Segregation of Avirulence/Virulence on Three Rice Cultivars in 16 Crosses of *Magnaporthe grisea*

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ABSTRACT

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Two isolates, 6-20 and Guy 11, both of which are pathogenic on rice, were crossed; the segregation for avirulence/virulence on three cultivars is given for 15 subsequent crosses. Both parents and all progenies were pathogenic on cultivar S201. Certain progenies were selected for intercrossing in each generation, and some progenies were backcrossed to Guy 11. Crosses between some isolates, both virulent on a particular cultivar, produced avirulent progenies. Crosses of isolates, one avirulent and the other virulent on a particular cultivar, usually segregated for avirulence/virulence. Crosses of two isolates, both avirulent on a particular cultivar, usually gave all avirulent progenies. The infection type ranges of parent isolates did not necessarily indicate the infection types of the progenies. The recovery of avirulent progenies in crosses between two virulent isolates suggests that one of the parent isolates classified as virulent contained an avirulence gene(s) whose expression was suppressed. The genes controlling avirulence and virulence on each of the three rice cultivars are different from one another. A postulated genotype of each of the parent isolates is presented.

The genetics of interactions between plants and fungal pathogens has been studied for many diseases. Most of the conclusions from the studies on avirulence and virulence of fungal pathogens have been based on the segregation ratios of avirulence/virulence on particular cultivars of the host species. Many of the studies have been with host-pathogen combinations in which the difference between alleles at a locus in the interaction phenotype is clearly avirulence vs. virulence of the two parent pathogen isolates and in which discontinuous variation in the segregating population exists. When the phenotypes of the two parents are not as distinctive, or even in some instances when the parents do give clearly distinguishable avirulence/virulence phenotypes, continuous variation, from highly avirulent to fully virulent progeny, is observed. The segregation ratios of avirulence/virulence are determined, in most instances, by a value judgment as to what is considered avirulent and what is considered virulent. In instances in which the actual variation in phenotype of the pathogen progenies is presented (11), it is obvious that progeny can differ considerably in the phenotype of the interaction with a particular cultivar but still be grouped in a reasonably logical manner to give a simple segregation ratio. But, are the individuals with different degrees of avirulence or virulence properly grouped into single postulated genotypes? This is basically an unanswered question, primarily because additional crosses with the segregants have rarely been made.

By classical gene-for-gene theory (1), crosses between isolates that are both virulent on a particular cultivar should give only virulent progenies. Crosses of a virulent isolate with an avirulent isolate should show segregation among the progenies. Crosses between avirulent isolates may or may not show segregation, because they may be avirulent on that cultivar based on different avirulence genes. In some crosses between two virulent isolates, avirulent progenies were recovered, because one of the parents contained an avirulence gene and the suppressor of that avirulence gene (7). The phenotype of the suppressor was detected only in the presence of the avirulence gene(s).

Magnaporthe grisea (T. T. Hebert) Yaegahsi & Udagawa (*Pyricularia oryzae* Cavara) is a haploid fungal pathogen of rice (*Oryza sativa*). This fungus is the causal organism of blast, one of the most destructive diseases of rice (10). Many races of *M. grisea* have been reported based on the cultivar specificity of the isolates tested (10). *M. grisea* has also been studied extensively because of the reported instability of some isolates (10). Single conidial isolates from one lesion on a rice plant can give rise to new races in each of several single conidial generations. The genetic basis of this extensive variability has not been established. The development of isolates that are pathogenic on rice, that can be intercrossed, and that give viable progenies, all of which are pathogenic on rice, provides an opportunity to analyze genetically the inheritance of cultivar specificity (2,3,5).

The objectives of this study were to determine the inheritance of avirulence/virulence of *M. grisea* on several cultivars through a series of crosses and test crosses and to answer the following questions. Does absence of segregation of avirulence/virulence indicate that the cross is homozygous for genes controlling interaction with that cultivar? Do differences in degrees of avirulence and virulence have an identifiable genetic basis?

