Inheritance of Virulence of *Puccinia recondita* f. sp. *tritici* on Durum and Spring Wheat Cultivars

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Published with the approval of the director of the North Dakota Agricultural Experiment Station as Journal Series Paper 1133.
Accepted for publication 21 May 1981.

ABSTRACT

Statler, G. D. 1982. Inheritance of virulence of *Puccinia recondita* f. sp. *tritici* on durum and spring wheat cultivars. Phytopathology 72:210-213.

Cultures 70-1 and 71-112 *Puccinia recondita* f. sp. *tritici* were crossed and the resulting F_1 selfed to study the inheritance of virulence on several isogenic lines of *Triticum aestivum* and cultivars of *T. durum*. Segregation of F_2 cultures on isogenic lines with genes *Lr*1, *Lr*2, *Lr*3*ka*, *Lr*11, *Lr*17, *LrEG*, and *LrT* fit a model in which single recessive genes conditioned virulence. Segregation on the durum cultivars Botno, Calvin, Edmore, Rugby, Ward, and Vic also fit a single recessive gene model. Segregation on Wells was closer to a 9:7 than a 3:1 ratio. Most of the single recessive genes for virulence were inherited independently except for a rather close linkage between *p*11 and *pEG* and linkage between *p*1 and *pEG*. Several associations were detected between genes for virulence on the durum cultivars. Genes for virulence on Edmore were apparently linked to those for virulence on *Lr*11, *LrEG*, Botno, and Calvin. Genes for virulence on Rugby were linked to those for virulence on *LrT*, Botno, and Edmore. Genes for virulence on Ward were linked to those for virulence on Botno, Edmore, and Rugby. Linkage was also indicated between genes for virulence on Botno and those for virulence on Calvin, Edmore, Rugby, and Ward. The F_2 segregations that fit 3:1 ratios on the durums also fit a 1:2:1 ratio when the infection types 2 and 2+ were placed in the intermediate category. Segregation for virulence on lines with *Lr*10, *Lr*16, and cultivars 5534, Waldron, and Olaf fit a digenic recessive ratio. These segregations on cultivar 5534 and *Lr*10 fit a ratio for two gene pairs in common. The F_2 cultures segregated for two recessive genes for virulence on the durum cultivars Crosby, Golden Ball, Leeds, Rolette, and Stewart 63. Segregation for virulence on the durums Cando and Wells and the *T. aestivum* line *Lr*3 fit a ratio for two recessive genes with either or both homozygous genes conditioning virulence. Segregation on the durum cultivar D6618 indicated a single dominant gene for virulence.

Additional key words: Leaf rust, *T. aestivum*, *T. durum*, linkage.

Leaf rust incited by *Puccinia recondita* Rob. ex. Desm. f. sp. *tritici* is one of the most serious diseases of wheat (*Triticum aestivum*) in the upper Mississippi Valley (10,11). Although the disease has not been as severe on durum wheats (*T. durum* Desf.), resistance has not been permanent because genetic variation in the natural rust population allows the fungus to increase rapidly on new wheat cultivars, which are not resistant to all races of the parasite. Several workers have studied the inheritance of *P. recondita* resistance in *T. aestivum* (2,3,4), but few have studied the inheritance of resistance by *T. durum* to this pathogen (1,6,9). In the lines D56-1 and D6733, resistance to a culture of UN race 1 was reported by Rashid et al (6) to be conditioned by one recessive gene. Two recessive genes were reported for the durum cultivars Ramsey and Leeds (6,9). Abdallah (1) found that seedling resistance to UN2 of *P. recondita* was conditioned by two recessive genes in *T. durum* cultivars Leeds, Ramsey, Stewart 63, and D56-1 (1). The fact that Rashid et al (6) found one gene in D56-1 using UN race 1 and Abdallah (1) found two genes using UN race 2 is difficult to explain because more genes should be evident using a culture with a narrow range of virulence, such as UN race 1. Abdallah (1) was also successful in transferring seedling resistance from several durums to hard red spring wheats. One such line, 5534, with genes for resistance from the durum Ramsey has been used in this study.

Most studies of virulence of *P. recondita* have involved virulence on single gene lines of *T. aestivum* (2,3,4,5,11). This study concerns the inheritance of virulence of *P. recondita* on several durum and hard red spring wheat cultivars.

