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Functional Characterization of Genes/QTLs for Increasing Rice Yield Potential

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1. Introduction

Rice (*Oryza sativa* L.) as the monocot model plant and an important food crop is cultivated worldwide. Due to the rapid growth of the world's population, rice yield is urgently required to increase to meet world food demand. In the last century, rice yield experienced rapid growth twice in China, which is mainly attributed to the exploitations of *semi-dwarf 1(sd1)* gene and heterosis of F_1 hybrid. Before the green revolution, rice varieties were tall and had a low harvest index. Introgression of *sd1* into the varieties significantly reduced the plant height and increased the harvest index, which resulted in a dramatic increase of rice productivity [1]. Heterosis breeding has been widely used to improve rice yield potential. Hybrid rice varieties usually have a yield advantage of 10-20% over the conventional inbred varieties, thus cover more than half of the total rice area in China at present [2, 3]. However, rice yield per unit area has not been much elevated and the arable land for rice cultivation has kept decreasing during the past two decades. New genetic improvement strategies are urgently required to break the bottleneck of yield potential of current varieties, which largely rely on the elucidation and exploitation of genetic and molecular basis for rice yield traits [4].

Rice yield traits are complex agronomic traits governed by multiple genes called as quantitative traits loci (QTLs), which usually show a continuous phenotypic distribution in a segregating population derived from a cross of a pair of inbred lines. Most QTLs for yield traits show small genetic effect and are difficult to be identified. These minor QTLs play a vital role in regulating yield trait and are widely utilized in commercial rice varieties, so that fine-mapping and map-based cloning of these QTLs will be beneficial for breeding. Number of panicles per plant, number of grains per panicle, and grain weight are three component traits which are determined by tiller, panicle and grain development. Dissecting the genetic basis of these traits by QTL mapping can facilitate breeding high yield varieties. However, it is rather

difficult to isolate these QTLs because each contributes little effect to yield traits, and the effect is strongly affected by the environment. In recent years, tremendous progress has been attained and many QTLs for rice yield traits have been isolated and functionally analyzed in detail, which provides new sights into the molecular mechanisms of the formation of rice yield traits. Meanwhile, mutant analysis has also functionally characterized many genes involved in yield traits because of the availability of rice genome and rice mutant collections. These studies greatly strengthen our understanding of regulatory mechanisms of these traits. In this chapter, we summarize the recent progress in the genetic and molecular mechanisms underlying rice yield traits and illustrate a strategy to develop varieties with higher yield potential.

2. Identification and validation of QTLs for rice yield traits

2.1. Identification of QTLs

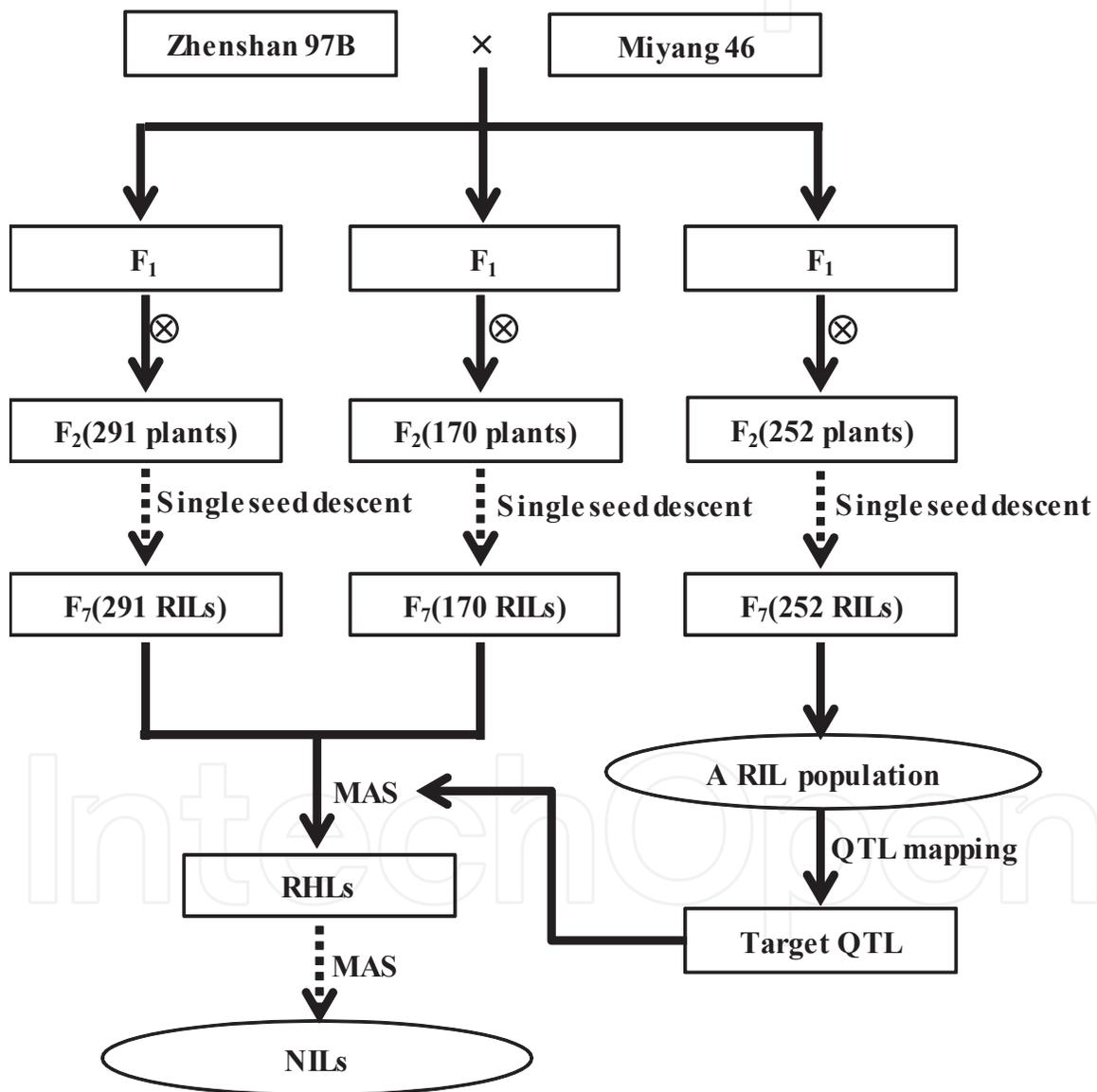
QTL mapping of a target trait is defined as the chromosomal location and genetic characterization of QTLs for the trait through the association between genetic markers and phenotypic variations. To facilitate this mapping, development of mapping population, construction of linkage map and phenotypic evaluation are essential for QTL analysis.

