary glumes are formed in place of the spikelet (45, 46). Instead of proceeding to floret formation as in the wild type, axillary meristems are formed in the axils of rudimentary glumes, which either arrest or develop into branches of higher order. The *fzp* mutant phenotype suggests that *FZP* is required for establishment of floral meristem identity by inhibiting the formation of axillary meristems within the spikelet meristem. The *FZP* gene was isolated by transposon tagging. *FZP* encodes a protein containing the ethylene-responsive element-binding factor (ERF) domain and is the rice ortholog of the maize *BD1* gene, which controls spikelet meristem identity (15).

Genes regulating the rate of spikelet formation. In addition to genes controlling the establishment of meristem identity, genes that control cell proliferation, which in turn affects meristem size and thus eventually regulates the rate of spikelet differentiation, are critical for panicle size and spikelet number. Two types of branches, rachis branches and spikelets, are produced during rice panicle development. In a mutant called *aberrant panicle organization 1 (apo1)*, the inflorescence meristem is precociously converted to a spikelet meristem after producing a small number of branch primordia (32). Another aberrant phenotype of this mutant involves inflorescence architecture, floral organ identity, and leaf production rate. Molecular studies (31, 33) revealed that *APO1* encodes an F-box protein, an ortholog of *Arabidopsis UNUSUAL FLORAL ORGAN* (*UFO*), which is a positive regulator of class-B genes. APO1 positively regulates spikelet number by suppressing the precocious conversion of inflorescence meristems to spikelet meristems. The panicle size is highly correlated with the expression level of *APO1*. Mutants with elevated levels of *APO1* expression produce an increased number of spikelets, primarily through increasing the rate of cell proliferation in the meristem, and overexpression of *APO1* increases branches and spikelets. In addition, *APO1* is associated with the regulation of the plastchron, floral organ identity, and floral determinacy. Unlike *UFO*, APO1 positively regulates class-C floral homeotic genes, rather than class-B genes, possibly indicating divergence of functions of *APO1* and *UFO* during evolution.

According to the concept of apical dominance, the relationship between shoot growth and branching is regulated by a balance between auxin that inhibits the growth of axillary buds and cytokinin that relieves the inhibition. It is reasonable to speculate that such phytohormonal balance also exists in the regulation of panicle branching. Indeed molecular cloning and analysis of a QTL for grain number, *GRAIN NUMBER1* (*Gn1a*), demonstrated the role of cytokinin in regulating panicle size (4). *Gn1a* encodes cytokinin oxidase/dehydrogenase (OsCKX2), an enzyme that degrades cytokinin. When the expression of *OsCKX2* is reduced, cytokinin accumulates in inflorescence meristems and increases the number of reproductive organs, which enhances the number of grains and leads to increased grain yield without affecting the phenology of the rice plant. A difference as much as 34 grains per main panicle was seen between two NILs homozygous for the two *Gn1a* alleles. In contrast, the *lonely guy* (*log*) mutant that has a defect in synthesis of active cytokinins produces a much smaller panicle than the wild type (49). Thus, cytokinins are critical in the control of panicle form in rice.

Other genes have also been reported to influence panicle form and size in rice, including

Short panicle 1 (*SP1*) and *DENSE AND ERECT PANICLE1* (*DEP1*), both of which show effects on spikelet number by enhancing meristematic activity and promoting cell proliferation. *SP1* encodes a putative peptide transporter (56). Interestingly, *DEP1* encodes a previously unknown protein containing a phosphatidylethanolamine-binding protein (PEBP)-like domain (29), sharing homology with the N terminus of GS3, which controls grain length (18).

