Genetics of Tan Necrosis and Extensive Chlorosis in Tan Spot of Wheat Caused by *Pyrenophora tritici-repentis*

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ABSTRACT

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Pyrenophora tritici-repentis (Ptr) induces tan necrosis and/or chlorosis, including extensive chlorosis in differential wheat cultivars. Reciprocal crosses were made in all combinations between the hexaploid wheats Glenlea (necrotic only), 6B365 (chlorotic only), and Salamouni (non-necrotic, nonchlorotic). F_1, F_2, and F_3 progenies were sequentially tested for extensive chlorosis by using isolate D308 (nec^-chl^+) and for tan necrosis with isolate 86-124 (nec^+chl^-) and/or the Ptr necrosis toxin. Reciprocal effects were not observed throughout the study. Resistance to tan necrosis and insensitivity to the Ptr necrosis toxin were recessive. Resistance to extensive chlorosis was dominant and incompletely dominant in crosses of line 6B365 with Glenlea and Salamouni, respectively. The F_2 and F_3 segregation ratios were consistent with the action of two independent genes, one controlling the development of tan necrosis, the second controlling the development of extensive chlorosis.

Additional keywords: *Drechslera*, *Helminthosporium*, host-pathogen interactions, inheritance.

Tan spot, caused by the ascomycete *Pyrenophora tritici-repentis* (Died.) Drechs. (anamorph *Drechslera tritici-repentis* (Died.) Shoemaker) is an important leaf disease of wheat (*Triticum aestivum* L.) worldwide. *P. tritici-repentis* has been reported to infect many gramineous species (8,9,12,13,20) and has the widest host range of any *Pyrenophora* species (24).

Resistance of wheat to tan spot has been previously reported (2,3,10,21–23). Lamari and Bernier (14) characterized wheat reaction to the fungus by using lesion types. Susceptibility consisted of small dark brown spots with tan necrosis and/or chlorosis, often extending to cover the entire leaf. Resistance was characterized by the absence, or the presence of slight amounts, of necrosis and/or chlorosis. Tan necrosis and chlorosis, which may be extensive, were found to be distinct components of the tan spot syndrome. Necrotic lesions were well defined and consisted of tan colored, collapsed tissue, whereas chlorotic lesions exhibited a gradual yellow discoloration, initially without collapse, of large areas of the leaf (14). Nitrogen fertilization reduced tan spot severity (11).

Isolates of *P. tritici-repentis* differ in virulence (3,6). Krupinsky (13) tested 27 isolates of *P. tritici-repentis* from smooth bromegrass on wheat and smooth bromegrass and found that these isolates varied in their ability to cause disease in wheat, as measured by lesion size and percentage of leaf area infected. Lamari and Bernier (15) identified three pathotypes in *P. tritici-repentis* based on the ability of isolates to induce, on appropriate differential cultivars, tan necrosis and extensive chlorosis (nec^+chl^+), tan necrosis only (nec^+chl^-), and extensive chlorosis only (nec^-chl^+). A fourth pathotype (nec^-chl^-) was later identified (L. Lamari, *unpublished data*).

P. tritici-repentis releases a host-selective toxin in culture (16,26), designated as the Ptr necrosis toxin (1). The toxin was produced by (nec^+) isolates only and found to be associated with the induction of tan necrosis in the host (16). It was subsequently purified and shown to be a protein of low molecular weight (1,27). Susceptibility of necrotic wheat genotypes to nec^+ isolates and sensitivity to the Ptr necrosis toxin were found to be controlled by the same dominant gene (16). Lines developing extensive chlorosis only were found to be insensitive to the Ptr necrosis toxin and resistant to (nec^+chl^-) isolates.

Resistance of wheat to *P. tritici-repentis* was reported to be quantitatively (4,21) and qualitatively inherited (16,18,25). Rees (23) found that resistance to tan spot was recessive and complex, involving at least four genes. Most of the above studies (4,18,21,25) were carried out before the recognition of tan necrosis and extensive chlorosis as two distinct symptoms in tan spot (14) and before the identification of pathotypes differing in virulence patterns (15). Lamari et al (17) reported the existence of wheat lines and cultivars capable of developing tan necrosis to nec^+ isolates and extensive chlorosis to chl^+ isolates, and suggested that the two symptoms were genetically distinct.

