Inheritance of Virulence of Culture 73-47 Puccinia recondita

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ABSTRACT

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Puccinia recondita, culture 73-47, virulent on *Triticum aestivum* 'Transfer', was selfed to study the inheritance of virulence. The 62 S_1 cultures resulting from selfing culture 73-47 were used to inoculate 11 isogenic wheat lines (*Lr*) and three tester cultivars. Culture 73-47 and all S_1 cultures were virulent on *Lr3* and *Lr9*, indicating that these genes were homozygous for virulence in the pathogen. Segregation of S_1 cultures on *Lr2c* and *Lr10* fit a model in which single dominant genes conditioning virulence were independently inherited. Culture 73-47 and all the S_1 cultures were avirulent on *Lr16, Lr19,* and Transec. Segregation ratios indicated that single recessive genes conditioned virulence on *Lr2, Lr17, Lr18, Lr24,* Waldron, and El Gaucho. The recessive genes conditioning virulence were independently inherited except between *Lr2* and Waldron and between *Lr17* and *Lr18* where linkage was indicated. Virulence on *Lr1* fit a model with two recessive genes for virulence.

Additional key words: leaf rust, virulence genes, segregation ratio, S_1 cultures, infection type, heterozygosity.

Losses from leaf rust caused by *Puccinia recondita* Rob. ex. Desm. f. sp. *tritici* have been reduced during the past 5-10 yr by growing resistant cultivars and by the use of fungicides. In 1965, six wheat cultivars were listed as resistant to all known, natural, uredial populations of *P. recondita* in North America (8). Six years later Shaner et al. (6) reported virulence on Transfer, one of the three remaining universally-resistant cultivars at that time. There are several examples of resistant cultivars rusting owing to changes in the natural *P. recondita* population (7). If most changes are due to mutations in the pathogen, as reported by Flor for *Melampsora lini* (3), virulence changes would take place more rapidly in a heterozygous population. Samborski and Dyck (4) reported a surprising degree of heterozygosity in *P. recondita*. This vast heterozygosity could explain much of the pathogenic variability found in *P. recondita*. In a heterozygous population of *P. recondita,* a single mutation for virulence would be expressed since the other dicaryotic nucleus would already possess a recessive virulence gene. Recombination via the sexual or parasexual cycle, although perhaps not as common as mutations, could also explain pathogenic changes in a heterozygous population.

This study was undertaken to determine the heterozygosity, and the inheritance of virulence and allelism of *P. recondita* culture 73-47 which is virulent on Transfer and *Lr9*.

MATERIALS AND METHODS

Puccinia recondita culture 73-47 was purified by three successive single-pustule isolations, and increased on host cultivar *Lr9*. Purity was checked on isogenic lines and differentials.

Teliospores were produced by injecting urediospores into culms of moderately-resistant plants in the boot stage (4). These plants were grown in greenhouses (19 ± 4 C) and teliospores collected while plants were still green.

Teliospores were conditioned to germinate by alternate wet-dry periods. After several cycles the telia were suspended over *Thalictrum speciosissimum* Loefl. (meadow rue), the alternate host of *P. recondita*. Spermatia from resulting spermogonial infections was separately transferred from one spermogonium to another spermogonium.

Because there were two mating types, only about half the spermatized spermogonia formed aeciospores. Aeciospores resulting from fertile crosses were used to inoculate Little Club wheat. Cultures were developed from single aecial clusters. The cultures were used to inoculate isogenic lines and tester cultivars. Sixty-two S_1 cultures were used to inoculate the 11 isogenic lines and three tester cultivars.

The infection type expressed on each differential was classified on the standard scale of 0-4, 10-12 days after inoculation. Infection types 0 to 2 were classified as avirulent and types 3 and 4 as virulent (4). 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 (4).

RESULTS AND DISCUSSION

One of the first self (S_1) cultures, resulting from selfing culture 73-47 of *P. recondita* was avirulent (infection type 1 to 2) on Little Club. I was unable to obtain sufficient

urediospores to inoculate the single-gene lines; therefore, this S_1 culture was not used in genetic ratios.

Culture 73-47 and all the S_1 cultures resulting from selfing culture 73-47 were virulent on isogenic lines that contained *Lr3* and *Lr9*. This indicated that culture 73-47 contained homozygous genes for virulence at the *Lr3* and *Lr9* loci (Table 1). A single recessive gene for virulence on *Lr3* labeled *p3* was previously reported (4).

Although the infection types observed when the isogenic lines were inoculated with S_1 cultures normally resembled the parental culture 73-47, a range of resistant infection types were observed (Table 1).

Samborski and Dyck (5) have observed a range of resistant reactions on most lines containing single genes for resistance. They explained these reactions by the fact that the action of a gene for resistance and a gene for virulence was influenced by modifiers in their own genetic background. These modifiers could explain the range of infection types observed for many of the resistant reactions in the current study. The variation in greenhouse temperatures during the course of the study could also affect infection types.

Segregation for virulence among the S_1 cultures was detected on several cultivars which were susceptible to 73-47. Segregation for virulence on cultivars containing *Lr2c* and *Lr10* fit a model with a single dominant gene for virulence.

Samborski and Dyck (4, 5) reported that dominant genes were not common in *P. recondita,* but reported dominant genes for virulence on *LrB, Lr3* and *Lr14b*. They (4) explained segregation of a culture of race 161, virulent on *Lr2c* by assuming that race 161 was heterozygous for gene *p2* and also for a gene that inhibited the expression of avirulence of *p2* on the Loros allele for resistance. A second gene inhibiting avirulence of *p2* may also be operating in this study since all cultures virulent on *Lr2* were also virulent on *Lr2c,* whereas many S_1 cultures were virulent on *Lr2c* but not *Lr2*.

