Analysis of Host Species Specificity of *Magnaporthe grisea* Toward Foxtail Millet Using a Genetic Cross Between Isolates from Wheat and Foxtail Millet

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ABSTRACT

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Host species specificity of *Magnaporthe grisea* toward foxtail millet was analyzed using F₁ cultures derived from a cross between a *Triticum* isolate (pathogenic on wheat) and a *Setaria* isolate (pathogenic on foxtail millet). On foxtail millet cvs. Beni-awa and Oke-awa, avirulent and virulent cultures segregated in a 1:1 ratio, suggesting that a single locus is involved in the specificity. This locus was designated as *Pfm1*. On cv.

Ki-awa, two loci were involved and one of them was *Pfm1*. The other locus was designated as *Pfm2*. Interestingly, *Pfm1* was not involved in the pathogenic specificity on cv. Kariwano-zairai. These results suggest that there is no "master gene" that determines the pathogenic specificity on all foxtail millet cultivars and that the species specificity of *M. grisea* toward foxtail millet is governed by cultivar-dependent genetic mechanisms that are similar to gene-for-gene interactions controlling race-cultivar specificity.

Additional keywords: *Pyricularia*, resistance.

The basis of host-parasite specificity between plants and microorganisms has been discussed by many phytopathologists. Day (1) distinguished two types of host-parasite interaction: one determines whether or not a plant is a host for a parasite, and the other determines the gene-for-gene specificity. Heath (3) termed the specificity determining host species range as plant species specificity, and the specificity determining cultivar range within a given host species as cultivar specificity. In the former, compatibility is specific, and the resistance involved (nonhost resistance) is genetically complex. In the latter, incompatibility is specific, and the resistance involved (cultivar resistance) is controlled by gene-for-gene interactions. However, data have been reported that may be interpreted as suggesting that gene-for-gene interactions underlie the plant species specificity (4).

Magnaporthe grisea (Hebert) Barr, the causal agent of blast disease, includes several subgroups that are pathogenic to a restricted range of plant species, i.e., *Oryza* pathotype pathogenic to rice (*Oryza sativa* L.), *Setaria* pathotype pathogenic to foxtail millet (*Setaria italica* Beauv.), *Panicum* pathotype pathogenic to common millet (*Panicum miliaceum* L.), *Eleusine* pathotype pathogenic to finger millet (*Eleusine coracana* (L.) Gaertn.), and *Digitaria* pathotype pathogenic to crabgrass (*Digitaria sanguinalis* (L.) Scop.) (7). In the 1980s, a new subgroup (*Triticum* pathotype) of *M. grisea* arose on wheat (*Triticum aestivum* (L.) Thell.) in Brazil and has spread to other countries in South America (18,19). This specificity between the pathotypes and gramineous plant species appears to be plant species specificity according to the traditional usage. However, there are some reports suggesting that the plant species specificity of *M. grisea* is governed by a rather simple mechanism. Yaegashi (20) crossed *Eleusine* isolates

with weeping lovegrass isolates and suggested that their pathogenicity to finger millet or weeping lovegrass is conditioned by a single gene. Kang et al. (5) and Sweigard et al. (10) found two unlinked major genes, *PWL1* and *PWL2*, which control species specificity to weeping lovegrass, and showed that they have characteristics similar to avirulence genes that condition cultivar specificity.

To elucidate the genetic mechanisms controlling the species specificity in *M. grisea*, we produced two hybrid populations. One was derived from a cross between a *Triticum* isolate and an *Avena* isolate. Using this population, Takabayashi et al. (11) showed that a gene-for-gene interaction underlies the avirulence of the *Avena* isolate on wheat. The other hybrid population was derived from a cross between a *Triticum* isolate and a *Setaria* isolate. Using this population, Murakami et al. (9) showed that the specific parasitism of the parental isolates on wheat is conditioned by two loci, *Pwt1* and *Pwt2*. Although this result suggested that the species specificity on wheat is controlled by a rather simple genetic mechanism, it was not clear whether these genes conditioned virulence or avirulence. The objective of this study was to determine genetic mechanisms of their specific parasitism on the other host species, foxtail millet. The results suggested that the specificity toward foxtail millet is governed by cultivar-dependent genetic mechanisms that are similar to gene-for-gene interactions controlling race-cultivar specificity.