Genes regulating the duration of panicle differentiation. The duration of panicle development refers to the period from the first bracket primordium to heading (30). Morphologically, the size of a panicle is determined to a large extent by the appearance of the terminal primary branch. Mutations in *Arabidopsis TERMINAL FLOWER 1* (*TFL1*) and its *Antirrhinum* ortholog *CEN* converted inflorescence meristem into terminal flower (2, 8, 9). Meanwhile, the period for transition from vegetative to reproductive phase is greatly reduced. Overexpression of the two genes delayed all growth phases, leading to late flowering and more branches in *Arabidopsis* (76). *TFL*, *CEN*, and *FT* belong to the family of PEBPs (8, 40, 72). Overexpression of *RCN1* and *RCN2*, rice *TFL1/CEN* homologs, delayed rice flowering up to 2 months with varied panicle morphology compared with wild-type plants. The increase of spikelets results mostly from an increased number of higher-order branches, whereas the number of primary branches is not very different from the wild-type plants. These results suggest *RNC1* and *RCN2* coordinate panicle development and flowering time (70).

Such concurrence of prolonged vegetative phase and reproductive phase seems to be common in rice. For example, cloning and molecular analysis of a QTL for grain number (114) showed that it has large pleiotropic effects on number of grains per panicle, heading date, and plant height and was thus named *Ghd7* (*g*rain number, plant *h*eight, and heading *d*ate). The *Ghd7* effect is regulated by day-length. Long day conditions enhance its expression resulting in delayed heading, increased plant height and panicle size, but no difference in tiller number, whereas under short-day conditions, the effects diminish. Detailed examination of the panicles revealed that *Ghd7* changes the numbers of both primary and secondary branches, and the magnitudes of such differences vary drastically with the genetic backgrounds. *Ghd7* encodes a protein having significant identity with the CCT domain of the CO protein in *Arabidopsis*, HD1 in rice, and a number of proteins found in a diversity of plant species that regulate processes such as photoperiodic flowering (74, 97, 116), vernalization (115), circadian rhythms (78, 89), and light signaling (39).

There is evidence indicating that the *Ghd7* effect on panicle size is related to the duration of panicle differentiation. The duration of panicle differentiation in Minghui 63 carrying a wild-type allele of *Ghd7* is 30 days compared with 23 in Zhenshan 97 in which the *Ghd7* locus is completely deleted (30). Based on the same dataset, however, this gene does not seem to accelerate the rate of panicle differentiation.

Grain Weight

Grain weight is determined by the volume (size) and the plumpness (filling) of the grain. The volume of a grain is the space bounded by the hull, measured by the length, width, and thickness. Grain size is also a highly important quality trait of the rice grain because long and slender grain is generally preferred for *indica* rice by the majority of consumers in China, the United States, and most Asian countries, although the preference for rice grain characteristics varies with consumer groups (37, 99). Although there has been a lack of systematic study on the biological process of the initiation, growth, and development of grains, recent studies in QTL mapping and cloning have made significant progress on identification of genes regulating grain weight for both grain size and filling. The results of such works are illustrated in **Figure 4**.

Genes for grain length. The first gene in this series is *GS3*, a major QTL for grain

length and weight, which also has a minor effect on grain width and thickness in rice, isolated using a map-based cloning approach (18). This QTL was consistently detected around the centromeric region of chromosome 3 in numerous studies across different genetic backgrounds and environments (1, 28, 48, 54, 77, 93, 95, 111, 117). This gene has a large effect on grain weight. In a BC_3-F_2 population derived by backcrossing the hybrid between Minghui 63 (long grain) and Chuan 7 (short grain) with Minghui 63, the 1000-grain weight of plants homozygous for the Minghui 63 allele (*GS3*-mh) was 25.6 g, which is nearly 50% higher than the 17.5 g for plants homozygous for the Chuan 7 allele (*GS3*-ch). Such difference in grain weight is mostly accounted for by the large difference in grain length (10.2 mm for *GS3*-mh homozygotes, and 7.32 mm for the *GS3*-ch homozygotes). *GS3* encodes a putative transmembrane protein, with a putative PEBP-like domain, a transmembrane region, a putative tumour necrosis factor receptor (TNFR)/nerve growth factor receptor (NGFR) family cysteine-rich domain, and a von Willebrand factor type C (VWFC) module. Comparative sequencing analysis identified a nonsense mutation (from nucleotide C to A), shared among all the large-grain varieties sequenced in comparison with the small- to medium-grain varieties. This mutation causes a 178–amino acid truncation in the C terminus of the predicted protein, suggesting that GS3 may function as a negative regulator for grain size. The increased grain size is largely due to a longitudinal increase in cell number (H. Mao & Q. Zhang, unpublished data). The effect of *GS3* on grain size has been detected in a diverse range of rice germplasm in the cultivated rice species *O. sativa*. (19, 91). Interestingly, however, no significant difference was observed between the two genotypic classes (A- and C-allele) of *O. rufipogon* in seed length, width, length/width ratio, or seed weight (91).

