REVIEW

A Genome-Wide Meta-Analysis of Rice Blast Resistance Genes and Quantitative Trait Loci Provides New Insights into Partial and Complete Resistance

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The completion of the genome sequences of both rice and *Magnaporthe oryzae* has strengthened the position of rice blast disease as a model to study plant–pathogen interactions in monocotyledons. Genetic studies of blast resistance in rice were established in Japan as early as 1917. Despite such long-term study, examples of cultivars with durable resistance are rare, partly due to our limited knowledge of resistance mechanisms. A rising number of blast resistance genes and quantitative trait loci (QTL) have been genetically described, and some have been characterized during the last 20 years. Using the rice genome sequence, can we now go a step further toward a better understanding of the genetics of blast resistance by combining all these results? Is such knowledge appropriate and sufficient to improve breeding for durable resistance? A review of bibliographic references identified 85 blast resistance genes and approximately 350 QTL, which we mapped on the rice genome. These data provide a useful update on blast resistance genes as well as new insights to help formulate hypotheses about the molecular function of blast QTL, with special emphasis on QTL for partial resistance. All these data are available from the OrygenesDB database.

Additional keywords: colocalization, defense, metaQTL, *Oryza sativa*.

Many different terms have been used to describe resistance. Moreover, the same term can sometimes have different meanings and interpretations. In order to be consistent, and to limit any misinterpretation of our results, we will provide the definitions used in this review to describe different forms of resistance.

i) Complete and partial resistance are defined phenotypically. Pathogen reproduction is fully inhibited for a genotype with complete resistance. A partial-resistance genotype allows limited but significantly reduced pathogen reproduction when

compared with a susceptible genotype (Parleviet and van Ommereen 1975).

ii) A resistance spectrum is defined according to interaction with pathogen strains. A specific resistance is effective against only some strains of the pathogen. Two definitions of broad-spectrum resistance are commonly used (Qu et al. 2006). The first one characterizes a resistance which is effective against different types of pathogens. The second characterizes a resistance which is effective against a large number of, but not all, strains of one pathogen. Because we will focus only on resistance to blast, in this review we will use the second definition of “broad spectrum.” A general resistance is effective against all strains of the pathogen (D’Arcy et al. 2001). Because it is almost impossible to demonstrate that a resistance is general, it can be used as a concept but not as a measurable trait.

iii) Durable resistance is defined sensu Johnson (1981); that is, “resistance that remains effective while a cultivar possessing it is widely cultivated.”

Rice (*Oryza sativa* L.) is the staple food of half of the world’s human population (Khush 2005). Rice blast disease, caused by the fungus *Magnaporthe oryzae* B. Couch is one of the most serious diseases of this crop. Its importance can be measured using several different parameters. First, its geographic distribution is wide; rice blast is present in all rice-growing areas worldwide (CAB International 1981). Second, yield losses can be very large; in upland conditions, complete yield loss is frequently observed, with dramatic impact on populations fed mainly on rice. Third, fungicides are often used to control rice blast and this generates additional costs in rice production. The sales of antiblast fungicides in Japan in 2000 were estimated at 26 billion yen (160 million euro) (Yamaguchi 2004). In China, from 1980 to 1990, the area treated with fungicides to prevent blast increased from 2.4 to 8.9 million ha (Shen and Lin 1994). As a consequence, independently of the rice-growing system or area, breeding for resistance to blast is needed to control the disease despite the availability of effective fungicides.

Rice blast is a well-studied disease and several recent reviews focused on the biology of the fungal infection (Caracul-Rios and Talbot 2007; Ebbole 2007; Veneault-Fourrey and Talbot 2005) and rice resistance (Dai et al. 2007). Moreover, with the completion of the rice (Goff et al. 2002) and *M. oryzae* (Dean et al. 2005) genome sequences, rice blast disease has strength-

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ened its position as a model for plant–pathogen interactions in monocotyledons (Ribot et al. 2007; Valent 1990; Veneault-Fourrey and Talbot 2005). Despite the relatively long history of genetic studies on rice resistance to blast (Takahashi 1965), examples of cultivars with durable resistance are very few (Ou 1985) and resistance breakdown shortly after cultivation of newly released cultivars is the rule. This phenomenon is due, in part, to pathogen evolution toward virulence. Reviewing breeding for resistance to blast, Ou (1985) concludes: “Some cultivars show breakdown in resistance after a few years, and others become susceptible in other geographic areas. The problem arises because of the variability in pathogenicity of the fungus, a factor which in the past has been underestimated by workers in this field.” Different strategies to breed durable resistance have been proposed to counter blast evolution. Some strategies, such as pyramiding (Bonman et al. 1992), lineage exclusion (Zeigler et al. 1994), multilines (Abe 2004), and mixtures (Zhu et al. 2000), are based on the use of complete and specific resistance genes. Others are based on the accumulation of partial resistance (Bonman et al. 1992), a strategy thought to be more durable because it is assumed to be more general. Last, based on the observation that some durably resistant cultivars display complete and partial resistance (Wang et al. 1994), a strategy combining both types of resistance was proposed (Bonman et al. 1992). The lack of durably resistant cultivars is due not only to pathogen evolution toward virulence but also to our limited knowledge of resistance mechanisms. In 1985, Ou stated: “The genetics of resistance are insufficiently known and are often oversimplified”. In the 20 years since then, the molecular function of some blast resistance genes has been described (Dai et al. 2007), and many quantitative trait loci (QTL) for resistance to blast have been mapped.