Typically, mapping population includes F_2 plants, doubled haploid lines (DHLs) and recombinant inbred lines (RILs). F_2 population that carries the complete genetic information from the parents can be easily developed, but its phenotypic evaluation cannot be replicated [5]. Due to the inherent homozygosity in the lines, both DHL and RIL populations can be planted repeatedly in different planting seasons and environment conditions as many times as necessary. DHL population is limitedly used in QTL mapping due to the difficulties in the plant regeneration from cultured anthers, especially for *indica* rice varieties [6, 7]. RIL population is widely used in QTL mapping although it is time-consuming and labor-intensive to prepare the population. Many RIL populations have been developed from inter-subspecies crosses [8, 9], intra-subspecies crosses [10-12], or crosses between commercial cultivars and wild rice [13].

Linkage map is composed of many linkage groups according to different chromosomes, which are constructed by genotyping using genome-wide polymorphic markers. DNA based molecular markers, such as restriction fragment length polymorphism (RFLP), simple sequence repeat (SSR), cleaved amplified polymorphic sequence (CAPS) and single nucleotide polymorphism (SNP), are widely applied in the construction of linkage map. Based on the complete genome-wide sequence of rice, it becomes easier to design genome-wide polymorphic markers and construct high density molecular linkage map [14].

Yield trait conditioned by QTLs usually varies continuously in a mapping population. Phenotypic values are difficult to be accurately measured due to environmental influences, especially for F_2 population without replication. The precision of phenotypic data greatly affects the resolution of QTL mapping [15].

Thousands of QTLs for rice yield traits have been detected and are distributed throughout the whole genome while many of them are co-localized (<http://www.gramene.org>). We use one of our studies to illustrate the general process for the classical characteristics of QTLs (Figure 1). A RIL mapping population is developed from an *indica-indica* cross between Zhenshan 97B and Milyang 46 using single seed descent method. QTL analysis shows that each yield trait is controlled by several QTLs. These QTLs are dispersedly distributed on chromosomes, and function on yield productivity not only by their own effects, but also by within-locus and inter-locus interactions [11].



RIL, recombinant inbred line; MAS, marker-assisted selection; RHL, residual heterozygous line; NIL, near isogenic line; QTL, quantitative trait locus

Figure 1. Flowchart for developing NILs through screening RHLs in a Zhenshan 97B/Miyang 46 RIL population.