MATERIALS AND METHODS

Fungal strains. Parental strains were two field isolates of *M. grisea*, Br48 and GFS11-7-2. Br48 was isolated from wheat in Brazil, whereas GFS11-7-2 was isolated from foxtail millet in Japan. They were crossed on oatmeal-agar media and a total of 80 F₁ cultures were obtained from 10 mature asci containing 8 ascospores (9). Preliminary cytological observation revealed that 11 of the 80 cultures were deficient mutants that lost the basic capacity of penetration. These mutants were excluded from further analysis.

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Plant materials. *Setaria italica* cv. Ki-awa (abbreviation, SI4), cv. Beni-awa (SI9), cv. Oke-awa (SI10), cv. Mosh-awa (SI12), and cv. Kariwano-zairai (SI17) were used as test plants for segregation analysis. Twelve seeds were sown in Sakata super-mix soil (Sakata, Yokohama, Japan) in a seedling case (5.5 × 15 × 10 cm) and grown at 26°C under natural light for 5 to 7 days.

Infection assays. *M. grisea* cultures were prepared as described previously (9). The conidia were suspended in water and adjusted to 1 × 10⁵ conidia per ml with 0.01% Tween 20. Twenty milliliters of the conidial suspension was sprayed on the adaxial surface of foxtail millet leaves with a glass sprayer connected to an air compressor. The inoculated seedlings were kept in a humid, dark box for 20 h at 20 to 22°C. They were then transferred to a controlled-environment room with a 12-h photoperiod of fluorescent lighting (4,000 lx) and incubated further at 20 to 22°C. Five days after inoculation, symptoms were evaluated based on affected area on the leaves. The affected area was rated using six progressive grades from 0 to 5: 0, no visible symptoms; 1, diseased leaf area of up to 5% of the whole leaf, mainly composed of brown pinpoint spots; 2, diseased leaf area of up to 10%, mainly composed of small brown lesions; 3, diseased leaf area of up to 50%, mainly composed of green lesions with an intermediate size; 4, diseased leaf area of up to 80%, mainly composed of green large lesions; and 5, diseased leaf area of more than 80%, almost completely shriveled. The mean disease severity on eight leaves was used as the virulence index (VI) of each culture. Isolates with VI less than 3 were considered avirulent, whereas those with VI greater than or equal to 3 were considered virulent. The inoculation assay was repeated at least five times with similar results.

RESULTS

Pathogenicity of the parental isolates toward foxtail millet cultivars. Primary leaves of foxtail millet cultivars were inoculated with the parental isolates. The *Triticum* isolate Br48 was avirulent on all the foxtail millet cultivars (Fig. 1; Table 1). In contrast, the *Setaria* isolate GFSII-7-2 produced many water-soaked lesions on SI4, SI9, SI10, and SI17. They enlarged and coalesced with time and, finally, the whole leaf shriveled with its color remaining green by 5 days after inoculation (Fig. 1). Virulence indices of Br48 and GFSII-7-2 on these four cultivars were approximately 1 and 5, respectively (Table 1). On SI12, however, both parental isolates produced no visible lesions (Table 1). This foxtail millet cultivar was assumed to carry a cultivar-specific resistance gene.

Segregation analysis of pathogenicity of F₁ cultures on foxtail millet cultivars. The foxtail millet cultivars were inoculated with the 69 F₁ cultures derived from the Br48 × GFSII-7-2 cross.

On SI12 all the F₁ cultures were avirulent (Table 2). On the other cultivars, the F₁ cultures showed varying disease severity levels, with VI ranging from 0 to 5, but could be classified into avirulent or virulent. Figure 1 shows reactions of SI4, SI9, and SI17 to a set of tetrads (1K1, 1L1, 1M1, and 1N1) as an example. Avirulent and virulent cultures segregated in 3:1, 1:1, and 3:1 ratios on SI4, SI9, and SI17, respectively. Interestingly, the patterns of reactions were various among the foxtail millet cultivars (Fig. 1).