Since the first publication of a QTL analysis of rice resistance to blast (Wang et al. 1994), many such studies have been published (Table 1). The attraction of QTL research can be explained by the fact that partial resistance implies more general mechanisms and, consequently, is thought to be more durable. But, is this assumption valid? Several studies have shown that partial resistance can be specific (Bonman et al. 1989; Lopez-Gerena 2006; Roumen 1992; Talukder et al. 2004). Moreover, four partial-resistance genes have been identified and have been

described as specific: *Pif* (Yunoki et al. 1970), *pi21* (Fukuoka and Okuno 1997), *Pb1* (Fuji et al. 1995), and *Pi34* (Zenbayashi-Sawata et al. 2002, 2005). An avirulence gene corresponding to *Pi34* (*AvrPi34*) was identified, similar to the case for complete resistance genes (Zenbayashi-Sawata et al. 2005). All these results suggest that partial resistance is sometimes specific and does not necessarily have a broader resistance spectrum than complete resistance. However, is this a general feature of partial resistance? Does this mean that partial resistance is controlled by the same genes as complete resistance? The first cloning of a gene for partial resistance to blast, *pi21*, has recently been achieved. Gene *pi21* encodes a protein with heavy-metal-binding and proline-rich domains but has no similarity to proteins of known function (Fukuoka et al. 2007). Thus, *pi21* does not support the hypothesis that classical resistance genes are responsible for partial resistance.

Despite the example provided by *pi21*, the question that underlies recent publications on partial resistance to rice blast remains: which types of genes are under a QTL? Some publications have demonstrated a relationship between QTL and different types of candidate genes: biotic stress expressed sequence tags (Wang et al. 2001), defense genes (Ramalingam et al. 2003; Wen et al. 2003), and resistance gene analogs (RGA) (Wisser et al. 2005). However, in our opinion, these analyses suffer from two limitations: i) these publications use only a limited subset of the QTL data or candidate genes available and ii) some of these studies considered that all QTL detected governed partial resistance. However, as previously shown for blast (Sallaud et al. 2003), QTL mapping can be used to efficiently detect complete resistance. Thus, using a QTL mapping strategy does not guarantee the identification of partial resistance.

To address these problems, we first attempted to compile an exhaustive list of QTL and then focused our analyses on partial resistance. In particular, we addressed the following two important questions: i) is partial resistance general (nonspecific) and ii) is partial resistance caused by defeated complete resistance genes?

The objectives of this review are to i) extensively review the information available on blast resistance genes and QTL; ii) provide accurate QTL data by a meta-analysis, and analyze the colocalization of the blast gene and QTL with candidate genes, using the rice genome sequence; iii) formulate hypotheses re-

Table 1. Literature sources used in the meta-analysis of quantitative trait loci (QTL) for resistance to rice blast

References	Population	Environment	Mapping ^a	Descendants used ^b	Partial resistance	Total no. of QTL detected ^c
Sirithunya et al. 2002	CT9993-5-10-1-m × KDML105	G and F	Yes	All	Nd ^d	6
Bagali et al. 2000	IR64 × Azucena	G and F	Yes	All	No	13
Fukuoka and Okuno 2001; Okuno and Fukuoka 2002	Nipponbare × Owarihatamochi	F	Yes	All	No	4
Miyamoto et al. 2001, 2003	Kahei × Koshihikari	F	Yes	All	No	2
Sallaud et al. 2003	IR64 × Azucena	G	Yes	All	No	9
Chen et al. 2003	Zhenshan 97 × Minghui 63	G	Yes	All	No	12
Huang et al. 2004	Tainung 69 × Koshihikari	G and F	Yes	All	No	2
Xu et al. 2004	ZYQ8 × JX17	G	Yes	All	No	77
Sato et al. 2006	URN12 × Koshihikari	F	No	All	No	2
Wang et al. 1994	Moroberekan × Co39	G and F	Yes	R, S	Yes	22
Tabien et al. 2002	Lemont × Teqing	G and F	Yes	All	No	11
Zenbayashi et al. 2002.	Norin29 × Chubu32	F	No	S	Yes	1
Loan et al. 2003	Lemont × Teqing	G	Yes	R, S	Yes	14
Talukder et al. 2004, 2005.	Bala × Azucena	G	No	All	Yes	41
Wu et al. 2005	Zhong 156 × Gumei 2	G and F	Yes	R, S	Yes	21
Lopez-Gerena 2006	Oryzica Llanos 5 × Fanny	G	Yes	All	Yes	21
A. Price, <i>unpublished</i>	Bala × Azucena	G	Yes	All	Yes	83
D. Tharreau and J. B. Morel, <i>unpublished</i>	IR64 × Azucena	G	No	All	Yes	2

^a G = greenhouse and F = field.

