Rice Mutants and Genes Related to Organ Development, Morphogenesis and Physiological Traits

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Recent advances in genomic studies and the sequenced genome information have made it possible to utilize phenotypic mutants for characterizing relevant genes at the molecular level and reveal their functions. Various mutants and strains expressing phenotypic and physiological variations provide an indispensable source for functional analysis of genes. In this review, we cover almost all of the rice mutants found to date and the variant strains that are important in developmental, physiological and agronomical studies. Mutants and genes showing defects in vegetative organs, i.e. leaf, culm and root, inflorescence reproductive organ and seeds with an embryo and endosperm are described with regards to their phenotypic and molecular characteristics. A variety of alleles detected by quantitative trait locus analysis, such as heading date, disease/insect resistance and stress tolerance, are also shown.

Keywords: Developmental mutant — Phenotype — Physiological mutant — Rice organ — Trait gene.

Abbreviations: ARGM, Asian rice gall midge; bHLH, basic helix–loop–helix; BPH, brown planthopper; CMS, cytoplasmic male sterility; EST, expressed sequence tag; GLH, green leafhopper; GO, gene ontology; GRH, green rice leafhopper; LRR, leucine-rich repeat; NBS, nucleotide-binding site; NIL, near isogenic line; PMS, photosensitive genic male sterility; PO, plant ontology; QTL, quantitative trait locus; RIL, recombinant inbred line; SAM, shoot apical meristem; TGMS, thermosensitive genic male sterility; TO, trait ontology; WBPH, whitebacked planthopper; WCVs, wide-compatibility varieties.

Introduction

The discovery and isolation of useful natural mutants and their variants in rice breeding has taken many years and considerable effort. In the latter quarter of the 20th century, many studies were conducted to generate artificial mutants by using γ-ray irradiation or chemical mutagens, e.g. methylnitrosourea (MNU) and ethyl methanesulfonate (EMS). These mutants have been used primarily for genetic studies such as identification of genes that regulate rice morphogenesis or physiological traits, and linkage analysis for gene mapping. In 1998, the rice genetics committee reported 571 mutant genes (Nagato and Yoshimura 1998). To date, nearly 2,000 trait genes including both single Mendelian loci/genes and quantitative trait loci (QTLs) have been recorded. Even at the present status where tens of thousands of cDNAs and expressed sequence tags (ESTs) have been isolated as genetic codes, most trait genes specific for rice mutants and variant/strains have not been isolated. In this review, 1,698 rice genes reported to date based on the mutant or variant phenotypes are listed. Most of these genes are mapped on one of the 12 rice chromosomes. Classification of the 1,698 genes into phenotypic categories is summarized in Table 1. Mutant phenotypes and gene classification fit into particular categories for rice development, as described by Itoh et al. in this issue. In this review, genes and mutants of key or interesting functions in relation to rice organ development and biological/physiological reactions have been selected from the gene list of Table 1. The genes described in this study should be important sources for functional genetic studies to connect gene structure with gene functions. In fact, enormous progress in rice genome mapping and sequencing has promoted map-based cloning of these trait genes during the last decade.

In addition to the positional cloning approach, systematic functional genomics approaches to screen mutants using insertion mutagenesis have been developed (Hirochika et al. 1996, An et al. 2003, Ito et al. 2004b, Kim et al. 2004, Sallaud et al. 2004). Several mutants and genes from Tos17-transposed lines and T-DNA insertion lines are included in this review. Tagged flanking sequence databases generated for most mutant populations are used in reverse genetics approaches. The MNU-induced mutant population established by Satoh and Omura (1981) is thought to possess a very frequent point mutation ratio and so could also be a powerful source for identifying mutant lines by applying reverse genetic approaches such as the TILLING system (Till et al. 2003). The mutant strains of both types are collected on a large scale, and their classifica-

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tion according to phenotype is underway; see Appendix 1 (1) for Tos17 tagging mutants and Appendix 1 (2) for the MNU-induced mutants.

In this review, rice trait genes important for the understanding of rice are documented. Detailed data including chromosomal locations, trait ontology (TO)/plant ontology (PO) and gene ontology (GO) IDs and reference for all 1,698 trait genes are available in the Oryzabase [see Appendix 1 (3)]. Such information could be useful for comparative studies of organ development, and for structural variation of genes among species and genera.

### Mutants and Genes in Vegetative Organ Development

In vegetative development, leaves are generated successively from flanks of the shoot apical meristem (SAM) with an alternate phyllotaxy. Culm, crown roots and lateral roots are also formed in this developmental phase. A large number of mutants defective in development of various organs have been reported in rice, and in several cases causal genes have been cloned. Mutants and genes associated with vegetative organ development are categorized into four groups: (i) genes specifically expressed in the SAM; (ii) leaf mutants; (ii) culm mutants; and (iv) root mutants.

**Genes specifically expressed in the SAM**

In the vegetative development of rice, the SAM maintains itself and simultaneously generates lateral organs such as leaves and tillers. Several different families of homeobox genes have been cloned and analysed in plants. One of them is a KNOX class 1 homeobox gene family which is a pivotal regulator of SAM formation and maintenance in plants. In rice there are six KNOX class 1 genes, OSH1, OSH3, OSH6, OSH15, OSH43 and OSH71, in its genome (Sentoku et al. 1999). Five of these genes are expressed in the SAM. Their constitutive expression results in inhibition of regeneration from the callus by maintaining the cells in an undifferentiated state or results in abnormal morphology of regenerated shoots mainly on the leaves (Sentoku et al. 2000, Ito et al. 2001). Among these genes, only the mutant of OSH15 has been analyzed (Sato et al. 1999). The osh15 mutant shows reduced plant height and is allelic to the dwarf mutant, d6. Thus, OSH15 is involved in the control of normal plant height in addition to SAM formation and maintenance. Another gene family that plays a role in SAM is the NAC gene family that shares an evolutionarily conserved NAC domain. Expression analysis suggests diverse functions for each member of this family (Kikuchi et al. 2000).

