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ORIGINAL PAPER

Fine genetic mapping of *xa24*, a recessive gene for resistance against *Xanthomonas oryzae* pv. *oryzae* in rice

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Abstract Bacterial blight, caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*), is the most devastating plant bacterial disease worldwide. Different bacterial blight resistance (*R*) genes confer race-specific resistance to different strains of *Xoo*. We fine mapped a fully recessive gene, *xa24*, for bacterial blight resistance to a 71-kb DNA fragment in the long arm of rice chromosome 2 using polymerase chain reaction-based molecular markers. The *xa24* gene confers disease resistance at the seedling and adult stages. It mediates resistance to at least the Philippine *Xoo* races 4, 6 and 10 and Chinese *Xoo* strains Zhe173, JL691 and KS-1-21. Sequence analysis of the DNA fragment harboring the dominant (susceptible) allele of *xa24* suggests that this gene should encode a novel protein that is not homologous to any known R proteins. These results will greatly facilitate the isolation and characterization of *xa24*. The markers will be convenient tools for marker-assisted selection of *xa24* in breeding programs.

Introduction

Bacterial blight, caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*), is one of the most economically destructive bacterial diseases of rice worldwide. Fortunately, rice has evolved resistance (*R*) genes to counteract this devastating disease. Utilization of resistant cultivars carrying a single *R* gene is relatively an effective method to control this disease. Pyramid lines incorporating two, three or four bacterial blight resistance genes show even a higher and a broad spectrum of resistance (Huang et al. 1997). About 30 *R* genes for bacterial blight resistance have been identified; these genes confer resistance to different *Xoo* strains (Zhang 2007). Six of these *R* genes, *Xa1*, *Xa3/Xa26*, *xa5*, *xa13*, *Xa21* and *Xa27*, have been isolated (Chu and Wang 2007), and another seven, *Xa4*, *Xa7*, *Xa10*, *Xa22*, *Xa23*, *Xa25* and *Xa29*, have been fine mapped in the molecular linkage map (Sun et al. 2003; Zhang 2007; Gu et al. 2008), which provides transgenic tools or tightly linked markers in marker-assisted selection for rice breeding programs.

Identification, fine mapping and characterization of *R* genes for *Xoo* resistance also provide a special model to study the interaction of host and pathogen. Unlike most intensively studied host–pathogen systems, such as *Arabidopsis–Pseudomonas syringae*, tomato: *Cladosporium fulvum*, and rice: *Magnaporthe grisea*, the rice: *Xoo* system is distinguished from other systems in two aspects. First, about one-third of the *R* genes for *Xoo* resistance are recessive in nature (Zhang 2007). Second, the *R* genes for *Xoo* resistance appear to encode diverse types of proteins. *Xa3/Xa26* and *Xa21* encode leucine-rich repeat (LRR) receptor kinase type proteins (Song et al. 1995; Sun et al. 2004; Xiang et al. 2006), which are the only two characterized plant LRR receptor kinase R proteins that mediate race-specific resistance. *Xa1* encodes a nucleotide binding–LRR protein (Yoshimura et al. 1998). The recessive gene *xa5* encodes the gamma subunit of transcription factor IIA (Iyer and McCouch 2004; Jiang et al. 2006). *Xa27* encodes a novel protein (Gu et al. 2005). The recessive gene *xa13*

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encodes a novel plasma membrane protein (Chu et al. 2006).

A recessive gene conferring resistance to Philippine *Xoo* race 6 (strain PXO99), which is virulent to most *R* genes, was first identified in DV86 by Mir and Khush (1990). This gene was then confirmed to be a new gene and designated as *xa24(t)* (Khush and Angeles 1999). The objective of the investigation reported here was to fine map the recessive gene *xa24(t)* in DV86 to provide tightly linked molecular markers for marker-assisted selection of this gene in breeding programs, and to provide candidates for isolation and characterization of this gene.