^b Complete resistance mapping allowed.

^c R, S = resistant and susceptible separately; S = susceptible.

^d Nd = type of resistance not determined.

garding the molecular function of some partial-resistance QTL; and iv) draw conclusions for breeding of durably resistant rice cultivars.

RICE BLAST RESISTANCE GENES AND QTL

A large amount of information is available on rice blast resistance. With a view to comparing partial and complete resistance, we first assembled this information by conducting a systematic survey of the literature.

Complete rice blast resistance genes.

Rice blast disease resistance genes were first described by Sasaki in 1923. An important contribution was then made by Kiyosawa and Ling (1984). In 1995, the Rice Genetic Newsletter (RGN) published a report including a list of 26 blast resistance genes and since then has published the registration of 15 additional genes. Resistance genes are described in databases such as Gramene and Oryzabase. Genetic maps of rice blast resistance genes have been proposed in recent articles (Monosi et al. 2004; Sallaud et al. 2003). However, with the increasing number of newly mapped blast resistance genes, data from all these sources turned out to be incomplete. Starting from the data available in the RGN publication of 1995 and in the Gramene and Oryzabase databases, we updated this list of genes by searching the literature for publications on blast resistance genes. Unpublished data were also provided by colleagues (referenced in Supplementary Table S1).

Despite the fact that they were identified after 1994, 18 genes were named without following the nomenclature of the Committee on Gene Symbolization (Kinoshita et al. 1994). More problematic, in some cases the same gene name was given to different genes, and nine redundant gene symbols are used (Supplementary Table S2). Thus, it is necessary to clarify the identity of these different genes and, possibly, to rename some of them. In the future, the rice blast research community should try to apply the existing nomenclature rules.

We identified a total of 73 complete resistance genes (i.e., twice the number reported in recent publications) (Monosi et al. 2004; Sallaud et al. 2003). As illustrated in Figure 1, we have physically mapped these genes on the 420 Mbp of the genome of Nipponbare, using the position of the flanking markers given in the Gramene database. When the position of a marker was not available, the next flanking marker was used. The physical position of the resistance genes and their references are available from the OryGenesDB database.

As observed previously (Monosi et al. 2004), most of the resistance genes are concentrated in certain genomic regions, particularly on chromosomes 6, 11, and 12. More than 80% of the genes described colocalize on the genome (i.e., are anchored in overlapping intervals) with another mapped resistance gene (Fig. 1). However, this apparent colocalization could be the result of several unrelated processes: the description of the same gene in different studies; the description of alleles of the same gene in different cultivars; the real clustering of resistance genes; or poor mapping resolution, which can generate colocalization by chance. Fifteen of these genes are finely mapped (to less than 500 kb) (Table 2) and the cloning of most of them is in progress. For the others, a more precise description is needed.

As expected, in Nipponbare, 80% of these complete resistance genes colocalize with nucleotide-binding site leucine-rich repeat (NBS-LRR) candidates, the most frequent type of RGA in rice. Eight complete resistance genes to blast have been cloned so far: *Pib* (Wang et al. 1999), *Pita* (Bryan et al. 2000), *Pikh* (Sharma et al. 2005), *Pi9* (Qu et al. 2006), *Pi2/Pizt* (Zhou et al. 2006), *Pid2* (Chen et al. 2006), *Pi36* (Liu et al. 2007), and *Pi37* (Lin et al. 2007a) (Table 3). Apart from *Pid2*, which encodes a serine/threonine-kinase membrane-spanning protein, all these genes belong to the NBS-LRR resistance gene family.

Partial rice blast resistance genes.

Apart from 11 examples of partial resistance that segregated as monogenic traits (Table 4), most of the partial rice blast resistance genes were described in QTL mapping studies. We created a database containing all available information on rice blast resistance QTL. We searched the literature for publications on QTL for resistance to *M. oryzae*. In all, 18 studies were analyzed, including 2 studies published for the first time in this article (Table 1). All the identified QTL were anchored to the rice genome, as described above for resistance genes. The physical position of the resistance genes and QTL and their references are available from the OryGenesDB database.

Altogether, we counted 347 QTL over the 12 chromosomes. Each QTL has a mean physical length of 4.9 Mbp. These QTL are inherited from 28 different parents used in crosses, mostly between one indica and one japonica parent. The position of these QTL is available in Supplementary Table S3.