Genes for grain width. Two QTLs for grain width have been cloned and molecularly

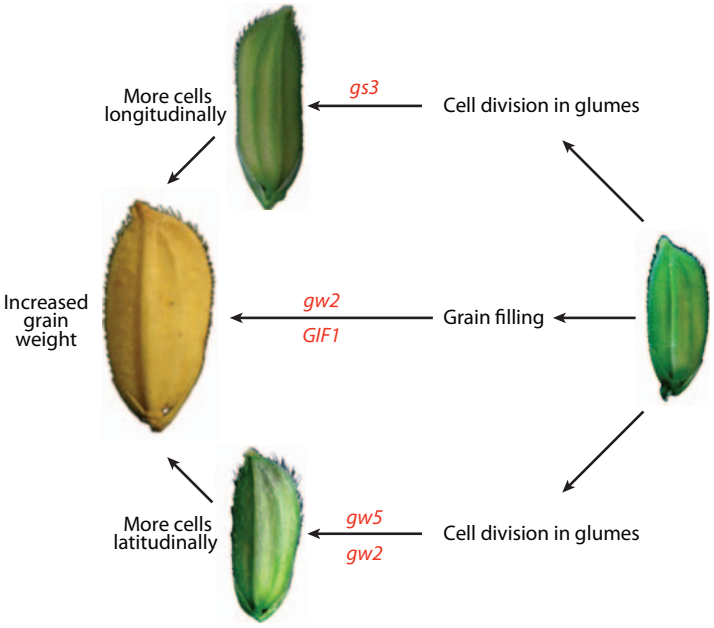

Figure 4

A schematic representation of genes involved in grain size regulation.

characterized. *GW2*, located on the short arm of chromosome 2, was cloned as a QTL controlling rice grain width and weight (86). The mutant arose as a point mutation causing premature termination of the predicted protein. Compared with the wild type, the mutant allele of *GW2* promotes cell division, which increases cell numbers resulting in wider glumes. Comparison of two homozygous NILs showed that the mutant allele *GW2* significantly increases grain weight (49.8%) by a large increase in grain width (26.2%), and slight increases in grain thickness (10.5%) and grain length (6.6%). The difference in grain size caused a significant difference in grain yield between the two NILs. *GW2* encodes a RING-type protein with E3 ubiquitin ligase activity, which is known to function in the degradation by the ubiquitin-proteasome pathway.

The second, *qSW5* (83) or *GW5* (105), is a QTL for grain weight and width consistently detected on chromosome 5 in a number of studies (55, 93, 100, 111, 117). The mutant allele resulted from a 1.2-kb deletion in the *GW5* genomic region. The grains of NIL homozygous

for the mutant allele are significantly heavier than the NIL homozygous for the wild-type allele, primarily owing to an increase in grain width as a result of an increase in cell number in the outer glume. Sequence analysis of diverse rice germplasms showed that this mutation is widespread and highly correlated with grain width. *GW5* encodes a previously unknown nuclear protein, which physically interacts with a polyubiquitin, indicating that GW5 may also function in the ubiquitin-proteasome pathway to regulate cell division during seed development. However, it remains to be investigated whether these two proteins, GW2 and GW5, work in the same pathway.