MATERIALS AND METHODS

Culture 70-1 of *P. recondita*, virulent on only one isogenic line (Table 1), and 71-112, virulent on six isogenic lines of *T. aestivum*, were purified through three successive single-pustule isolations. Purity was evaluated on isogenic lines and differential cultivars of *T. aestivum*. Infection types are listed in Table 1.

Teliospores were produced by injecting urediospores of each culture into culms of moderately resistant plants of *T. aestivum* 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*. Honeydew and pycniospores were transferred from a pycnium of culture 70-1 to a pycnium of culture 71-112. Aeciospores resulting from the cross were used to inoculate Little Club wheat, and the purity of the resulting urediospore culture labeled X59 was evaluated on lines of *T. aestivum* and *T. durum* listed in Tables 1 and 2.

Teliospores of the F_1 culture X59 were produced, conditioned to germinate, and suspended over meadow rue. Selfing to produce F_2 cultures was accomplished by separately transferring honeydew and pycniospores from one pycnium to another. Seventy-two single-aecial cultures from selfing X59 were used to inoculate the isogenic lines of *T. aestivum* and cultivars of *T. durum* listed in Tables 1 and 2.

Infection types were classified on a standard scale of 0–4 at 10–12 days after inoculation. Infection types 0–2 were classified as avirulent and types 3 and 4 as virulent (7). For some analyses, the type 2 or 2+ reactions were classified as intermediate. Chi-square tests were used to estimate probabilities for segregation ratios and tests for independence. The recombination values were estimated by the product method.

RESULTS

The parental cultures (70-1 and 71-112), the F_1 (X59), and all the F_2s were avirulent on cultivars with genes *Lr*9, *Lr*19, *Lr*21, and cultivars Agent and Transec, indicating that X59 was homozygous avirulent on those cultivars (Table 1).

Segregation occurred in the F_2 on cultivars with genes *Lr*1, *Lr*2, *Lr*3, *Lr*3*ka*, *Lr*10, *Lr*11, *Lr*16, *Lr*17, *LrEG*, *LrT*, and cultivars Waldron, Olaf, and 5534. The ratios approximated three avirulent to one virulent on cultivars with genes *Lr*1, *Lr*2, *Lr*3*ka*, *Lr*11, *Lr*17, *LrEG*, and *LrT*. Because probability values were all >0.05, single recessive genes for virulence were indicated (Table 1).

Single recessive genes for virulence corresponding to host genes

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0031-949X/82/02021004/$03.00/0

TABLE 1. Segregation for virulence on *Triticum aestivum* cultivars among F_2 cultures derived from selfing culture X59, the F_1 of a cross between *Puccinia recondita* f. sp. *tritici* culture 70-1 and culture 71-112

Host isogenic line or cultivar	Response of parent		F1	Number of F2 cultures of infection type								Expected ratio[b]	Goodness of fit (*P*)
	70-1	71-112	X59	0–0;	0;1–0;2	1	2	3	4	Avirulent[a]	Virulent		
*Lr*1	0; 1	4	0	56	3	0	0	3	10	59	13	3:1	>0.10
*Lr*2	0;	4	0	50	3	1	0	3	15	54	18	3:1	>0.99
*Lr*3	0; 1	0; 3	4	14	12	14	3	15	14	43	29	9:7	>0.50
*Lr*3ka	0;	0; 1	0;	17	11	13	10	12	9	51	21	3:1	>0.25
*Lr*9	0;	0;	0	66	6	0	0	0	0	72	0	HA	–
*Lr*10	0;	4	2+	22	20	19	6	4	1	67	5	15:1	>0.75
*Lr*11	2+	4	12	5	9	25	21	6	6	60	12	3:1	>0.10
*Lr*16	2N	2+	2	1	3	14	48	5	1	66	6	15:1	>0.25
*Lr*17	01	4	2+	8	17	24	4	10	9	53	19	3:1	>0.75
*Lr*19	0;	0;	0;	72	0	0	0	0	0	72	0	HA	–
*Lr*21	0; 1	2+	0;	19	26	19	8	0	0	72	0	HA	–
*Lr*23	3–	12	4	0	0	0	0	37	35	0	72	HV	...
*Lr*EG	2+	3–	12	9	16	21	14	5	7	60	12	3:1	>0.10
Agent	0; 1	12	01	26	33	13	0	0	0	72	0	HA	...
*Lr*T	2+	0; 2	2+	5	10	11	22	18	6	48	24	3:1	>0.10
Transec	0; 2	0; 2	0; 2	21	31	16	4	0	0	72	0	HA	–
Waldron	0;	2+	0;	54	11	2	2	3	0	69	3	15:1	>0.25
Cv 5534	0;	12	1C	45	16	3	4	3	1	68	4	15:1	>0.75
Olaf	0	12	0;	44	7	1	0	1	1	52	2	15:1	>0.25
Thatcher	4	4	4	0	0	0	0	0	72	0	72	HV	–