### Leaf mutants

In leaf development, various types of mutants have been identified and can be categorized into two large groups; a leaf

<table>
<thead>
<tr>
<th>Class of genes</th>
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<tr>
<td>Vegetative organ</td>
<td>276</td>
<td>Seed</td>
<td>236</td>
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<tr>
<td>SAM</td>
<td>30</td>
<td>Morphological traits</td>
<td>55</td>
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<tr>
<td>Leaf</td>
<td>53</td>
<td>• Embryo</td>
<td>28</td>
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<tr>
<td>Culm</td>
<td>141</td>
<td>• Endosperm</td>
<td>3</td>
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<tr>
<td>Root</td>
<td>52</td>
<td>• Grain shape</td>
<td>24</td>
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<tr>
<td>Reproductive organ</td>
<td>464</td>
<td>Physiological traits</td>
<td>181</td>
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<tr>
<td>Heading date</td>
<td>115</td>
<td>• Dormancy</td>
<td>30</td>
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<tr>
<td>Inflorescence</td>
<td>111</td>
<td>• Longevity</td>
<td>3</td>
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<td>Spikelet</td>
<td>46</td>
<td>• Storage substances</td>
<td>80</td>
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<tr>
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<td>238</td>
<td>• Shattering</td>
<td>28</td>
</tr>
<tr>
<td>• Fertility restoration</td>
<td>16</td>
<td>• Taste</td>
<td>40</td>
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<tr>
<td>• Segregation distortion</td>
<td>15</td>
<td>Tolerance, resistance</td>
<td>288</td>
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<td>• Hybrid sterility,</td>
<td>58</td>
<td>Disease resistance</td>
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<td>Insect resistance</td>
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<tr>
<td>• Male sterility</td>
<td>107</td>
<td>Stress tolerance</td>
<td>82</td>
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<tr>
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<tr>
<td>• Meiosis</td>
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<td>Characters as QTLs</td>
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<tr>
<td>• Other sterilities</td>
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<td>• Grain quality</td>
<td>47</td>
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<td>Heterochrony</td>
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<td>• Yield and productivity</td>
<td>134</td>
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<td>133</td>
<td>• Plant growth activity</td>
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<td>Root activity</td>
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<tr>
<td>Others</td>
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<td>Seed sterility</td>
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<td>Total</td>
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morphology group and a leaf color group. The leaf morphology group contains seven types of mutants: rolled leaves, drooping-wet leaves, narrow leaves, drooping leaves, blade–sheath boundary defect leaves, glabrous leaves and hairy leaves; and QTLs controlling leaf angle, leaf length and leaf width.

Mutants of three loci have been categorized in the drooping leaf subgroup. Among them, DROOPING LEAF (DL) has been thoroughly analyzed (Nagasawa et al. 2003, Yamaguchi et al. 2004). DL is expressed in the central region of the developing young leaf and its loss-of-function mutation results in a defective midrib formation and drooping leaves. DL encodes a putative transcription factor of the YABBY family. DL is a pleiotropic gene and also specifies carpel identity during flower development (see Organ Identity section). Detailed analyses of other mutants remain to be carried out.

Seven lamina joint angle QTLs, two leaf length QTLs, five leaf width QTLs and one leaf length mutant have been identified. In a coleoptile photomorphogenesis1 (cppml) mutant, coleoptiles elongate more than wild-type coleoptiles under red light or in the darkness (Biswas et al. 2003).

There are three mutants affecting leaf blade–sheath boundary, auricless (aul), ligureless (lg) and collarless (col). The auricless (aul) mutant lacks auricles and the ligule is rudimentary, whereas ligureless (lg) lacks ligule, auricle and leaf collar (Maekawa 1988). The collarless mutant lacks collar (Sanchez and Khush 1998). Another two classes of mutants, two glabrous leaf and two hairy leaf mutants were also found in the leaf mutants. The other leaf mutant group has abnormalities in leaf color including lesion mimic spotted phenotype.

**Culm mutants**

Culm mutants can be categorized into eight groups; dwarfism/elongation, floating, tiller formation, tiller angle, brittleness, thickness, twisted and others. The dwarfism/elongation group includes mutants impaired in gibberellin synthesis, gibberellin signaling, brassinosteroid synthesis and brassinosteroid signaling.

**DWARFISM/ELONGATION**

Since plant height in rice is an agronomically important trait for breeding high-yielding cultivars, numerous dwarf and semi-dwarf mutants have been collected. Some of them have been shown to be gibberellin- or brassinosteroid-related mutants.

D35, SD1 and D18 genes encode gibberellin biosynthesis enzymes which catalyze specific steps. D35 encodes ent-kau rene oxidase which catalyzes the early three steps of gibberellin synthesis (ent-kaurene→ent-kaurenol→ent-kaurenal→ent-kaurenoic acid) (Itoh et al. 2004). SD1, the ‘green revolution’ gene, encodes GA20 oxidase which catalyzes three steps (GA53→GA44→GA19→GA20) (Sasaki et al. 2002). D18 encodes GA3 β-hydroxylase that catalyzes the step from GA20 to GA1, which is an active form of gibberellin (Itoh et al. 2001). Mutants lacking each of these gene activities show specific patterns in reducing internode length. These gibberellin synthesis genes form small gene families in the rice genome, and the members of each gene family show different expression patterns.

In addition to gibberellin synthesis genes, several gibberellin signaling genes have been cloned and a signaling mechanism has been studied. These genes are, D1, GA-INSENSITIVE DWARF2 (GID2) and SLENDER RICE1 (SLR1). Loss-of-function mutants of D1 and GID2 show a dwarf phenotype, whereas a loss-of function mutant of SLR1 resembles the phenotype of an exogenous application of gibberellins on plants (Ashikari et al. 1999, Fujisawa et al. 1999, Ikeda et al. 2001). D1 and GID2 encode positive regulators for gibberellin signaling, whilst SLR1 encodes a negative regulator. GID2 is involved in the degradation of SLR1 (Sasaki et al. 2003). GID2 encodes an F-box protein in a component of ubiquitin ligase E3 named SCF<sub>GID2</sub>.

D1 encodes an α-subunit of the trimeric G protein, which is localized in the plasma membrane as a complex with the β- and the γ-subunits (Ashikari et al. 1999, Fujisawa et al. 1999, Kato et al. 2004). The d1 mutant is insensitive to low concentrations of gibberellins, but still shows a response to high concentrations of gibberellins (Ueguchi-Tanaka et al. 2000).

Two genes have been cloned which encode proteins catalyzing brassinosteroid synthesis. In d2 mutants, the second internode from the top has been shown to be shortened, whereas elongation of the other internodes is rarely affected (Hong et al. 2003). The d2 mutant also shows an erect leaf phenotype. D2 encodes brassinosteroid C-3 oxidase (also called CYP90D2, a member of cytochrome P450), which catalyzes a C-3 oxidation step of brassinosteroid synthesis. In the rice genome, one homolog of D2 has been found, which is expressed mainly in the root.