Genes for grain filling. Grain filling is a key determinant of grain weight. A gene regulating grain filling, *GIF1*, was recently cloned (102). It was mapped to chromosome 4 and encodes a cell-wall invertase required for carbon partitioning during early grain filling. A loss-of-function mutant showed slower grain filling and markedly more grain chalkiness than the wild type owing to abnormally developed and loosely packed starch granules, leading to a large reduction in grain weight. Compared with wild rice that produces small grains, the *GIF1* allele in cultivated rice has a more restricted expression pattern during grain filling. Moreover, transgenic rice that overexpressed *GIF1* with its native promoter had larger and heavier grains than the wild-type plants, whereas transgenic plants ectopically expressing *GIF1* with the 35S promoter or the rice *Waxy* promoter had smaller grains. It was interpreted that the restricted expression pattern of the *GIF1* gene in the ovular vascular trace is the key to increased grain weight. This also suggests that tissue specifically enhancing the expression of *GIF1* may provide a means for increasing grain filling.

In addition, the results of Song et al. (86) show that *GW2* also has a significant effect on grain filling. The wider grains exhibit an accelerated rate of grain filling compared to the narrower ones.

THE MOLECULAR NATURE OF THE GENES FOR YIELD TRAITS

The genes controlling rice yield traits can tentatively be divided into two classes. The first class consists of the genes for the basic biological processes; defects of such genes would cause deleterious effects on the plants such as severe growth retardation, abnormality in morphology, and very low rate of seed setting. Examples of these genes include all the genes reviewed for tillering and some of those for panicle development. Most of them were identified as natural mutations and, in recent years, via insertional mutagenesis. The other class is composed of the genes for quantitative changes of the traits; different alleles at the respective loci, including loss-of-function alleles, would cause variation in the measurements of the traits. Examples of this class are those for number of grains per panicle or panicle size and those for grain dimension, which are identified and isolated by map-based cloning.

Botanically, tillering and panicle development share substantial commonality in the sense that they both involve apical growth and branching. Thus, similar regulatory mechanisms should be involved in both processes, and the final plant architecture and panicle size are largely the outcome of a balance between apical dominance and branching, at various levels. Moreover, many of the processes may involve similar or the same genes regulated by complex networks of hormones, photoperiod, internal developmental signals, and external environmental cues.

The relationships of genes and hormones involved in tillering and panicle development based on the available literature are illustrated in **Figure 5**. According to the present understanding, tillering is the outcome of an interaction among the three hormones, cytokinin, auxin, and the newly identified strigolactone, with cytokinin promoting branching and the other two inhibiting it. For panicle development, the effect of cytokinin has also been elucidated as to promote branching, thus increasing the spikelet number. However, whether the

