Genetic Control of Resistance to Tan Necrosis Induced by *Pyrenophora tritici-repentis*, Races 1 and 2, in Spring and Winter Wheat Genotypes

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ABSTRACT

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The symptoms of tan spot of wheat, caused by *Pyrenophora tritici-repentis*, include a tan necrosis component and an extensive chlorosis component. Since tan spot has become the major component of the leaf-spotting disease complex of wheat in western Canada, the need for resistant cultivars has increased. This study was conducted to determine whether the resistance to tan spot found in a diverse set of spring and winter wheat genotypes was due to resistance genes not previously reported. The genetic control of resistance to necrosis induced by *P. tritici-*

repentis race 1 and race 2 was determined, under controlled environmental conditions, for spring wheat genotypes Erik and 86ISMN 2137 and winter wheat genotypes Hadden, Red Chief, and 6B-365. Plants were inoculated at the two-leaf stage and disease reaction was assessed based on lesion type. Tests of the F₁ and F₂ generations, and of F_{2:3} and F_{2:8} families, indicated that one recessive gene controlled resistance to the necrosis component of tan spot caused by both race 1 and race 2 in each cross studied. Lack of segregation in crosses between the resistant cultivars indicated that the resistance gene was the same in all of the cultivars.

Additional keywords: host resistance, *Triticum aestivum*.

Tan spot of wheat, caused by the fungus *Pyrenophora tritici-repentis* (Died.) Drechs., is a worldwide disease that causes loss both in quality and quantity of grain (5,14). Increases in the incidence and severity of tan spot have been attributed to changes in cultural practices including shifts from conventional tillage and stubble burning to conservation tillage systems, shorter crop rotations, continuous wheat cultivation, and use of susceptible cultivars (5,6,15).

The tan spot disease syndrome consists of two phenotypically distinct and independent symptoms: tan necrosis and extensive chlorosis (20). Resistance is expressed as small, dark brown lesions that do not increase in size, while susceptibility is expressed as dark brown spots surrounded by tan necrosis and/or extensive chlorosis that may involve the entire leaf. Necrotic lesions are well defined and tan in color, while chlorotic lesions are less well defined and exhibit gradual yellow discoloration (20).

Initially, isolates of *P. tritici-repentis* were grouped into four pathotypes based on their ability to induce tan necrosis and/or extensive chlorosis (22,23). Later Lamari et al. (24) proposed a race-based system to describe isolates of *P. tritici-repentis* and currently 11 races of *P. tritici-repentis* have been identified (2,3,25,26).

Resistance to tan spot of wheat has been reported to be inherited quantitatively (10,13,28,29) and qualitatively (4,8,9,17,18). A gene, *tsn1*, controlling resistance to the necrosis component of tan spot has been located on chromosome 5BL (12,30). Recent genetic studies (8,17) have indicated that single recessive genes control resistance to necrosis caused by races 1 and 2 in durum and in common wheat. Because these results suggest a narrow genetic base for resistance to tan spot in wheat, this study was initiated using a diverse range of winter and spring cultivars

to identify new resistance genes. The specific objectives of this study were to (i) study the genetic control of resistance to the necrosis component of tan spot caused by *P. tritici-repentis*, race 1 and race 2; (ii) determine the allelic relationship among the resistance gene(s) identified; and (iii) determine the relationship between the genes controlling resistance to necrosis caused by race 1 and race 2.

MATERIALS AND METHODS

Population development. Seven wheat genotypes, selected for this study based on their reaction to race 1 and race 2 of *P. tritici-repentis* (Table 1), were used to produce 16 cross populations. Each F₁ plant grown to produce F₂ seed was harvested separately, and the F₂ populations derived from each F₁ plant were tested individually for disease reaction. All F_{2:3} families, F_{2:4} families, and F_{2:8} families were produced by single seed descent.