Materials and methods

Plant materials

An F_2 population, developed from the cross between a resistant rice line DV86 (*Oryza sativa* ssp. *indica*) carrying the target gene and susceptible line IR24 (*O. sativa* ssp. *indica*), was used to map the bacterial blight resistance gene *xa24(t)*. This population was randomly divided into two groups. The first group, consisting of 300 individuals, was used for genetic mapping of *xa24(t)*. For further fine mapping of the gene, the second group consisting of 4,860 individuals was supplemented to analyze the recombination events that occurred on the either sides of the *xa24(t)* locus. For confirmation of fine mapping results, 20 F_3 plants of each key recombinant F_2 individual were further examined for both the performance to *Xoo* infection and marker genotypes.

Bacterial inoculation and disease evaluation

Parental lines DV86 and IR24 were inoculated with Philippine *Xoo* strains PXO61 (race 1), PXO86 (race 2), PXO79 (race 3), PXO71 (race 4), PXO112 (race 5), PXO99 (race 6), PXO145 (race 7), PXO280 (race 8), PXO339 (race 9) and PXO341 (race 10), and with Chinese *Xoo* strains JL691, Zhe173 and KS-1-21 at the seedling (5- to 6-leaf) or booting stages by the leaf clipping method (Sun et al. 2004). Disease was scored as lesion length (cm) of three to five leaves from each plant at 14–21 days after inoculation.

Genetic analysis

The first group of the F_2 population was genotyped with simple sequence repeat (SSR) markers (http://www.gramene.org; International Rice Genome Sequencing Project 2005). A molecular linkage map was constructed with the computer program MAPMAKER/EXP 3.0 (Lincoln et al. 1992). The maximum-likelihood map order for markers and the chromosomal location of the target gene on the molecular linkage map were determined with a logarithm of odds threshold of 3.0. The genetic distance (centimorgan) between markers for fine mapping of the target gene using 700 highly resistant and susceptible individuals from the second group of the F_2 population was calculated using equation $(N_1 + N_2/2)/N \times 100$, in which N is the total number of the highly resistant and susceptible plants surveyed, N_1 is the number of individuals homozygous for the SSR band from the resistant or susceptible parent, and N_2 is the number of individuals heterozygous for bands from the two parents (Zhang et al. 1994). Chi-square (χ^2) tests were used to evaluate segregation distortion for the target gene in both the F_2 and the $F_{2:3}$ generations.

Sequence analysis

Gene prediction analysis was performed with the programs GENSCAN (http://genes.mit.edu/GENSCAN.html), FGENESH (http://www.softberry.com/berry.phtml) and GeneMark (http://opal.biology.gatech.edu/GeneMark/). The potential identities of predicted coding sequences were determined by BLAST analysis (Altschul et al. 1997).

Results

Resistance of experimental materials to different *Xoo* strains

A total of 13 *Xoo* strains, including Philippine races 1–10 and three Chinese strains, were used for the resistance comparison between resistant variety DV86 carrying *xa24(t)* and susceptible variety IR24, which were used for the construction of F_2 populations (Table 1). DV86 was highly resistant to Philippine strains PXO61, PXO86 and PXO99 at the seedling and booting stages, and highly resistant to Philippine strain PXO71 and Chinese strain JL691 at the booting stage, but moderately resistant to the two *Xoo* strains at the seedling stage. DV86 was also highly resistant to Philippine strain PXO341 and Chinese strains Zhe173 and KS-1-21, at least at the seedling stage (Table 1). IR24 was susceptible to all of the *Xoo* strains tested at the adult and seedling stages (Table 1).