Because the information generated by different studies was likely to be redundant, but also because further analysis could not be done on such a large number of QTL, and in order to

Table 2. Finely mapped resistance genes (<500 kb)^a

Gene symbol	Chr.	Size (kbp)	Synonyms	Genotype	Candidate genes in Nipponbare	Reference
<i>IPi</i>	12	292.1	<i>Rmg56</i> , <i>IPi(t)*</i>	Nd	1 RPM1-like	Causse et al. 1994
<i>IPi3</i>	12	292.1	<i>Rmg57</i> , <i>IPi3(t)*</i>	Nd	1 RPM1-like	Causse et al. 1994
<i>Pb1</i>	11	349.7	<i>RMg58</i> , <i>Pbst</i>	Modan		Fujii et al. 1995
<i>Pi15</i>	9	44.6	<i>Pi15(t)</i> , <i>Pi14</i> , <i>RMg34</i>	GA25	2 RPM1-like	Pan et al. 2003; Lin et al. 2007b
<i>Pi24</i>	1	313.7	<i>RMg61</i>	Azucena	1 non RD kinase	Sallaud et al. 2003
<i>Pi25(t)</i>	2	263.3	<i>RMg62</i>	IR64		Sallaud et al. 2003
<i>Pi33</i>	8	237.0	<i>Pi33(t)</i> , <i>RMg70</i>	IR64	3 RPM1-like	Berruyer et al. 2003
<i>Pi34</i>	11	67.0	<i>RMg71</i>	Chubu 32	5 RPM1-like and 1 Xa21-like	Zenbayashi et al. 2002
<i>Pi39</i>	12	37.7	...	Q15	1 RPM1-like and 1 Pto-like	Liu et al. 2007
<i>Pi42</i>	9	105.6	...	Nd	2 RPM1-like	Q. Pan, unpublished
<i>Pi5</i>	9	168.6	<i>RMg22</i> , <i>Pi5(t)</i> , <i>Pi3</i> , <i>RMg21</i>	C104PKT	3 RPM1-like	Wang et al. 1994; Jeon et al. 2003
<i>Pigm(t)</i>	6	53.8		Gumei4	4 RPM1-like	Deng et al. 2006
<i>Pik</i>	11	218.0	<i>RMg8</i> , <i>Pik-s</i> , <i>Pik-p</i> , <i>Pik-m</i> (<i>Pi m</i>), <i>Pik-h</i> , <i>Pi k-g</i>	K60	5 RPM1-like	Kiyosawa 1970
<i>Pi-x(t)</i>	2	336.0	...	Nd	1 RPG1-like	Huang, unpublished (Lei et al. 2005)
<i>Pi-y1(t)</i>	2	110.4	...	Yanxian 1	...	Lei et al. 2005
<i>Pi-y2(t)</i>	2	110.4	...	Yanxian 1	...	Lei et al. 2005

^a Chr. = chromosome and Nd = not determined.

Fig. 1. Physical map of resistance genes and meta-quantitative trait loci (QTL) of resistance to rice blast. Chromosome size is shown by the scale on the left (in million base pairs) based on Gramene. The positions and names of markers are indicated by labeled bars on the chromosomes. The positions and names of resistance genes are indicated to the right of each chromosome. MetaQTL are symbolized by filled (partial-resistance metaQTL) or open (other resistance metaQTL) bars. Field QTL and greenhouse QTL are analyzed separately: the letters F, G, and P in QTL names represent field, greenhouse, and partial, respectively. MetaQTL represented by only one study and one repetition are not represented in this figure; these QTL were considered too specific to the population used or to the experiment.

refine their positions, we decided to summarize the information by conducting a meta-analysis. Having QTL from different populations and using different traits to measure resistance, a meta-analysis allows statistical estimation of whether some QTL in fact correspond to one single QTL, and provides an estimation of the position of this metaQTL (Goffinet and Gerber 2000). This technique has been successfully applied, for example, to dissection of the genetic basis of flowering time in maize (Chardon et al. 2004), fiber development in cotton (Rong et al. 2007), and earliness in bread wheat (Hanocq et al. 2007).

The meta-analysis allowed detection of 165 metaQTL, thus reducing significantly the initial dataset of 347 QTL. These data are available in Supplementary Table S4 and on the Ory-

GenesDB database by activating the “All Rice MetaQTL” annotation layer in the genome browser. A graphical overview of the position of these metaQTL is presented in Figure 1. The average size of a metaQTL is 3.3 Mbp. In all, 11% of the metaQTL are finely mapped (<500 kb) and 5% are large (>12 Mbp). On average, a metaQTL is the consensus of 1.9 initial QTL—only 10% come from four QTL or more. Because of their presence in more than four independent studies, these metaQTL can be considered as more robust. They could be found in both indica and japonica rice subgroups (Table 5). Nine of these robust metaQTL present less than two RGA and six do not colocalize with a mapped resistance gene. These metaQTL could be considered for a functional analysis. Most

Fig. 1. Continued from preceding page

proximal microsatellite markers were identified as a first step toward using this information for marker-assisted selection (Table 5).