2.2. Validation of QTLs

Primary mapping cannot delimit an individual QTL in a precise location, so that further experiments are necessary to validate the biological function of target QTLs individually. Development of near isogenic lines (NILs) is an efficient strategy for QTL validation. NILs contain the segregated target QTL region in a homogeneous genetic background. In general, NILs are produced by consecutive backcrosses with a recurrent parent aided by molecular marker assisted selection (MAS). Only the plants carrying the target QTL in the recurrent parent background will be selected to further develop NILs. Many QTLs controlling rice yield traits have been validated by this classical method. However, successful backcross combining with MAS is laborious and time-consuming. During the process of developing Zhenshan 97B / Miyang 46 RIL population, we form a new method for developing NILs by screening residual heterozygous lines (RHLs). The RHLs should contain a heterozygous chromosomal segment at the target QTL region in a nearly homozygous genetic background [2] (Figure 1). Following MAS in a high density marker linkage map, a series of RHLs with overlapping segregated segments for the target QTL are selected from F₇ RILs. This method has proven to be efficient, and several yield trait QTLs, such as *qGY6* and *qGL7-2*, have been successfully fine-mapped and validated [2, 16].

Introgression lines (ILs) and chromosome segment substitution lines (CSSLs), which are developed by backcrossing repeatedly with the recurrent parent, can also be used for QTL validation, fine-mapping and breeding superior rice varieties [17,18].

3. QTLs/genes for rice yield traits

3.1. QTLs/genes for tillering

Rice tillers are mainly composed of primary, secondary and tertiary tillers, which are shoot branches arising from the unelongated basal internodes. Tillering starts with the appearance of the fourth leaf from the main culm. Usually, the duration of tillering will last about 25-30 days. The number of panicles and yield potential are determined by panicle-bearing tillers, and grain yield are mainly contributed by primary and secondary tillers. Therefore, tiller number is considered a key component in determining rice yield. Some key genetic factors responsible for rice tillering have been molecularly characterized (Figure 2 and Table 1).

Rice tillering undergoes two major processes, the formation and outgrowth of tiller bud. Isolation and characterization of *MONOCULM* (*MOC1*) provide a new sight for the formation of tiller bud. The *moc1* mutant phenotypically exhibits only one main culm without any tillers due to the deficiency to form axillary bud. *MOC1* encodes a member of *GAI*, *RGA* and *SCR* (GRAS) family nuclear proteins to function on the formation of axillary buds [19].