Genetic mapping of *xa24*

The *xa24(t)* was identified and named based on its resistance to Philippine *Xoo* strain PXO99 (Khush and Angeles 1999). PXO99 was used to inoculate the first group of the F_2 population for mapping *xa24(t)* at the booting stage. The distribution of the lesion length caused by PXO99 infection in the 300 individuals was bimodal with an apparent valley at

Table 1 Lesion lengths (cm) of parents of the F_2 population after inoculation with *Xoo*

Strain	Seedling stage		Booting stage	
	DV86	IR24	DV86	IR24
Philippine strains				
PXO61	1.29 ± 0.42	19.23 ± 0.69	1.25 ± 0.23	26.27 ± 0.38
PXO86	1.09 ± 0.26	18.27 ± 1.33	1.47 ± 0.26	25.36 ± 0.66
PXO79	17.2 ± 0.37	19.56 ± 0.57	22.43 ± 0.99	24.8 ± 0.59
PXO71	4.93 ± 0.05	17.23 ± 0.61	1.95 ± 0.23	24.13 ± 0.90
PXO112	15.64 ± 0.45	16.85 ± 0.34	19.8 ± 0.35	20.33 ± 0.52
PXO99	0.94 ± 0.46	17.98 ± 0.28	1.74 ± 0.19	23.00 ± 0.59
PXO145	18.39 ± 0.33	18.84 ± 0.06	21.00 ± 0.43	25.27 ± 0.90
PXO280	10.73 ± 0.39	11.71 ± 0.99	22.55 ± 0.30	24.27 ± 0.50
PXO339	10.52 ± 0.17	11.14 ± 0.17	24.73 ± 0.25	28.2 ± 0.59
PXO341[a]	1.25 ± 0.26	9.88 ± 0.85		
Chinese strains				
JL691	5.27 ± 0.02	16.48 ± 0.33	2.29 ± 0.17	25.27 ± 0.38
Zhe173[a]	1.4 ± 0.40	9.70 ± 1.01		
KS-1-21[a]	1.23 ± 0.17	12.00 ± 0.82		

Each datum represents mean (five replicates) ± standard deviation

[a] Plants generated from rice stubs

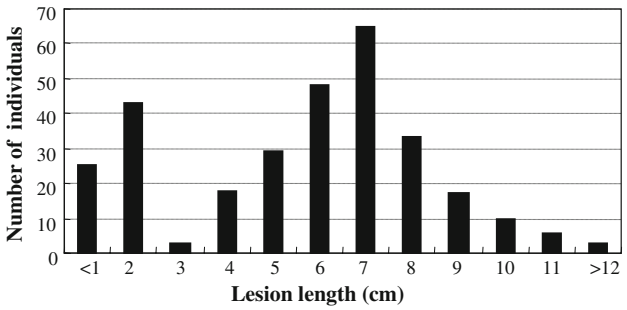

Fig. 1 The distribution of lesion length after PXO99 infection in a sample containing 300 random individuals from the first group of the F_2 population, which was derived from the cross between resistant DV86 and susceptible IR24. The average lesion length of DV86 and IR24 was 0.85 ± 0.02 and 9.69 ± 0.17 cm, respectively

approximately 3 cm (Fig. 1). With a lesion length of 3 cm used as the dividing point, the number of resistant (lesion length < 3 cm) and susceptible (lesion length ≥ 3 cm) F_2 individuals was 71 and 229, respectively, which fits the expected 1:3 ratio ($\chi^2 = 0.28$, $P > 0.5$). The F_1 plants were susceptible to PXO99, with an average lesion length of 21.9 ± 0.81 cm, compared with 1.7 ± 0.19 and 23.0 ± 0.59 cm measured for parents DV86 and IR24, respectively. These results indicate the involvement of a major fully recessive gene for resistance to PXO99 in DV86.

A total of 612 SSR markers were used to screen DV86 and IR24; 182 markers showed polymorphism between DV86 and IR24. From the first group of the F_2 population, 210 individuals were randomly chosen for construction of a molecular linkage map. A total of 122 SSR markers distributed evenly on the 12 chromosomes were mapped to this linkage map. The number of markers distributing on chromosomes 1–12 were 14, 10, 12, 11, 9, 9, 8, 10, 9, 10, 12 and 8, respectively. With the lesion length of the first group of the F_2 population used as the criterion, the *xa24(t)* gene in DV86 was mapped to the terminal region of long arm of chromosome 2, located between SSR markers RM482 and RM138 (Fig. 2a). The gene locus was 8 cM from RM482 on one side and 0.9 cM from RM138 on the other side. Because no major bacterial blight resistance gene has been mapped on rice chromosome 2 (Zhang 2007), the gene against PXO99 in DV86 is a new *R* gene. Thus, we refer to it as *xa24* in the following text.

