A Type of Stable Resistance to Blast Disease of Rice

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ABSTRACT

Pyricularia oryzae isolated from a few lesions appearing on certain resistant cultivars under epiphytotic conditions consistently produced only a few lesions when the isolates were inoculated back to the same cultivars. Pathogenicity tests on single conidial subcultures derived from these isolates showed that they consisted of many races. From one isolate, 43 races were identified from 189 single conidial subcultures. From other isolates, 28 races from 160 subcultures, 19 races from 52 subcultures, and eight races from 45 subcultures were found. Most of these races were nonpathogenic to the cul-

tivars from which they were isolated. Among the subcultures belonging to the same pathogenic races, many also did not infect the resistant cultivars while others produced only a few lesions. The resistance shown by these cultivars against the pathogenic isolates is the result of the constant change of the fungus into many races and the broad spectrum of resistance in the cultivars against most of the new races. The phenomenon seems to promise more stable resistance to blast. Phytopathology 61:703-706

Additional key words: variability of Pyricularia oryzae, varietal resistance to rice blast.

After more than 180 tests in the international blast nurseries in 26 countries during the last 6 years, several cultivars having a broad spectrum of resistance to blast have been identified (4, 6, 8). Two of these, Tetep and Carreon, were also tested in our blast nurseries more than 40 times during the past 8 years. They showed a resistant reaction, but sometimes a few Type-4 lesions (susceptible type) (5) occurred on the plants. The fungus in these lesions was isolated and then inoculated back to the two cultivars. In repeated tests, fewer lesions were produced on Tetep and Carreon than on the susceptible variety (Khao-teh-haeng 17) inoculated at the same time. The pathogenicity of seven of these isolates and their progenies was studied both qualitatively (pathogenic races) and quantitatively (number of lesions).

MATERIALS AND METHODS.—From the few Type-4 lesions appearing on the leaves of Tetep in our blast nursery, four cultures of *P. oryzae*, designated as FR-1, FR-78, FR-79, and FR-80, were isolated. From Carreon, one culture, designated as FR-13, was isolated. A number of monoconidial subcultures was obtained from each of the cultures: 160 from FR-1, 189 from FR-13, 45 from FR-78, 52 from FR-79, and 45 from FR-80.

In addition, 48 monoconidial subcultures were obtained from FR-1-138, a monoconidial subculture of FR-1, and 100 from FR-78-16, one of the most virulent monoconidial subcultures of FR-78 on Tetep. Thirty-four other isolates and reisolates from Tetep were also used.

All sub-subcultures were inoculated on Tetep, Carreon, and on the eight international and 12 Philippine differential cultivars (1, 2) at the same time.

Methods of isolation, culture, inoculation, and identification of pathogenic races were described earlier (7). To quantitatively evaluate resistance, we counted the numbers of both the Type-3 (intermediate type reaction) and Type-4 (susceptible type reaction) lesions

(5) on 20 plants. Normally, about 25 to 250 conidia are produced on Type-3 lesions each night for 1 week or less, while Type-4 lesions produced 2,000 to 6,000 conidia each night for about 2 weeks. Thus, Type-4 lesions produced vastly more conidia than Type-3 lesions. The Type-3 lesions are not epidemiologically significant, so for brevity they are not reported in the results. Lesions larger than standard Type 3 but smaller than Type 4 were also counted.

The blast nursery test followed the procedures for the international uniform blast nurseries (5). Lesions on 100 seedlings of each cultivar and the susceptible check cultivar in next row, 10-cm away, were counted every other day.

RESULTS.—Most of the 37 isolates from Tetep when inoculated back to Tetep produced 0 to less than 5 lesions/seedling, except two isolates which produced 14.1 (isolate FR-4A10) and 16.1 (FR-76-16) lesions (Fig. 1). No lesion was produced on Carreon. The average number of lesions on Tetep is 2.2/seedling in comparison with 32.7 on Khao-teh-haeng 17 (KTH), which is one of the most susceptible cultivars. Since KTH was inoculated at the same time with Tetep, yet had many more lesions, many of the conidia apparently failed to infect Tetep. Several other resistant cultivars were similarly inoculated with cultures isolated from them, and similar results were obtained.