Inheritance studies. Inheritance studies involved testing for tan spot reaction in crosses between resistant and susceptible genotypes (R/S crosses). The F₁ and F₂ generations and the F_{2:3} families were screened with race 1 isolate Ptr 200 and race 2 isolate Ptr 92-164 to determine the inheritance of resistance to the necrosis component of tan spot. F_{2:4} families were also tested with race 2. A family size of 16 to 18 plants was used to give a 99% probability of correctly distinguishing between a segregating and a nonsegregating family if a single gene controlled resistance (19). However, since the cross Glenlea/6B-365 segregated for both the necrosis and the chlorosis components when tested with race 1, it was hypothesized that resistance to tan spot in this cross was controlled by two genes. In this cross, at least 36 plants per F_{2:3} family were tested to ensure a 90% probability of correctly identifying all genotypic classes (19). A minimum of 60 families per cross were tested.

Allelism studies. Allelic studies involved testing for tan spot reaction in crosses between resistant × resistant genotypes (R/R

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crosses) and between susceptible $\times$ susceptible genotypes (S/S crosses). The six possible crosses among the resistant genotypes Red Chief, 86ISMN 2137, Erik, and Hadden and the cross between susceptible cvs. Kenyon and Glenlea were studied. F₁ and F₂ plants and F_{2:3} families (12 plants per family) were screened with race 2. At least 40 F_{2:3} families per cross were tested.

F_{2:8} lines of the cross Erik/Glenlea were tested with race 1 and race 2 to establish the relationship between the genetic control of resistance to the necrosis component of tan spot caused by these two races. Eighty lines with at least 16 plants per line were screened with each race.

Inoculum production. Isolates Ptr 200 (race 1), provided by G. R. Hughes, University of Saskatchewan, and Ptr 92-164 (race 2), provided by L. Lamari, University of Manitoba, were used in this study. Single-spore cultures were produced for each of the two isolates and stored at 4°C for inoculum production.

Inoculum was produced using a modification of the method of Lamari and Bernier (20). Mycelial plugs, 0.5-cm diameter, from the stock cultures were transferred to 10-cm petri plates containing V8-potato dextrose agar (PDA) (150 ml of V8 juice, 10 g of PDA, 10 g of agar, 3 g of CaCO₃, and 850 ml of distilled water). These cultures were incubated in the dark at 20 to 22°C for 6 days. The plates were then flooded with sterile distilled water, the mycelium was flattened with the base of a sterile test tube, and excess water was poured off. The plates were incubated under continuous light at room temperature for 2 days to induce conidiophore production, and then for 1 day in the dark at 15 to 16°C to induce conidial production.

To produce inoculum, the plates were flooded with distilled water and the conidia were suspended in the distilled water by gently brushing the mycelium with a camel-hair brush to dislodge the conidia from the conidiophores. Tween 20 (polyoxyethylene sorbitan monolaureate) was added to the spore suspension (5 to 7 drops per liter), and the spore concentration was adjusted to 3,000 conidia per ml.

Disease screening. To optimize uniform emergence, seed of the populations to be screened and the parental genotypes, which were grown as resistant and susceptible checks and were pregerminated, and seeds at a similar stage of germination were planted in 6-in.-diameter pots containing soilless mix (Redi-Earth, W.R. Grace and Co. of Canada Ltd., Ajax, ON, Canada). In the allelism studies, 12 seeds per pot were planted, whereas 9 seeds per pot were planted in all other studies. All disease tests were conducted using a completely randomized design. Using a hand sprayer, plants at the two-leaf stage were sprayed with a conidial suspension of the appropriate isolate until runoff. Following inoculation, the seedlings were incubated for 24 h in continuous leaf wetness in a mist chamber located in a growth room at 22/17°C (day/night) with a 16 h photoperiod.

Eight days after inoculation, seedlings were rated for reaction to tan spot using the 1 to 5 lesion type rating scale developed by

Lamari and Bernier (20). Plants with ratings of 1 to 2 were considered resistant, and those with ratings of 3 to 5 were considered susceptible.