The frequency distribution of VI in the 69 F₁ cultures is summarized in Table 2. On SI9 and SI10, the segregation of avirulent and virulent cultures fitted a 1:1 ratio, suggesting that a single locus is involved in the specific pathogenicity of the parental isolates. The segregation patterns on these cultivars were identical; all F₁ cultures avirulent on SI9 were avirulent on SI10, whereas all F₁ cultures virulent on SI9 were virulent on SI10 (Table 3). This result suggests that the specific pathogenicity to these cultivars is controlled by the same gene. This locus was designated as *Pfm1* after pathogenicity on foxtail millet. On another cultivar SI4 avirulent and virulent cultures segregated in a 3:1 ratio (Table 2), suggesting that two unlinked loci are involved. The segregation pattern on SI4 was compared with that on SI9 to determine whether one of the two loci was *Pfm1*. If one locus is identical to *Pfm1*, F₁ cultures that are avirulent on both SI9 and SI4, avirulent on SI9 only, avirulent on SI4 only, and virulent on both SI9 and SI4 should segregate in a 2:0:1:1 ratio. If both of the two loci are different from and independent of *Pfm1*, they should segregate in a 3:1:3:1 ratio. The combined segregation on these cultivars fitted the former ratio (Table 3), suggesting that one of the two loci is *Pfm1*. The other locus was designated as *Pfm2*. On SI17 avirulent and virulent cultures also segregated in a 3:1 ratio although distribution of VI was somewhat continuous (Table 2). This result suggests that the pathogenicity to SI17 is conditioned by two loci. The combined segregation on SI9 and SI17 fitted a 3:1:3:1 ratio (Table 3), indicating that both of the two loci are different from

TABLE 1. Pathogenicity of parental isolates of *Magnaporthe grisea* to foxtail millet cultivars

Cultivar	Abbreviation	Virulence index ^a	
		GFSII-7-2	Br48
Ki-awa	SI4	4.6	1.0
Beni-awa	SI9	4.8	1.1
Oke-awa	SI10	4.8	1.2
Mosh-awa	SI12	0.6	0.2
Kariwano-zairai	SI17	4.5	1.0

^a The mean disease severity on eight leaves was used as the virulence index (VI) of each culture. Isolates with VI less than 3 were considered avirulent, whereas those with VI greater than or equal to 3 were considered virulent.

Fig. 1. Pathogenicity of *Magnaporthe grisea* field isolates (Br48 and GFSII-7-2) and their F₁ progeny (1K1, 1L1, 1M1, and 1N1) on foxtail millet cvs. Ki-awa (SI4), Beni-awa (SI9), and Kariwano-zairai (SI17). 1K1, 1L1, 1M1, and 1N1 are tetrads derived from a single ascus.

Pfm1 and, therefore, that *Pfm1* is not involved in the specificity on SI17. The segregation pattern on SI17 was also compared with that on SI4 to determine if *Pfm2* is involved in the specificity on SI17. The combined segregation was not significantly different from 5:1:1:1, which is expected when one of the two loci is *Pfm2*, nor from 9:3:3:1, which is expected when both loci are different from *Pfm2* (12).

Although dominance of alleles at the *Pfm1* and *Pfm2* loci cannot be unequivocally determined in this haploid fungus, we designate avirulence alleles from the *Triticum* isolate as *PFM1* and *PFM2* and virulence alleles from the *Setaria* isolate as *pfm1* and *pfm2*. This is based on the fact that the segregation of avirulence to virulence was not 1:3 but 3:1 on SI4 (Table 2; Fig. 1). The results described previously can be summarized by assigning these gene symbols to the tetrads in Figure 1. 1L1 and 1N1 are avirulent on SI9 because they carry *PFM1*, whereas 1K1 and 1M1 are virulent on it because they carry *pfm1*. 1L1 and 1N1 are also avirulent on SI4 because *PFM1* operates on SI4. 1M1 is avirulent on SI4 due to *PFM2*. On SI17, the *Pfm1* locus should not be involved because the *PFM1* carrier 1N1 is virulent and the *pfm1* carrier 1M1 is avirulent.

The segregation data on SI4, SI9, and SI17 were combined with previously reported data (9) on wheat cv. Norin 4 (N4). In all combinations of cultivars there occurred hybrid cultures that were virulent on both wheat and foxtail millet (data not shown). As reported previously (9), the specificity of the *Setaria* isolate GFSI1-7-2 and the *Triticum* isolate Br48 on wheat is mainly controlled by two loci, *Pwt1* and *Pwt2*. Avirulence alleles from the *Setaria* isolates were designated as *PWT1* and *PWT2*, and virulence alleles from the *Triticum* isolates were designated as *pwt1* and *pwt2* (11). When the segregation data of *Pfm1* were combined with those of *Pwt1*, the possible four genotypes occurred in a

1:1:1:1 ratio (Table 4), indicating that those loci are inherited independently. When *Pfm1* was combined with *Pwt2*, the possible four genotypes also occurred (Table 4), but the recombinants (P;Q and p;q) were more frequent than the parental types for unknown reasons. Such analyses were not possible for *Pfm2* because *F₁* cultures carrying *PFM1* alone and those carrying both *PFM1* and *PFM2* showed the same phenotype (avirulent on both SI9 and SI4) and were not distinguishable.