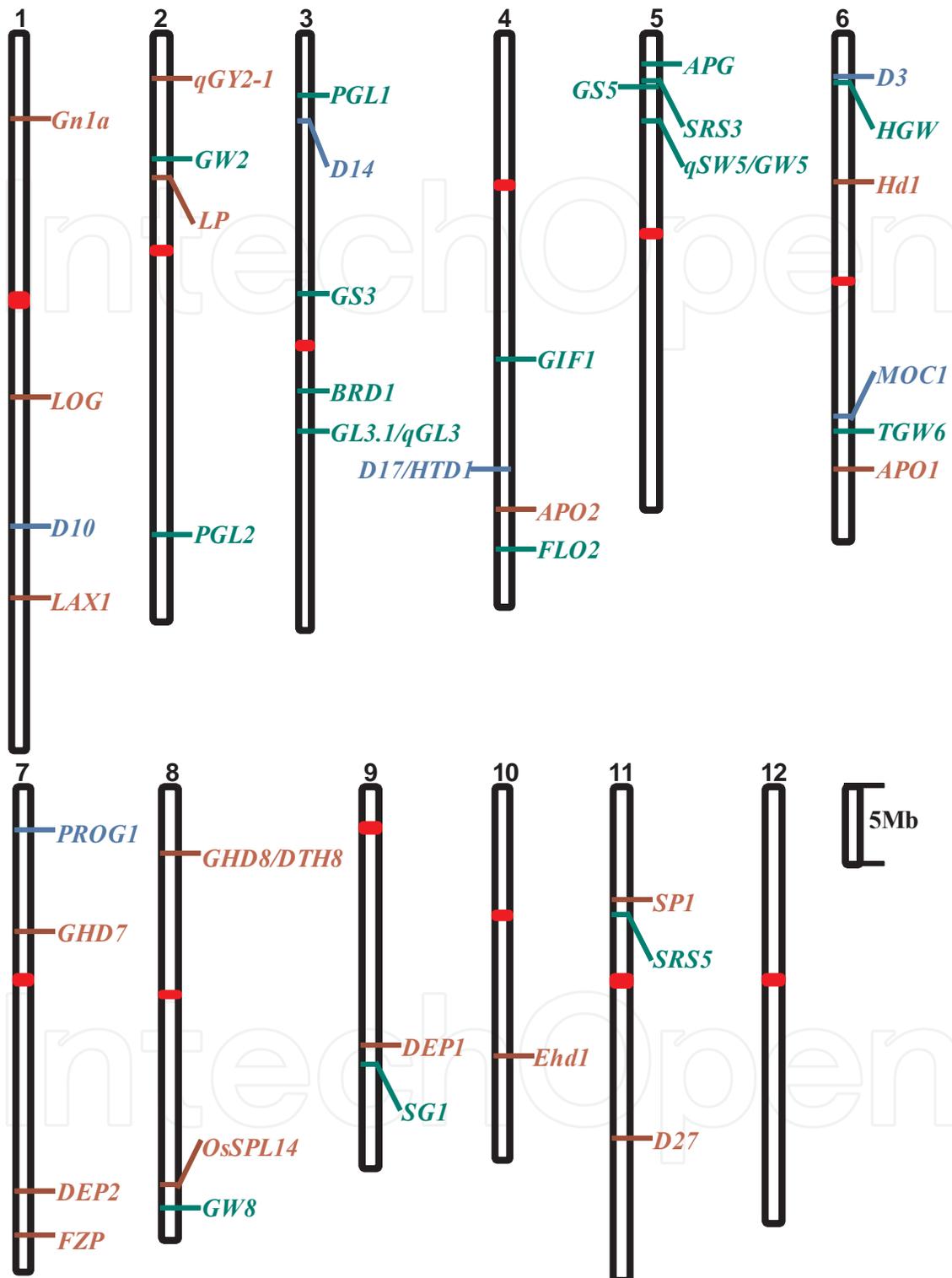


Figure 2. Distribution of the cloned genes/QTLs for rice yield traits in a physical map based on Nipponbare genome sequence; Red ellipses indicate the centromere region according to the data from the International Rice Genome Sequencing Project (<http://rgp.dna.affrc.go.jp/IRGSP/>); the color horizontal lines represent the locations of each gene/QTL on the chromosome for each trait; blue for number of panicles per plant (NPP); crimson for number of grains per panicle (NGP); green for grain weight (GW).

Traits	Gene/QTL	MSU-ID	Encoded protein	M/QTL	Refs
NPP	<i>D27</i>	LOC_Os11g37660	Iron-containing protein	M	[24]
NPP	<i>D10</i>	LOC_Os01g54270	Carotenoid cleavage dioxygenase 8	M	[23]
NPP	<i>D14</i>	LOC_Os03g10620	Alpha/beta-fold hydrolase superfamily protein	M	[26]
NPP	<i>D17/HTD1</i>	LOC_Os04g46470	carotenoid cleavage dioxygenase	M	[22]
NPP	<i>D3</i>	LOC_Os06g06050	F-box leucine-trich repeat protein	M	[25]
NPP	<i>MOC1</i>	LOC_Os06g40780	GRAS family nuclear protein	M	[19]
NPP, NGP	<i>OsSPL14</i>	LOC_Os08g39890	Souamosa promoter binding protein-like 14	QTL	[27, 28]
NPP, NGP	<i>PROG1</i>	LOC_Os07g05900	Zinc-finger nuclear transcription factor	QTL	[29, 30]
NPP, NGP	<i>qGY2-1</i>	LOC_Os02g05980	Leucine-rich repeat receptor-like kinase	QTL	[31]
NGP	<i>Gn1a</i>	LOC_Os01g10110	Cytokinin oxidase/dehydrogenase	QTL	[39]
NGP	<i>LOG</i>	LOC_Os01g40630	Cytokinin-activating enzyme	M	[40]
NGP, NPP	<i>LAX1</i>	LOC_Os01g61480	A bHLH transcription factor	M	[32]
NGP	<i>LP</i>	LOC_Os02g15950	Kelch repeat-containing F-box protein	M	[41]
NGP, NPP	<i>APO2</i>	LOC_Os04g51000	Plant-specific transcription factor	M	[35]
NGP	<i>APO1</i>	LOC_Os06g45460	F-box protein	M	[34]
NGP, NPP	<i>DEP2</i>	LOC_Os07g42410	Plant-specific protein	M	[38]
NGP, NPP	<i>FZP</i>	LOC_Os07g47330	Ethylene-responsive element-binding factor	M	[33]
NGP	<i>SP1</i>	LOC_Os11g12740	Transporter of the peptide transporter family	M	[36]
NGP	<i>Hd1</i>	LOC_Os06g16370	Protein with a zinc finger domain	QTL	[67]
NGP	<i>Ghd7</i>	LOC_Os07g15770	CCT domain protein	QTL	[62]
NGP	<i>Ghd8/DTH8</i>	LOC_Os08g07740	OsHAP3 subunit of a CCAAT-box binding protein	QTL	[63, 64]
NGP	<i>EHD1</i>	LOC_Os10g32600	B-type response regulator	QTL	[65, 66]
NGP	<i>DEP1</i>	LOC_Os09g26999	PEBP-like domain protein	QTL	[37]
GW, GS	<i>TGW6</i>	LOC_Os06g41850	indole-3-acetic acid (IAA)-glucose hydrolase	QTL	[45]
GW, GS	<i>GW2</i>	LOC_Os02g14720	RING-type E3 ubiquitin ligase	QTL	[53]
GW, GS	<i>PGL2</i>	LOC_Os02g51320	Atypical bHLH protein	Hom*	[49]
GW, GS	<i>PGL1</i>	LOC_Os03g07510	Atypical bHLH protein	Hom*	[48]
GW, GS	<i>GS3</i>	LOC_Os03g29380	Trans-membrane protein	QTL	[42]
GW, GS	<i>BRD1</i>	LOC_Os03g40540	Brassinosteroid-6-oxidase	M	[51]