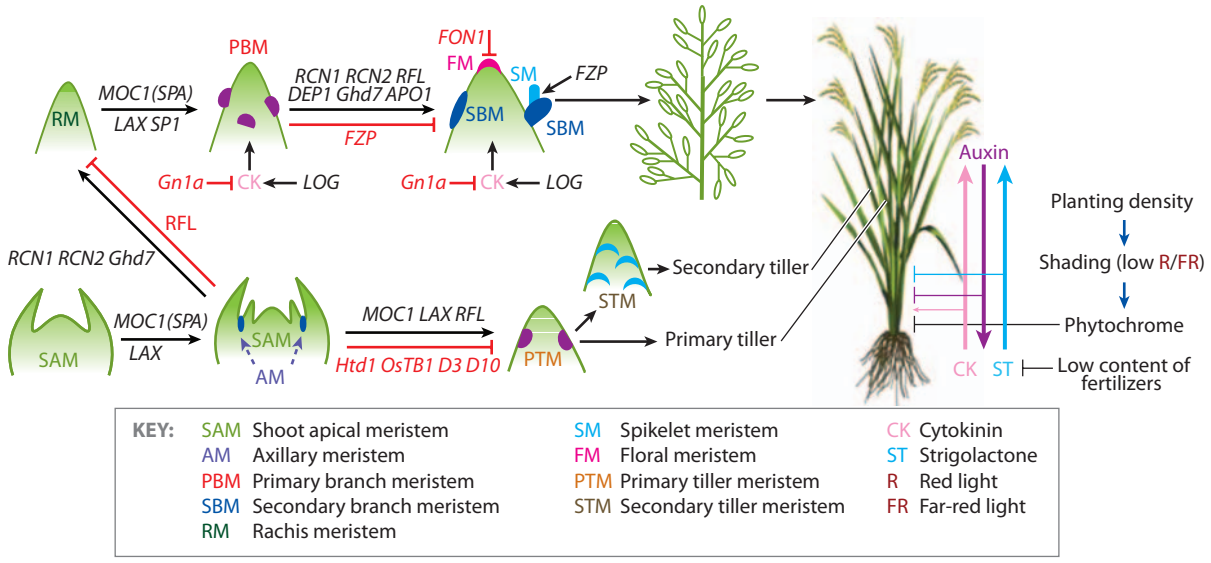

Figure 5

A schematic representation of genes involved in tillering and panicle formation.

other two hormones or some other growth regulators have any role in panicle size remains to be identified.

As illustrated in **Figure 5**, the two branching processes involve a diversity of genes with various biochemical functions. Some of the genes, such as *MOC1*, *LAX1*, and *RFL* (75), function in both tillering and panicle development. Other genes may specifically function only in one of the branching processes. At present, the pathways and the network underlying these processes have just started to be elucidated; identification of more genes involved in the processes is essential for characterizing the regulatory network.

The three genes identified for grain size (length and width), although differing in biochemical functions, also share high similarity in regulation of grain size. They are all negative regulators of grain size such that loss-of-function mutants produce larger (longer or wider) grains by increasing cell numbers of the glumes, presumably by promoting cell proliferation, whereas the wild-type alleles produce smaller grains. Most likely, these are important regulators of cell cycles during growth and development of the glumes. Because grain weight is mainly determined by the content of the endosperm, we speculate that these genes may also function in grain filling, as in the case of *GW2* reported by Song et al. (86).

IMPLICATIONS OF QTL CLONING IN QUANTITATIVE GENETICS AND CROP BREEDING

Cloning and understanding the molecular nature of the genes may have fundamental implications for quantitative genetics. It was hypothesized in classical quantitative genetics that quantitative traits are controlled by large numbers of genes with small, similar, and supplementary effects, a theory known as the multiple factor hypothesis (17), and polygenes and major genes are regarded as accounting for different types of variation, continuous and discontinuous (64). Recent progress in the analysis of NILs based on QTLs for typical quantitative traits, such as number of grains per panicle and grain size, showed that in near-isogenic genetic backgrounds, these traits can be inherited

as discrete classes in the same manner as in qualitative traits. The genes underlying QTLs can also have major effects on the traits, rather than merely minor effects as typically assumed in classical quantitative genetics. Thus, there may be no meaningful distinction in the genetic and molecular nature between QTLs and major genes with respect to the continuity of phenotypic variation.

One consequence of the complex regulation of the gene functions during the process of development is that many genes would have pleiotropic effects, as they often play roles in multiple processes thereby producing morphological changes that are measured as different traits. Thus, the QTL main effects, widespread pleiotropic effects, and prevalent epistatic effects combine to provide the genetic and molecular bases of the "netlike" structure among genes and characters in the system as perceived by Wright (107).

Progress in understanding the genetic and molecular bases of the yield traits also has significant implications in breeding for yield improvement. In particular, the major effects observed between the NILs and the cloning of QTLs suggests that yield, like other traits, can also be improved by genetic manipulation of the component traits. This implies that it is now practically feasible to modify any of the yield traits individually using either molecular marker-assisted selection or transformation. Indeed, Asihkari et al. (4) transferred the *Gn1a* allele for increasing grain number and the *sd1* allele for reducing plant height from Habataki into Koshihikari, which produced the expected phenotype. The *GS3* marker has now been widely used in rice breeding programs in China to change grain size and shape, which has improved both the yield and grain quality (Y. He and G. Ren, personal communications). Transgenic rice harboring the wild-type allele of *Ghd7* in the backgrounds of a range of *japonica* varieties produced panicles with two to three times the grain numbers of the wild-type plants (114). Such strategies should also be applicable in the genetic improvement of other cereals, and much of the framework for this approach has been outlined by Xu et al. (113).

