***Rmg8*, a New Gene for Resistance to *Triticum* Isolates of *Pyricularia oryzae* in Hexaploid Wheat**

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ABSTRACT

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Blast, caused by *Pyricularia oryzae*, is one of the major diseases of wheat in South America. We identified a new gene for resistance to *Triticum* isolates of *P. oryzae* in common wheat ‘S-615’, and designated it “resistance to *Magnaporthe grisea* 8” (*Rmg8*). *Rmg8* was assigned to

chromosome 2B through molecular mapping with simple-sequence repeat markers. To identify an avirulence gene corresponding to *Rmg8*, *Triticum* isolate Br48 (avirulent on S-615) was crossed with 200R29 (virulent on S-615), an F₁ progeny derived from a cross between an *Eleusine* isolate (MZ5-1-6) and Br48. Segregation analysis of their progeny revealed that avirulence of Br48 on S-615 was conditioned by a single gene, which was designated AVR-*Rmg8*. AVR-*Rmg8* was closely linked to AVR-*Rmg7*, which corresponded to *Rmg7* located on chromosome 2A of tetraploid wheat.

Development of resistant cultivars carrying resistance (*R*) genes is the most effective and economical way to control disease. Historically, however, many *R* genes have been rendered ineffective only a few years after their use in farmers’ fields, resulting in a “breakdown of resistance”, because of the high pathogenic variability of pathogens (Kiyosawa 1982; Leach et al. 2001). To prevent such breakdowns, cultivars with durable *R* genes are required.

Pyricularia oryzae, a blast fungus of gramineous plants, is composed of several genus-specific subgroups or pathotypes (Kato et al. 2000), such as the *Oryzae* isolates pathogenic on rice (*Oryza sativa*), *Setaria* isolates pathogenic on foxtail millet (*Setaria italica*), *Eleusine* isolates pathogenic on finger millet, and *Triticum* isolates pathogenic on wheat (*Triticum aestivum*). Wheat blast caused by *Triticum* isolates (Urashima et al. 1993) significantly reduces wheat yields and grain quality; yield losses reach to 70 to 100% when favorable climatic conditions occur at the critical developmental stages for infection (Cruz et al. 2012; Kohli et al. 2011). The first epidemic of this disease occurred in Paraná State, southern Brazil in 1985 (Igarashi et al. 1986). Since then, it has become widely distributed across most of the wheat-producing areas in Brazil, Argentina, Paraguay, and Bolivia (Cruz et al. 2012; Kohli et al. 2011) because of a lack of resistant cultivars and effective fungicides (Maciel et al. 2014). Recently, wheat blast was also found in North America. In May 2011, *P. oryzae* was isolated from a single severely blasted wheat head at a research plot at the University of Kentucky Research and Education Center in Princeton. This infection was possibly caused by a host jump from an annual ryegrass pathogen (Pratt 2012). Although no serious outbreaks have been reported outside of South America, there is an increasing threat of wheat blast epidemics in other parts of the world (Kohli et al. 2011).

To date, seven blast *R* genes have been identified in wheat. The first *R* gene, designated *Rmg1* (*Rwt4*), was effective against an *Avena* isolate but not against *Triticum* isolates (Takabayashi et al. 2002). *Rmg4* and *Rmg5* conditioned resistance to a *Digitaria* isolate but not to *Triticum* isolates (Nga et al. 2009). *Rmg6* was effective against a *Lolium* isolate but not against *Triticum* isolates (Vy et al. 2014). These four *R* genes governed resistance to inappropriate subgroups. On the other hand, the other three genes were effective against isolates of an appropriate subgroup (*Triticum* isolates), including *Rmg2* and *Rmg3*, identified in common wheat ‘Thatcher’ (Zhan et al. 2008), and *Rmg7*, identified in tetraploid accessions, St24, St17, and St25 (Tagle et al. 2015). These genes may be useful for wheat breeding but have not yet been mapped.

In the present study, we identified a novel gene for resistance to *Triticum* isolate Br48 on chromosome 2B of hexaploid wheat ‘S-615’ (S615) and designated it “resistance to *Magnaporthe grisea* 8” (*Rmg8*). We also identified its corresponding avirulence (AVR) gene by using progeny derived from a cross between Br48 and laboratory strain 200R29.