Traits	Gene/QTL	MSU-ID	Encoded protein	M/QTL	Refs
GW, GS	<i>GL3.1/qGL3</i>	LOC_Os03g44500	Phosphatase with Kelch-like repeat domain	QTL	[43, 44]
GW, GF	<i>GIF1</i>	LOC_Os04g33740	Cell-wall invertase	M	[58]
GW, GF	<i>FLO2</i>	LOC_Os04g55230	Protein with a tetratricopeptide repeat motif	M	[60]
GW, GS	<i>APG</i>	LOC_Os05g04740	Typical bHLH protein	Hom*	[48]
GW, GS	<i>SRS3</i>	LOC_Os05g06280	Kinesin 13 protein	M	[46]
GW, GS	<i>GS5</i>	LOC_Os05g06660	Putative serine carboxypeptidase	QTL	[56]
GW, GS	<i>qSW5/GW5</i>	LOC_Os05g09520	Nuclear protein	QTL	[54, 55]
GW, GF	<i>HGW</i>	LOC_Os06g06530	ubiquitin-associated domain protein	M	[59]
GW, GS	<i>GW8</i>	LOC_Os08g41940	Squamosa promoter-binding protein-like 16	QTL	[57]
GW, GS	<i>SG1</i>	LOC_Os09g28520	Novel protein	M	[52]
GW, GS	<i>SRS5</i>	LOC_Os11g14220	Alpha-tubulin protein	M	[47]

NPP, number of panicles per plant; NGP, number of grains per panicle; GW, grain weight; GS, grain size; GF, grain filling; M/QTL, mutant/QTL; Hom*, Homolog.

Table 1. Genes/QTLs for rice yield traits are reviewed in this chapter

Phytohormone pathways play a crucial role in controlling the outgrowth of tiller bud from leaf sheath. Plant hormones interact to regulate axillary bud outgrowth. It is well known that auxin maintains shoot apical dominance and inhibit axillary bud outgrowth, whereas cytokinins promote branches development [4]. Strigolactones, as a new kind of terpenoid plant hormones, might act as the downstream of auxin to inhibit axillary bud outgrowth. Several genes involved in the synthesis and signaling pathway of strigolactones are isolated and functionally characterized through analyzing a serious of tillering dwarf mutants [20, 21]. *DWARF17 (D17)/HIGH-TILLERING DWARF1 (HTD1)*, *DWARF10 (D10)* and *DWARF27 (D27)* are involved in the biosynthesis of strigolactones, while *DWARF3 (D3)* and *DWARF14 (D14)* act in the signaling pathway in rice [22-26]. Their loss-of-function causes similar phenotype of enhanced shoot branches accompanying with reduced plant height. Although the relationship among phytohormones in regulating axillary bud outgrowth is complex and requires more proof to substantiate, recent advances in the regulatory mechanisms involved in phytohormones help further understand rice tillering.

Tiller number and angle are major determinants of rice plant architecture. New plant type known as ideal plant architecture (IPA) is proposed with reduced tiller number with almost no unproductive tillers to improve cultivar yield potential. A major QTL for IPA encoding SOUAMOSA PROMOTER BINDING PROTEIN-LIKE 14 (*OsSPL14*) has been cloned. *OsSPL14* is regulated by microRNA *OsmiR156*, and increasing level of the *OsSPL14* transcript and protein results in an IPA phenotype and higher grain productivity [27, 28]. *PROSTRATE*

GROWTH 1 (*PROG1*) controlling wide tiller angle and great number of tillers in wild rice species encodes a zinc-finger nuclear transcription factor and is highly expressed in the axillary meristems. An amino acid substitution caused by a SNP in *PROG1* leads to the transition from prostrate growth of the wild rice *O. rufipogon* to erect growth of the domesticated rice *O. sativa* [29, 30]. In addition, *qGY2-1*, a major QTL for grain yield per plant, encodes leucine-rich repeat receptor-like kinase (LRK), and over-expression of *LRK1* causes more tillers and greater grain yield than the wild type [31].

3.2. QTLs/genes regulating number of grains per panicle

Number of grains per panicle is an important agronomic trait for grain productivity, which is determined by the panicle formation. During the past two decades, many genes/QTLs controlling panicle development have been characterized (Figure 2 and Table 1). Rice panicle developed from a terminal inflorescence at the top of a stem contains panicle axis, primary and secondary branches, pedicel and spikelets. Pedicels arise from the primary and secondary branches and bear spikelets on the top. Panicles and the bearing spikelets on them directly determine the rice yield.