Zhang (119) proposed Green Super Rice as a new goal for rice improvement for sustainable rice production. On the premise of continued yield increase and quality improvement, Green Super Rice should possess resistances to multiple insects and diseases, high nutrient-use efficiency, and drought resistance, promising to greatly reduce the consumption of pesticides, chemical fertilizers, and water. He also outlined the strategies and described resources available for developing Green Super Rice. In this perspective, improvement of yield traits should be integrated with improvement in grain quality, disease and insect resistances, nutrient-use efficiency, and tolerance to abiotic stresses.

FUTURE PERSPECTIVES

Currently, there are still large knowledge gaps on the molecular controls of the basic biological processes related to yield traits. This is especially the case for grain development. With the global development of functional genomic resources, many tools and genetic stocks have now become available for identifying genes underlying these processes (120). Quantitative differences of the traits controlled by the QTLs represent variation amendable in breeding programs. Map-based cloning continues to play a major role in cloning QTLs, and with the accumulation of global resources in mutant libraries, mutation analyses may also have a role in identifying QTLs. In particular, analyses combining QTL mapping based on bin maps developed using next-generation sequencing (29), with insertional mutant libraries (47) and expression profiles (103), provide a powerful strategy for QTL cloning. Once the genes are identified, they will need to be placed in pathways and networks that regulate the developmental processes of tillers, panicles, and grains, not only the genesis and development of the organs but also the numbers and sizes.

Agronomically, grain yield of rice is the outcome of three constituents: the sink, which is

the capacity represented by the mathematical product of the three component traits discussed above, including number of panicles, number of grains per panicle, and grain weight; the source, which is the biological productivity of the plants to provide sufficient carbohydrates to the sink via photosynthesis; and the flow, which is the ability to transport the photosynthetic products and other nutrients to fill up the sink. Less than optimal results in the latter two constituents would result in the gain in one component trait of the sink being compensated by loss in the other traits. Unfortunately, however, all the progress reviewed here has only dealt with the traits comprising the sink, whereas there have been very limited studies in the other two constituents, which should be targets of future work.

For improving the transport capacity, the very first step might be to investigate the genetic variation in the capacity for translocation of the carbohydrates and other nutrients to the sink, which may involve the vascular systems and a number of proteins such as enzymes and transporters. Ultimately, improving biological productivity requires an increase in photosynthetic product. There is currently an international effort to convert rice from C3 photosynthesis to C4, and the feasibility for such an endeavor has been intensively evaluated (81). In the near term, it may be rewarding to explore genetic variation that may exist in the rice germplasm in photosynthesis-related traits, including canopy development, plant architecture, the ability to maintain green leaves, and photosynthetic rates under different environmental conditions. The various opportunities for increasing photosynthesis discussed by Long et al. (60) may be explored.

The ultimate goal of rice biology research is to realize the ideal situation of breeding by design to breed for cultivars to meet the diverse needs of global rice production. The full version of breeding by design as outlined by Zhang et al. (120) comprises four different levels: (*a*) the yield limit achieved through a population structure that makes maximum use of the solar energy in given ecological conditions; (*b*) the plant architecture to realize the population structure; (*c*) the traits to construct the plant architecture and to achieve high quality, high nutrient-use efficiency, and resistances to multiple biotic and abiotic stresses; and (*d*) the genes to produce the traits. Further development on a number of fronts, such as functional genomics, proteomics, and metabolomics, and the integration of these developments into a systems approach involving multiple disciplines are essential to realize the goals of rice biology research.

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Annual Review of Plant Biology
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Contents

Indexes

Errata

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