Fine mapping of *xa24*

The second group of the F_2 population was inoculated with *Xoo* strain PXO99 and disease was evaluated. Markers RM482 and RM138 were first used to screen 400 highly resistant individuals (average lesion length < 1 cm) and 300 highly susceptible individuals (average lesion length > 10 cm) in this group. Eighteen recombinants were identified by RM482 and another two recombinants were identified by RM138 (Table 2). Six additional polymorphic SSR markers, RM14201, RM14206, RM14212, RM14213, RM14222 and RM14226, which are known to be located between RM482 and RM138 (http://www.gramene.org; International Rice Genome Sequencing Project 2005), were further used to examine the 20 recombinant individuals detected by RM482 and RM138. Markers RM14201, RM14206, RM14212, RM14213 and RM14222 detected 12, 9, 9, 4 and 1 recombination events, respectively, in the 18 plants identified by RM482 and co-segregated with the

Table 2 Marker genotypes of 20 recombinant individuals from highly resistant and susceptible F_2 individuals ($n = 700$) and their reactions to the *Xoo* strain PXO99

Individuals	Phenotype	Marker genotype							
		RM482	RM14201	RM14206	RM14212	RM14213	RM14222	RM14226	RM138
D11-3	Susceptible	R	R	R	R	H	H	H	H
L15-4	Susceptible	R	R	R	R	H	H	H	H
A6-14	Resistant	H	H	H	H	H	H	R	R
B1-12	Resistant	H	H	H	H	H	R	R	R
B17-9	Resistant	H	H	H	H	H	R	R	R
C8-19	Resistant	H	H	H	H	H	R	R	R
D3-10	Resistant	H	H	H	H	R	R	R	R
E2-9	Resistant	H	H	H	H	R	R	R	R
E12-4	Resistant	H	H	H	H	R	R	R	R
F9-3	Resistant	H	H	R	R	R	R	R	R
F17-3	Resistant	H	H	R	R	R	R	R	R
G12-5	Resistant	H	H	R	R	R	R	R	R
H5-11	Resistant	H	R	R	R	R	R	R	R
H19-2	Resistant	H	R	R	R	R	R	R	R
I4-9	Resistant	H	R	R	R	R	R	R	R
J13-1	Resistant	H	R	R	R	R	R	R	R
J16-14	Resistant	H	R	R	R	R	R	R	R
K15-17	Resistant	H	R	R	R	R	R	R	R
L20-11	Resistant	R	R	R	R	R	R	R	H
L1-9	Resistant	R	R	R	R	R	R	H	H

R and H stand for the homozygous resistant and heterozygous genotypes, respectively, of the F_2 recombinants from the cross between resistant DV86 and susceptible IR24

two plants identified by RM138 (Table 2). Marker RM14226 detected 1 recombination event in the 2 plants identified by RM138 and co-segregated with the 18 plants identified by RM482 (Table 2). Thus, *xa24* was mapped between RM14222 and RM14226. The locus was 0.07 cM from both markers (Fig. 2b).