[a]Avirulent = 0–2 and virulent = 3–4.
[b]HV = homozygous virulent, HA = homozygous avirulent. *P* values not provided when cultures all virulent or avirulent.

TABLE 2. Segregation for virulence on *Triticum durum* cultivars among F_2 cultures derived by selfing the F_1 culture (X59) of a cross between *Puccinia recondita* f. sp. *tritici* culture 70-1 and culture 71-112

Durum cultivar	Response of parent		F1	Number F2 cultures infection type								Expected ratio[b]	Goodness of fit (*P*)
	70-1	71-112	X59	0–0	0;1–0;2	1	2	3	4	Avirulent[a]	Virulent		
Botno	3	12	2+	3	6	8	39	16	0	56	16	3:1	>0.50
Calvin	2+	2+	12	0	6	17	34	11	4	57	15	3:1	>0.25
Cando	2	3–	2+	1	3	3	28	28	9	35	37	9:7	>0.10
Coulter	2	02	12	8	20	32	12	0	0	72	0	HA	–
Crosby	2	12	2+	6	12	21	28	5	0	67	5	15:1	>0.75
D56-1	2+	12	01	6	23	27	16	0	0	72	0	HA	–
D6618	4	4	4	1	2	3	6	25	35	12	60	1:3	>0.10
D6733	2	2+	12	10	22	27	13	0	0	72	0	HA	–
Edmore	2+	2+	2+	4	4	6	33	23	2	47	25	3:1	>0.05
Golden Ball	2	2+	2+	2	8	19	35	8	0	64	8	15:1	>0.05
Leeds	12	3–	2+	5	9	15	35	7	1	64	8	15:1	>0.05
Ramsey	21	12	1	17	27	28	0	0	0	72	0	HA	–
Rolette	2+	2+	2	9	12	17	31	3	0	69	3	15:1	>0.25
Rugby	2	12	2+	6	9	17	28	12	0	60	12	3:1	>0.10
Stewart 63	2	12	2+	4	18	31	16	3	0	69	3	15:1	>0.25
Ward	2+	3–	2+	7	6	10	32	17	0	55	17	3:1	>0.75
Wells	2+	3	2+	2	4	7	33	24	2	46	26	9:7	>0.10
Vic	3–	2+	2+	1	4	14	29	14	0	48	14	3:1	>0.10

[a]Avirulent = 0–2 and virulent = 3–4.
[b]HV = homozygous virulent, HA = homozygous avirulent. *P* values not provided when cultures all virulent or avirulent.

TABLE 3. Chi-square test for independence between genes for virulence in F_2 cultures of *Puccinia recondita* f. sp. *tritici*

Genes[a] for virulence	*P* value	Recombination estimate (%)	Standard error (%)
*p*1 vs *pEG*	<0.025	26.5	6.2
*p*11 vs *pEG*	<0.005	7.0	3.1
p3ka vs *pT*	<0.005	21.0	5.5
*p*11 vs *pEd*	<0.050	29.1	6.6
*p*11 vs *pWe*	<0.050	30.1	6.7
pEG vs *pEd*	<0.025	29.5	6.6
pEG vs *pWe*	<0.050	30.1	6.7
pT vs *pRu*	<0.025	28.5	6.5
pBo vs *pCa*	<0.005	25.8	6.1
pBo vs *pEd*	<0.005	14.0	4.4
pBo vs *pRu*	<0.005	24.5	6.0
pBo vs *pWa*	<0.005	16.0	4.7
pCa vs *pEd*	<0.100	27.1	6.3
pCa vs *pWe*	<0.005	22.6	5.7
pEd vs *pRu*	<0.005	23.0	5.8
pEd vs *pWa*	<0.005	23.6	5.8
pEd vs *pWe*	<0.005	17.5	5.0
pRu vs *pWa*	<0.005	8.5	3.4
pRu vs *pWe*	<0.005	19.5	5.3
pWa vs *pWe*	<0.005	14.0	4.4

[a] Virulence genes correspond to resistance genes from Edmore (Ed), Wells (We), Rugby (Ru), Botno (Bo), Calvin (Ca), Ward (Wa).