MATERIALS AND METHODS

The *M. grisea* isolates used in this study were derived from a series of crosses that originated with a cross between an isolate pathogenic on rice with an isolate pathogenic on weeping love grass (*Eragrostis curvula*) (6). Sib selection for fertility and pathogenicity on rice was practiced for four generations. Isolate 6-20 is pathogenic on rice and was crossed with Guy 11, an isolate that is also pathogenic on rice (6,9). This cross was labeled cross 12. (Tables 1–3). Three progenies from cross 12 were used in crosses 28, 29, and 31. Crosses were also made between selected progenies from these latter three crosses. Five progenies from cross 57 were used in backcrosses to Guy 11.

Isolates to be stored were grown on a complete agar medium (12) for 1 wk, dried, and stored at −20 C. Isolates prepared in this manner can be stored for several years. Isolates were designated by the cross from which each came and a progeny number. For example, isolate 12-7 was the seventh progeny from cross 12.

Crosses were made by placing two isolates (mycelia in agar) about 5 cm apart on oatmeal agar at 20–22 C (3,8) in a petri plate. Asci usually formed in 16–21 days. Individual perithecia were removed, crushed, and dragged across the agar surface. Individual asci were separated with a bent, sterile glass needle. The spores in the asci began to germinate within about 6 h, and

small colonies producing conidia developed in about 2–3 days. One conidium was isolated from each ascus colony, thus ensuring the collection of one progeny from each meiotic event (3,8).

Isolates to be tested for pathogenicity were grown either on autoclaved corn leaves or on oatmeal agar in petri dishes at 20–22 C for about 1 wk. The colonies were flooded with sterile H_2O, and then conidia were scraped from these colonies. The suspension was adjusted to approximately 10^5 conidia per milliliter and atomized onto rice seedlings that were at the two- to three-leaf stage. Inoculated plants were incubated in a moist chamber at 20–22 C for 20 h and then transferred to either a growth chamber or greenhouse at 25–27 C. Similar results were obtained in a growth chamber or greenhouse. The reactions of the plants were scored at 7–10 days after inoculation. Four infection phenotypes were recognized: 1 = minute black spots; 2 = 2–3-mm-length black lesions; 3 = usually circular, variable lesions with gray centers; and 4 = large, commonly diamond-shaped lesions with gray centers, which commonly coalesced into long stripes with gray centers (6). In some isolate-cultivar combinations, only one lesion type was observed. In other combinations, a range of lesion types, including combinations with a complete range of infection types, occurred. The data are presented as the maximum range of lesion types observed for the isolate-host cultivar interactions (Tables 1–3).

Isolates were tested for their avirulence/virulence phenotype on 15 cultivars of rice. The results for cultivars Bluebelle (CI 9544), L202 (PI 483097), and Leah (CI 9979) are given in this paper. Seeds of these cultivars were kindly supplied by A. Marchetti, USDA-ARS, Beaumont, Texas. All isolates were virulent on cultivar S201 (CI 9974).

The tests of the avirulence/virulence phenotype were either done three times for each isolate with at least three plants each time

TABLE 1. Cross number, parents, infection type of parents, and numbers of progeny with a given infection type range on the cultivar Bluebelle

	Parent no.		Infection type of parent		Number of progeny with each infection type range[a]										Apparent ratio	Chi-square
Cross no.	1	2	1	2	0–1	1–2	2	1–3	1–4	2–3	2–4	3	3–4	4	Avir/vir[b]	1:3
12	6-20	Guy 11	3–4	3–4							3^{+c}	1	16	9	0:29	9.45**[d]
28	12-7	12-20	3–4	3–4							1		6		0:7	
29	12-7	12-26	3–4	3–4	1		1			4	3		6	5	2:18	2.4
31	12-26	Guy 11	3–4	3–4				1^+		2^+	5^+	5	16		0:29	
33	31-1	31-34	3	3	1					1		4			1:5	0.21
34	31-4	31-16	3	3	1	1				1		1		1	2:3	0.59
41	31-3	31-10	3–4	3–4								5	6	5	0:16	
42	31-3	31-21	3–4	3–4								2	2	3	0:7	
56	28-2	29-4	2–4	4						3			3	3	0:9	
57	28-2	29-2	2–4	3–4	1		2			2	2		2	10	3:16	0.85
61	29-15	29-19	4	4						2				5	0:7	
65	57-4	Guy 11	2–3	3–4	1		2			12			5	3	3:20	1.75
66	57-10	Guy 11	4	3–4			1			12	10	4			1:26	6.4*
67	57-17	Guy 11	4	3–4						6			2		0:8	
68	57-15	Guy 11	4	3–4	1		1			4			2	4	2:10	0.44
69	57-9	Guy 11	2–4	3–4						7	1		1	1	0:10	

[a] Range of infection types observed.
[b] Isolates with infection types 0–2 are usually considered to be avirulent (avir). Infection type ranges from 2–3 to 4 are usually considered to be virulent (vir).
[c] A + or − indicates that the predominant infection type was the higher or lower infection type, respectively.
[d] * Indicates $P < 0.005$; ** indicates $P < 0.01$.