DISCUSSION

Two loci were identified that are involved in the pathogenic specificity of the *Triticum* isolate and the *Setaria* isolate toward foxtail millet. Their specific parasitism on SI9 and SI10 was controlled by the same single locus, *Pfm1*, whereas that on SI4 was controlled by two loci, *Pfm1* and *Pfm2*. The segregation data on SI9, SI10, and SI4 were summarized in Table 5 with genotypes of the *F₁* cultures. Genotypes of the parental *Triticum* isolate (Br48)

TABLE 5. A hypothesis for genetic mechanisms of the interaction between foxtail millet cultivars and *F₁* cultures derived from the cross Br48 × GFSI1-7-2

Genotype of <i>F₁</i> cultures	Reaction with ^a	
	SI9 and SI10 (<i>R₁</i> ; <i>r₂</i>) ^b	SI4 (<i>R₁</i> ; <i>R₂</i>)
<i>PFM1</i> ; <i>PFM2</i>	A [<i>PFM1</i> - <i>R₁</i>] ^c	A [<i>PFM1</i> - <i>R₁</i> , <i>PFM2</i> - <i>R₂</i>]
<i>PFM1</i> ; <i>pfm2</i>	A [<i>PFM1</i> - <i>R₁</i>]	A [<i>PFM1</i> - <i>R₁</i>]
<i>pfm1</i> ; <i>PFM2</i>	V [-]	A [<i>PFM2</i> - <i>R₂</i>]
<i>pfm1</i> ; <i>pfm2</i>	V [-]	V [-]

^a A, avirulent; V, virulent.
^b Plant genotype.
^c Avirulence gene-resistance gene pair involved in the reaction.

TABLE 2. Pathogenicity to foxtail millet cultivars of *F₁* cultures derived from the cross Br48 × GFSI1-7-2

Cultivar	Number of <i>F₁</i> cultures						<i>X</i> ^{2b}			
	0– ^a	1–	2–	3–	4–5	Total	Avirulent (index < 3)	Virulent (index ≥ 3)	1:1	3:1
SI4	20	31	2	15	1	69	53	16	19.84**	0.12
SI9	11	23	2	18	15	69	36	33	0.13	19.17**
SI10	9	24	3	17	16	69	36	33	0.13	19.17**
SI12	63	6	0	0	0	69	69	0	–	–
SI17	18	28	7	13	3	69	53	16	19.84**	0.12

^a The affected area was rated using six progressive grades from 0 to 5: 0, no visible symptoms; 1, diseased leaf area of up to 5% of the whole leaf, mainly composed of brown pinpoint spots; 2, diseased leaf area of up to 10%, mainly composed of small brown lesions; 3, diseased leaf area of up to 50%, mainly composed of green lesions with an intermediate size; 4, diseased leaf area of up to 80%, mainly composed of green large lesions; and 5, diseased leaf area of more than 80%, almost completely shriveled. The mean disease severity on eight leaves was used as the virulence index (VI) of each culture. Isolates with VI less than 3 were considered avirulent, whereas those with VI greater than or equal to 3 were considered virulent.
^b ** indicate significant at the 1% level.

TABLE 3. Combined segregation of pathogenicity to two cultivars of foxtail millet in *F₁* cultures derived from the cross Br48 × GFSI1-7-2

Cultivar		Number of <i>F₁</i> cultures					<i>X</i> ^{2b}	
p	q	ApAq ^a	ApVq	VpAq	VpVq	Total	2:(0):1:1	3:1:3:1
SI9	SI10	36	0	0	33	69	...	...
SI9	SI4	36	0	17	16	69	0.16	23.76**
SI9	SI17	30	8	23	8	69	7.46**	1.07

^a A, avirulent; V, virulent. For example, ApVq represents cultures that are avirulent on p and virulent on q.
^b ** indicate significant at the 1% level.