Based upon their reactions on either the international or the Philippine differentials, the monoconidial subcultures of isolates from Tetep and Carreon, FR-1, FR-13, FR-78, FR-79, and FR-80, consisted of many pathogenic races. These races vary greatly in pathogenicity. Table 1 summarizes the results based upon the numbers of the 12 Philippine differential cultivars infected by the different races.

The 160 subcultures from FR-1 were separated into 28 races based upon Philippine differentials. The pathogenicity varied from one race which infected only two cultivars to other races which infected 11 or all 12

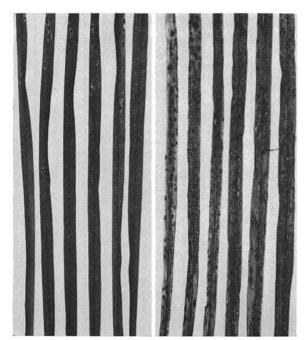


Fig. 1. Number of lesions produced on leaves of rice cultivars Tetep (6 at left) and Khao-teh-haeng 17 (6 at right) when inoculated with a virulent culture of *Pyricularia oryzae* isolated from Tetep.

differentials. The monoconidial subcultures from FR-13, FR-78, FR-79, FR-80 and from the sub-subcultures, FR-1-138 and FR-78-16, behaved similarly. The 189 monoconidial subcultures from FR-13 were separated into 43 races. Eight races were found in the 45 subcultures of FR-78, 19 races in the 52 subcultures of FR-79, and seven races in the 45 subcultures of FR-80. The 100 monoconidial subcultures from the single conidial culture of FR-78-16 consisted of 51 races. In the 48 subcultures of FR-1-138, we found 12 races.

The frequency of distribution of the subcultures among the races varied (Table 1). Often one or a few of the races had a large number of subcultures, but each isolate usually had a different predominant race. For instance, from FR-1, the predominant races were *P-8* and *P-12*; from FR-13, *P-15*; from FR-78, *P-92*; and from FR-79 and FR-80, *P-8*. The frequency of distribution of subcultures among the races is only an indication of the distribution, since relatively few of the large numbers of conidia produced in the cultures were tested.

The results of using the international differentials were very similar to the results obtained from using the Philippine differentials. Table 2 shows the number of pathogenic races of the monoconidial subcultures from the above seven isolates differentiated by the international differentials, by the Philippine differentials, and by combinations of the two sets of differentials and two complementary varieties, Tetep and Carreon (20 cultivars). Many more races were separated when more differential cultivars were used. Possibly, none of the monoconidial subcultures would have the same pathogenicity if enough additional differentials were used.

All subcultures produced fewer lesions on Tetep and Carreon than on KTH. Many subcultures failed to infect the cultivars from which they were originally isolated; e.g., 88% from isolate FR-1, 94% from FR-1-138, 2% from FR-78, 57% from FR-78-16, and 67% from FR-79. Among these, FR-78 was most pathogenic to Tetep. FR-78-16, however, one of the most pathogenic subcultures from FR-78, produced many nonpathogenic subcultures in its progeny. None of the subcultures of FR-13 produced any susceptible type lesions on Carreon, the original cultivar, though a few Type-3 lesions occurred. Possibly, the original lesion from which the isolate was obtained was a Type-4 minus lesion.

Most of the pathogenic subcultures produced less than five lesions/plant on Tetep and Carreon, while on KTH most produced more than 20 lesions. But even though KTH is one of the most susceptible cultivars we found, it seems to have some resistance to race groups of FR-78-16. In several instances, inoculations of FR-78-16 produced more lesions on varieties Caloro,

Table 1. Pathogenic races of *Pyricularia oryzae* derived from isolates FR-1, FR-1-138, FR-13, FR-78, FR-78-16, FR-79, and FR-80 grouped by the number of the Philippine differential cultivars infected