TABLE 2. Cross number, parents, infection type of parents, and numbers of progeny with a given infection type range on the cultivar L202[a]

	Parent no.		Infection type of parent		Number of progeny with each infection type range										Apparent ratio	Chi-square	
Cross no.	1	2	1	2	0–1	1–2	2	1–3	1–4	2–3	2–4	3	3–4	4	Avir/vir[b]	1:1	1:3
											3^{-c}						
12	6-20	Guy 11	3–4	3–4				2	1		3^+		10	10	6:23		0.29
28	12-7	12-20	3–4	3–4					2^+	2	2^+	1			0:7		
29	12-7	12-26	3–4	3–4	3	2	1		3^+	2	1		2	5	6:13		0.44
								1^-									
31	12-26	Guy 11	3–4	3–4				4^+	1^+	4^+	3^+	5	9	2	1:28		7.17*[c]
33	31-1	31-34	3–4	3	1	2	3					1			6:1	3.57	
34[d]	31-4	31-16	$2–3^+$	$2–3^+$	1	1	2							1	4:1		
41	31-3	31-10	3–4	3–4								6	8		0:14		
42	31-3	31-21	3–4	4						1		3	3		0:7		
56	28-2	29-4	2–3	3–4					1^+	2	1^+			4	0:8		
57[d]	28-2	29-2	2–3	3–4			1		2		2		2	13	1:19		4.26*
61	29-15	29-19	4	4						1			1	5	0:7		
65	57-4	Guy 11	2	3–4	1		1			4		1	11	6	2:22		3.54
66	57-10	Guy 11	4	3–4					3	5	10		8	2	0:28		
67	57-17	Guy 11	4	3–4		1				6			3		1:9		0.12
68	57-15	Guy 11	4	3–4						1				11	0:12		
69	57-9	Guy 11	2–4	3–4	1					2			2	5	1:9		0.12

[a] Range of infection types observed.
[b] Isolates with infection types 0–2 are usually considered to be avirulent (avir). Infection type ranges from 2–3 to 4 are usually considered to be virulent (vir).
[c] A + or − indicates that the predominant infection type was the higher or lower infection type, respectively.
[d] See text for discussion of this cross.
[e] Indicates $P < 0.05$.

or by a single test on at least 8–12 plants. Isolates that gave unexpected phenotypes (e.g., low infection type progenies from the cross of two parents that gave high infection types on that cultivar) were tested for their pathogenicity at least three times. Although an effort was made to test all isolates on all three cultivars, reactions with certain isolate-cultivar combinations were not determined for various reasons. Therefore, the numbers of segregants observed for interactions with different host cultivars in each cross may be different.

Progenies from crosses are commonly grouped into the two categories of avirulent and virulent. Isolates with interaction phenotypes of 1 or 2 on a particular cultivar are usually considered to be avirulent on that cultivar, whereas isolates with interaction phenotypes of 3 or 4 usually are considered to be virulent on that cultivar (6). Isolates that give a wide range of infection types on a given cultivar are considered, in this work, to be avirulent or virulent depending on the predominant infection type. The calculations of observed ratios in Tables 1–3 are based on the above arguments. Determining if this interpretation is correct is part of the subject of this study.

RESULTS

The segregation of avirulence/virulence on cultivar Bluebelle in 16 crosses is given in Table 1. In cross 12, both parents, 6-20 and Guy 11, gave an infection type range of 3–4 on Bluebelle. Three progenies gave an infection type range of 2–4, one gave infection type 3, 16 gave infection type range of 3–4, and nine gave an infection type of 4 on Bluebelle. In this and subsequent crosses, the "+" designation of certain progenies, such as the three with a rating of 2–4, indicates that the predominant lesions were of the high infection type class, yet lesions with infection types 2 and 3 were also observed. A "−" indicates the lower infection type was predominant. From this cross between two isolates, both of which are virulent on Bluebelle, all progenies were classified as being virulent on Bluebelle. The results suggest the cross is homozygous for virulence on Bluebelle.