BRASSINOSTEROID-DEPENDENT1/BRASSINOSTEROID-DEFICIENT DWARF1 (BRD1) encodes a brassinosteroid C-6 oxidase (OsDWARF), which catalyzes the C-6 oxidation step of brassinosteroid synthesis (Hong et al. 2002, Mori et al. 2002). The brd1 mutant shows almost no internode elongation, a reduced ratio of leaf sheath to leaf blade, and a short root. The brd1 mutant also shows constitutive photomorphogenesis in the dark.

In the brassinosteroid signaling pathway in rice, only one gene has been cloned. D61 encodes a receptor-like protein kinase with extensive sequence identity to the Arabidopsis brassinosteroid receptor BR1 (Yamamuro et al. 2000). This suggests that D61 is the receptor for brassinosteroid in rice. The d61 mutant shows less sensitivity to exogenously applied brassinosteroid. More endogenous brassinosteroids accumulate in the d61 mutant than the wild-type plant. The relative ratio of leaf blade to sheath length is also smaller compared with the wild type. The results of these phenotypic analyses are consistent with the idea that D61 is the brassinosteroid receptor.
Another group of mutants are related to floating characters or deep water tolerance. These genes exist in the floating rice strains, and are involved in reactions mainly for extreme elongation of the internodes. These genes are analyzed as QTLs and are described in the Stress tolerance section.

TILLER FORMATION

Seven mutants with a reduced number of tillers have been identified. Among them, *MONOCCULM1* (*MOC1*) has been shown to be involved in tiller bud initiation (Li et al. 2003a). The moc1 mutant shows no tillering owing to a defect in tiller bud formation during the vegetative phase, and produces no rachis branches in the inflorescence. *MOC1* encodes a transcription factor of the plant-specific GRAS family. Expression of *MOC1* starts in the presumptive region of the axillary bud formation, and continues until maturation of the tiller buds.

Brittle

Six mutants with brittle culm have been identified. Brittle mutants are easily broken by bending. Among them, *BRITTLE CULM1* (*BC1*) has been studied most extensively (Li et al. 2003b). The bc1 mutant has less cellulose and more lignin than the wild-type plant. Monosaccharide composition is also affected in the bc1 mutant. Thus, *BC1* controls the mechanical strength of plants through the cell wall composition. *BC1* encodes a COBRA-like protein and is expressed mainly in developing sclerenchyma cells and in vascular bundles.

TILLER ANGLE AND THICKNESS

Seven genes identified as mutations or QTLs are categorized in the tiller angle group. However, detailed morphological and molecular analyses of these genes have not yet been reported. *Big uppermost culm* (*Buc*) is categorized in the thickness group.

Root mutants

In contrast to the understanding of shoot development, analysis of root development lags behind, even though a number of mutants have been isolated which affect various aspects of root development. These include mutants for root number, root branching, root length, root thickness, root weight, root pulling force and rhizome formation. The *crl1* (*crown rootless1*) mutant forms radicle and lateral roots normally, but is impaired in the initiation of crown root primordia (Fig. 1A, B) (Inukai et al. 2001a). Thus, *CRL1* specifically regulates initiation of crown root primordia. *CROWN ROOTLESS2* (*CRL2*) regulates various aspects of root development including initiation and subsequent growth of crown root primordia (Fig. 1C, D, Inukai et al. 2001a). The reduced root length1 (*rrl1*) and reduced root length2 (*rrl2*) mutations cause short roots (Inukai et al. 2001b). In the *rrl1* mutant, only the cortical cell length is significantly reduced, whilst in the *rrl2* mutant, the root apical meristem is also small (Fig. 1E, G).
Inflorescence and flower mutants can be categorized into seven groups according to the developmental step at which mutant phenotypes are observed, such as lateral branching, lateral meristem identity, flower organization, organ number, organ identity, organ development and others.

LATERAL BRANCHING
This group of mutants has defects in the formation and elongation of lateral branches. The mutants of this group form fewer numbers of rachis branches and spikelets or compact panicle(s). In this group, LAX PANICLE (LAX) has been studied most extensively. In lax mutants, spikelets are formed only on the apices of rachis branches, and no lateral spikelets are formed (Fig. 2A, B, Komatsu et al. 2001, Komatsu et al. 2003a). LAX is necessary for initiation of lateral meristem formation. LAX encodes a putative transcription factor with a plant-specific basic helix–loop–helix (bHLH) domain and is expressed at the boundary between the lateral meristem and apical meristem.

LATERAL MERISTEM IDENTITY
This group of mutants fails to specify the identity of the lateral branch meristem as the spikelet or flower meristem. This group includes FRIZZY PANICLE (FZP). In fzp mutants, primary rachis branch meristems develop normally, but spikelets are replaced by branch meristems (Fig. 2C–E, Komatsu et al. 2003b). As a result, branch meristems are continuously generated in the inflorescence of the fzp mutant. FZP encodes a transcriptional activator with the ERF domain, and is expressed in a region where the rudimentary glume primordia develop.

FLOWER ORGANIZATION
This group of mutants has defects in flower organization. A mutant flower of this group has an abnormal number or arrangement of whorls. Although some mutants are known, such as extra glume-1 (eg1) and extra glume-2 (eg2), their detailed analyses remain to be carried out.

ORGAN NUMBER
This group of mutants has defects in controlling the number of organs developed in each whorl. In these mutants, only the number of floral organs is affected, whereas flower organization and organ identity are normal. floral organ number1 (fon1) and fon2 are mutants in this group. In these mutants, the number of floral organs such as carpel, stamen and lodicule is increased, whereas the number of glumes such as lemma and palea is not affected (Fig. 3B, Nagasawa et al. 1996). fon1 and fon2 control organ numbers by regulating meristem size or number of meristem cells. The FON1 gene encodes a receptor-like protein kinase with a high homology to Arabidopsis CLAVATA1 (CLV1) (Suzaki et al. 2004).