*Lr*1, *Lr*2, *Lr*3, *Lr*3*ka*, *Lr*10, *Lr*11, *Lr*17, *LrEG*, *LrT*, and the resistance gene of cultivar Waldron had been reported previously (2,4,7,8,10,11). These genes for virulence were labeled *p*1, *p*2, *p*3, *p*3*ka*, *p*10, *p*11, *p*17, *pEG*, *pT*, and *p Waldron* to correspond with the respective host genes.

Segregation for virulence on cultivars with *Lr*10, *Lr*16, and cultivars Waldron, 5534, and Olaf fit the 15:1 ratio expected for two recessive genes (all *P* values >0.05) (Table 1). Segregation ratios for virulence on both *Lr*10 and cultivar 5534 fit a ratio for the same two genes for virulence on each ($P > 0.25$). Chi-square tests did not demonstrate any associations for the other two gene combinations.

Segregation for pathogenicity on *Lr*3 fit a 9:7 ratio with $P > 0.05$ for duplicate recessive epistasis, in which either homozygous gene pair would cause virulence. Both parental cultures, the F_1 and all F_2 cultures, were avirulent on durum cultivars Coulter, D56-1, D6733, and Ramsey. The lack of virulent segregants in the F_2 cultures indicated that X59 was homozygous avirulent on these cultivars.

The F_2 cultures segregated for virulence on durum cultivars Botno, Calvin, Cando, Crosby, D6618, Edmore, Golden Ball, Leeds, Rolette, Rugby, Stewart 63, Ward, Wells, and Vic (Table 2). The segregation ratio approximated three avirulent to one virulent on the durum cultivars Botno, Calvin, Edmore, Rugby, Ward, and Vic ($P > 0.05$ for all cultivars), indicating single recessive genes for virulence (Table 2).

The durum cultivars are characterized by many intermediate infection types (infection type 2 to 2+). When infection types 2 and 2+ were grouped as intermediates, segregation on cultivars Botno, Calvin, Edmore, Ward, Wells, and Vic fit a 1:2:1, avirulent/intermediate/virulent ratio with all *P* values >0.05. Rugby would fit a 1:2:1 ratio ($P > 0.05$), but only if infection types 1 and 2 were classified as intermediate.

Segregation on Cando and Wells fit the 9:7 ratio expected when two recessive gene pairs exist, with either or both homozygous recessive gene pairs conditioning virulence.

The F_2 cultures segregated approximately 15:1, avirulent/virulent on durum cultivars Crosby, Golden Ball, Leeds, Rolette, and Stewart 63 ($P > 0.05$ for all cultivars), indicating two recessive gene pairs for virulence. Chi-square tests did not indicate any association between these genes.

The F_1 was virulent on cultivar D6618 and the F_2 segregated approximately 1:3, avirulent/virulent ($P > 0.10$), indicating a single dominant gene for virulence.

Chi-square tests indicated linkage between *p*1 and *pEG* and between *p*11 and *pEG* (Table 3). Genes *p*11 and *pEG* are apparently closely linked in coupling, with an estimated percentage recombination of $7.0 \pm 3.1\%$. Genes for virulence on Edmore were apparently linked to those for virulence on host genes *Lr*11, *LrEG*, Botno, and Calvin. Chi-square tests indicated that genes for virulence on Rugby were linked to those for virulence on *LrT*, Botno, and Edmore. Linkage was also indicated between genes for virulence on Ward and genes for virulence on Botno, Edmore, and Rugby. Genes for virulence on Botno were apparently linked to those on Calvin, Edmore, Rugby, and Ward. All other single recessive genes for virulence were apparently inherited independently.

Because some of the genes for virulence on the durum cultivars appeared to be associated, Chi-square tests were used to determine gene associations. However, none of the combinations fit a ratio for identical genes; rather, most appear to be linked in coupling.