The objectives of this study were to determine the mode of inheritance of the extensive chlorosis response and its relationship to the tan necrosis response to further our understanding of the genetics of resistance to tan spot.

MATERIALS AND METHODS

Inoculation. Inoculum was produced on V8-PDA (150 ml of V8-juice, 10 g of Difco potato-dextrose agar [PDA], 3 g of $CaCO_3$, 10 g of Bacto agar, 850 ml of distilled water) as described previously (14). Cultures were incubated in the dark at 20 C until the colonies reached about 4–5 cm in diameter. They were then flooded with sterile distilled water, the mycelium was flattened with a flamed test tube bottom, and the excess water was decanted. The cultures were incubated for 18–24 h at room temperature (20–24 C) under light (about 90 $\mu E \cdot m^{-2} \cdot s^{-1}$) provided by three cool white fluorescent tubes, followed by 18–24 h in the dark at 15 C. Spores were suspended in distilled water by using a wire loop and inoculum concentration was measured with a cell counter (Hausser Scientific, Blue Bell, PA) and adjusted to 3,500–4,000 conidia per milliliter with distilled water. Ten drops per liter of Tween 20 (polyoxyethylene sorbitan monolaurate) were added to the spore suspension to reduce surface tension. Seedlings at the two-leaf stage were sprayed with the spore suspension until runoff, with a DeVilbis-type sprayer connected to an air outlet and operated at 69 kP (10 lb/in^2). The seedlings were incubated for 24 h under continuous leaf wetness, at 22 C, and a 16-h photoperiod. Leaf wetness was provided by two computer-controlled ultrasonic humidifiers filled with distilled water (14). The plants were then transferred to a growth room

bench and kept at 22/18 C (day/night) and a 16-h photoperiod (about 180 $\mu E \cdot m^{-2} \cdot s^{-1}$) for 8 days at which time they were rated for the presence (+) or absence (−) of tan necrosis or extensive chlorosis. Seedlings were also infiltrated with about 100 μl of a 1:50 dilution of cell-free culture filtrates from isolate 86-124 (nec^+ chl^-), known to contain the Ptr necrosis toxin. Culture filtrates were produced and processed as described previously (1,16) and were infiltrated into wheat leaves by using a Hagborg device (7). Sensitivity to the toxin was recorded 48 h after infiltration as "+" or "−", respectively, for the presence or absence of symptoms.

Crosses of host lines. Line 6B365 (University of Manitoba accession 6B365) (susceptible to extensive chlorosis only) was reciprocally crossed to Salamouni (University of Manitoba accession 6B359) (resistant to necrosis and chlorosis) and cultivar Glenlea (pedigree [Pembina[3] × Bage] × CB100 [5]) (necrotic with little, restricted chlorosis), previously characterized for their reactions to *P. tritici-repentis* (15). Seeds from the F_1 and F_2 generations and parental lines were planted in 13-cm-diameter pots containing a 2:1:1 soil mix (soil/sand/peat) at the rate of six to eight seeds per pot. F_1 and F_2 progenies from all crosses were sequentially inoculated, at 10-day intervals, with isolate D308 (nec^- chl^+) to test for the presence of extensive chlorosis and with 86-124 (nec^+ chl^-) for tan necrosis. Infected leaves from the first inoculation were discarded before the second inoculation, and only the two youngest leaves were used. Preliminary tests with sequential inoculation did not reveal changes of reactions nor isolate contamination (L. Lamari, *unpublished data*). Seedlings were rated with lesion types of 1–5, as described previously (14). Seedlings with lesion types 1–2 were considered to be resistant (−), whereas those with scores of 3–5 were classified as susceptible to a given trait (+). For analysis, the seedlings were assigned the following binomials (+,−), (+,+), (−,−), and (−,+) to indicate the presence (+) or absence (−) of necrosis and chlorosis, respectively. Twenty-five to 30 seedlings of each of 123 F_3 families from the cross between Glenlea and 6B365 were tested with chlorosis isolate D308 for extensive chlorosis and with the Ptr necrosis toxin for necrosis reaction. They were rated for each symptom as resistant, segregating, or susceptible.