ORGAN IDENTITY
This group of mutants has defects in specification of the identity of organs. In rice, several genes involved in organ identification have been identified. Among them, SUPERWOMAN (SPW) specifies lodicules and stamens. In spw mutant flowers, lodicules and stamens are homeotically transformed into organs of the lower whorl (SPW1); the homeoieties of organs is determined in the uppermost whorl by PAPILLON (PAP). PAP specifies lodicules and stamens when expressed in the uppermost whorl, and is homeotic when expressed in the second whorl (PAP1).
into palea-like organs and carpels, respectively (Fig. 3C, Nagasawa et al. 2003). The SPW gene has been shown to be identical to OsMADS1, a rice MADS-box gene. SPW is expressed in the lodicules and stamens. These analyses indicate that SPW is a class B gene and the ABC model is applicable, at least in part, to rice.

**DROOPING LEAF (DL)**, which plays a role in leaf development (see Leaf mutants section), also specifies carpel identity (Nagasawa et al. 2003, Yamaguchi et al. 2004). In the dl mutant, the carpel has been homeotically transformed into the stamens (Fig. 3D). In flower development, expression of DL starts in the presumptive region where carpel primordium is initiated, and continues in the carpel primordium. Thus, DL specifies the identity of only a single whorl. In this respect, the ABC model is modified in rice.

Another gene involved in specification of organ identity is LEAFY HULL STERILE1 (LHS1) (Jeon et al. 2000). In the lbs1 mutant flower, lemma and palea show a leaf-like appearance. Lodicules also become leafy. The number of stamens is reduced. In some flowers, a new abnormal flower is formed in the whorl of the stamens, indicating that specification of floral organs when they are ectopically expressed or their anti-sense is expressed (Kang et al. 1998, Kyozuka and Shimamoto 2002).

**Organ Development and Others**

Mutants of the organ development group have defects in development of each organ and show abnormal development of awn, lemma and palea after specification of their organ identity. Twenty genes or QTLs involved in awn development are described below. Awn, lemma and palea after specification of their organ identity (Nagasawa et al. 2003, Yamaguchi et al. 2004). The gene has been shown to be identical to OsMADS1, a close member of Arabidopsis AP1. LHS promotes determination of the floral meristem identity and specification of the lemma, palea and lodicules. Other genes have been cloned which have resulted in homeotic transformation of floral organs when they are ectopically expressed or their anti-sense is expressed (Kang et al. 1998, Kyozuka and Shimamoto 2002).

**Sexual Reproduction Processes**

In this section, we describe the genes and mutations associated with gamete or seed sterility.

**Male sterility and fertility restoration**

Male sterility, for which a large number of mutants are found, is classified into four major groups: male sterility caused by cytoplasmic male sterility (CMS), photoperiod-sensitive genic male sterility (PMS), thermosensitive genic male sterility (TGMS) and other genic male sterilities. CMS, PMS and TGMS can be used for practical hybrid production. The CMS lines require combination with the fertility restorer lines to maintain a hybrid system, whereas alteration of environmental conditions, such as day length and temperature shift, can restore fertility in PMS and TGMS (Liu et al. 2001, Wang et al. 2003).

In *Oryza sativa*, only a single CMS system has been thoroughly studied using cybrids with the cytoplasm of cv. Chinsurah Boro II (Boro, indica rice) and the nuclear genome of cv. Taichung 65 (T65, japonica rice), called ms-bo type or BT type CMS (Shinjo 1975, Shinjo 1984). A mitochondrial *atp6* and a unique sequence *orf79* downstream of *atp6* are known to relate to CMS (Kadowaki et al. 1990, Iwabuchi et al. 1993, Akagi et al. 1994). The *Rf-1* gene has been reported to encode a protein with a mitochondrial transit peptide and pentatricopeptide repeat (PPR) (Kazama and Toriyama 2003, Komori et al. 2004). On the other hand, neither PMS nor TGMS genes have been isolated as yet. Ku et al. (2003) reported that programmed cell death of premature tapetum is associated with TGMS in rice.

Several other genic mutations exhibiting pollen sterility or abnormal anther development have also been reported (mostly designated *ms*). Recently, the *aid1* mutant showing a defect in anther dehiscence was identified using an Ac/Ds transposon tagging system (Zhu et al. 2004). The *AID1* gene encodes a novel protein with a single MYB DNA-binding domain.

**Hybrid sterility and reproductive barrier**

Although there are several genic models for hybrid sterility, only two models are applied to interspecific rice hybrids. One is the ‘single locus sporo-gametophytic interaction’ model, and the other is the ‘duplicate loci gametic lethal’ model (Oka 1974, Sano et al. 1979).

Gamete eliminator genes play key functions in preferential inheritance of one of the alleles in the heterozygous *F1* plants, and support the former model. The Mendelian segregation of marker genes linked with the gamete eliminator is distorted in progenitors. Many gamete eliminator loci, designated as *S* in most cases, have been identified in various cross-combinations among the *Oryza* species. In the cross-combination between *O. sativa* and *O. glaberrima*, at least seven *S* loci have been identified using NILs of a T65 background (Sano 1983, Sano 1990, Doi et al. 1998, Doi et al. 1999, Taguchi et al. 1999). The loci *S1*, *S3*, *S19* and *S20* act to abort pollen carrying the T65 haplotype, and the loci *S2* and *S21* act to abort pollen carrying the *glaberrima* haplotype (Fig. 4A–D). Heterozygous plants in the *S18* locus probably exhibit a defect in male sporocyte development. In addition to the gamete eliminator alleles, Ikehashi and Araki (1986) have reported the existence of a neutral *S* allele (*S5*) in javanicas, called wide-compatibility varieties (WCVs). The WCVs enable generation of fertile *F1* plants when crossed with japonica and indica, whilst japonica–indica hybrids exhibit high sterility and are primarily attributed to the *S5* locus. Thus the WCVs have actually contributed to overcoming hybrid sterility (Wan et al. 1993).
addition to \( S \) genes, many loci or genes distorting Mendelian segregation of flanking marker genes in interspecific hybrids have been identified, many of which are designated \( g_a \). Recently, Harushima et al. (2002) reported that many map positions for numerous segregation distorer loci of japonica and indica strains differed with cross-combinations, suggesting rapid evolution of reproductive barrier genes.