MATERIALS AND METHODS

Fungal materials. Isolates used for a preliminary screening were 14 *Triticum* isolates collected in Brazil in 1990 to 1992 (Supplementary Table S1). After screening, a representative *Triticum* isolate, Br48, was used for segregation analyses of wheat F₂ and F₃ populations. To identify an AVR gene, Br48 (*MAT1-1*) was crossed with 200R29 (*MAT1-2*), a highly virulent F₁ culture derived from a cross between *Eleusine* isolate MZ5-1-6 and Br48 (Tagle et al. 2015), as described previously (Murakami et al. 2000). A random hybrid population was produced by sampling one ascospore from each ascus. These cultures were maintained on barley seed media, as described previously (Hirata et al. 2007).

Plant materials. Plant materials used for the preliminary screening were 31 cultivars of common wheat (*T. aestivum* L.) provided by Dr. K. Tsunewaki, formerly of Kyoto University, and Dr. U. Hiura, formerly of Okayama University. After the screening, three representative cultivars were selected for further analyses.

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Common wheat S615 was resistant to Br48, whereas ‘Shin-chunaga’ (Sch) and ‘Hope’ were susceptible (Table 1). Tetraploid accession *T. dicoccum* KU-120 (St24) was employed for a comparison of *R* genes because it carried *Rmg7* (Tagle et al. 2015). Wheat seed were pregerminated by incubation on moistened filter papers at 22°C for 24 h, sown in vermiculite supplied with liquid fertilizer in a seedling case (5.5 by 15 by 10 cm), and grown at 22°C in a controlled-environment room with a 12-h photoperiod of fluorescent lighting for 7 days. Leaves were then fixed onto a hard plastic board for inoculation.

Infection assays. Inocula were prepared following the protocol described by Tagle et al. (2015). The inoculum suspension (1 × 10⁵ conidia/ml) was sprayed onto adaxial surfaces of the 7-day-old primary leaves using an air compressor. The inoculated plants were incubated for 24 h in darkness in humid trays at 22 or 25°C, after which they were transferred to dry conditions under fluorescent lighting and further incubated at the same temperature. Disease symptoms were evaluated 4 or 5 days after inoculation based on the color of lesions and the affected leaf area. The affected area was rated by six progressive grades from 0 to 5, where 0 = no visible evidence of infection, 1 = pinhead spots, 2 = small lesions (<1.5 mm), 3 = scattered lesions of intermediate size (<3 mm), 4 = large typical lesions, and 5 = complete blighting of leaf blades. A disease score is composed of a number denoting the size of lesions and a letter or letters indicating the lesion color: “B” for brown and “G” for green. Infection types with brown lesions were considered to be resistant or avirulent while those with infection types 3G, 4G, and 5G were taken as susceptible or virulent. Sometimes lesions of size 3 and 4 were accompanied by both brown and green tissue. These types, designed as 3BG and 4BG, were considered moderately resistant or moderately avirulent (Hyon et al. 2012). All infection assays at the seedling stage were repeated at least three times.

For infection assays of wheat plants at the heading stage, stems with spikes at the stage of full head emergence were cut from wheat

plants that had been sown in an experimental field in the autumn of the previous year, and put in flasks with water. Spikes of the susceptible cultivar (Sch) could not be harvested simultaneously with the resistant cultivar (S615) because of different heading times; Sch was early heading while S615 was late heading. Therefore, late-heading Hope was used as a susceptible control for assays at the heading stage. Harvested spikes were inoculated with conidial suspensions (1 × 10⁵ spores/ml), covered with a plastic bag, and placed in darkness at 25°C for 24 h. The plastic bag was removed and the set-up was incubated further at the same temperature with 12 h each of alternate light and dark conditions. Five days after inoculation, spike infections were rated with six progressive grades from 0 to 5, where 0 = no visible evidence of infection, 1 = pinhead spots, 2 = small lesions (<1.5 mm), 3 = scattered lesions of intermediate size (<3 mm), 4 = mixture of green and white tissues with no apparent browning caused by hypersensitive reaction, and 5 = complete blighting of the spike. Infection types 0 to 3 were considered resistant and 4 to 5 were susceptible. Three spikes were used for each wheat cultivar per replication. All infection assays at the heading stage were repeated twice.

Molecular mapping. To map *Rmg8* and *Rmg7*, 93 F₂ plants derived from a cross between S615 and Sch and 94 F₂ plant derived from a cross between St24 and Tat14 (*T. paleocolchicum* KU-156, highly susceptible to Br48) (Tagle et al. 2015) were grown in a field and selfed. From each of the resulting F₃ lines, 20 seeds were used for disease assays with Br48. Another 20 seeds were grown at 22°C for 5 to 7 days, and pooled leaf tissue samples were used for DNA extraction.