RESULTS

The reactions of the three parental lines were as described previously (14–16) and are shown in Table 1. Salamouni was resistant to both isolates (−,−), and developed only small brown to black spots. Line 6B365 was resistant to isolate 86-124, but developed extensive chlorosis (−,+) to isolate D308. Glenlea developed tan necrosis to 86-124, and typically small brown spots surrounded by a thin ring of chlorosis to isolate D308 (+,−). Only Glenlea was sensitive to the Ptr necrosis toxin. Reciprocal effects were not observed throughout this study.

6B365 × Glenlea. All the F_1 seedlings from the reciprocal cross 6B365 × Glenlea developed symptoms similar to those of Glenlea, when tested to isolates 86-124, D308, and the Ptr necrosis toxin (Table 1). All seedlings developed tan necrosis to 86-124, but were resistant to extensive chlorosis when tested to D308, indicating that resistance to necrosis was recessive but that resistance to extensive chlorosis was dominant. Seedlings from the F_2 populations segregated for necrosis in a 1:3 (resistant/susceptible) ratio and in a 3:1 ratio for extensive chlorosis (Table 2), indicating control by a single gene for each of the two symptoms. Seedlings that developed tan necrosis were sensitive to the Ptr necrosis toxin. When reactions for necrosis and extensive chlorosis were combined for analysis, the F_2 populations segregated in a 9(+,−):3(+,+):3(−,−):1(−,+) ratio (Table 3), which is consistent with the action of two independent loci, where resistance to necrosis is recessive, and resistance to extensive chlorosis is dominant. The segregation ratio of the F_3 populations fit a ratio of 1:8:7 (homozygous resistant to both necrosis and chlorosis, segregating for either or both traits, homozygous susceptible to either or both traits), indicating the involvement of two independent genes (Table 4), with necrosis being dominant and extensive chlorosis recessive.

Salamouni × Glenlea. All the F_1 seedlings derived from this

TABLE 1. Reactions of Glenlea, Salamouni, and 6B365 and their F_1 and F_2 progenies to the necrosis isolate of *Pyrenophora tritici-repentis* and the Ptr necrosis toxin

Lines/crosses	Generation[a]	Toxin[b]		86-124 (nec^+chl^-)[c]					D308 (nec^-chl^+)[c]				
		−	+	1	2	3	4	5	1	2	3	4	5
Glenlea	P	0	177	0	0	0	75	102	23	154	0	0	0
6B365	P	212	0	120	92	0	0	0	0	0	0	21	185
Salamouni	P	133	0	80	53	0	0	0	130	3	0	0	0
Glenlea × 6B365	F_1	0	9	0	0	0	2	7	6	3	0	0	0
6B365 × Glenlea	F_1	0	11	0	0	0	0	11	5	6	0	0	0
Glenlea × 6B365	F_2	53	183	6	47	15	86	82	18	160	1	43	14
6B365 × Glenlea	F_2	43	156	10	33	20	52	84	21	124	0	24	30
Salamouni × Glenlea	F_1	0	10	0	0	0	0	10	7	3	0	0	0
Salamouni × Glenlea	F_2	73	200	23	50	24	71	105	185	88	0	0	0
6B365 × Salamouni	F_1	12	0	10	2	0	0	0	0	0	12	0	0
6B365 × Salamouni	F_2	191	0	125	66	0	0	0	21	29	95	7	39
Salamouni × 6B365	F_2	222	0	198	24	0	0	0	32	23	99	25	43

[a] P = parental.
[b] Ptr necrosis toxin (+, sensitive; −, insensitive).
[c] Lesion types of 1–5 in which 1–2 represent resistance and 3–5 susceptibility.

TABLE 2. Segregation of F_2 populations from crosses between Glenlea, 6B365, and Salamouni for tan necrosis and extensive chlorosis induced by *Pyrenophora tritici-repentis*

Cross	Ratios[a]					
	Tan necrosis			Extensive chlorosis		
	Observed	Expected	Probability[b]	Observed	Expected	Probability[b]
Glenlea × 6B365	53:183	1:3	$0.50 > P > 0.25$	178:58	3:1	$0.90 > P > 0.75$
6B365 × Glenlea	43:156	1:3	$0.50 > P > 0.25$	145:54	3:1	$0.75 > P > 0.50$
Salamouni × Glenlea	73:200	1:3	$0.75 > P > 0.50$	200:0	1:0	...