Cross 28 was between two progenies from cross 12 that had infection types 3–4 on Bluebelle. Only seven progenies were analyzed from this cross, and all gave high infection types on Bluebelle. Cross 29 was a cross also between two progenies from cross 12 that each gave a high infection type on Bluebelle. Recovered among the progenies of cross 29 were two strains that gave low infection types on Bluebelle. The recovery of avirulent progenies from the cross between two virulent isolates was unexpected and suggested that isolates 12-7 and 12-26 differ by at least two genes, even though both parents are virulent on Bluebelle.

Cross 31 is a backcross of 12-26 to Guy 11. Both isolates were virulent on Bluebelle, and the predominant infection type of the progenies would suggest that all progenies should be considered virulent on Bluebelle. Crosses 33 and 34 were each crosses between two progenies of cross 31 that gave infection type 3 on Bluebelle. Although infection type 3 is usually considered indicative of virulence, both crosses yielded avirulent progenies. Crosses 41 and 42 were between progenies of cross 31 that gave an infection type range of 3–4 on Bluebelle, and all progenies from these crosses were considered to be virulent on Bluebelle.

Crosses 56 and 57 were between progeny 28-2 from cross 28, which gave an infection type of 2–4 on Bluebelle, and two progenies from cross 29. Isolates 29-4 and 29-2 gave infection types 4 and 3–4, respectively, on Bluebelle. All progeny from cross 56 gave an infection type of 2–3 or greater and were tentatively designated as virulent. Cross 57, however, yielded progenies that gave low infection types on Bluebelle. The recovery of only virulent progeny from crosses 28 and 56 suggests that 28-2 is not the source of the avirulence gene(s) and that 29-2 contains one or more avirulence factors, even though it gave an infection type of 3–4 on Bluebelle.

Cross 61 was between two progenies from cross 29, which each gave infection type 4 on Bluebelle. Five of the progeny gave infection type 4, and two gave infection type 2–3 on Bluebelle.

Crosses 65–69 were backcrosses of five different progenies from cross 57 to Guy 11. The progenies from cross 57 represented three different infection type ranges. Isolate 57-4 was one of two progenies from cross 57 that gave infection type 2–3 on Bluebelle. Isolate 57-4 apparently passed a gene(s) for low infection type onto some of its progenies. Crosses 66 and 68 also yielded some progenies with low infection type on Bluebelle, but crosses 67 and 69 did not. Although small numbers of progeny were tested, the results with crosses 67 and 69 suggest that it is not Guy 11 that brought genes for low infection type into crosses 12, 31, 65, 66, and 68. The progenies from cross 57, which were used in crosses 65–69, suggest that the range of infection types of the parent (i.e., 2–3, 2–4, and 4) is not a good predictor of the segregation of the range of infection types that was actually observed in crosses with Guy 11.

The results of the inheritance of avirulence/virulence on cultivar

TABLE 3. Cross number, parents, infection type of parents, and numbers of progeny with a given infection type range on the cultivar Leah[a]

	Parent no.		Infection type of parent		Number of progeny with each infection type range										Apparent ratio	Chi-square
Cross no.	1	2	1	2	0–1	1–2	2	1–3	1–4	2–3	2–4	3	3–4	4	Avir/vir[b]	1:1
12	6-20	Guy 11	0–1	3–4	11	1		3+[c]	1+		3		7	2	12:16	0.57
28	12-7	12-20	3–4	0–1	2			1	2	1			1		2:5	1.29
29	12-7	12-26	3–4	0–1	3	2	2	1− 3−		3	1		5	3	8:12	0.80
31	12-26	Guy 11	0–1	3–4	11	3		1+	2	2	1+	2	4		17:12	0.86
33	31-1	31-34	3	3	1	1	2					3			4:3	0.14
34[d]	31-4	31-16	0–1	0–1	3									1	3:1	
41	31-3	31-10	3	0–1	6					1		4	4	1	6:10	1.00
42	31-3	31-21	3	3–4								2	2	2	0:6	
56	28-2	29-4	1	1		4	4								8:0	
57	28-2	29-2	1	3–4	4		6			1	1		2	4	10:8	0.22
61	29-15	29-19	4	4						5			2		0:7	
65[d]	57-4	Guy 11	3–4	3–4	1		1			10			5	2	2:17	
66	57-10	Guy 11	4	3–4						8	11		8		0:27	
67	57-17	Guy 11	1	3–4	2		3						1		5:1	2.66
68	57-15	Guy 11	2	3–4	1		3			3			1	4	4:8	2.66
69	57-9	Guy 11	2–4	3–4						3	1		3	3	0:10	