Based on phenotypic data, five homozygous resistant (*RR*) and five homozygous susceptible (*rr*) F₃ lines were selected for bulked segregant analysis (Michelmore et al. 1991). Resistant and susceptible bulks (10 ng/μl) were constructed by mixing equal amounts of DNA of the five *RR* and five *rr* F₃ lines, respectively. Simple-sequence repeat (SSR) loci reported by Somers et al. (2004) and Torada et al. (2006) were amplified from DNA of the parental cultivars and the two bulks. Polymerase chain reaction (PCR) products were separated in 2% agarose or in 13% nondenaturing polyacrylamide gels using a high-efficiency genome scanning system (Nippon Eido, Tokyo) (Hori et al. 2003). Markers showing a possibility of linkage were then subjected to analyses on the F₃ lines to construct a linkage map. A genetic map was constructed using software MAPMARKER (Lander et al. 1987). Thresholds of linkage for map construction were a minimum log-of-likelihood ratio of 4.0 and a maximum recombination fraction of 0.4. The Kosambi mapping function was employed to compute map distances in centimorgans.

Fig. 1. Symptoms on **A**, seedlings and **B**, spikes of resistant ‘S615’ and susceptible ‘Shin-chunaga’ (Sch) or ‘Hope’, 5 days after inoculation with *Triticum* isolate Br48 at 25°C.

TABLE 1. Infection types of wheat seedlings with *Triticum* isolate Br48, *Eleusine* isolate MZ5-1-6, and F₁ culture 200R29 derived from the cross MZ5-1-6 × Br48

Wheat line	Lab code	Infection type with ^a					
		Br48		200R29		MZ5-1-6	
		22°C	25°C	22°C	25°C	22°C	25°C
<i>Triticum aestivum</i>							
‘S-615’	S615	0–1B*	1–2B*	3BG*	5G	1B*	2B*
<i>T. aestivum</i>							
‘Shin-chunaga’	Sch	5G	5G	3–4G	5G	1B*	1B*
<i>T. aestivum</i>							
‘Hope’	Hope	5G	5G	5G	5G	0–1B*	0*
<i>T. dicoccum</i>							
KU-120	St24	0*	1–2B*	1–2B*	5G	1–2B*	5G

^a Evaluated 5 days after inoculation, where 0 = no visible evidence of infection, 1 = pinhead spots, 2 = small lesions (<1.5 mm), 3 = scattered lesions of intermediate size (<3 mm), 4 = large typical lesions, and 5 = complete blighting of leaf blades; B and G indicate brown and green lesions, respectively. Asterisks indicate resistant reactions.

RESULTS

Infection assays at the seedling stage. Screening of the common wheat cultivars revealed that S615 was resistant to all *Triticum* isolates at both 22 and 25°C while Sch and Hope were susceptible (Table 1; Fig. 1A). Consequently, we selected these three cultivars for further analyses. When an F₂ population derived from a cross between S615 and Sch was inoculated with Br48, resistant and susceptible seedlings segregated (Fig. 2) in a 3:1 ratio (164:42, $\chi^2 = 2.34$). In F₃ populations, nonsegregating resistant, segregating, and nonsegregating susceptible lines were distributed in a 1:2:1 ratio (21:52:20, $\chi^2 = 1.32$), indicating that the resistance of S615 to Br48 was controlled by a single dominant gene. This gene was designated *Rmg8*.

Infection assays at the heading stage. To check whether the resistance of S615 is expressed at the heading stage, its spikes were inoculated with Br48 together with those of the susceptible control Hope and incubated at 25°C. The spikes of S615 showed infection type 2, with small brown lesions caused by a hypersensitive reaction (Fig. 1B), as expected. By contrast, the spikes of Hope were completely blighted with infection type 5 (Fig. 1B).