As confirmation of the above results, the phenotypes and genotypes of four recombinant F_2 individuals (D11-3, L15-4, A6-14 and L1-9) (Table 2), which define the fine location of *xa24* locus, were further verified by examining the lesion lengths and marker genotypes of their F_3 progenies (20 F_3 plants from each of the F_2 individuals). After inoculation with the *Xoo* strain PXO99, no phenotypic segregation was observed within the F_3 plants from the resistant F_2 individuals A6–14 and L1–9 (Supplemental Table 1). The genotypes of the F_3 plants from A6–14 segregated in the expected ratio of 1:2:1 (homozygous for the resistant DV86 allele, the heterozygous, and homozygous for the susceptible IR24 allele, respectively) for locus RM482 ($\chi^2 = 0.3$, $P > 0.5$) and did not show any segregation for locus RM138. The genotypes of the F_3 plants from L1–9 segregated in the expected ratio of 1:2:1 (homozygous for the resistant DV86 allele, the heterozygous, and homozygous for the susceptible IR24 allele, respectively) for RM138 ($\chi^2 = 1.2$, $P > 0.25$) and did not show any segregation for RM482 (Supplemental Table 1). Also, plants in each F_3 family from the two susceptible F_2 individuals, D11-3 and L15-4, segregated into susceptible and resistant plants (Supplemental Table 1). F_3 plants from D11-3 ($\chi^2 = 0.3$, $P > 0.5$) and L15-4 ($\chi^2 = 0.3$, $P > 0.5$) also co-segregated at RM14226 and RM138 loci with the expected ratio of 1:2:1 (homozygous for the resistant DV86 allele, the heterozygote, and homozygous for the susceptible IR24 allele, respectively). These results further confirm that the F_2 individuals used for the fine physical mapping of *xa24* (t) were reliable.

Based on BLAST analysis, the end nucleotide positions of markers RM14222 and RM14226, which flank *xa24* locus, are 35729170 and 35800135, respectively. Thus, the genomic region containing *xa24* locus is narrowed to a fragment approximately 71 kb in length.

Resistance spectrum of *xa24*

For determination of the resistance spectrum of plants as conferred by *xa24*, 500 random F_2 plants from the second group were cut at about 12 cm above ground level after

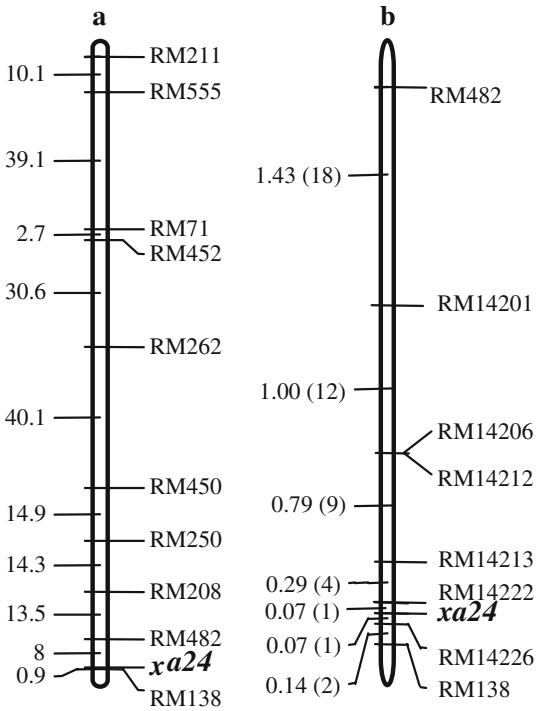

Fig. 2 The location of bacterial blight resistance gene *xa24* on the molecular linkage map of chromosome 2. The numbers between molecular markers indicate the genetic distances in centimorgans, and the numbers in parentheses indicate recombination events detected between the *xa24* locus and the corresponding markers. **a** Mapping of *xa24* based on the 210 individuals of the first group of the F_2 population. **b** Fine mapping of *xa24* based on the 700 individuals of the second group of the F_2 population

their performance to PXO99 infection had been evaluated. The ratoons (at 5-leaf stage) generated from each 100 stubs were inoculated with PXO71, PXO341, Zhe173, JL691 and KS-1-21, respectively (Fig. 3). As controls, seedlings generated from another 100 random stubs were inoculated with PXO99. The lesion lengths of five random resistant and susceptible plants from the first round of infection caused by PXO99 were compared with the lesion lengths from the second round of infection (Supplemental Table 2). In light of the performance of the controls, cutting the plants did not influence their response to *Xoo* infection. The comparison showed that, if a plant was resistant or susceptible to PXO99, it was also resistant or susceptible to PXO71, PXO341, Zhe173, JL691 or KS-1-21 in the second round of inoculation (Supplemental Table 2).