We then addressed the problem of identifying partial resistance among QTL. As pointed out by Sallaud and associates (2003), QTL mapping can be used to efficiently detect complete resistance to blast. In fact, most QTL studies reveal loci for partial resistance and loci for complete resistance. We assumed that if one of the parents was completely resistant to the strain used, then at least one complete resistance gene will segregate in the mapping population. In such cases, for the identification of partial-resistance QTL, completely resistant descendants should be excluded from the analysis or a detailed phenotype of the resistance of QTL should be available (as in the case of *pi21*) (Fukuoka and Okuno 2001). Because this information is rarely provided in the literature, we decided to consider the QTL detected in such studies as a mixture of partial and complete resistance (Table 1). Similarly, field studies without control of strains are likely to detect both partial and complete resistance QTL. These studies rely on natural inocula, which are mixtures of races of differing virulence spectrum. Such mixtures can mimic partial resistance. Therefore, in our analysis, we decided to separate QTL obtained in the field from QTL obtained in the greenhouse where the rice blast strains used for inoculation were controlled. This approach allowed us to identify a total of 49 metaQTL as partial-resistance metaQTL. For the other metaQTL, the nature (partial or complete) was considered undetermined, either because of

insufficient information or because they are likely to mix both types of resistance.

IS PARTIAL RESISTANCE DIFFERENT FROM COMPLETE RESISTANCE?

To date, all described rice genes for complete resistance to blast are specific. On the contrary, partial resistance is thought to be general. Nevertheless, some publications provide examples of specific partial resistance (Bonman et al. 1989; Fuji et al. 1995; Fukuoka and Okuno 1997; Lopez-Gerena 2006; Roumen 1992; Talukder et al. 2004; Yunoki et al. 1970; Zenbayashi et al. 2002). The only gene for partial resistance to blast cloned so far does not encode a classical NBS-LRR (discussed above). Thus, the functions of genes controlling partial resistance remain unknown. The 49 QTL for partial resistance to blast assembled here provide an opportunity to test, on a relatively large sample, the specificity of these QTL and genes, as well as colocalization with candidate genes.

Is partial resistance specific or general?

Direct comparison of the resistance spectrum of partial-resistance metaQTL with that of complete resistance genes is difficult to realize because resistance spectrum evaluation differs (in particular regarding numbers of blast strains and choice of strains) between the two types of data. Comparison with metaQTL of unknown resistance is also tedious because other metaQTL are likely to be a mixture of different types of resistances. However, if partial resistance is general, then the

Table 3. Summary of information on cloned resistance genes^a

Gene symbol	Chr.	Accession	Avr cloned ^b	Direct interaction	RGA type ^c	Ref. ^d	Length (aa) ^e	Localization	Expression	Locus structure	Alleles	Origin
<i>Pib</i>	2	AB013448	No	ND	NBS-LRR	Wang	1,251	ND	Circadian, inducible by stress	2 genes	3 S mutants	indica
<i>Pita</i>	12	AF207842	Metall.	Yes	NBS-LRR	Bryan	928	Cytoplasm	Constitutive	2 genes	1 aa with S indica, 5 aa with S japonica	indica
<i>Pikh</i>	11	AY914077	No	ND	NBS-LRR	Sharma	330	ND	Inducible, difference R/S	6 genes	1 substitution in the promoter	indica
<i>Pi9</i>	6	ABB88855	No	ND	NBS-LRR	Qu	1,032	ND	Constitutive	9 genes		<i>Oryza minuta</i>
<i>Pi2/Pizt</i>	6	ABC94599/ DQ352040	No	ND	NBS-LRR	Zhou	1,032 to 1,033	ND	Constitutive	9 genes	8 aa between <i>Pi2</i> and <i>Pizt</i>	indica
<i>Pid2</i>	6	...	No	ND	B lectin	Chen	825	Membrane	Constitutive	Simple	1 aa in TM with S	indica
<i>Pi36</i>	8	DQ900896	No	ND	NBS-LRR	Liu	1,056	ND	Constitutive	Simple	1 aa	indica
<i>Pi37</i>	1	DQ923494	No	ND	NBS-LRR	Lin	1,290	Cytoplasm	Constitutive	4 genes	6 aa with S	japonica

^a Chr. = chromosome; ND = type of resistance not determined; and R and S = resistant and susceptible, respectively.

^b Avr = avirulence; Metall. = metalloprotease.

^c RGA = resistance gene analogue; NBS-LRR = nucleotide-binding site leucine-rich repeat; B lectin = B lectin receptor Ser/Thr kinase.

^d References: Wang = Wang et al. 1999, Bryan = Bryan et al. 2000, Sharma = Sharma et al. 2005, Qu = Qu et al. 2006, Zhou = Zhou et al. 2006, Chen = Chen et al. 2006, Liu = Liu et al. 2007, and Lin = Lin et al. 2007a.