Only a few pairs of loci involved in complementary \( F_2 \) hybrid sterility support another genic model. Recently, Kubo and Yoshimura (1999) reported a pair of complementary loci for \( F_2 \) hybrid sterility, \( hsa1 \) on chromosome 12 and \( hsa2 \) on chromosome 8. The double recessive plant causes female sterility (Fig. 4E, F), whilst the single recessive homozygotes of each locus exhibit moderate sterility.
Hybrid weakness

Hybrid weakness or hybrid breakdown is not directly related to sexual reproduction, but is worth noting in this section because they are one of the components of the reproductive barriers. All hv loci reported so far (Hwa, hwu, Hwc, hwe and hwe) complementarily affect viability of F1 or F2 plants (Oka 1957, Amemiya and Akamine 1963, Chu and Oka 1972, Fukuoka et al. 1998, Kubo and Yoshimura 2002). The plants affected by the complementary gene sets generally show a small number of tillers, short culms and panicles, chlorosis or necrosis of leaves, absence of seed sets, root growth inhibition, and so on (Fig. 4G).

Meiosis

In the majority of eukaryotes, chromosome synapsis is mediated by an evolutionarily conserved, tripartite, protein structure called the synaptonemal complex (SC) (Wettstein et al. 1984, Zickler and Kleckner 1999). Synaptic mutants are classified into two categories, asynaptic and desynaptic mutants (Li et al. 1945). The former class is characterized by partial or complete inhibition of the synopsis of homologous chromosomes, whereas the latter shows precocious separation of bivalents following chromosome pairing. In O. sativa, many asynaptic (as) and desynaptic (ds) mutants have been isolated so far (Katayama 1963, Kitada and Omura 1983).

Recently, Nonomura et al. (Nonomura et al. 2004a, Nonomura et al. 2004b) identified the rice meiotic genes PAIR1 and PAIR2 from sterile mutant lines tagged with Tos17 (Hirochika et al. 1996). The loss-of-function mutations of two PAIR genes result in asynapsis and chromosome non-disjunction in male and female meiocytes (Fig. 5A–D). The PAIR1 gene encodes a novel nuclear protein (Nonomura et al. 2004a). PAIR2 encodes a protein homologous to the yeast HOP1 and Arabidopsis ASY1 (Nonomura, K., Eiguchi, M., Nakano, M., Suzuki, T. and Kurata, N. unpublished data).

Other sterilities

There are only a few other genes or mutations that are involved in sexual reproduction but not classified in the above categories. In the msp1 mutant flowers, the number of male and female sporocytes increases abnormally (Fig. 5F). The Msp1 gene encodes a putative leucine-rich repeat (LRR) receptor-like protein kinase, and is expressed in the female sporocytes increases abnormally (Fig. 5F) (Nonomura et al. 2004b). Lee et al. (2004) identified the OsCP1 gene from T-DNA-tagged mutant lines. The oscp1 mutant showed a significant defect in pollen development, in addition to dwarfism. OsCP1 expression is observed mainly in anther locules but not in vegetative tissues. OsCP1 encodes the papain family cysteine protease.

Kaneko et al. (2004) identified a MYB domain gene OsGAMYB, from the Tos17-tagged lines. The osgamyb mutant exhibits defects especially in anther and pistil development, but not in vegetative tissues. GAMYB is a positive transcriptional regulator of gibberellin-dependent α-amylase (Gubler et al. 1995). Even though OsGAMYB is also highly expressed in the aleurone layer of rice seeds, one of its essential functions might be in pollen development (Kaneko et al. 2004).

Large-scale monitoring of specific gene expression in reproductive organs

Using cDNA microarray analyses and in situ hybridization techniques, Endo et al. (2004) revealed 259 non-redundant cDNA clones specifically or predominantly expressed in rice anthers, and classified into four groups. Lan et al. (2004) reported the 253 ESTs exhibiting differential expression during pollination and fertilization.

Genes and Mutants Identified as Seed Characters

Embryo and endosperm mutants are divided into categories of embryo, endosperm and grain shape for the morphological characters, and dormancy, longevity, storage substances, shattering and taste for the physiological characters.

Embryo

A large number of mutations affecting various aspects of seed development have been reported in rice. Rice seeds consist of two distinct parts, an embryo and an endosperm. Over 200 embryo mutants reported to date (Nagato et al. 1989, Kitano et al. 1993, Hong et al. 1995a) are categorized into five groups: embryoless, deletion of embryonic organs, altered embryo size, modified organ position and aberrant morphology.

Embryoless and organ deletion

The embryoless1 (eml1) mutant develops seeds that lack an embryo (Fig. 6A) (Hong et al. 1995a, Hong et al. 1995b). The eml1 embryo degenerates at the early stage (∼3 d after pollination), but endosperm development seems normal. Notably, manifestation of an embryoless phenotype depends on the growing temperature.

The globular embryo (gle) mutants develop embryos with a globular shape and completely lack embryonic organs (Fig. 6B). At least four loci (gle1–gle4) cause this phenotype. Analysis of gle4 embryo using molecular markers indicates that GLE4 plays a role in radial pattern formation during rice embryogenesis, but not in formation of embryonic organs (Kamiya et al. 2003). The club-shaped embryo (cle) mutants fail to form both a shoot and a radicle, as does the gle mutations (Fig. 6C). Unlike the gle embryos, palisade-shaped cells characteristic of the scutellar epithelium are formed in the whole epidermis of cle. Since the palisade-shaped cells of cle express an α-amylase gene, it is considered that the most cle embryo comprises the scutellum (Hong et al. 1995a).

The organless (orl) mutants also lack both a shoot and a radicle (Fig. 6D). Unlike cle1, the orl1 embryo differentiates palisade-shaped cells in the epidermis-facing endosperm, suggesting that embryonic polarity is not affected in orl1. Consist-
ently, tissue specificity of OSH1 gene expression is maintained in orl1 (Sato et al. 1996).

Four shootless (shl) mutants, shl1, shl2, shl3 and shl4, have been reported to be indispensable in shoot formation during embryogenesis (Satoh et al. 1999). Of these, shl1, shl2 and shl4 exhibit indistinguishable phenotypes; complete loss of shoots, coleoptile and epiblast and normal radicle formation (Fig. 6E). In another mutant, shl3, in addition to deletion of the shoot, the radicle is exogenously produced and cells in the remaining tissues are highly vacuolated.

SHL1, SHL2 and SHL4 function upstream of OSH1 (Satoh et al. 1999). At least two loci, RAL1 and RAL2, are required for radicle formation. The ral embryo seems to be truncated in the apical–basal direction but develops a shoot. After germination, the ral plant produces normal crown roots. Thus, it is supposed that the ral phenotype bears root-producing ability, but is caused by deletion of the basal embryonic region where the radicle develops. In addition, ral1 affects vascular pattern formation (Scarpella et al. 2003).