Inflorescence development determines the formation of rice panicle. Inflorescence meristem generates primary and secondary branches meristems, and subsequent spikelet meristems. Several genes involved in the formation of inflorescence branch and spikelet meristems are identified through mutant analysis. *LAX1* encodes a basic helix-loop-helix (bHLH) transcription factor and is required for the initiation/maintenance of inflorescence branch meristems. The *lax1* mutant produces severely reduced primary and secondary branches and spikelets [32]. *FRIZZY PANICLE* (*FZP*), which encodes an ethylene-responsive element-binding factor (ERF), is responsible for the establishment of floral meristem identity through suppressing the formation of axillary meristems within the spikelet meristem. The *fzp* mutant is deficient in spikelet development and exhibits sequential rounds of branching instead of the formation of florets [33]. *ABERRANT PANICLE ORGANIZATION* (*APO1*), which encodes an F-box protein, functions in preventing the precocious transition from branch meristems to spikelet meristems. The *apo1* loss-of-function mutants produce small panicles with greatly reduced branches and spikelets [34]. In addition, *APO2* interacts with *APO1* to regulate panicle development [35].

Rice panicle size is largely determined by the number and length of primary and secondary branches. *SHORT PANICLE 1* (*SP1*) encodes a putative transporter of the peptide transporter family and participates in the elongation of rice panicle. The mutation of *SP1* causes a short-panicle phenotype due to the defect in the elongation of inflorescence branches in the *sp1* mutant [36]. *OsSPL14* not only controls tillering, but also promotes panicle branching and produces larger panicles with more spikelets [27, 28].

Rice panicle architecture is mainly determined by the arrangement of primary and secondary branches and grain density. Erect panicle is an important agronomic trait closely related to grain yield. *DENSE AND ERECT PANICLE1* (*DEP1*) encodes a phosphatidylethanolamine-binding (PEBP) protein-like domain protein and controls panicle branches, grain density and erect panicle. The gain-of-function mutation in *DEP1* resulted in the phenotype of increased primary and secondary branches and number of grains per panicle, and decreased panicle

length [37]. *DEP2*, which encodes a plant-specific protein and is strongly expressed in young panicles, is responsible for panicle outgrowth and elongation. The *dep2* mutant displays a dense and erect panicle phenotype [38].

Cytokinins regulate number of spikelets per panicle. A major QTL, *GRAIN NUMBER1 (Gn1a)*, which encodes cytokinin oxidase/dehydrogenase (*OsCKX2*), controls number of grains per panicle. Repression of *OsCKX2* leads to cytokinin accumulation, which finally results in the increase of number of grains per panicle and grain yield [39]. *LONELY GUY (LOG)* is responsible for shoot meristem activity and encodes cytokinin-activating enzyme for the conversion from inactive cytokinin nucleotides to the free-base forms. Loss of function of *LOG* results in producing small panicles with reduced panicle branches and grains in the *log* mutant [40]. *LARGER PANICLE (LP)* encoding a Kelch repeat-containing F-box protein regulates panicle architecture. Larger panicle with more primary branches and grains is observed in the *LP* loss-of-function mutants. *LP* could regulate panicle architecture by modulating cytokinin level due to the significant down-regulation of *OsCKX2* expression level in the mutants [41]. Furthermore, *DEP1* might control the number of panicle branches through cytokinin pathway because expression level of *OsCKX2* is clearly down-regulated in NIL-*dep1* plant [37]. These studies imply that the phytohormone cytokinin plays a vital role in regulating panicle development.

3.3. QTLs/genes controlling grain weight

Rice grain is closely enclosed by a hull which is composed of one palea, lemma, rachilla and two sterile lemmas. A brown rice mainly consists of bran, endosperm and embryo. During the process of grain filling, endosperm cells expand and accumulate a massive amount of nutrients, mainly starch. Rice grain weight is largely determined by the endosperm size. Dozens of genes/QTLs involved in rice grain weight have been isolated and molecularly characterized (Figure 2 and Table 1).

