Inheritance of Pathogenicity of Culture 70-1, Race 1, of *Puccinia recondita tritici*

Glen D. Statler

Professor, Department of Plant Pathology, North Dakota State University, Fargo, 58105.
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ABSTRACT

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Culture 70-1 (race 1) of *Puccinia recondita tritici* was selfed to study the inheritance of virulence. The 60 S_1 cultures resulting from selfing culture 70-1 were used to inoculate 13 isogenic wheat lines (*Lr*) and six tester cultivars. Segregation of S_1 cultures on *Lr1, Lr2, Lr2c, Lr3, Lr3ka, Lr17, Lr18,* and *Lr21,* fit a model in which single recessive genes conditioned virulence. Segregation for pathogenicity on Terenzio closely approached, but did not fit, a 3:1 ratio; virulence probably was conditioned by a single recessive gene. The recessive genes were inherited independently, but chi-square tests indicated linkage associations between *p2* and *p2c*, *p17* and *p18*, and *p3* and *pT*. Pathogenicity on Cultivar El Gaucho fit a model with two recessive genes for virulence. Culture 70-1 and all the S_1 cultures were avirulent on *Lr9, Lr10, Lr16, Lr19, Lr24,* and cultivars Waldron, Transec, and Kenya Farmer, indicating that the corresponding genes for pathogenicity were homozygous avirulent in culture 70-1, race 1.

Additional key words: heterozygosity, leaf rust, *Triticum aestivum.*

Leaf rust of wheat *(Triticum aestivum)* which is incited by *Puccinia recondita* Rob. ex. Desm. f. sp. *tritici* can become epidemic and severely reduce wheat yields. Roelfs *(personal communication)* reported many thousand hectares of wheat destroyed by a leaf rust epidemic in Mexico during 1977.

The development of cultivars containing major genes for resistance has been one of the most successful methods of controlling plant diseases (4). Unfortunately, that kind of resistance is not long lasting because most pathogens have the ability to change and attack previously resistant cultivars. This ability of the pathogen to change, combined with an ability to increase rapidly and become widely distributed places the rust fungi among the most potentially damaging plant pathogens (1). Flor (4) concluded that most changes of virulence in *Melampsora lini* were due to mutations followed by sexual recombination. Virulence changes would take place more rapidly in a heterozygous population of *P. recondita tritici* than in a homozygous one since a single recessive mutation for virulence would be expressed immediately without undergoing sexual recombination.

Previous studies have demonstrated that *P. recondita tritici* is heterozygous at certain loci (5,7). Studies of heterozygosity can be used to determine which genes are most useful in a breeding program. If cultures from the natural population are heterozygous for a pathogenicity gene, the corresponding gene for resistance probably would have only short-term value in a breeding program.

This study was conducted to determine the amount of heterozygosity, inheritance of virulence, and linkage relationships of genes for pathogenicity of *P. recondita tritici* race 1. This information is important in determining which host genes would provide the best combinations of resistance genes to incorporate into new cultivars.

MATERIALS AND METHODS

A culture of *P. recondita tritici* race 1, was purified by three successive single pustule isolations. Purity was evaluated on isogenic lines and differentials. These cultivars are listed in Table 1.

Teliospores were produced by injecting urediospores of race 1 into culms of moderately-resistant plants in the boot stage. Teliospores were conditioned to germinate by alternate wet-dry periods. After several cycles of conditioning the telia were suspended over *Thalictrum speciosissimum* Loefl. (meadow rue), the alternate host of *P. recondita tritici.* Selfing was conducted by separately transferring honeydew and pycniospores from one pycnium to another.

Since there are two mating types, only about half of the fertilized pycnia formed aeciospores. Aeciospores resulting from fertile crosses were used to inoculate Little Club wheat. Cultures were developed from single aecial clusters. These first self (S_1) cultures were used to inoculate isogenic lines and tester cultivars. Sixty S_1 cultures were used to inoculate the 13 isogenic lines and the six tester cultivars listed in Table 1.

The infection type expressed on each differential was classified on a standard scale of 0–4 at 10–12 days after inoculation. Infection types 0 to 2 were classified as avirulent and types 3 and 4 as virulent (5). Chi-square tests were used to determine the probabilities of the segregating cultures fitting hypothetical ratios. Chi-square tests for independence were used to determine if genes were independently inherited. The recombination values were estimated by the product method.