[a] Range of infection types observed.
[b] Isolates with infection types 0–2 are usually considered to be avirulent (avir). Infection type ranges from 2–3 to 4 are usually considered to be virulent (vir).
[c] A + or − indicates that the predominant infection type was the higher or lower infection type, respectively.
[d] See text for discussion of this cross.

L202 from the 16 crosses are given in Table 2. In cross 12, 6-20 and Guy 11 gave infection type 3–4 on L202, but progenies that represent a wide range in infection types were obtained. No clear segregation for avirulence/virulence was observed. Three progenies gave a range of infection types (either 1–3 or 1–4) on L202, and the lesions with high and low infection types were in approximately equal frequencies on each leaf. They were tentatively grouped with the avirulent progenies for the calculation of an apparent ratio in Table 2. Two progenies from cross 12 that gave infection type 3–4 were crossed (cross 28), and all seven progenies had predominantly high infection types. In cross 29, both parents also gave infection type 3–4 on L202, but six progenies gave low infection types on L202. Clearly, at least one of the parents brought a gene(s) for avirulence into the cross. Cross 31, a backcross of a progeny from cross 12 to Guy 11, gave only one progeny with predominantly low infection type. Crosses 33, 34, 41, and 42 were intercrosses of progenies from cross 31. Cross 33 was made between isolates with infection types 3–4 and 3, respectively; nevertheless, six out of seven progenies from this cross gave low infection types on L202. Cross 34, between two isolates with infection type 2–3$^+$, gave four out of five progenies with infection type lower than either parent. Crosses 41 and 42 were made between two virulent parents, and all progenies were virulent on L202. Crosses 65–69 were backcrosses to Guy 11, and they showed that the phenotype of the parent (progenies from cross 57) was not a dependable predictor of the segregation of progenies with a range of infection types. Although crosses 65 and 69 might be expected to give progenies with low infection types, because the one parent has low infection type lesions, cross 67 was not expected to give progenies with low infection types on L202. The avirulence most likely did not come from Guy 11, because crosses 66 and 68 gave progenies with a predominance of high infection types.

The infection type ranges among the parents and progenies of the 16 crosses on cultivar Leah are given in Table 3. In each of the first four crosses, one parent gave a high infection type, and the other gave a low infection type on cultivar Leah. The two progenies from cross 31 that gave infection type 3 on Leah were crossed (cross 33), and four of the seven progenies gave low infection types on Leah. Although both isolates 31-1 and 31-34 were classified as virulent, one or more genes for avirulence must be present in one or both of these isolates, which permit them to give clearly avirulent progenies. Both parents in cross 34 gave low infection types on Leah, and three of the four progenies tested showed low infection types. Crosses 41 and 42 have one parent in common, 31-3, which gave infection type 3 on Leah. Crossed with 31-10 (cross 41), progenies segregated for low and high infection types. Cross 42 did not give progenies with low infection types. When 28-2 was crossed with 29-2, the segregation ratio of 8:10 of low/high infection types was observed. Cross 61 was between two isolates that gave high infection types on Leah, and all progenies derived from this cross also gave high infection types on Leah. Crosses 65–69 were of progenies of cross 57 with different infection types on Leah backcrossed to Guy 11. The recovery of two avirulent progeny from cross 65 was unexpected. The segregation in crosses 66–69 was in agreement with the expectations that a cross between avirulent × virulent isolates will give progenies that segregate for avirulence/virulence and that a cross of two virulent isolates will give only virulent progenies. The results from cross 69 suggested that 57-9 is, in fact, properly classified as virulent on Leah, because crosses 66 and 69 gave similar types of progenies.