RESULTS

Erik, Red Chief, Hadden, and 86ISMN 2137 were resistant to both the necrosis and the chlorosis component. However, these genotypes differed in the level of resistance expressed. Erik and Red Chief were rated 1 and 86ISMN 2137 was rated 2, whereas Hadden was more variable, giving a 1 to 2 rating for resistance to the necrosis component. Glenlea and Kenyon consistently produced susceptible necrotic reactions (ratings of 4 to 5). Line 6B-365 was resistant to necrosis but susceptible to the chlorosis component.

Genetics of resistance to necrosis caused by race 2. F₁ plants of all eight R/S crosses studied were susceptible, indicating that resistance was recessive (Table 2). These plants showed a range of susceptible reaction types (ratings of 3 to 5), although the F₁ plants of crosses involving resistant parent Hadden were less susceptible than F₁ plants of the other crosses.

The F₂ generations of these R/S crosses segregated in a 1 resistant/3 susceptible ratio indicating single gene control and confirming that resistance to necrosis was recessive (Table 2). The F_{2:3} family segregation of 1 homozygous resistant/2 segregating/1 homozygous susceptible supported the hypothesis of monogenic control of resistance (Table 3). This hypothesis was further confirmed by the F_{2:4} family segregation which fitted a 3 homozygous resistant/2 segregating/3 homozygous susceptible ratio in each cross studied (Table 4). All F₁ and F₂ plants and the F_{2:3} families of the S/S cross between susceptible cvs. Kenyon and Glenlea were susceptible, indicating that both parents share the same susceptibility gene(s) (Tables 2 and 3).

F₁ plants of the six possible R/R crosses among the four resistant parents were resistant to race 2. However, while most F₂ plants of these crosses were resistant, a few plants showed a moderately susceptible reaction (rating 3). With the exception of cross Hadden/Red Chief, F₂ plants of each cross involving only resistant genotypes did not fit a 15 resistant/1 susceptible segregation ratio, suggesting that each genotype carried the same resistance gene (Table 2). None of the F_{2:3} families of the R/R crosses studied were susceptible, thus confirming the presence of the same resistant gene among the resistant parents (Table 3). The F₂ generation of the cross Hadden/Red Chief segregated in a 15 resistant/1 susceptible ratio indicating that Hadden and Red Chief possessed different resistance genes. This hypothesis could not be verified because seed of F_{2:3} families of the cross Hadden/Red Chief had not been produced.

Genetics of resistance to necrosis caused by race 1. F₁ plants of the R/S crosses Erik/Glenlea and Hadden/Glenlea were susceptible to necrosis indicating that resistance to the necrosis component of tan spot was recessive (Table 2). Segregation of the

TABLE 1. Growth habit, origin, reaction to *Pyrenophora tritici-repentis* race 1 and race 2, and pedigrees of the cultivars selected for study of the genetics of resistance to tan spot of wheat

Wheat cultivar	Growth habit	Origin	Reaction to <i>P. tritici-repentis</i> ^a		Pedigree
			Race 1	Race 2	
Erik	Spring	USA	Resistant (1)	Resistant (1)	Kitt/2/Walron/Era
86ISMN2137	Spring	CIMMYT	Resistant (2)	Resistant (2)	Not known
Glenlea ^b	Spring	Canada	Susceptible (4–5)	Susceptible (4–5)	Pembina*2/Bage/2/CB100
Kenyon	Spring	Canada	Susceptible (4–5)	Susceptible (4–5)	Neepawa*5/Buck Manantial
6B-365	Winter	Lebanon	Susceptible (4–5)	Resistant (1–2)	Not known
Red Chief	Winter	USA	Resistant (1)	Resistant (1)	Red Clawson/Red Arcadian
Hadden	Winter	USA	Resistant (1–2)	Resistant (1–2)	Trumbull*2/Red Wonder/2/T. timopheevii/ Steinwedel/3/Asosan/Hardired/4/Chancellor/ 5/Coker 47-27/6/ Coastal

^a Rating scale (1 to 5) based on lesion type where 1 = resistant and 5 = fully susceptible (20).

^b Glenlea is susceptible to necrosis but resistant to chlorosis induced by race 1.