RESULTS AND DISCUSSION

Two of the S_1 cultures derived from selfing culture 70-1 of race 1, *P. recondita tritici* were avirulent (infection type 1) on Little Club wheat. I was unable to obtain sufficient urediospores to inoculate the isogenic lines; therefore, these S_1 cultures were not used in genetic analyses. However, this observation may indicate that Little Club has a gene for resistance or several minor genes that are sufficient to give resistance to a few weakly pathogenic progeny. The remainder of the S_1 cultures were virulent on Little Club. The infection types observed when the isogenic lines were inoculated with the S_1 cultures usually resembled the parental culture 70-1. However, a range of low infection types was observed, especially when a 0;1, 1 or 2 was observed on a cultivar inoculated with the parental culture 70-1. Samborski and Dyck (6) explained the range of resistant reactions in their studies by suggesting that the action of a gene for resistance and a gene for virulence could be influenced by modifiers in the genetic background of host, pathogen, or both.

The parental culture 70-1 and all the S_1 progenies tested were avirulent on *Lr9, Lr10, Lr16, Lr19, Lr24,* and cultivars Waldron, Transec, and Kenya Farmer. The failure to observe virulent segregants in S_1 cultures indicated that culture 70-1 was homozygous avirulent on those cultivars.

Segregation for pathogenicity occurred among the S_1 cultures of 70-1 on *Lr1, Lr2, Lr2c, Lr3, Lr3ka, Lr17, Lr18, Lr21,* El Gaucho, and Terenzio. Culture 70-1 was avirulent on these isogenic lines or cultivars. The S_1 cultures segregated approximately three avirulent

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to one virulent on *Lr1, Lr2, Lr2c, Lr3, Lr3ka, Lr17, Lr18, Lr21,* and Terenzio (Table 1). Since the *P* values were all >0.05 except for Terenzio, single recessive genes for virulence were indicated.

With one more culture virulent on Terenzio, the data would fit a 3:1 ratio. A single recessive gene may condition virulence and the failure to fit a 3:1 ratio may be due to chance alone. A single recessive gene for virulence was previously reported for Terenzio and labeled *pT* (6).

Single recessive genes for virulence *p1, p2,* and *p3* corresponding to host genes *Lr1, Lr2,* and *Lr3* were previously reported in race 1 of *P. recondita tritici* by Samborski and Dyck (5).

Segregation for pathogenicity on El Gaucho would fit a 15:1 ratio for two recessive genes. It is possible, therefore, that El Gaucho contains a second gene for resistance in addition to the single gene previously reported (6) or that two genes for virulence are required to overcome the one gene for resistance in El Gaucho.

All the single recessive genes for virulence were inherited independently except for *p2* and *p2c, p17* and *p18,* and *p3* and *pT* for which the chi-square test indicated linkage (Table 2).

Results of this study indicate a single recessive gene for virulence on *Lr2* which apparently was linked closely to a single gene for virulence on *Lr2c* ($P < 0.005$) (Table 2) and appeared to be coupled. These two genes for resistance were reported to be allelic in the host (6), but this study indicates that the two genes for virulence, *p2* and *p2c,* were closely linked on the same chromosome in the fungus. The percentage recombination estimate was 5.4 ± 3.0% between *p2* and *p2c.* Dyck and Samborski (3) reasoned that there was a single recessive gene *p2* for virulence on the various alleles at the *Lr2* locus and another gene (s) for virulence or a gene that modifies or inhibits the action of the *p2* alleles. A single recessive gene for virulence plus a modifier may be the most plausible explanation of the gene(s) for virulence on host genes *Lr2* and *Lr2c* since all the S_1 cultures virulent on *Lr2* were also virulent on *Lr2c* and only two cultures were virulent on *Lr2c* but not *Lr2.*

Genes for virulence on *Lr17* and *Lr18* were linked in coupling with an estimated percentage recombination of 3.0 ± 2.3% (Table 2). Earlier, I (7) reported linkage of these loci in culture 73-47, but Dyck and Samborski (2) reported that the genes for virulence *p17* and *p18* segregated independently in their studies. Host genes *Lr17* and *Lr18* are not linked and apparently occur on different chromosomes. This study indicates that the genes for virulence *p17* and *p18* are closely linked and on the same chromosome in my culture of race 1 ($P < 0.005$).

Genes for virulence on *Lr3* and *LrT* were linked in coupling with an estimated percentage recombination of 11.0 ± 4.3%. The genes for resistance *Lr3* and *LrT* are reported to be independently inherited in the host (6). However, linkage between *p3* and *pT* indicates that these genes for virulence are on the same chromosome in the fungus.

The culture of race 1 that was studied was heterozygous for virulence on 10 of the 19 cultivars used in the study. Other cultures also have been reported to be heterozygous (2,7). This study supports the view that the heterozygosity in *P. recondita tritici* accounts for the vast amount of pathogenic variation in the natural population. In a heterozygous population, a single deletion or sexual recombination could cause virulence at several loci if the loci are linked, making several host genes ineffective.