No. differential — cultivars infected	No. races derived from isolates: no. subcultures in each race group (in parentheses)						
	FR-1	FR-1-138	FR-78	FR-78-16	FR-79	FR-80	FR-13
1				1 (1)			
2	1(1)			1(1)			2 (11)
3	1(1)			6 (6)			6 (68)
4	4 (4)			7 (9)	5 (12)		7 (45)
5	7 (15)	2(2)		5 (7)	1(1)		9 (31)
6	5 (22)	5 (11)		7 (10)	5 (17)	2 (3)	8 (12)
7	1 (61)	1 (19)		6 (12)	2 (11)	1 (37)	6 (14)
8	3 (34)	3 (12)	2 (3)	6 (13)	2 (2)	2 (3)	4(7)
9	3 (17)	1 (4)	3 (6)	7 (11)	3 (5)	2 (2)	1 (1)
10		- (.)	1 (33)	3 (17)	1 (4)	2 (2)	1 (1)
11	2 (3)		2 (3)	2 (13)	- (1)		
12	1(2)		- (0)	2 (20)			
Total no. races	28	12	8	51	19	7	43
Total no. subcultures	160	48	45	100	52	45	189

TABLE 2. Number of pathogenic races of *Pyricularia* oryzae differentiated from the single conidial subcultures of seven single conidial parental isolates by inoculation on three different sets of differential cultivars

Isolate and total no. subcultures	By 8 inter- national differential cultivars	By 12 Philippine differential cultivars	By combination of 2 sets and Tetep & Carreon (20 cultivars)	
FR-1 (160)	20	28	59	
FR-1-138 (48)	6	12	22	
FR-13 (189)	20	43	95	
FR-78 (45)	3	8	11	
FR-78-16 (100)	23	51	63	
FR-79(52)	25	19	37	
FR-80 (45)	3	7	12	

Kataktara DA 2, and P.I. 8970(s) than on KTH. Furthermore, three of the 100 subcultures of FR-78-16 produced less than one lesion/seedling on KTH, so they are considered not pathogenic to KTH.

The number of pathogenic races, number of pathogenic subcultures, and average number of lesions produced by the subcultures of the seven cultures on Carreon, Tetep, and KTH are presented in Table 3. Carreon and Tetep consistently had significantly smaller numbers of lesions than KTH.

Despite the large number of pathogenic races and pathogenic subcultures from certain isolates, the number of lesions on Tetep or Carreon was nevertheless very small. For instance, from FR-1, 11 of the 28 races and 60 of the 160 subcultures were pathogenic to

Carreon, but very few lesions were produced. FR-78 was the most pathogenic isolate to Tetep; seven of eight races and 44 of 45 subcultures were pathogenic to it. Even so, Tetep had only small number of lesions/plant.

Several experiments were made in blast nurseries to measure the numbers of lesions that developed on Carreon, Tetep, and Tjeremas (susceptible check) inoculated with isolates FR-1, FR-78-16, and FR-13. The results consistently showed that under 300 lesions occurred on 100 seedlings of resistant varieties, while 7,000 to 8,000 lesions/100 seedlings were found on susceptible check. The data from one of the experiments are shown in Fig. 2.

Discussion.—The experiments further demonstrated the great pathogenic variability of the fungus reported earlier (3, 7). Such variation should be considered in working on varietal resistance, genetics of resistance, and other aspects of the disease.

In most studies of races of *P. oryzae*, the reaction is evaluated on a qualitative basis; i.e., a cultivar that has two or three susceptible lesions on each plant is considered as susceptible as another cultivar having 50 or 100 lesions. Total resistance to blast should be evaluated both qualitatively (susceptible or resistant) and quantitatively (the number of lesions or severity).

We also found that among the many isolates belonging to the same race, some infected Tetep or Carreon while others did not. Pathogenicity of the isolates in the same races may not behave similarly on cultivars other than the differentials.

Varieties Tetep and Carreon have been tested in our blast nursery over 40 times in 8 years. They have usu-

TABLE 3. Qualitative (pathogenic races) and quantitative (number susceptible lesions) pathogenicity of monoconidial subcultures of isolates FR-1, FR-1-138, FR-78, FR-78-16, FR-79, and FR-80 of *Pyicularia oryzae* from Tetep and FR-13 from Carreon, artificially inoculated on Tetep, Carreon, and Khao-teh-haeng 17 (KTH) cultivars