F₂ generation fitted a 1 resistant/3 susceptible ratio and the F_{2:3} families of these crosses segregated in a 1 homozygous resistant/2 segregating/1 homozygous susceptible ratio indicating that a single recessive gene controlled resistance (Tables 2 and 3).

F₁ plants of the cross Glenlea/6B-365 showed only necrotic symptoms indicating that resistance to the chlorosis component and to the necrosis component caused by race 1 was dominant

and recessive, respectively (Table 2). Since rating of individual plants for resistance to both necrosis and chlorosis was difficult, plants were rated for the presence or absence of necrosis and/or chlorosis. The F₂ generation segregated in a 3:13 (absence/presence) ratio (Table 2). The F_{2:3} families segregated in a 1 homozygous resistant (necrosis and chlorosis)/8 segregating (necrosis and/or chlorosis)/7 homozygous susceptible (necrosis and/or

TABLE 2. F₁ plant disease reaction to isolates Ptr 92-164 (race 2) and Ptr 200 (race 1) and chi-square tests of F₂ segregation ratios of crosses used to study the inheritance and allelic relationships among the gene(s) controlling resistance to necrosis caused by races 1 and 2

Cross	F ₁ plants ^a		F ₂ plants ^a		Ratio tested	χ ² value	Probability value ^b
	R	S	R	S			
Resistant/susceptible tested with Ptr 92-164 (race 2)							
86ISMN 2137/Kenyon	0	9	102	255	1:3	2.43	0.12
Erik/Kenyon	0	9	90	264	1:3	0.03	0.85
Red Chief/Kenyon	0	9	72	254	1:3	1.48	0.22
Hadden/Kenyon	0	8	95	305	1:3	0.33	0.56
86ISMN 2137/Glenlea	0	9	80	274	1:3	1.09	0.30
Erik/Glenlea	0	9	96	256	1:3	0.97	0.33
Red Chief/Glenlea	0	9	112	320	1:3	0.20	0.65
Hadden/Glenlea	0	8	92	248	1:3	0.77	0.38
Susceptible/susceptible tested with Ptr 92-164 (race 2)							
Kenyon/Glenlea	0	9	0	340	0:1	...	...
Resistant/resistant tested with Ptr 92-164 (race 2)							
Erik/86ISMN 2137	9	0	315	7 ^c	15:1	9.13	<0.01
Erik/Hadden	8	0	338	5 ^c	15:1	13.44	<0.01
Erik/Red Chief	8	0	311	9 ^c	15:1	6.45	0.01
86ISMN 2137/Hadden	9	0	312	8 ^c	15:1	7.68	<0.01
86ISMN 2137/Red Chief	8	0	336	3 ^c	15:1	16.65	<0.01
Hadden/Red Chief	8	0	308	13 ^c	15:1	2.65	0.10
Resistant/susceptible tested with Ptr 200 (race 1)							
Erik/Glenlea	0	9	94	266	1:3	0.24	0.63
Hadden/Glenlea	0	9	54	252	1:3	0.74	0.39
Susceptible/susceptible tested with Ptr 200 (race 1)							
Glenlea/6B-365	0	8	54	252	3:13	0.24	0.88

^a R = resistant (ratings of 1 to 2), S = susceptible (ratings of 3 to 5).

^b Probability of obtaining deviations from the expected ratio by chance alone. A probability value greater than 0.05 indicates that segregation in the observed population does not differ significantly from the expected ratio.

^c Reaction was moderately susceptible (rating of 3).