^e Length in amino acids (aa).

Table 4. Monogenic resistance genes mapped for partial resistance

Gene symbol	Synonyms	Chromosome	Gramene accession	Genotype	Reference
<i>Pb1</i>	<i>RMg58, Pbst</i>	11	GR:0060596	Modan	Fujii et al. 1995
<i>pi21</i>	<i>rMg40</i>	4	GR:0060650	Owarihatamochi	Fukuoka and Okuno 1997
<i>Pi21</i>	<i>RMg41</i>	12	GR:0060651	Suweon 365	Ahn et al. 1997
<i>Pi22</i>	<i>RMg42</i>	6	GR:0060652	Suweon 365	Ahn et al. 1997
<i>Pi23</i>	<i>RMg43</i>	5	GR:0060653	Suweon 365	Ahn et al. 1997
<i>Pi34</i>	<i>RMg71</i>	11	GR:0061457	Chubu 32	Zenbayashi et al. 2002
<i>Pi35(t)</i>	...	1	...	Hokkai 188	Nguyen et al. 2006
<i>Pif</i>	<i>RMg3</i>	11	GR:0060614	St No.1	Yunoki et al. 1970
<i>Pikur1</i>	<i>RMg9</i>	4	GR:0060620	Kuroka	Shinoda et al. 1971
<i>Pikur2</i>	<i>RMg10</i>	11	GR:0060621	Kuroka	Goto 1988
<i>Pise1</i>	<i>RMg11, Rb1</i>	11	GR:0060622	Sensho	Goto 1970

resistance detected by partial-resistance metaQTL should be effective against most of the races tested. We developed a broad-spectrum resistance (BSR) index. When several strains were tested, no partial-resistance metaQTL was effective against all strains. Only three partial-resistance metaQTL are associated with broad-spectrum resistance (BSR ratio >50%). Thus, the resistance spectrum of metaQTL selected as being associated with partial resistance is not particularly broad.

The first resistance gene for partial resistance to blast to be cloned, *pi21*, is not a classical resistance gene. However, *pi21* is specific. Defense mechanisms following recognition of the pathogen by the plant are likely to be triggered via different signaling pathways (Jones and Dangl 2006). Some of these pathways might be specific to some complete resistance gene families. Thus, if *pi21* is one effector or regulator of one such specific pathway, it will appear to be specific. Further characterization of *pi21* function will allow testing of this hypothesis.

Molecular mechanisms of partial resistance?

It has been proposed that partial and complete resistance could be governed by different types of genes. Several complete resistance genes to blast have been cloned: most of them (7 of 8) are members of the NBS-LRR family (discussed above). Genes controlling partial resistance to blast remain poorly documented. The only quantitative resistance gene cloned, *pi21*, is recessive (Fukuoka and Okuno 2001) and is not a classical RGA (discussed above). It has also been proposed that specific partial resistance could be controlled by defeated complete resistance genes (Talukder et al. 2004). This mechanism was demonstrated for the rice complete resistance gene to bacterial blight *Xa4*. This gene shows residual resistance against virulent strains (Li et al. 1999). An alternative hypothesis could be that partial resistance is governed by defense genes. This hypothesis is supported by recent experiments showing the successful use of defense-gene-derived markers to improve selection for quantitative resistance to blast (Liu et al. 2004; Wu et al. 2004).

If partial resistance is controlled by either defeated complete resistance genes or defense genes, then metaQTL for partial resistance would be expected to colocalize with one of these gene categories. The colocalization of QTL of resistance to rice diseases and genes of the NBS-LRR family has already been attested (Wisser et al. 2005). However, this latter analysis did not differentiate between partial and complete resistance, putatively introducing a bias toward colocalization between QTL and RGA. Colocalization between QTL and defense genes or defense regulators was also investigated (Wen et al. 2003; Wisser et al. 2005) but the number of genes tested was limited. We mapped RGA in the genome of the japonica cv. Nipponbare (Tables 6 and 7). We constructed an RGA map (available on the OryGenesDB database: annotation layer "All RGA"). A map of approximately 4,100 defense genes compiled by Vergne and associates (2008) is also available on the OryGenesDB database (annotation layer "All rice defense genes").

We next compared the localization on the genome of the complete resistance genes mapped, the 49 selected metaQTL for partial resistance, and the other metaQTL with the localization of RGA and defense genes (Tables 6 and 7).

We first tested the colocalization between metaQTL and mapped resistance genes. These results show no significant differences between partial-resistance metaQTL and other metaQTL (Table 6). Despite a tendency of the metaQTL to colocalize with mapped resistance genes (61 and 58-% for partial-resistance metaQTL and other metaQTL, respectively) (Table 7), this differs significantly from the proportion of mapped resistance genes that colocalize with another mapped resistance gene (84%). Colocalization between resistance genes is expected because resistance genes are clustered. Redundancy in the mapped resistance genes, some of which are probably identical or allelic, also contributes to colocalization.