**Organ Morphogenesis**

Two shoot organization (sho) mutants, sho1 and sho2, show severe defects in embryonic organ development. In sho embryos, the first to the third leaves are malformed, but the radicle develops normally (Fig. 6F). The sho1 plant produces malformed leaves with a random phyllotaxy and a short plasto-chron, resulting in deformed shoot architecture (Itoh et al. 2000).

**Embryo Size**

Two types of mutations causing either a reduction or enlargement of embryo size have been reported (Hong et al. 1996). The giant embryo (ge) mutants have a 1.2–1.5 times longer embryo and reduced endosperm (Fig. 6G). The scutellum is enlarged but the sizes of the shoot and radicle are not affected (Hong et al. 1996). Three reduced embryo (re) mutants, re1, re2 and re3, exhibit a reduction in embryo size. The re embryo is less than half the length of the wild-type embryo, and shows a reduction in all embryonic organs including the apical meristems and the enlarged endosperm (Fig. 6G) (Hong et al. 1996). Anatomical and double mutant analyses using ge, re and other mutants suggest that the primary function of GE and RE resides in endosperm development, not in embryogenesis (Hong et al. 1996).

**Organ Position**

The APD1 locus is related to organ position. In the apical displacement (apd) mutant embryo, the shoot is formed at the apex of the embryo and the radicle at the center of the embryo (Fig. 6H). This phenotype is due mostly to an enlargement of the basal region of the embryo and underdevelopment of the scutellum (Hong et al. 1995a).


Endosperm

Because the cereal endosperm is an important staple diet in many countries, studies on genes acting in the endosperm are important issues for both basic research and plant breeding. Although a large number of genes that are associated with storage substances in the endosperm, such as protein and starch, have been identified, analysis of genes regulating endosperm development is limited. Only one mutant affecting endosperm development has been reported. The *endospermless* (*enl*) mutant fails to form an endosperm (Fig. 6I) (Kageyama et al. 1991). After fertilization, the *enl* endosperm starts to develop but degenerates at an early stage (∼3 d after pollination), resulting in the production of a very large embryo (Kageyama et al. 1991, Miyoshi et al. 2000). The mutations associated with embryo size, *ge* and *re*, are also supposed to affect endosperm growth (Hong et al. 1996).

SEED MATURATION

To date, a large number of rice genes related to seed maturation processes, mainly starch and storage protein synthesis and their accumulation, have been identified.

Rice seeds accumulate various kinds of substances as nutrients to support seedling growth. Starch, which consists of amylose and amylpectin, is the major source of nutrition for seedling growth. The *Waxy* (*Wx*) gene encodes a granule-bound starch synthase involved in amylose synthesis (Nelson and Pan 1995). Two naturally occurring *Wx* alleles, *Wxa* and *Wxb*, are known (Sano 1984, Sano et al. 1986). Rice cultivars (mainly japonica cultivars) with a *Wxb* allele produce lower amounts of *Wx* protein than those (mainly indica cultivars) carrying *Wxa* (Sano et al. 1986). *Wxb* carries a substitution mutation at the 5′ spliced site of the first intron, and causes a low content of amylose (Cai et al. 1998, Ishihiki et al. 1998). The *dull* (*du*) mutations also affect amylose content. In *du*-1 and *du*-2, the amount of spliced *Wxb* mature transcript, but not *Wxa* transcript, is reduced (Ishihiki et al. 2000).

Cereal seed proteins have been classified into four types, water-soluble albumin, salt-soluble globulin, alcohol-soluble prolamin and acidic or basic solution-soluble glutelins. Among them, glutelin has been studied intensively because it is the most abundant storage protein occupying 60–80% of the total endosperm protein. Glutelin precursors are encoded by a multigene family that consists of the *GluA* and *GluB* subfamilies (Takaika et al. 1991). Mutations showing a reduction or lack of a particular subunit or precursor polypeptide have been identified. Three loss-of-function mutants, *gulutelin1* (*glu1*), *glu2* and *glu3*, have defects in subunits 1a, 2a and 3a, respectively. A dominant mutation of *Low glutelin content1* (*Lgc1*) causes a reduction in glutelin content. Unlike the above *glu* mutations, the *Lgc1* mutation has been found to influence the amount of most glutelin subunits (Iida et al. 1997). It is revealed that *Lgc1* suppresses accumulation of glutelin gene family transcripts via RNA silencing (Kusaba et al. 2003).

Seed dormancy

Since seed dormancy is a complex and quantitative character influenced by both genetic and environmental factors, the regulatory mechanism of dormancy is not well understood. *OsVP1* is a rice ortholog of *VP1* in maize and *ABI5* in *Arabidopsis* (Hattori et al. 1994). Molecular analysis and in situ expression patterns indicate the involvement of *OsVP1* in seed maturation processes including dormancy, as in maize and *Arabidopsis* (Hattori et al. 1995, Miyoshi et al. 2002). The *rice vivipary* (*riv*) mutants, *riv1* and *riv2*, exhibit precocious germination before harvesting and reduced sensitivity to abscisic acid (Miyoshi et al. 2000).

QTL analysis has detected dormancy-related genes. By using RILs (recombinant inbred lines) between cultivated and wild rice strains, 17 QTLs have been detected (Cai and Morishima 2000). In addition, five QTLs have been detected using BILs (backcross inbred lines) derived from a backcross of Nipponbare/Kasalath/Nipponbare (Miura et al. 2002).

Heterochrony

The heterochronic genes control the temporal regulation of development and greatly affect plant architecture. Two heterochronic genes have been identified and characterized in rice. One is *PLASTOCHRON1* (*PLA1*) (Itoh et al. 1998). In the *pla1* mutant, primary rachis branches in the panicle are converted into vegetative shoots (Fig. 7B). Because vegetative to reproductive phase transition occurs normally, the vegetative phase is elongated and both vegetative and reproductive programs are expressed simultaneously in the *pla1* mutant. Thus, *PLA1* is a heterochronic gene controlling proper termination of the vegetative phase. The *pla1* mutant also shows short plastochron, an enlarged SAM, small leaves, bending of the lamina joint and dwarfism (Fig. 7A). *PLA1* encodes a member of CYP78A, a subfamily of the large cytochrome P450 family, and is expressed in young leaves and bracts (Fig. 7C, D, Miyoshi et al. 2004).