DISCUSSION

The postulated genotypes of each of the parent isolates for the 16 crosses are presented in Table 4. The postulated genotypes are based primarily on the minimum number of genes that are required to explain the performance of the progenies, rather than on the segregation ratios. For example, the recovery of progenies, all of which gave high infection types on Bluebelle in cross 12, would suggest that 6-20 and Guy 11 contain the same genes for virulence on Bluebelle. However, the recovery of avirulent progenies in cross 29 and six other crosses suggests that either 6-20 or Guy 11 has genes for avirulence on Bluebelle. The recovery of progenies, all of which had high infection types in crosses 31, 67, and 69, suggests that Guy 11 is not the source of the avirulence.

The recovery of avirulent progenies in a cross between two virulent isolates has been observed in *Melampsora lini*. Flor (4) observed what he interpreted as an example of dominance for virulence on the flax cultivar Willston Brown. However, the segregation ratios fit neither a 3:1 nor a 15:1 ratio for this dikaryotic pathogen. The data do fit a 13:3 (virulent/avirulent) ratio. Lawrence et al (7) have shown that the segregation is due to a dominant gene for avirulence and a dominant suppressor, which give a 13:3 segregation ratio. In fact, Lawrence et al have shown the segregation of at least two suppressors, one that suppressed the expression of one avirulence gene and another that suppressed the expression of four avirulence genes. They also observed that there was a higher incidence of intermediate phenotypes with the progeny from crosses in which segregation of suppressors occurred. These results indicate that the suppressor did not totally suppress the expression of avirulence; however, crosses to test their hypotheses were not done.

By postulating that 6-20 contains a gene for avirulence on Bluebelle, *Pl*, and a suppressor of that avirulence gene, *Sul*, one can explain the origin of avirulent progenies in the intercross of the progenies of cross 12 (Table 1). If the parents in a cross differ by two genes, one avirulence gene and a suppressor of that avirulence gene, then the progenies should segregate 1:3, avirulent/virulent. If 6-20 contained both an avirulence gene and a suppressor, segregation would have been expected in cross 12 if the genes were not linked and segregated independently.

Isolate 6-20 was derived from a cross between two isolates from different host species (rice and weeping love grass). The

TABLE 4. Postulated genotypes of the parents in each cross[a]

Cross no.	Parents	Genotype per cultivar		
		Bluebelle	L202	Leah
12	6-20	***Pl Sul***[b]	***P2Su2***	***P3***
	Guy 11	*p1su1*	*p2su2*	*p3*
28	12-7	*p1su1*	*p2su2*	*p3*
	12-20	*p1*[c]	*p2*	***P3***
29	12-7	*p1su1*	*p2su2*	*p3*
	12-26	***Pl Sul***	***P2Su2***	***P3***
31	12-26	***Pl Sul***	***P2Su2***	***P3***
	Guy 11	*p1su1*	*p2su2*	*p3*
33	31-1	***Pl Sul***	*p2su*	
	31-34	*p1su1*	***P2su***	
34	31-4	***Pl Sul***		
	31-16	*p1su1*		
41	31-3	*p1*	*p2*	*p3*
	31-10	*p1*	*p2*	***P3***
42	31-3	*p1*	*p2*	*p3*
	31-21	*p1*	*p2*	*p3*
56	28-2	*p1*	*p2su2*	***P3***
	29-4	*p1*	*p2*	***P3***
57	28-2	*p1su*	*p2su2*	***P3***
	29-2	***Pl Sul***	***P2Su2***	*p3*
61	29-15	*p1*	*p2*	*p3*
	29-19	*p1*	*p2*	*p3*
65	57-4	***Pl Sul***	***P2su2***	
	Guy 11	*p1su1*	*p2su2*	*p3*
66	57-10	***Pl Sul***	*p2*	*p3*
	Guy 11	*p1su1*	*p2su2*	*p3*
67	57-17	*p1*	***P2Su2***	***P3***
	Guy 11	*p1su1*	*p2su2*	*p3*
68	57-15	***Pl Sul***	*p2*	***P3***
	Guy 11	*p1su1*	*p2su2*	*p3*
69	57-9	*p1*	***P2Su2***	*p3*
	Guy 11	*p1su1*	*p2su2*	*p3*

[a] Based on the minimal number of genes needed to explain the observations.
[b] ***Psu*** = avirulent; ***PSu*** = virulent; *psu* = virulent; *pSu* = virulent.
[c] A blank indicates that the allele at the second locus can not be deduced.

segregation of pathogenicity on rice, avirulence/virulence on specific rice cultivars, and mating competence in subsequent generations suggest that the original cross was the equivalent of an interspecific or intergeneric cross (3,6). Guy 11 has its origin from rice in Guyana (9). Segregation is commonly distorted in interspecific crosses. Therefore, it is probable that progenies from 6-20 × Guy 11 may not represent a random sample of possible recombinants (3).