The distributions of the lesion length caused by PXO99, PXO71, PXO341, Zhe173, JL691 and KS-1-21 in each group of 100 regenerated seedlings were bimodal with an apparent valley at approximately 3–4 cm (Fig. 3). With a lesion length of 3 cm used as the dividing point for the groups inoculated with PXO99, PXO71 and PXO341, the number of resistant (lesion length < 3 cm) plants was 22, 21 and 21, respectively, and the number of susceptible (lesion length ≥ 3 cm) plants was 78, 79 and 79, respectively. With a lesion length of 4 cm used as the dividing point for the groups inoculated with Zhe173, JL691 and KS-1-21, the number of resistant (lesion length < 4 cm) plants was 30, 32 and 33, respectively, and the number of susceptible (lesion length ≥ 4 cm) was 70, 68 and 67, respectively. The distributions of lesion length caused by PXO99, PXO71, PXO341, Zhe173, JL691 and KS-1-21 all fit the expected 1:3 ratio ($\chi^2 = 0.85$, $P > 0.25$; $\chi^2 = 0.48$, $P > 0.25$; $\chi^2 = 0.85$, $P > 0.25$; $\chi^2 = 1.33$, $P > 0.1$; $\chi^2 = 2.61$, $P > 0.1$; $\chi^2 = 3.41$, $P > 0.05$). These results suggest that the resistance of DV86 to PXO99, PXO71, PXO341, Zhe173, JL691 and KS-1-21 was conferred by a single recessive gene, which is *xa24*.

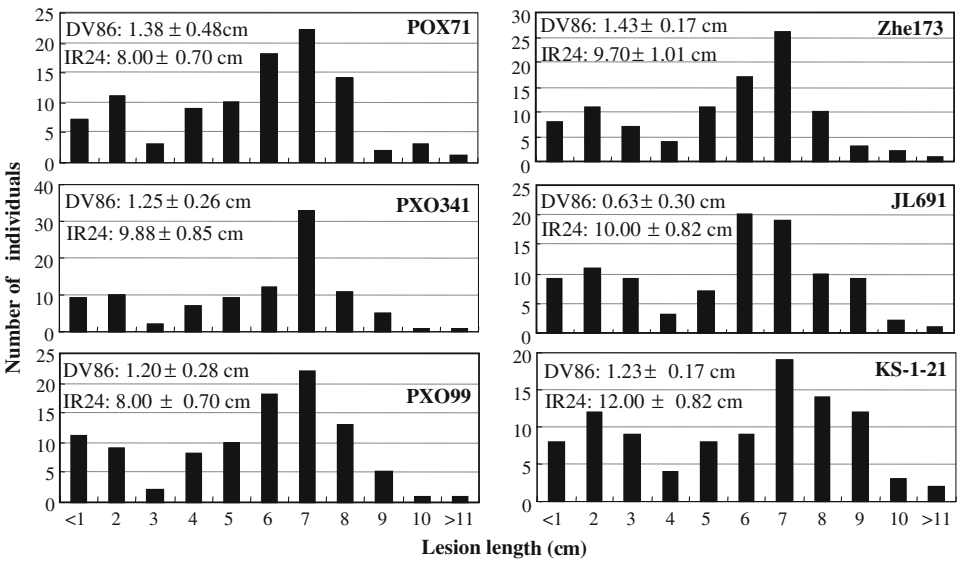

Fig. 3 The distribution of lesion lengths after PXO71, PXO341, PXO99, Zhe173, JL691 or KS-1-21 infection in a sample containing 100 random individuals from the second group of the F_2 population, which was derived from the cross between resistant DV86 and susceptible IR24. The average lesion lengths of DV86 and IR24 are indicated after infection with each *Xoo* strain