Identification of an AVR gene in a *Triticum* isolate. In order to identify an AVR gene corresponding to *Rmg8*, an isolate virulent on the *Rmg8* carrier is required as a mating partner of Br48. However, S615 was resistant to all *Triticum* isolates tested. To find a mating partner, S615 was inoculated with isolates from other

Fig. 2. Frequency distribution of infection types with *Triticum* isolate Br48 at 22°C in F₂ seedlings derived from the cross between S615 and Sch. Infection type 0 = no visible evidence of infection, 1 = pinhead spots, 2 = small lesions (<1.5 mm), 3 = scattered lesions of intermediate size (<3 mm), 4 = large typical lesions, and 5 = complete blighting of leaf blades; B and G indicate brown and green lesions, respectively.

host-specific subgroups (e.g., *Eleusine* isolates and *Setaria* isolates) and their descendants, and incubated at 22 and 25°C. Several virulent cultures were found in an F₁ population derived from MZ5-1-6 (*Eleusine* isolate) × Br48. Their mating types were checked using *MAT1-1*- and *MAT1-2*-specific primers (Zheng et al. 2008). Finally, 200R29 was selected as the mating partner because it carried the *MAT1-2* allele and was highly virulent on S615 at 25°C (Table 1). Br48 (*MAT1-1*) was crossed with 200R29, resulting in 73 random hybrid cultures. When they were sprayed on S615 and incubated at 25°C, 31 and 40 cultures were stably avirulent and virulent, respectively (Table 2). There were two exceptional cultures whose infection types were unstable (i.e., fluctuated among replications). Regardless of their categorization, however, segregation of avirulent and virulent cultures fitted a 1:1 ratio (Table 2), suggesting that a single gene is involved in the avirulence of Br48 on S615. This AVR gene was designated *AVR-Rmg8*.

When the 73 cultures were sprayed onto St24 carrying *Rmg7*, avirulent and virulent cultures segregated in a 1:1 ratio (Table 2). This gene was considered to be *AVR-Rmg7*, corresponding to *Rmg7* (Tagle et al. 2015). To check whether *AVR-Rmg8* and *AVR-Rmg7* are located at the same locus, the segregation patterns on S615 and St24 were compared. When the unstable two cultures were classified as “virulent”, the segregation patterns on S615 and St24 were perfectly concordant (Table 3). When these two cultures were classified as “avirulent”, however, they were regarded as recombinants (Table 3).

Molecular mapping of *Rmg8*. To determine the chromosomal position of *Rmg8*, bulked-segregant analysis was performed with the F₃ lines derived from a cross between S615 and Sch. Of 725 SSR markers tested, 290 were polymorphic between the parental accessions. Twelve polymorphic markers located on chromosome 2B appeared to be associated with *Rmg8* in bulked segregant analysis. Genotyping of all F₃ lines indicated that *Rmg8* was flanked by SSR markers *Xwmc317* and *Xbarc159* (Fig. 3), assigned to the distal region in the long arm of chromosome 2B (Somers et al. 2004).

To determine the chromosomal position of *Rmg7*, 94 F₃ lines derived from a cross between St24 (*Rmg7*) and Tat14 (*rmg7*) were employed. When they were inoculated with Br48, nonsegregating resistant, segregating, and nonsegregating susceptible lines were distributed in a 1:2:1 ratio (26:45:23, $\chi^2 = 0.36$). Molecular mapping with these F₃ lines indicated that *Rmg7* was flanked by SSR markers *Xcfd50* and *Xhbg327* (Fig. 3), assigned to the distal region in the long arm of chromosome 2A (Somers et al. 2004).

TABLE 3. Combined segregation of virulence on two wheat lines in a population derived from the cross Br48 × 200R29

Categorization of unstable cultures	Number of cultures ^a				Total
	A _{S615} A _{St24} ^a	A _{S615} V _{St24}	V _{S615} A _{St24}	V _{S615} V _{St24}	
Avirulent	31	2	0	40	73
Virulent	31	0	0	42	73

^a A = avirulent and V = virulent. For example, A_{S615}V_{St24} represents cultures that are avirulent on S615 and virulent on St24.

TABLE 2. Responses of wheat seedlings to a population derived from the cross, Br48 × 200R29

Wheat lines	Number of cultures ^a							Subtotal	A	U	V	Total	χ^2 (1:1)
	0/1B	2B	3B/3BG	4B/4BG/5B	3G	4G	5G						
S615	9	16	6	0	0	0	40	71	31	2	40	73	0.67 ([A+U]:V) ^b
St24	...	...	...	...	...	...	...	...	...	...	...	...	1.66 (A:[U+V]) ^b
Sch	14	11	6	0	0	0	42	73	31	0	42	73	1.66 (A:V) ^b
Sch	0	0	0	0	0	5	68	73	0	0	73	73	...