The segregation for avirulence/virulence on cultivar L202 can also be explained if one avirulence gene, *P2*, and one suppressor for that avirulence gene, *Su2*, are assumed. Segregation for avirulence on Leah can be explained by a single avirulence gene, *P3*, for 14 of the 16 crosses. Crosses 34 and 65 are the apparent exceptions. It is possible, however, that a different suppressor, *Su7*, which affects interactions with cultivar Newbonnet (data not shown), is also responsible for suppression of *P3* in crosses 34 and 65.

Two strains deserve special consideration. One progeny from cross 34 is unique. The parents for cross 34 gave intermediate infection types on cultivars Bluebelle and L202 and low infection type on Leah. One progeny, however, was highly virulent on each of these three cultivars, as well as on 12 other cultivars tested. The recovery of one progeny that was highly virulent on many cultivars on which the parents were either avirulent, intermediate in virulence, or virulent is an enigma. It is like no other strain in our stock collection. A postulated genotype for the parents of cross 34 for their interaction with cultivars L202 and Leah is not presented here. The second strain is 57-4. Segregation of avirulence/virulence on Leah appears to be due to a single locus, *P3*, with two alleles, except for strains 34-4 and 57-4. Strain 57-4 was clearly virulent on Leah, yet when crossed with Guy 11 gave two progenies that were avirulent on Leah. Cross 65 was expected to give only virulent progenies on Leah. The progenies that were avirulent on Leah were not the same progenies that were avirulent on Bluebelle and L202. None of the many crosses made with Guy 11 suggest that Guy 11 has, by itself, genes for avirulence on Leah. Genes for avirulence on Leah most likely came from 57-4, and these genes are expressed after being crossed with Guy 11.

The inheritance of avirulence/virulence on five additional cultivars in these same crosses provided evidence that the patterns observed for cultivars Bluebelle and L202 are not unique (data not shown). For four of five genes controlling avirulence/virulence on five other cultivars, it was necessary to postulate a second gene that controlled expression of each of the four avirulence/virulence genes.

Cross 57 gave progenies with a range of phenotypes on each of the three rice cultivars. Progenies that represented the range of infection types on each of the three cultivars were crossed to Guy 11, a common parent that is virulent on all three cultivars (crosses 65–69). The segregation in these crosses clearly shows that virulence on these three cultivars is controlled by different genes. For example, the recovery in crosses 66 and 68 of progenies that are avirulent on Bluebelle and none that are avirulent on L202 or Leah shows that avirulence on Bluebelle is controlled by genes different from those that control avirulence on L202 or Leah. These crosses also show that the infection type of the isolate crossed to Guy 11 did not predict if the progenies recovered would be avirulent on that cultivar. Lawrence et al have suggested that the progenies of *M. lini* that had intermediate infection types might have an avirulence gene plus a suppressor of the avirulence. An intermediate phenotype might result if the suppressor does not completely negate the expression of avirulence. Lawrence et al did not make crosses to test their hypothesis. Data presented herein, in particular, crosses 65–69, do not support the hypothesis that the intermediate infection types are due to suppression of the avirulence alleles.

The genetic basis of the range in infection types remains largely unsolved. Crosses between highly virulent cultures may give progeny that are highly virulent and also intermediate in virulence (Table 1; crosses 66 and 68). Crosses between isolates of intermediate virulence may give progenies that are highly avirulent to highly virulent (Table 1; cross 34). Crosses between isolates of intermediate virulence with isolates of high virulence (Table 1; crosses 65 and 69) can give progenies indistinguishable from crosses of two isolates with high virulence (Table 1; cross 67). An extensive series of test crosses to identify the genes responsible for the range in infection types and the environmental conditions that give maximum differences between the alternate alleles is now underway.

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