TABLE 3. Chi-square tests for F_{2:3} family segregation ratios of crosses studied to determine the inheritance and allelic relationships for resistance to necrosis caused by Ptr 92-164 (race 2) and Ptr 200 (race 1)

Cross	F _{2:3} family reaction			Ratio tested	χ ² value	Probability value ^d
	Resistant ^a	Segregating ^b	Susceptible ^c			
Resistant/susceptible tested with Ptr 92-164 (race 2)						
86ISMN 2137/Kenyon	27	56	37	1:2:1	2.20	0.33
Erik/Kenyon	28	60	32	1:2:1	0.27	0.88
Red Chief/Kenyon	23	61	36	1:2:1	2.85	0.24
Hadden/Kenyon	26	61	33	1:2:1	0.85	0.65
86ISMN 2137/Glenlea	28	54	38	1:2:1	2.87	0.24
Erik/Glenlea	31	55	34	1:2:1	0.98	0.61
Red Chief/Glenlea	25	64	31	1:2:1	1.13	0.57
Hadden/Glenlea	28	63	29	1:2:1	0.32	0.85
Susceptible/susceptible tested with Ptr 92-164 (race 2)						
Kenyon/Glenlea	0	...	40	0:1	...	...
Resistant/resistant tested with Ptr 92-164 (race 2)						
Erik/86ISMN 2137	40	...	0	1:0	...	...
Erik/Hadden	40	...	0	1:0	...	...
Erik/Red Chief	40	...	0	1:0	...	...
86ISMN 2137/Hadden	40	...	0	1:0	...	...
86ISMN 2137/Red Chief	40	...	0	1:0	...	...
Resistant/susceptible tested with Ptr 200 (race 1)						
Erik/Glenlea	26	53	21	1:2:1	0.86	0.65
Hadden/Glenlea	26	36	18	1:2:1	2.40	0.30
Susceptible/susceptible tested with Ptr 200 (race 1)						
6B-365/Glenlea	3	33	24	1:8:7	0.64	0.73

^a Family has individuals expressing a resistant reaction (ratings of 1 to 2).

^b Family is segregating for resistant and susceptible reactions.

^c Family has individuals expressing a susceptible reaction (ratings of 3 to 5).

^d Probability of obtaining deviations from the expected ratio by chance alone. A probability value greater than 0.05 indicates that segregation in the observed population does not differ significantly from the expected ratio.

chlorosis) ratio (Table 3). These ratios can be explained by the presence of two independent genes controlling resistance to race 1 in the cross Glenlea/6B-365, a recessive gene for resistance to the necrosis component contributed by 6B-365 and a dominant gene for resistance to the chlorosis component contributed by Glenlea.

Relationship between genes controlling resistance to necrosis caused by races 1 and 2. F_{2:8} lines of the R/S cross Erik/Glenlea were tested with isolates Ptr 200 (race 1) and Ptr 92-164 (race 2) to study the relationship between the single recessive genes controlling resistance to necrosis caused by race 1 and race 2. When tested with race 1 and with race 2, segregation of F_{2:8} lines of the cross Erik/Glenlea fitted the ratio of 63 homozygous resistant/2 segregating/63 homozygous susceptible, as expected for single-gene control (Table 5).

These genes controlling resistance to the necrosis induced by race 1 and race 2 were not independent (Table 5). Forty lines were resistant to both isolates Ptr 92-164 (race 2) and Ptr 200 (race 1) and 37 lines were susceptible to both isolates. Two lines segregated for resistance to both isolates and one line was susceptible to isolate Ptr 92-164 but segregated for resistance to isolate Ptr 200. All but one line gave similar disease reactions to isolates Ptr 200 (race 1) and Ptr 92-164 (race 2). This indicated that the same gene controlled resistance to necrosis caused by race 1 and by race 2.

DISCUSSION

Throughout this study, a single recessive gene was found to control resistance to the necrosis component of tan spot. A similar

finding has also been reported by others (7–9,18,21,23). Duguid (7) observed that the F₂ segregation in cross Erik/Katepwa fitted a 5 resistant/11 susceptible ratio, while in cross Erik/BH1146, the F₂ segregation ratio was 1 resistant/3 susceptible. However, the F_{2:3} family segregation for both crosses indicated single gene control. In this study, crosses involving resistant genotype Erik segregated for single gene control, also reported by Lamari and Bernier (21). These differing results demonstrate the undesirability of basing segregation data for tan spot on single plant ratings. Single plant data are useful to derive preliminary results but misclassification or disease escape can cause incorrect genetic interpretation. These results should be verified by family segregation data, which is less liable to such errors.