^a Infection type, 5 days after inoculation at 25°C, where 0 = no visible evidence of infection, 1 = pinhead spots, 2 = small lesions (<1.5 mm), 3 = scattered lesions of intermediate size (<3 mm), 4 = large typical lesions, and 5 = complete blighting of leaf blades; B and G indicate brown and green lesions, respectively; A = avirulent (infection type 0 to 3BG), U = unstable (fluctuate from 4B/4BG to 4G), and V = virulent (infection type 3G to 5G).

^b Not significant at $P = 0.05$.

DISCUSSION

In the present study, we identified *Rmg8*, a dominant gene conferring strong resistance to *Triticum* isolate Br48 in common wheat S615. This gene was located on the long arm of chromosome 2B (Fig. 3). *Rmg8* should be different from *Rmg2* and *Rmg3*, two of the three *R* genes previously identified as effective against *Triticum* isolates, because *Rmg2* and *Rmg3* are located on chromosomes 7A and 6B, respectively (Zhan et al. 2008). *Rmg8* also should be different from the other (third) *R* gene, *Rmg7*, which was identified in a tetraploid wheat accession (St24) by Tagle et al. (2015), because *Rmg7* is located on chromosome 2A (Fig. 3).

Segregation analysis of a fungal hybrid population suggested that their corresponding AVR genes, *AVR-Rmg7* and *AVR-Rmg8*, are closely linked or located at the same locus (Table 3). If these AVR genes are the same, this means that a single AVR gene corresponds to *Rmg7* in tetraploid wheat and *Rmg8* in hexaploid wheat. It should be noted that *Rmg8* and *Rmg7* are located in the distal regions of long arms of homeologous chromosomes 2B and 2A, respectively (Fig. 3). There is a possibility that *Rmg8* in hexaploid wheat and *Rmg7* in tetraploid wheat are homeologous genes. To verify the homeologous relationship between *Rmg8* and *Rmg7*, fine mapping of these genes should be performed in further studies.

R genes to be used in breeding for resistance to wheat blast must fulfill some requirements. First, they must be expressed at the heading stage because wheat blast in the field is primarily a spike disease. Variation in blast response among wheat cultivars has been recognized at both the seedling and adult plant stages (Cruz et al. 2012; Prestes et al. 2007; Urashima et al. 2004). Cruz et al. (2012) suggested that seedling infection assays were not reliable in predicting the severity of spike infection. Maciel et al. (2014) showed that there was not a strong correlation between seedling and spike reactions to the wheat blast pathogen. On the other hand, Tagle et al. (2015) showed that tetraploid accessions carrying *Rmg7* (St17,

St24, and St25) were highly resistant even at the heading stage. In the present study, we found that S615 carrying *Rmg8* conferred resistance not only at the seedling stage but also at the heading stage. If resistance at the heading stage is conferred by *Rmg8*, then it is considered to fulfill the first requirement.

Second, *R* genes must be durable. The durability of *R* genes may be predicted by evaluating a fitness cost imposed by mutation of or constraints imposed on corresponding AVR genes (Janzac et al. 2009; Leach et al. 2001; Vera Cruz et al. 2000), or by estimating the stability of corresponding AVR genes based on their chromosomal location and the molecular structure around them (Chuma et al. 2011; Tosa and Chuma 2014). In any case, identification of AVR genes is a prerequisite to evaluate durability of new *R* genes. To identify an AVR gene corresponding to an *R* gene (*R1*), we need an isolate virulent on *R1*. However, this requirement includes an intrinsic antinomy; durable *R* genes should be resistant to all prevailing *Triticum* isolates. In this respect, *Rmg7* and *Rmg8* were effective against all *Triticum* isolates tested. Tagle et al. (2015) solved this problem by using temperature sensitivity of avirulence of an *Eleusine* isolate on the *Rmg7* carriers. In the present study, we overcame this problem by producing a culture virulent on *Rmg8* (200R29) through crossing a *Triticum* isolate (Br48) with an *Eleusine* isolate (MZ5-1-6). By backcrossing this virulent isolate with the *Triticum* isolate, we successfully identified *AVR-Rmg8*. Cloning of *AVR-Rmg8* is under way to reveal its chromosomal location, surrounding molecular structure, and the fitness cost imposed by its mutations.

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Fig. 3. Genetic map of the *Rmg8* (left) and *Rmg7* (right) regions constructed using F₃ lines derived from crosses between two common wheat cultivars (S615 and Sch) and two tetraploid wheat accessions (St24 and Tat14), respectively; cM = centimorgans.

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