We then tested possible colocalizations with RGA or defense genes. Most of the mapped resistance genes and metaQTL colocalized with an RGA or a defense gene (Table 6). Based on the

Table 5. Broad-spectrum meta-quantitative trait loci (QTL)^a

MetaQTL name	Chr.	Size (Mb)	Closest flanking markers		Stud. ^b	Tested ^c	Recog. ^d	Isolates (%) ^e	Donor cultivars	R genes ^f	RGA ^g	Defense ^h
q11F2	11	3.15	RM26869	RM27030	4	Field	Field	Nd	Chubu 32, Moroberekan, Lemont	6	26	10
q11G5	11	0.51	RM26816	RM26840	5	39	5	13	Moroberekan, Azucena, Oryzica Llanos 5, Teqing, Gumei 2	3	2	1
q11G7	11	2.45	RM27057	RM27222	4	50	6	12	Gumei 2, ZYQ8, Bala, Lemont	5	15	10
q12G8	12	3.63	RM27785	RM27936	5	36	10	28	Oryzica Llanos 5, IR64, Azucena	15	8	19
q12G9	12	1.84	RM28060	RM28127	7	57	14	25	Moroberekan, Azucena, Bala, Oryzica Llanos 5, ZYQ8	9	2	4
q2G4	2	0.71	RM13626	RM1307	4	48	4	8	JX17, Bala, Oryzica Llanos 5, Gumei 2	0	1	12
q3G4	3	2.91	RM3716	RM14760	4	43	4	9	Gumei 2, Bala, ZYQ8, Azucena	0	3	36
q3G5	3	1.67	RM14798	RM14886	4	38	8	21	ZYQ8, Teqing, Fanny, Bala	0	1	20
q5P5	5	8.13	RM18408	RM18882	4	32	7	22	Azucena, Lemont, Moroberekan, Azucena	2	11	76
q6G4	6	1.22	RM19574	RM19665	4	45	6	13	Azucena, Bala, JX17	5	0	18
q6G9	6	0.33	RM2049	RM20508	4	44	7	16	Bala, JX17, Azucena	1	0	2
q7G10	7	0.78	RM22041	RM22068	4	42	6	14	ZYQ8, Bala	0	0	6
q7G2	7	2.92	RM7161	RM21328	4	32	5	16	Teqing, Bala, Azucena	0	1	17
q8G1	8	1.96	RM22453	RM6208	7	53	12	23	ZYQ8, Gumei 2, Oryzica Llanos 5, Bala, Moroberekan, Minghui 63	2	10	18
q8G8	8	0.53	RM23132	RM23170	5	59	10	17	Bala, ZYQ8, Lemont, Fanny	1	1	2
q9G6	9	0.91	RM3533	RM242	4	30	7	23	Zhenshan 97, ZYQ8, Oryzica Llanos 5, Azucena	0	7	9

^a Chr. = chromosome and Nd = type of resistance not determined.

^b Number of independent studies.

^c Number of isolates tested.

^d Number of isolates recognized.

^e Percentage of isolates recognized.

^f Number of colocalizing mapped blast resistance (R) genes.

^g Number of colocalizing resistance gene analogues (RGAs).

^h Number of colocalizing defense genes.

Table 6. Colocalization of resistance genes, meta-quantitative trait loci (QTL), and candidate genes; frequency of mapped genes^a

Mapped ^b	Mean length (Mbp)	Number	Genome (%)	Mapped blast <i>R</i> gene (%)	RGA (%)	Defense gene (%)
Mapped blast <i>R</i> gene	4.4	88	46	84	86	89
Partial metaQTL	4.5	48	47	61	84	98
Other metaQTL	2.9	117	53	58	78	98

^a Frequency of mapped *R* genes or metaQTL that colocalize with at least one other mapped *R* gene or with one or more candidate genes.

^b To test the colocalization of resistance genes or metaQTL with resistance (*R*) gene analogues (RGAs), we considered six RGA types: nucleotide-binding site leucine-rich repeat (NBS-LRR) (e.g., RPM1), LRR-kinase XII (e.g., Xa21), LRR-kinase LRK10L-2 (e.g., RLK10), Waka kinase (e.g., RPG1), Sd2b kinase (e.g., Pid2), and CrRLK1L-1 RD-kinase (e.g., Pto). These different families were established thanks to additional data on non-RD kinase (Dardick and Ronald 2006) and using the tools domain search in the OryGenesDB for the Pfam domain NB-ARC. We also mapped other non-RD and RD kinases for which no proof of implication in resistance exists at present. To test the colocalization of *R* genes or metaQTL with defense genes, we used the map of 4,100 defense genes compiled by Vergne and associates (2008), which is available on the OryGenesDB database.