In this study, plants of crosses involving resistant winter wheat cultivars were not vernalized, resulting in loss of winter-habit families in the F₃ and later generations. If linkage between genes for vernalization and resistance to necrosis existed, the segregation patterns observed in the inheritance study of crosses involving spring and winter cultivars would be different from those involving only spring wheat. This was not observed and indicated that the resistance gene was not linked to genes for vernalization. These results also indicated that the same recessive gene for resistance was present in both spring and winter resistant cultivars and that this gene controlled resistance to necrosis caused by race 1 and by race 2.

Preliminary studies by Anderson et al. (4) suggested that the same recessive gene controlled resistance to necrosis in both durum and common wheat. Based on results from this study and from that of Anderson et al. (4), it is hypothesized that resistance

TABLE 4. Chi-square tests of F_{2:4} family segregation ratios of crosses used to study the inheritance of resistance to necrosis caused by Ptr 92-164 (race 2)

Cross	F _{2:4} family reaction			Ratio tested	χ^2 value	Probability value ^d
	Resistant ^a	Segregating ^b	Susceptible ^c			
86ISMN 2137/Kenyon	33	24	23	3:2:3	2.73	0.25
Erik/Kenyon	29	23	28	3:2:3	0.62	0.73
Red Chief/Kenyon	27	24	29	3:2:3	1.13	0.57
Hadden/Kenyon	26	23	31	3:2:3	1.02	0.60
86ISMN 2137/Glenlea	24	22	34	3:2:3	1.95	0.38
Erik/Glenlea	25	23	32	3:2:3	1.40	0.50
Red Chief/Glenlea	29	25	26	3:2:3	1.82	0.40
Hadden/Glenlea	34	33	23	3:2:3	2.62	0.27

^a Family has individuals expressing a resistant reaction (ratings of 1 to 2).

^b Family is segregating for resistant and susceptible reactions.

^c Family has individuals expressing a susceptible reaction (ratings of 3 to 5).

^d Probability of obtaining deviations from the expected ratio by chance alone. A probability value greater than 0.05 indicates that segregation in the observed population does not differ significantly from the expected ratio.

TABLE 5. Chi-square tests and test of independence of F_{2:8} family segregation ratios in the cross Erik/Glenlea for resistance to the necrosis component induced by isolates Ptr 92-164 (race 2) and Ptr 200 (race 1)

Isolate	F _{2:8} families			Ratio tested	χ^2 value	Probability value ^d
	Resistant ^a	Segregating ^b	Susceptible ^c			
Ptr 92-164	40	2	38	63:2:63	0.51	0.78
Ptr 200	40	3	37	63:2:63	2.60	0.27
Ptr 92-164						
	R ₂	Seg ₂	S ₂			
Ptr 200						
R ₁	40	0	0	R.T. ^e	276.63	0.00
Seg ₁	0	2	1			
S ₁	0	0	37			

^a Family has individuals expressing a resistant reaction (ratings of 1 to 2).

^b Family is segregating for resistant and susceptible reactions.

^c Family has individuals expressing a susceptible reaction (ratings of 3 to 5).

^d Probability of obtaining deviations from the expected ratio by chance alone. A probability value greater than 0.05 indicates that segregation in the observed population does not differ significantly from the expected ratio.

^e R.T. = ratio tested for test of independence = 3,969 R₁R₂/126 R₁Seg₂/3,969 R₁S₂/126 Seg₁R₂/4 Seg₁Seg₂/126 Seg₁S₂/3,969 S₁R₂/126 S₁Seg₂/3,969 S₁S₂, where R₁ and R₂ = resistant to race 1 and race 2, respectively; Seg₁ and Seg₂ = segregating to race 1 and race 2, respectively; and S₁ and S₂ = susceptible to race 1 and race 2, respectively.

to necrosis caused by race 1 and race 2 in both durum and common wheat is controlled by the same single gene. This suggests that the genetic base for resistance to the necrosis component of tan spot is narrow. The relationship of this resistance gene to *tsn1* (12,30) is not known since both studies involved genotypes different to those used in this study. However, since this and other studies have shown this resistance gene to be widespread in the common wheat gene pool, it is likely that the recessive gene identified in this study is also *tsn1*.