Combinations of genes for resistance are vulnerable when the corresponding genes for virulence are closely linked in coupling, so these combinations should be avoided in breeding programs. How-

TABLE 1. Segregation of S_1 cultures derived from selfing culture 70-1 of *Puccinia recondita tritici* race 1

Host cultivar or isogenic Line	Response to culture 70-1	Number of S_1 cultures								Expected ratio[b]	Goodness of fit
		Infection type						Number cultures[a]			
		0 to 0;	0;1 to 0;2	1	2	3	4	Avirulent	Virulent		
Lr1	0;	36	3	0	0	4	17	39	21	3:1	$P > 0.05$
Lr2	0;	36	6	0	0	8	10	42	18	3:1	$P > 0.25$
Lr2c	0;	29	8	3	0	5	15	40	20	3:1	$P > 0.10$
Lr3	0;1	24	20	5	2	4	5	51	9	3:1	$P > 0.05$
Lr3ka	2	11	19	7	16	7	4	49	11	3:1	$P > 0.05$
Lr9	0	58	2	0	0	0	0	60	0	HA	...
Lr10	0;1	35	21	3	1	0	0	60	0	HA	...
Lr16	2	1	7	24	28	0	0	60	0	HA	...
Lr17	0;1	10	25	6	0	11	8	41	19	3:1	$P > 0.10$
Lr18	0;1	9	26	6	2	9	8	43	17	3:1	$P > 0.50$
Lr19	0	60	0	0	0	0	0	60	0	HA	...
Lr21	0;1	25	17	8	1	7	2	51	9	3:1	$P > 0.05$
Lr24	0;1	31	21	7	1	0	0	60	0	HA	...
Waldron	0;	57	3	0	0	0	0	60	0	HA	...
Transec	12	18	36	3	3	0	0	60	0	HA	...
El Gaucho	0;	44	7	2	4	1	2	57	3	15:1	$P > 0.50$
Terenzio	0;	38	11	0	3	6	2	52	8	3:1	$P < 0.05$
Kenya Farmer	0;	55	5	0	0	0	0	60	0	HA	...
Little Club	4	0	0	0	0	0	60	0	60	HV	...

[a]Avirulent = 0 to 2; Virulent = 3 to 4.
[b]HV = homozygous virulent; HA = homozygous avirulent.

TABLE 2. *P* values for chi-square tests for independence of genes for virulence in cultures obtained from selfing culture 70–1, *Puccinia recondita tritici* race 1

	p1	*p2*	*p2c*	*p3*	*p3ka*	*p17*	*p18*	*p21*	*pT*
p1		$P > 0.50$	$P > 0.10$	$P > 0.05$	$P > 0.10$	$P > 0.05$	$P > 0.05$	$P > 0.05$	$P > 0.50$
p2			$P < 0.005$	$P > 0.10$	$P > 0.50$	$P > 0.50$	$P > 0.75$	$P > 0.10$	$P > 0.10$
p2c				$P > 0.10$	$P > 0.25$	$P > 0.50$	$P > 0.75$	$P > 0.10$	$P > 0.10$
p3					$P > 0.10$	$P > 0.10$	$P > 0.10$	$P > 0.05$	$P < 0.005$
p3ka						$P > 0.25$	$P > 0.25$	$P > 0.10$	$P > 0.50$
p17							$P < 0.005$	$P > 0.10$	$P > 0.05$
p18								$P > 0.10$	$P > 0.50$
p21									$P > 0.75$
pT									

ever, more data are needed before this concept can be applied to the natural population of *P. recondita tritici*.

Data from this study are compatible with the gene-for-gene theory outlined by Flor (4); the segregation on the single gene lines always indicated a single gene for virulence corresponding to the single gene for resistance in the single-gene lines. One exception to this was cultivar El Gaucho in which race 1 of *P. recondita tritici* segregated for two recessive genes but only one gene for resistance was reported previously. El Gaucho may have an additional gene for resistance to our culture of race 1, or two genes for virulence may be required to overcome the single gene in El Gaucho.

Other exceptions to the gene-for-gene theory also are noted when data from this study are compared to those of previous studies. For example, Samborski and Dyck (5) found virulence to Mediterranean and Democrat, (which contain *Lr3*) to be dominant in race 161 of *P. recondita tritici* and recessive in race 1. This study also indicated a recessive gene for virulence in race 1. Data from my previous study with culture 73-47, *P. recondita tritici* indicated that a single dominant gene conditioned virulence on *Lr2c* (7), but data from this study indicated a single recessive gene conditioning virulence in race 1. These apparent inconsistencies may indicate that different loci are involved in different cultures to overcome the resistance in *Lr2c* and *Lr3*.

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