Linkage was indicated in a number of cases where only one class was low and probably caused the Chi-square test to indicate linkage. This linkage was between genes for virulence on Edmore with those on *LrEG*, *Lr*11, Wells, Cando, and Rugby; between genes for virulence on *Lr*11 and Wells; and between genes for virulence on Rugby and *LrT*. In most cases, except for comparisons between genes for virulence on Edmore and those on Calvin and Rugby, *P* values were only <0.05.

DISCUSSION

The recessive digenic ratios for virulence on *Lr*10, *Lr*16, cultivar 5534, Waldron, and Olaf are difficult to reconcile with published results (10,11). Although two genes for resistance were reported in Waldron (10), a single recessive gene for virulence on Waldron was found in culture 73-47 (10). However, 73-47 could be homozygous for virulence at the other locus. Host lines *Lr*10 and *Lr*16 are both near-isogenic lines, and a single gene for virulence would be expected in the fungus unless the two lines have undetected closely linked genes for resistance.

Segregation ratios for virulence on both *Lr*10 and cultivar 5534 fit a ratio for two gene pairs in common. Therefore, the same two gene pairs apparently condition virulence to both cultivars.

A single gene for resistance has been reported for *Lr*3 (5), but segregation for virulence on *Lr*3 was closer to a 9:7 ratio ($P > 0.50$) than a 3:1 ratio ($P > 0.005$). The 9:7 ratio could be due to duplicate recessive epistasis, in which either homozygous gene pair would produce a virulent response. However, the F_1 is virulent, so virulence is dominant and the ratio should have been 9:7, virulent/avirulent. This aberrant 3:1 ratio could also be explained by a partially dominant gene as previously reported (3). Haggag et al (5) reported a recessive gene *p*3 for virulence in Democrat and an additional dominant gene for virulence on Sinvalocho in race 11 *P. recondita*. Different cultures of *P. recondita* must react differently on *Lr*3. This indicates that *Lr*3-*p*3 is a complex association.

All the segregating cultures that fit a 3:1 ratio on the durum cultivars also fit a 1:2:1, avirulent/intermediate/virulent ratio when infection types 2–2+ were grouped as intermediates. Incomplete dominance could be used to explain this 1:2:1 ratio. Dyck and Samborski (3) previously reported a partially dominant gene for virulence. The culture may also be heterozygous for intermediate infection types.

Segregation on Cando and Wells also fit a 9:7 ratio for two recessive gene pairs, with either or both homozygous recessive gene

pairs conditioning virulence. A 9:7 segregation ratio is uncommon for an F_2 in *P. recondita*.

A single dominant gene for virulence was indicated for D6618 in this study. Dominant genes for virulence are not common in *P. recondita* but have been previously reported (7,8,10).

Two recessive genes for virulence were indicated on durum cultivars Crosby, Golden Ball, Leeds, Rolette, and Stewart 63. Two recessive genes for resistance were previously reported for Leeds (9).

Chi-square tests indicated linkage between *p*1 and *pEG* and between *p*11 and *pEG*. Genes *p*11 and *pEG* are apparently closely linked in coupling. All the other single recessive genes for virulence on the hard red spring wheat cultivars were inherited independently. Linkages between genes for virulence in *P. recondita* are not common but have been reported in a few cases (8,11).

Although linkage is not common between genes for virulence in *P. recondita* on *T. aestivum*, linkage was indicated between several genes for virulence on *T. durum* cultivars. These linkage patterns indicate that a mutation or deletion of a small segment carrying linked genes could simultaneously cause virulence on more than one gene for resistance. This would be very important in breeding for resistance to *P. recondita* in *T. durum* wheat. The use of different *T. durum* genes may not provide the diversity that different *T. aestivum* genes do.

Some of the genes for virulence on the durum cultivars appeared to be the same. But when ratios were tested against a 3:0:0:1 ratio for the same genes, none of the combinations fit ($P<0.05$); rather, most appear to be linked in coupling.

Cultivar 5534 has seedling resistance genes from Ramsey. However, the F_2 segregated 15:1 on cultivar 5534 but was homozygous avirulent on Ramsey. This is difficult to reconcile unless Ramsey has an additional gene for resistance.

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