Table 7. Colocalization of resistance genes, meta-quantitative trait loci (QTL), and candidate genes; frequency of candidate genes^a

Mapped ^b	Number	Mapped blast <i>R</i> gene (%)	Partial metaQTL (%)	Other metaQTL (%)	MetaQTL specific (%)	MetaQTL broad spectrum (%)
RGA	959	59***	42	61***	38	9
Defense gene	4,100	36	45	53	Nd	Nd
Random cDNA	2,000	38	44	53	35	11

^a Frequency of candidate genes located in a position where mapped blast *R* genes, partial-resistance metaQTL, other meta QTL, specific metaQTL, or broad-spectrum metaQTL were mapped. A random set of 2,000 cDNAs was generated and their colocalization with *R* genes and metaQTL was tested. Frequencies of colocalizations with these random cDNA were compared with frequencies of colocalization with RGAs and defense genes using a χ^2 test. *P* values are indicated as follows: *** = $P < 0.001$, ** = $0.001 < P < 0.01$, and * = $0.01 < P < 0.05$. Nd = not determined.

^b To test the colocalization of resistance genes or metaQTL with resistance (*R*) gene analogues (RGAs), we considered six RGA types: nucleotide-binding site leucine-rich repeat (NBS-LRR) (e.g., RPM1), LRR-kinase XII (e.g., Xa21), LRR-kinase LRK10L-2 (e.g., RLK10), Waka kinase (e.g., RPG1), Sd2b kinase (e.g., Pid2), and CrRLK1L-1 RD-kinase (e.g., Pto). These different families were established thanks to additional data on non-RD kinase (Dardick and Ronald 2006) and using the tools domain search in the OryGenesDB for the Pfam domain NB-ARC. We also mapped other non-RD and RD kinases for which no proof of implication in resistance exists at present. To test the colocalization of *R* genes or metaQTL with defense genes, we used the map of 4,100 defense genes compiled by Vergne and associates (2008), which is available on the OryGenesDB database.

number of RGA and the size of the genome, the distribution of RGA can be estimated at approximately 9 RGA per 3.3 Mbp (i.e., the average size of metaQTL we identified). Thus, any QTL or resistance gene mapped within an interval of 3.3 Mb will colocalize with RGA purely by chance. To try to detect significant differences, we compared colocalization of metaQTL and genes with colocalization of a set of 2,000 random positions (with an average size similar to QTL and genes) with candidate genes. As expected, the frequency of RGA was significantly greater at the positions of mapped complete resistance genes than for the random set of genes (Table 7). In contrast, the frequency of RGA is not significantly greater at the positions of partial-resistance metaQTL. These results show significant differences between partial-resistance metaQTL and mapped complete-resistance genes with regards to colocalization with RGA. The frequency of defense genes in the genome intervals defined by mapped resistance genes or by partial-resistance metaQTL is not higher than in other parts of the genome (Table 7). These results do not support the hypotheses that partial resistance to blast is controlled by defeated resistance genes or by classical defense genes. In contrast, like mapped resistance genes, the other metaQTL colocalize with RGA. This result supports the idea that other metaQTL are mainly corresponding to complete resistance genes detected by a QTL method.

CONCLUSION

This review provides an updated overview of the mapped resistance genes and QTL for resistance to rice blast. This information was used to try to clarify relationships between previously described resistance genes and QTL. Extensive and curated analyses of published QTL data do not support the idea that partial resistance is more general than complete resistance. Our analyses confirm previous published results obtained on smaller scales (Bonman et al. 1989; Lopez-Gerena 2006; Roumen 1992; Talukder et al. 2004). The results suggest that differences in pathogenicity can be selected and that partial

resistance is likely to be overcome. Hence, partial resistance could turn out not to be durable. Our analyses do not support either of the following hypotheses: that partial resistance is controlled by defeated complete resistance genes or that it is controlled by classical defense genes. The first resistance gene for partial resistance to blast to be cloned, *pi21*, is neither an RGA nor a classical defense gene. Further characterization of partial resistance is likely to reveal new functions in plant defenses. This will first require a finer mapping of QTL for partial resistance.

No method of simplifying breeding for durable resistance emerges directly from this global analysis. If partial resistance is targeted (alone or in combination), the large number and wide distribution of QTL controlling this trait are major constraints to the maintenance of favorable alleles for this character and to the incorporation of other favorable characters (yield, grain quality, abiotic stress tolerance, and so on). However, the global analysis presented here allows us to identify a limited number (less than 10) of partial-resistance QTL with desirable characteristics: efficiency against most of the strains tested, identification in several independent experiments, and significant impact on resistance. These QTL are, in our opinion, interesting targets for future breeding programs.

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AUTHOR-RECOMMENDED INTERNET RESOURCES

Gramene database: www.gramene.org/
 OrygenesDB database: orygenesdb.cirad.fr
 Oryzabase Integrated Rice Science database:
www.shigen.nig.ac.jp/rice/oryzabase/top/top.jsp