Given that each grain in a rice panicle can be fully filled, grain weight is determined by grain size, which can be measured with grain length, width and thickness. *GS3*, *GL3.1/qGL3* and *TGW6*, three major QTLs controlling grain length, are map-based cloned and functionally analyzed [42-45]. *GS3* encodes a putative trans-membrane protein containing four putative domains, a plant-specific organ size regulation (OSR) domain, a trans-membrane domain, a tumor necrosis factor receptor/nerve growth factor receptor (TNFR/NGFR) family cysteine-rich domain and a von Willebrand factor type C (VWFC). Loss-of-function or deletion of plant-specific OSR domain results in long grain phenotype [42]. *GL3.1/qGL3* encodes Ser/Thr phosphatase of phosphatase kelch family to regulate grain length and yield. *GL3.1/qGL3* directly down-regulates Cyclin-T1;3 to dephosphorylate Cyclin-T1;3 and results in short grain shape [43,44]. *THOUSAND-GRAIN WEIGHT 6 (TGW6)* controls grain length and weight, which expression is especially high around the endosperm in the pericarp. *TGW6* possesses indole-3-acetic acid (IAA)-glucose hydrolase to decompose IAA-glucose into IAA and glucose, which influences the transition timing from the syncytial to the cellular phase and results in short grain phenotype [45]. *SMALL AND ROUND SEED (SRS3)*, which encodes a protein of the kinesin 13 subfamily containing a kinesin motor domain and a coiled-coil structure, is strongly expressed in developing organs and regulates rice grain length. The *srs3* mutant

shows shorter cells compared to the wild type, which causes the small and round seed phenotype [46]. *Srs5* encodes alpha-tubulin protein and its mutation produces a semi-dominant mutant exhibiting similar phenotype with the *srs3* mutant [47]. *POSITIVE REGULATOR OF GRAIN LENGTH 1 (PGL 1)* and *ANTAGONIST OF PGL1 (APG)* encode an antagonistic pair of bHLH proteins and interact to regulate rice grain length [48]. *PGL 1* and *PGL 2* redundantly suppress the function of *APG* to form elongated grains [49]. In addition, brassinosteroid (BR) pathway affects rice grain size. A series of mutants related to the synthesis and signaling pathway of BR such as *d61*, *brd1* and *short grain1 (sg1)* display shorter grain phenotype than their wild types [50-52].

Four QTLs conditioning grain width, *GW2*, *qSW5/GW5*, *GS5* and *GW8*, have been isolated and characterized. *GW2* encodes a RING-type protein with E3 ubiquitin ligase activity to function in the protein degradation through the ubiquitin-proteasome pathway. *GW2 E3* ligase negatively regulates cell division and the mutant allele of *GW2* promotes spikelet hull cell division to result in an increase of grain width and weight [53]. *GW5* and *qSW5* are the same QTL on chromosome 5 in fact, identified by two research groups separately [54, 55]. *qSW5/GW5* encodes a novel nuclear protein, physically interacting with polyubiquitin and acting in the ubiquitin-proteasome pathway to regulate cell division. *qSW5/GW5* is also a negative regulator for grain width and its mutant allele causes an increase of grain width [54]. *GS5* encodes a putative serine carboxypeptidase and positively regulates grain width. Over expression of *GS5* promotes cell division and results in increased grain width [56]. *GW8*, synonym with *OsSPL16*, encodes a positive regulator of cell proliferation and conditioning grain width and yield. Enhanced expression level of *GW8* promotes cell division and grain filling, while its loss-of-function forms a slender grain [57].

Grain thickness largely depends on the ability of grain filling. *GRAIN INCOMPLETE FILLING 1 (GIF1)*, which encodes a cell-wall invertase to download sucrose in the ovular and stilar vascular tissues and hydrolyzes sucrose to glucose and fructose for the starch synthesis in the endosperm, is responsible for rice grain-filling and yield. Mutation in the *GIF1* causes slower grain-filling to result in reduced levels of glucose, fructose and sucrose in the *gif1* mutants. Compared to the wild type *GIF1*, the cultivated *GIF1* displays a restricted expression during the filling stage to bring about grain weight increase [58]. Expression level of *GIF1* is substantially low in the *heading and grain weight (hgw)* mutant, which delays the heading date and reduces grain weight. *HGW* encodes a novel plant-specific ubiquitin-associated domain protein and acts through *GIF1* to regulate grain width and weight [59]. *FLOURY ENDOSPERM2 (FLO2)* encodes a protein harboring a tetratricopeptide repeat motif and preferentially expresses in developing seeds. *FLO2* positively regulates the expression of genes involved in production of storage starch and proteins in the endosperm, so mutation of *FLO2* causes significantly smaller grain size phenotype [60].

4. QTLs/genes for rice yield-related traits

Plant height and heading date are two important agronomic traits closely related to rice yield. The Green Revolution has made a tremendous contribution to solve the global food crisis, and

this mark achievement in rice is caused by the application of *sd1* gene. *SD1* encodes an oxidase enzyme involved in the biosynthesis pathway of gibberellin, which is one of the most important determining factors of plant height. Mutation of *SD1* produces semi-dwarf phenotype and significantly increase rice yield [61].