The candidates of *xa24*

On the basis of the annotation of the Institute for Genomic Research (TIGR, http://rice.tigr.org), analysis of the 71-kb sequence harboring the *xa24* locus indicated that this region contains 16 genes. Thirteen of the 16 genes have full-length cDNA support. No cDNA or expressed sequence tag has been identified in GenBank for the other three genes. The putative encoding products of 13 of the 16 genes contain a known domain or motif, and the encoding products of the other three genes provide no clue as to the proteins' mode of action.

Discussion

Our results suggest that *xa24* confers whole-growth-stage resistance to bacterial blight. This gene mediates resistance to at least Philippine *Xoo* races 4, 6 and 10, and Chinese *Xoo* strains Zhe173, JL691 and KS-1-21. The representative strain PXO99 of Philippine *Xoo* race 6 is virulent to most of the identified *R* genes. Chinese *Xoo* strain Zhe173 is a prevalent race in the major rice-growing areas of China. Bacterial blight resistance gene *Xa21* has been widely used for rice breeding programs (Chen et al. 2000, 2001; Chen and Zhang 2000; Tu et al. 2000; Zhai et al. 2002). However, the activity of *Xa21* is developmentally controlled; *Xa21*-mediated resistance increases progressively from the susceptible juvenile stage to full resistance at the later adult stage (Century et al. 1999). Furthermore, Philippine *Xoo* race 10 is virulent to plants carrying *Xa21*. Thus, *xa24* is promising for rice breeding programs. The fine mapping of *xa24* using polymerase chain reaction-based molecular markers provide a convenient tool for marker-assisted selection of *xa24* in breeding programs.

Another accomplishment of this study is fine mapping of *xa24* to a region of 71 kb. This result will greatly facilitate the isolation and characterization of *xa24*. Race-specific recessive resistance is seldom observed in bacterial and fungal systems except of *Xoo* system (Iyer-Pascuzzi and McCouch 2007). However, the molecular mechanisms of the recessive interaction between rice and *Xoo* are largely unknown, because only two recessive *R* genes, *xa5* and *xa13*, for *Xoo* resistance have been characterized (Iyer and McCouch 2004; Chu et al. 2006). The *xa5*-mediated resistance is due to two nucleotide substitutions resulting in an amino acid change of the gamma subunit of transcription factor IIA, compared with its dominant (susceptible) allele (Iyer and McCouch 2004). Three different models for xa5 function have been suggested and they have one point in common that the function of either dominant XA5 or recessive xa5 is influenced by the direct binding of bacterial effector (Nino-Liu et al. 2006; Iyer-Pascuzzi and McCouch 2007). A recent report indicates that *xa5*-mediated recessive resistance is the result of restricted bacterial movement, but not restricted multiplication (Iyer-Pascuzzi et al. 2008). The *xa13* and its dominant allele can encode identical protein, a plasma membrane protein with unknown mode of action; promoter mutations in the susceptible allele cause down-regulation of expression during rice–*Xoo* interaction, resulting in the fully recessive *xa13* (Chu et al. 2006). The dominant *Xa13* is required for bacterial growth. *Xa13* expression is induced by bacterial infection. A region of 18 nucleotides corresponding to the -69 to -86 region of *Xa13* promoter may be responsible for this induction (Chu et al. 2006). A type III effector gene of *Xoo* may be involved in this regulation (Yang et al. 2006). Analysis of the 71-kb DNA sequence harboring *xa24* locus or its dominant allele *Xa24* revealed 16 predicted genes. None of the 16 genes encode proteins similar to any of the characterized *R* gene that mediates race-specific resistance. Thus, *xa24* may be a novel *R* gene that confers a new type of plant disease resistance. Characterization of *xa24* will provide another model to understand the molecular mechanisms of recessive resistance in rice-*Xoo* system.

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