Isolate	Cultivar	No. races pathogenic to respective varieties	No. sub- cultures pathogenic to respective cultivars	Avg. no. lesions per plant by all subcultures	Avg. no. lesions per plant by pathogenic subcultures
FR-1	Carreon	11 (28) ⁿ	60 (160) ^b	0.3	1.1
	Tetep	5 (28)	19 (160)	0.1	1.4
	KTH	28 (28)	160 (160)	33.9	33.9
FR-1-138	Carreon	6 (12)	15 (48)	0.8	2.8
	Tetep	1 (12)	3 (48)	0.1	6.2
	KTH	12 (12)	48 (48)	56.6	56.6
FR-78	Tetep	7 (8)	44 (45)	5.2	6.1
	KTH	8 (8)	45 (45)	22.6	22.6
FR-78-16	Carreon	1 (51)	1 (100)	0.1	8.7
	Tetep	17 (51)	43 (100)	3.6	8.9
	KTH	48 (51)	97 (100)	17.4	18.0
FR-79	Carreon	1 (19)	1 (52)	0.01	0.5
	Tetep	11 (19)	17 (52)	0.7	2.5
	KTH	19 (19)	52 (52)	46.3	46.3
FR-80	Carreon	3 (7)	7 (45)	0.6	4.1
	Tetep	1 (7)	1 (45)	0.2	7.9
	KTH	7 (7)	45 (45)	47.9	47.9
FR-13	Carreon	0 (43)	0 (189)	0	0
	Tetep	17 (43)	43 (189)	0.1	0.8
	KTH	43 (43)	189 (189)	50.3	50.3

a Total no. races.

b Total no. subcultures.

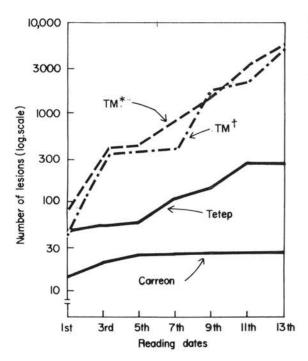


Fig. 2. Number of lesions per 100 rice seedlings on resistant cultivars (Tetep, Carreon) and on susceptible cultivar Tjeremas adjacent to Tetep (TM*) and adjacent to Carreon (TM+) inoculated with isolate (FR-78-16) of Pyricularia oryzae from Tetep in blast nursery.

ally shown a resistant reaction. Under very severe disease conditions, a few lesions have appeared. Infection of new hosts occurred because of the variability of the fungus. On the other hand, because of the constant change in pathogenicity, the fungus fails to build up the population of particular race or races efficiently on cultivars that have a broad spectrum of resistance. This is an inherent weakness of the fungus.

Only 19 out of the 160 monoconidial subcultures tested, or 12%, from FR-1 were pathogenic (producing one lesion/plant or more) to the original cultivar Tetep. Even when the subcultures were pathogenic, few lesions occurred on Tetep. The percentages of pathogenic subcultures from most other isolates were also low: 4% from FR-1-138, 25% from FR-79, and 2% from FR-80. The percentage of pathogenic subcultures from FR-78 was relatively high: 87%. However, only 37% of the progeny of the most pathogenic subculture, FR-78-16, were pathogenic.

Among the conidia naturally present in a field or blast nursery, hundreds of races may be represented. Sixty races were identified among 363 single conidia from our blast nurseries (9). On a susceptible variety such as KTH, nearly all conidia in contact with the plants produce a lesion, since almost all races can infect the cultivar. On resistant cultivars like Tetep and Carreon, however, only a few conidia belong to a pathogenic type that will produce lesions. The broader the

spectrum of resistance to the races, the fewer the lesions that develop.

The number of lesions that develop on resistant cultivars depends on the conidial population of the pathogenic races. The population distribution of each race is not uniform. At one time, more lesions may be found on the resistant cultivars when the population of one or more pathogenic races is high. However, since these pathogenic races tend to separate into many other races continuously, the population of the pathogenic races is diluted in each generation and does not build up rapidly, as shown for FR-78-16, which was the most pathogenic subculture in our study.

We also noticed that 37 isolates from Tetep and 99 out of 100 monoconidial subcultures from FR-78-16 did not infect Carreon. It may be possible to combine the resistance genes of two or more such resistant cultivars to further increase the spectrum of resistance.

From the above experiments, it seems possible to obtain more stable resistance to blast. Other resistant cultivars identified through the international blast nurseries (8) may also be evaluated for their quantitative resistance. These varieties apparently do not have the true horizontal resistance (10, 11). The stable resistance is mainly due to the constant change of the fungus into many new races and to the broad spectrum of resistance in these varieties against most of the new races. This is perhaps the first report of such host-pathogen relationship resulting in a more stable type of resistance.

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