Genes/QTLs controlling heading date usually prolong the duration of panicle differentiation to produce more spikelets per panicle and enhance grain yield potential. *Ghd7* and *Ghd8/DTH8* are key genes regulating heading date to enhance grain yield and plant height under long-day conditions. *Ghd7* encodes a CCT domain protein and pleiotropically controls an array of traits such as number of grains per panicle, plant height and heading date. Increased expression level of *Ghd7* under long-day conditions suppresses the expression of *Hd3a*, which results in delaying heading date and prolongs the duration of panicle differentiation [62]. Similarly, *Ghd8/DTH8* encodes the OsHAP3 subunit of a CCAAT-box binding protein and simultaneously regulates grain yield, heading date, and plant height. *Ghd8/DTH8* can down-regulate the express level of *Early heading date 1(Ehd1)* and *Hd3a* under long-day conditions, which leads to delay heading date and produce 50% more grains per plant [63, 64]. *Ehd1* and *Hd1* can regulate panicle development. Increased expression level of *Hd3a* and *RFT1* reduces number of primary branches per panicle in the line combining *Hd1* and *Ehd1* [65, 66]. In addition, *Hd1* increases number of spikelets per panicle and grain yield by suppressing *Hd3a* expression and delaying heading date [67].

5. Future perspectives

As mentioned above, cloning and functional characterization of genes/QTLs have greatly strengthened our understanding in the genetic and molecular mechanisms underlying rice yield traits, which has facilitated the breeding efforts for higher yield potential varieties. Pyramiding of favorable genes/QTLs has become an efficient strategy in rice genetic improvement and is widely adopted by rice breeders. For instance, combination of *Gn1* (*Gn1a+Gn1b*) and *sd1* into Koshihikari has simultaneously improved two traits with increased grain numbers per plant by 23% and reduced plant height by 18% as compared to wild type Koshihikari [39]. The NIL (*GW8/gS3*) with a pyramiding of *GW8* and *gs3* produces longer and wider grains than the wild type NIL (*gw8/GS3*) [57].

Although tremendous progress has been made in the studies of rice yield trait, there is still a long way to clearly elucidate the molecular mechanisms responsible for the formation of rice yield traits. Almost all the rice yield traits including number of panicles per plant, number of grains per panicle and grain weight exhibit comprehensive and continuous variations in the genetic population or among the commercial varieties, typically due to the function of multiple genes called as QTLs. According to the Gramene database, thousands of QTLs conditioning rice yield traits have been detected by QTL mapping and majorities of them are minor QTLs with small genetic effect, which are difficult to be identified through mutant analysis. However, minor QTLs may participate in different molecular pathways to regulate rice yield traits and play a vital role in improving yield potential. During the long domestication process, these

minor QTLs have been selected and combined relying on the breeders' experience to develop cultivated varieties. Therefore, more efforts are necessary to isolate minor QTLs and elucidate the functional mechanisms in the future.

Natural variation exists widely in the genes/QTLs, resulting in many alleles for each gene/QTL. For example, *Ghd7* has at least five alleles including *Ghd7-0*, *Ghd7-0a*, *Ghd7-1*, *Ghd7-2* and *Ghd7-3* which enable rice to be cultivated in different ecotype regions. Till now, it is still rather difficult to combine favorable alleles freely in breeding higher yield potential varieties. Mining the alleles is a key base to combine the alleles. Based on the affordable next-generation sequencing technology, association mapping is a promising strategy to mine favorable alleles using a set of diverse germplasm accessions. On the basis of available and favorable alleles, an efficient breeding strategy has been proposed to exploit rice yield potential, involved in identifying the genes/QTLs for rice yield traits, mining alleles of target genes/QTLs through candidate-gene association mapping, developing functional markers and combining favorable alleles in cultivated varieties (Figure 3).

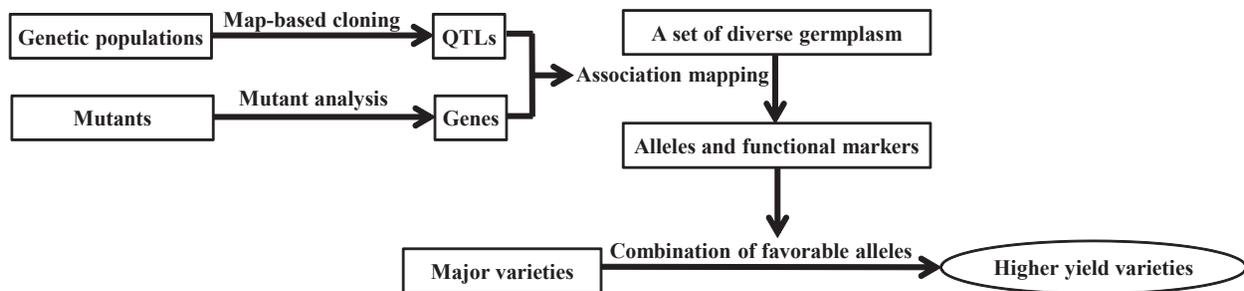


Figure 3. Flowchart for depicting a new strategy to breed higher yield varieties

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