[a] Resistant/susceptible.
[b] A fit to the expected ratio is accepted if $P > 0.05$ (chi-square test).

cross were resistant to extensive chlorosis but susceptible to necrosis and sensitive to the Ptr necrosis toxin (Table 1). The F_2 seedlings were all resistant to extensive chlorosis and segregated for necrosis in a 1:3 (resistant/susceptible) ratio (Table 2), indicative of a single gene controlling the expression of necrosis. All seedlings susceptible to necrosis were also sensitive to the Ptr necrosis toxin.

6B365 × Salamouni. All F_1 plants developed a faint chlorosis over large portions of the leaf, but were resistant to tan necrosis and insensitive to the Ptr necrosis toxin (Table 1). The F_2 progenies were all resistant to tan necrosis (and insensitive to the Ptr necrosis toxin), but segregated for extensive chlorosis in a ratio of 1:2:1 (resistant/intermediate/susceptible) (Table 5). The F_3 populations segregated as nine resistant/16 segregating/six susceptible, and fit a 1:2:1 ratio, indicative of the action of a single gene locus.

DISCUSSION

The F_1, F_2, and F_3 data from crosses between resistant, necrotic only, and chlorotic only lines indicated that the development of necrosis and extensive chlorosis in wheat was controlled by two independent loci, each associated with a single symptom. The results of this study support previous findings about the qualitative inheritance of wheat reaction to tan spot (16,18,25). The monogenic and recessive nature of resistance to tan necrosis and insensitivity to the Ptr necrosis toxin confirms the results of a previous study (16), suggesting that the Ptr necrosis toxin (1) could be used as a surrogate for necrosis-inducing isolates of *P. tritici-repentis* to screen large host populations.

The inheritance of reaction to extensive chlorosis has not been previously reported. The expression of this symptom in the heterozygous condition appears to vary with the parental lines used, and can be recessive or incompletely dominant. The intermediate chlorotic reaction observed in 6B365 × Salamouni, but not in 6B365 × Glenlea, suggests the possibility of minor gene(s) action. Additional studies with more parental lines and larger F_2 and F_3 populations are required to resolve this question. This condition, in addition to the presence of tan necrosis in some crosses, may add complexity to observed genetic ratios and be suggestive of quantitative inheritance. The separation of tan necrosis and extensive chlorosis for genetic studies, as done in this study, helps avoid such complications but requires the use of isolates from appropriate pathotypes. The use of (nec^+chl^+) isolates would be useful for breeding purposes, but may not be reliable in differentiating between (+,+) and (−,+) reactions.

The epistatic effect of the incompatible interaction observed in systems that follow the gene-for-gene model (19) was not present in the wheat-*P. tritici-repentis* system. In gene-for-gene systems, resistance may be conferred by a single incompatible interaction between a gene for resistance in the host and a gene for avirulence in the pathogen, regardless of the number of compatible interactions present in the system. In tan spot of wheat, however, an incompatible interaction for a symptom (necrosis or chlorosis) does not override a compatible interaction at the second locus. This is supported by the identification, in the Glenlea × 6B365 F_2 populations, of seedlings with both necrosis and extensive chlorosis (+,+) and also by the recent identification of two wheat lines capable of developing tan necrosis to nec^+ isolates and extensive chlorosis to chl^+ isolates (17). It would appear from the present results that wheat cultivars must carry at least one gene for resistance to necrosis and a second gene for resistance to chlorosis to ensure the expression of "full" resistance. The existence of two nonallelic and independent genes determining the reaction to tan spot is demonstrated for the first time. It appears from the present findings that *P. tritici-repentis* is a highly specialized pathogen, possessing genes that are matched by specific genes in the host. The necrotic subsystem was previously shown to involve the Ptr necrosis toxin (1,16) and follows the interaction for susceptibility model, where specificity in the host-parasite system is based on compatibility (susceptibility), as opposed to incompatibility (resistance) in gene-for-gene systems (19). The present results suggest that the chlorosis subsystem follows the same model because susceptibility (compatibility) is the basis for specificity. However, the involvement of a toxin capable of differentially inducing extensive chlorosis in known chlorotic wheat genotypes has not been conclusively demonstrated.