TABLE 4. Combined segregation of *Pfm1* and *Pwt* alleles in *F₁* cultures derived from the cross Br48 × GFSI1-7-2

Locus		Number of <i>F₁</i> cultures				
P/p	Q/q	P;Q ^a	P;q	p;Q	p;q	Total
<i>Pfm1</i>	<i>Pwt1</i>	18	18	19	14	69
<i>Pfm1</i>	<i>Pwt2</i>	24	12	8	25	69

^a Genotypes. The capital and small letters represent avirulence and virulence alleles, respectively.

and *Setaria* isolate (GFSI1-7-2) are presumed to be *PFM1*;*PFM2* and *pfm1*;*pfm2*, respectively. A cross between them should yield four genotypes in the F_1 population, i.e., *PFM1*;*PFM2* (I), *PFM1*;*pfm2* (II), *pfm1*;*PFM2* (III), and *pfm1*;*pfm2* (IV). On SI9 and SI10, the *Pfm2* locus has no effect on the phenotype and only the *Pfm1* locus is involved in the specificity. So, *PFM1* progeny (I and II) are avirulent while *pfm1* progeny (III and IV) are virulent, resulting in a 1:1 segregation in the F_1 population. On SI4 both loci are involved, resulting in a 3:1 segregation. Do *Pfm1* and *Pfm2* govern avirulence or virulence? Are functional alleles *PFM1* and *PFM2* (avirulence alleles from the *Triticum* isolate) or *pfm1* and *pfm2* (virulence alleles from the *Setaria* isolate)? We assume that they are both avirulence loci because of the way they interact epistatically; one copy of either gene is sufficient to cause avirulence in the F_1 population (Fig. 1; Tables 2 and 5).

How do these genes govern the specificity? A clue may be in our data shown in Table 3 and Figure 1. These data indicate that *Triticum* and *Setaria* isolates carry no "master gene" that operates on all foxtail millet cultivars commonly for determining their parasitic specificity on *Setaria italica*. Fungal genes involved in the specificity are different from cultivar to cultivar. This variation can be explained by gene-for-gene interactions (2); the variation of fungal genes involved may be due to the variation of resistance genes carried by the cultivars. A very similar situation has been already reported in the forma specialis-genus specificity in the powdery mildew (*Blumeria graminis*)-cereal system. Tosa and coworkers crossed *B. graminis* f. sp. *tritici* (the wheat mildew fungus) with *B. graminis* f. sp. *agropyri* (the wheatgrass mildew fungus) and identified four fungal genes that are involved in their parasitic specificity on wheat (12,13,15). They subsequently identified their corresponding resistance genes in wheat cultivars (15–17) and demonstrated that the forma specialis-genus specificity is governed by gene-for-gene interactions (13–15). The fungal genes were all avirulence genes from the wheatgrass mildew fungus that are involved in its specificity on the genus *Triticum*. However, they were cultivar-specific in their effects and operated in various combinations on each wheat cultivar (15). This cultivar-specific operation of the avirulence genes could be attributed to the difference of resistance genes carried by each cultivar (15). The specificity between foxtail millet cultivars and *Setaria/Triticum* isolates of *M. grisea* can be conditioned by a similar genetic mechanism and may be an example that species specificity is governed by gene-for-gene interactions.

With this hypothesis, the interactions between the F_1 cultures and the foxtail millet cultivars are explained as follows (Table 5). Resistance genes corresponding to *PFM1* and *PFM2* are tentatively designated as *R1* and *R2*, respectively. On SI4 both *PFM1* and *PFM2* are involved in the specificity because this cultivar carries both *R1* and *R2*. On SI9 *PFM1* is involved but *PFM2* is not, because this cultivar carries *R1* but not *R2*.

Kato (6) found cultivar specificity between three *Setaria* isolates and 20 foxtail millet cultivars. The resistance of SI12 to GFSI1-7-2 reported in the present study may be an example of such specificity; SI12 may carry a resistance gene and GFSI1-7-2 may carry its corresponding avirulence gene. It should be noted that the cross between Br48 and GFSI1-7-2 did not produce progeny that is virulent on SI12. A simple interpretation is that Br48 also carries the avirulence gene corresponding to the resistance gene in SI12. This is comparable to the fact that the rye mildew fungus, *B. graminis* f. sp. *secalis*, carries avirulence genes corresponding to wheat genes for resistance to races of the wheat mildew fungus, *B. graminis* f. sp. *tritici* (8).

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