The *mor1* mutant reiterates the wild-type second leaf stage and fails to induce adult phase (Asai et al. 2002). *MOR1* is a heterochronic gene playing an important role in juvenile to adult phase transition.

Tolerance and Resistance

Rice has evolved many kinds of resistance or tolerance genes against biotic and abiotic stresses. Almost all of the resistance or tolerance genes have been found as variant alleles in a variety of cultivated and wild strains.

Disease resistance

A large number of resistant genes or alleles specific to individual races of fungi and bacteria have been identified. Two serious and well-studied rice diseases are rice blast, a fungal disease caused by *Magnaporthe grisea*, and bacterial bright
caused by Xanthomonas oryzae. Most resistance genes identified for bacterial and fungal pathogens have nucleotide-binding sites/LRRs (NBS-LRR) and/or serine/threonine receptor kinase domains. In total, >20 resistance genes to Xanthomonas are identified, and three genes are cloned; Xanthomonas resistance gene 1 (Xa1), Xa21 and Xa26, encoding LRR receptor kinase proteins (Yoshimura et al. 1998, Wang et al. 1998, Sun et al. 2004). Among >60 blast resistance genes/alleles including QTLs, two genes are cloned; Pyricularia oryzae (Magnaporthe grisea) resistance gene ta (Pi-ta) encoding NBS-LRR and a gene found in a resistant strain Zhai Ye Qing8 (ZYQ8) encoding a protein carrying a low homology to the serine/threonine kinase domain and a calmodulin-binding domain (Wang et al. 1999, Zheng et al. 2004).

A single base change in Pi-ta confers a difference between resistance and susceptibility (Bryan et al. 2000). Avirulence gene that is interactive with Pi-ta has also been isolated (Orback et al. 2000). In addition, an intensive gene-for-gene analysis has been carried out of the multiple steps of the infectious process of Magnaporthe in rice (Sesuma and Osbourn 2004), and a variety of cDNAs induced during the defense steps in the rice blast-resistant mutant have also been identified (Han et al. 2004).

Recently, an approach has been used to identify mutants which confer breakdown of Xa21-mediated resistance from thousands of mutant strains (Wang et al. 2004). Xa21 is a wide spectrum resistance gene for multiple varieties of X. oryzae. Therefore, findings of the breakdown mutants for Xa21 resistance would clarify genes composed of the Xa21 multispectrum defense pathway.

A hundred disease-resistant or defense-response gene-like sequences have been mapped into several clusters on the rice chromosomes, and some of them coincide with QTLs or major resistance genes (Wang et al. 2001). The NBS family has around 400 members in the rice genome (Monosi et al. 2004), and the receptor-like kinase (RLK) family has about 1,200 members (Shiu et al. 2004).

Lesion mimic mutants

The lesion mimic mutants show symptom-like spots on leaves and sometimes on the panicles. Over 11 mutants have been identified and are classified as spotted leaves (spl) or cell death and resistance (cdr) (Fig. 8A). Most of them are thought to have defects in genes with roles in the disease defense pathways against attack from fungi, bacteria or viruses, although a few spl mutants do not show resistance. Some mutant genes which produce lesions mimicking symptoms are well characterized (Takahashi et al. 1999, Yamanouchi et al. 2002). Two genes cloned from spl mutants are the heat stress transcription factor (HSF) gene (Kawasaki et al. 1999), and a U-box/Armadillo repeat protein gene involved in the ubiquitination pathway (Zeng et al. 2004).

Insect resistance

More than 200 species of insects are associated with rice plants as pests. Among them, the brown planthopper (BPH; Nilaparvata lugens Stål), whitebacked planthopper (WBPH; Sogatella furcifera Horvath), green leafhopper (GLH; Nephotettix virescens Distant), green rice leafhopper (GRH; Nephotettix cincticeps Uhler) and Asian rice gall midge (ARGM; Orseolia oryzae Wood-Mason) are the worst pests for which the major host resistance genes have been well studied. Most of the >70 resistance genes reported so far have been identified using RILs, NILs or detected as QTLs. More than 13 resistance genes against BPH and six genes against WBPH have been identified, and several BPH resistance genes have been tagged on a molecular linkage map. An example of antibiosis is shown in Fig. 8B–D. Rice ovicidal response to WBPH is characterized by the formation of watery lesions and production of an ovicidal substance, benzyl ben-
zoate, which causes high egg mortality of WBPH (Seino et al. 1996, Suzuki et al. 1996). A gene with ovicidal activity to WBPH, Ovc, and four ovicidal QTLs, qOVA-1–3, qOVA-4, qOVA-5–1 and qOVA-5–2, have been identified (Yamasaki et al. 2003). Ovc was the first gene to be identified that kills insect eggs in plants.

Another type of antibiosis is found in resistant varieties against GLH and GRH. The resistant plants cause delayed growth and eventual death of infesting insects. The resistance may be concerned with sucking inhibition after their infestation. So far, six loci for resistance to GRH, Grh1–Grh6, are known. NILs carrying single resistance genes show weak resistance (Grh1 and Grh2) and susceptibility (Grh4). On the other hand, NILs carrying two resistance genes, Grh2 and Grh4, express strong resistance to GRH and GLH. The interaction of Grh2 and Grh4 expresses a strong resistance against two leafhopper species in rice.

Gm2 is a dominant gene conferring resistance to biotype 1 of ARGM (Diptera: Cecidomyiidae), the major dipteran pest. Tissue necrosis, represented by a typical hypersensitive reaction accompanied by maggott mortality, is observed within 4 d after infestation of avirulent biotype 1 of the ARGM (Bentur and Kalode 1996). Two other resistance genes, Gm6 and Gm7 (both may be identical), are tightly linked to Gm2 (Katiyar et al. 2001, Sardesai et al. 2002).

Wild rice species are excellent genetic resources for resistance genes and can also be used to integrate their resistance loci into cultivated rice (Brar and Khush 1997). Some loci have been confirmed as being transferred successfully into cultivated rice via homologous chromosome recombination. None of the rice resistance genes to insects have yet been cloned.