The presence of two independent subsystems in tan spot of wheat may have confounded some early genetic studies. Lee and Gough (18) reported that in crosses between susceptible and resistant wheat genotypes, 30 out of 97 clearly segregating F_3 populations followed a 3:1 ratio (resistant/susceptible) and 67 populations segregated in a 1:3 ratio. The authors concluded that resistance was recessive based on the observation that two thirds of the populations segregated in a 1:3 ratio. Their observation could be partly explained on the basis of the results of this study, because 25% of the total F_3 segregating populations (assuming (+,−),(−,+) and (+,+) = susceptible reaction) will, theoretically, segregate for chlorosis in a ratio of 3:1 (resistant/susceptible) and 75% will follow ratios of 1:3 and 3:13. Sykes and Bernier (25) reported a genetic study in which they used an isolate of type (nec^+chl^+), capable of inducing both necrosis and chlorosis, to test F_2 seedlings from hexaploid crosses and found a 3:13 ratio (resistant/susceptible), indicative of the involvement of two genes. Their ratio is consistent with the necrosis/chlorosis model and can be generated from Table 3 by pooling all classes that develop necrosis and/or chlorosis, yielding the following ratio: 3(−,−):9(+,−) + 3(+,+) + 1(−,+) (i.e., 3 resistant/13 susceptible). The 3:13 ratio in the study of Sykes and Bernier (25) was likely due to the fact that the susceptible parent Columbus developed both necrosis and chlorosis (15).

In addition to the qualitative inheritance of wheat reaction

TABLE 3. F_2 combined segregation ratios for necrosis and extensive chlorosis induced by *Pyrenophora tritici-repentis*

Cross	Ratios[a]		Probability[b]
	Observed	Expected	
6B365 × Glenlea	139:39:44:14	9:3:3:1	$0.90 > P > 0.80$
Glenlea × 6B365	118:27:38:16	9:3:3:1	$0.25 > P > 0.10$

[a]Phenotypes are: (necrosis, no extensive chlorosis), (no necrosis, no extensive chlorosis), (necrosis, extensive chlorosis), (no necrosis, extensive chlorosis).
[b]A fit to the expected ratio is accepted if $P > 0.05$ (chi-square test).

TABLE 4. Segregation of F_3 populations derived from a cross between necrotic (Glenlea) and chlorotic (6B365) wheat genotypes

	Segregation ratios								Probability[b]
F_2 genotypes[a]	NNCC	NNCc	NNcc	NnCC	NnCc	Nncc	nnCC	nnCc:nncc	
Expected F_3	1 :	2 :	1 :	2 :	4 :	2 :	1 :	2 : 1	
Observed F_3	4 :	17 :	10 :	13 :	26 :	25 :	5 :	14 : 9	
Expected F_3[c]	1 R : 8 Seg : 7 S								
Observed F_3	5 : 53 : 65								$0.50 > P > 0.10$

[a]NN, Nn = necrotic; nn = resistant to necrosis; CC, Cc = resistant to extensive chlorosis; cc = susceptible to extensive chlorosis. Necrosis is dominant (N) over resistance to necrosis (n); resistance to extensive chlorosis (C) is dominant over susceptibility (c).
[b]A fit to the expected ratio is accepted if $P > 0.05$ (chi-square test).
[c]Categories are grouped as homozygous resistant (R = nnCC), segregating for either or both traits (Seg= NnCc + nnCc + NnCC), and homozygous susceptible to either or both traits (S= NNCC + NNCc + NNcc + Nncc + nncc).

TABLE 5. Segregation of F_2 for extensive chlorosis caused by isolate D308 (nec^-chl^+) of *Pyrenophora tritici-repentis*

		Ratio[a]		
		Observed	Expected	Probability[b]
6B365 × Salamouni	F_2	50:95:46	1:2:1	$0.95 > P > 0.90$
Salamouni × 6B365	F_2	55:99:68	1:2:1	$0.25 > P > 0.13$

[a]Resistant (no chlorosis, no necrosis) intermediate/susceptible (extensive chlorosis).
[b]A fit to the expected ratio is accepted if $P > 0.05$ (chi-square test).

to tan spot, this study has shown that it is possible to identify resistant wheat genotypes from crosses between two susceptible lines, provided that one line is susceptible to necrosis only and the second to chlorosis only. This would allow for the development of resistant cultivars from crosses between some susceptible parental lines. The need to screen large numbers of accessions for high levels of resistance would be reduced.

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