A few moderately susceptible plants (rating 3) were observed in the F₂ generation of the R/R crosses. These moderately susceptible plants were not progeny tested, but testing of random F_{2:3} families showed that all families were homozygous resistant, thus confirming that the resistant parents possessed the same resistance gene. Although F₂ segregation data indicated that Hadden and Red Chief possessed different genes for resistance, F_{2:3} families of crosses Erik/Hadden and Erik/Red Chief failed to segregate indicating the presence of the same resistance gene in Erik, Hadden, and Red Chief. These conflicting results are viewed as further evidence of the unreliability of single plant segregation data. However, these few moderately susceptible plants could be expected if a cluster of tightly linked resistance genes existed. The population sizes used in this study were not large enough to conclusively identify the moderately susceptible plants as rare recombinants and thus the presence of such a cluster.

Lamari et al. (25) believe that the wheat-*P. tritici-repentis* pathosystem is an example of the toxin model originally proposed by Ellingboe (11). In this model, a mirror image of the classical gene-for-gene model of host-pathogen interaction (16), a compatible interaction between host plant and pathogen, leads to susceptibility, the result of the presence of a "toxin-receptor" site in the host. If such a site is considered the product of a susceptibility gene, resistance in the wheat-*P. tritici-repentis* pathosystem could be viewed as due to absence of a susceptibility gene rather than presence of a resistance gene. Resistance would be expected to be lost when a mutation in the host results in production of the receptor site or the pathogen produces a new toxin which would be recognized by a different receptor site in the host.

This suggests that the fact that only one gene has been identified for resistance to necrosis caused by race 1 and race 2 does not necessarily imply that the resistance caused by this gene will be short-lived. Both race 1 and race 2 produce the necrosis-inducing toxin Ptr ToxA (21), which would have a specific host receptor site. Absence of this site would result in resistance which to be defeated would require the pathogen to produce a new toxin, a gain of function. Gain of a function, which did not exist before, is usually associated with the occurrence of a back mutation, normally a much less common event than forward mutations which are usually associated with loss of function. A similar type of mutation in the resistant host would be needed to produce a receptor site for Ptr ToxA. Since either mutation event would be rare, use of this resistance gene in breeding tan spot-resistant cultivars is unlikely to result in the occurrence of new virulent races and subsequent breakdown of resistance.

Genotype 6B-365 is resistant to the necrosis and susceptible to the chlorosis induced by race 1. When tested with isolate Ptr 200 (race 1), the segregation data for cross Glenlea/6B-365 indicated that resistance was due to two independent genes. However, in crosses Erik/Glenlea and Hadden/Glenlea, which segregated for the necrosis component only, resistance to race 1 was controlled by a single gene. Therefore, depending on whether a cross was segregating for one or both components of the tan spot disease syndrome, resistance to race 1 would appear to be controlled by either one or two genes.

Because this pathosystem follows the toxin model, development of wheat cultivars resistant to all known races requires selection against those host susceptibility genes which result in a compatible reaction. For resistance to the necrosis component in

common wheat, this involves selection against the susceptibility genes related to three groups of races of tan spot, races 1, 2, 7, and 8 (25), race 10 (2), and race 11 (26). In durum wheat, necrosis-inducing races 3, 5, and 6 also must be considered. To ensure that this selection proceeds efficiently, the racial structure of the pathogen population should be monitored on a regular basis. As well, a pre-emptive breeding strategy should be considered (27). Race 5, initially identified in North Africa (24), has recently been found in North America (1,31). Recently identified races 9 and 10, from South America, possess virulence not yet found in the current North American races (2,3) and could be used in confined tests of North American cultivars and breeding material to identify sources of resistance.

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