The parental culture 73-47 and all the S_1 progenies tested were avirulent on *Lr16, Lr19,* and Transec. The failure to observe virulent segregants in S_1 cultures indicated that culture 73-47 was homozygous avirulent on Transec and the cultivars containing the *Lr16* and *Lr19* genes.

Segregation for virulence was found among the S_1 cultures of 73-47 on cultivars Waldron, El Gaucho, and cultivars containing the *Lr2, Lr17, Lr18,* and the *Lr24* genes. Since culture 73-47 was avirulent on these cultivars and the S_1 cultures segregated approximately three avirulent to one virulent, single recessive genes for virulence were indicated (Table 1).

The single genes for virulence on *Lr2, Lr17, Lr18,* and El Gaucho previously have been assigned symbols *p2, p17, p18,* and *pEG* (4, 5) to correspond to genes for resistance in the host. The symbols *p24* and *pW* should be used to represent genes for virulence on *Lr24* and Waldron.

All the single recessive genes in culture 73-47 that condition virulence on *Lr2, Lr17, Lr18, Lr24,* El Gaucho, and Waldron appeared to be independently inherited except those between *Lr2* and Waldron and between *Lr17* and *Lr18* where the chi-square test indicated linkage ($P < 0.005$).

The chi-square test for independence also indicated linkage between *p17* and *p24*, and between *p18* and *p24*. Since only one class caused most of the variation, the non-significant chi-square ($P < 0.010$) may be variation due to chance only and not linkage.

The relatively high chi-square value between *p2* and *pW* ($P < 0.005$) appeared to be due to a higher-than-normal number of cultures avirulent on Waldron, but virulent on *Lr2* and a higher-than-expected number virulent on both host genes. The percentage recombination estimate was $13.9 \pm 4.82\%$ between *p2* and *pW*.

With only a few exceptions, all cultures virulent on *Lr17* were also virulent on *Lr18*. The chi-square value was high ($P < 0.005$) and the percentage recombination was $6.1 \pm 3.18\%$ between *p17* and *p18*. The genes for virulence appear to be coupled.

TABLE 1. Segregation of S_1 cultures derived from selfing culture 73-47 *Puccinia recondita tritici*

Host, cultivar, or isogenic line	Response to culture 73-47	Number of S_1 cultures: Infection type							Avirulent	Virulent	Expected ratio	Goodness of fit
		0 to 0;	0; 1	0; 2	1	2	3	4				
Lr1	0;	22	0	9	0	1	1	29	32	30	9:7	$P > 0.25$
Lr2	0; 1	21	2	4	2	12	0	21	41	21	3:1	$P > 0.10$
Lr2c	4	2	3	7	0	5	7	38	17	45	1:3	$P > 0.10$
Lr3	4	0	0	0	0	0	7	55	0	62	HV[b]	...
Lr9	4	0	0	0	0	0	10	52	0	62	HV	...
Lr10	3	2	1	2	1	8	14	34	14	48	1:3	$P > 0.50$
Lr16	2	4	0	3	0	55	0	0	62	0	HA[b]	...
Lr17	0; 1	10	5	15	2	9	5	16	41	21	3:1	$P > 0.10$
Lr18	0; 1	17	5	9	1	9	4	17	41	21	3:1	$P > 0.10$
Lr19	0;	60	2	0	0	0	0	0	62	0	HA	...
Lr24	0; 1	24	8	6	0	5	3	16	43	19	3:1	$P > 0.25$
Waldron	0; 1	30	5	10	1	6	3	7	52	10	3:1	$P > 0.10$
Transec	0; 1	38	8	11	0	5	0	0	62	0	HA	...
El Gaucho	0; 1	28	4	7	1	1	8	13	41	21	3:1	$P > 0.10$

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

Dyck and Samborski (1) previously found virulence of race 11 conditioned by a single recessive gene, *p17*. They found race 11 heterozygous for a single gene *p18* which was partially dominant. The two genes for virulence were inherited independently. In this study, single recessive genes conditioned virulence on *Lr17* and *Lr18* and were apparently closely linked.

The only clear cases of linkage previously reported for *P. recondita* was between *p3ka* and *pT* (Terenzio) and between *p3* and *p14a* (5). Results of this study would support the observation of Samborski and Dyck (5) that the limited linkage suggested that genes for virulence in *P. recondita* are not clustered at specific locations.

Culture 73-47 also was avirulent on cultivars containing *Lr1*. The data would fit a 9:7 ratio for two recessive genes ($P > 0.50$). However, unless a second gene for resistance was contained in *Lr1*, two recessive genes in the parasite would be interacting with one gene in the host, a contradiction to the gene-for-gene relationship (2). A single recessive gene in race 1 which conditioned resistance on *Lr1* has been previously reported and labeled *p1* (4).

Samborski and Dyck previously reported a surprising amount of heterozygosity in cultures of race 1, 15, and 161 (4). In the current study, *P. recondita* culture 73-47 was heterozygous on 10 of the 15 isogenic lines and cultivars studied. This heterozygosity probably accounts for the vast amount of pathogenic variation in the natural *P. recondita* population (7). In a heterozygous population, a single mutation would be expressed as virulent whereas in a population containing homozygous genes for virulence, two mutations or a single mutation followed by genetic recombination would be required for the change to be expressed.

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