Stress tolerance

Sensitivities to various stresses such as drought, cold (low temperature), salt/osmotic stress, herbicides and metals vary from strain to strain. QTL analysis using an F₂ population, RILs or NILs is the most convenient way to detect multiple loci for tolerance and sensitivity (Nguyen et al. 2004). Instead of identification of stress tolerance genes using genetic methods, another molecular approach to identify and isolate such tolerance-related genes is employed (de los Reyes et al. 2003, Dubouzet et al. 2003). Dubouzet et al. (2003) isolated five stress responsible rice genes of the DREB/COF transcription factor by homolog hunting of Arabidopsis genes. The cloned genes are expressed under cold or dehydration and high-salt stress and then activate a number of target genes. Attempts to generate stress-tolerant transgenic rice with stress tolerance-related genes have also been made (Garg et al. 2002, Mukhopadhyay et al. 2004). Recently, a more sophisticated genomic approach has become available involving combinatorial analysis of expression profiling using microarray analysis with segregated progeny from a cross of tolerant and sensitive parent strains (Cooper et al. 2003, Hazen et al. 2004).

Stress tolerance is tightly related to the developmental stages and specific organs. For instance, metal ion and salinity tolerance is expressed mainly in the root during the process of ion absorption through water uptake. Cold tolerance is especially needed during the germinating and flowering stages. It might be possible to identify tolerant mutants/gens in the mutant/variant groups of these organs. Submergence tolerance may be specifically important in rice, a character enabling survival during deep water stress. A group of wild rice strains and landraces show submergence tolerance, which results in rapid internode elongation. This specific character must also be regulated by a genetic program for culm development. A recent review and a QTL analysis for submergence sensitivity have shown various aspects of this reaction (Jackson and Ram 2003, Toojinda et al. 2003).

Coloration

Coloration is one of the most important characters of plants, especially in relation to the chlorophyll biosynthesis pathway. Tissue-specific coloration with anthocyanin is an interesting phenomenon, which is partly integrated in the organ developmental program. A large number of mutants and variants have been identified in these categories.

Anthocyanin and other colorations

More than 40 mutants and/or variants have been found to possess purple coloration genes/alleles by modifying anthocyanin biosynthesis in rice. The divergent pattern of anthocyanin coloration in specific organs, e.g. coleoptile, leaf axil, leaf sheath, leaf blade, leaf margin, midrib, leaf apex, internode, nodal ring, pericarp and stigma, is detected in the mutants/variants. It has been reported that these specific patterns in expression of anthocyanin are attributed to dysfunction of the key regulator of anthocyanin biosynthesis R genes/alleles in rice. R genes encode bHLH protein transcription factors and regulate pigmentation in specific organs. One of the rice R loci, purple
leaf (Pl), has a complex allele Pl(w) composed of at least two genes of the bHLH proteins (Sakamoto et al. 2001). The complex nature of multiple alleles of the R loci may be involved in a variety of organ/tissue-specific regulations of anthocyanin biosynthesis. Sixteen mutants for coloration with substances other than anthocyanin have been reported: Brown furrows of hull (Bf), gold furrows of hull (gf1and2), gold hull and internode (gh1-3), red pericarp and seed coat (Rd), and so on. These mutants also show organ-specific coloration.

Chlorophyll

Chlorophyll synthesis is very important to all plants in relation to photosynthesis. Chlorophyll and its encasing organelle, the chloroplast, are both synthesized from chloroplast- and nucleus-encoded genes. Studies on chlorophyll and chloroplast biosynthesis pathways have been extensively compiled for many plants. In rice, >70 chlorophyll mutants exhibiting albino, chlorina, stripe, virescent, yellow-green and zebra leaves have been recorded. Albino has no chlorophyll, completely lacks any green color and dies soon after germination. Chlorina mutants have light green leaves and a low ability for photosynthesis, thus growing weakly and depending on the gene locus or allele. The stripe phenotype appears as a white sectored leaf in the longitudinal direction, and is different in size and number of white stripes among the loci. On the contrary, zebra has a white or yellow transverse banding phenotype in the green leaves. This phenotype is highly variegated in band size, frequency and color at every stage of plant growth. Virescent mutants also show conditional chlorosis in the leaf to different degrees depending on the temperature, strength and wavelength of light. One of the virescent mutant inhibits the translation of plastid transcripts during chloroplast differentiation (Sugimoto et al. 2004). These analyses indicate there are several components used in chlorophyll synthesis and the photosynthesis pathways. Most classes of chlorophyll mutants are composed of >10 independent loci, suggesting that each biosynthesis pathway is constructed with at least 10 proteins that are necessary to complete chlorophyll/chloroplast biogenesis. One chrolina mutant has been revealed to encode OsCHLH, a key enzyme in the chlorophyll branch biosynthesis pathway (Jung et al. 2003).

For photomorphogenesis in rice, coleoptile photomorphogenesis 1 (cpm1) has been reported which shows impairment of phytochrome-mediated inhibition of coleoptile growth and impairment of anthesis (Biswas et al. 2003). However, this mutant expresses almost the same level of phytochrome A, B and C genes as in the wild type, suggesting that the CPM1 gene is involved in phytochrome signal transduction that specifically leads to alterations in leaf and anther growth. This is one of the classes of pleiotrophic mutants in which defects are observed in leaves and other organs. A large number of other mutant genes with different loci could provide genetic links to the chlorophyll biosynthesis pathway.

Mutant and Variant Genes in Future Functional Genomics of Rice

As shown in this mini-review, many trait genes revealed by mutant and QTL analyses have been accumulated. In the next decade, full genome sequence information should help to promote the rapid growth in isolation and characterization of these trait genes. The collection and positioning of many pieces of genes in the developmental and physiological pathways will decipher the many gene networks and thus determine the morphological and developmental regulations for those gene networks. Morphological mutants play an indispensable role in the study of rice development and of QTLs with regards to plant growth reactions. However, individual gene characterization is not enough to achieve such kinds of studies, but systematic work using genomic and bioinformatic approaches is required. Generation of a search system for knock-out mutants for all rice genes, microarray analysis for mutant and variant strains, gene/genome comparative studies with Arabidopsis, wild rice and other cereal species, and an ideal analytical system for handling large quantities of data using bioinformatic tools will together promote functional genomic studies in the very near future.

Appendix 1

(1) http://tos.nias.affrc.go.jp/~miyao/pub/tos17/
(2) http://www.shigen.nig.ac.jp/rice/oryzabase/nbrpStrains/kyushu-Grc.jsp
(3) http://www.shigen.nig.ac.jp/rice/oryzabase/genes/geneClasses.jsp

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References

Rice mutants and trait genes
