Inheritance of Stripe Rust Resistance in Wheat Cultivars Postulated to Have Resistance Genes at *Yr*3 and *Yr*4 Loci

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ABSTRACT

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It has been postulated that wheat (*Triticum aestivum*) cultivars Cappelle Desprez, Druchamp, Hybrid 46, Minister, Nord Desprez, Stephens, Vilmorin 23, and Yamhill have genes for resistance to stripe rust at the *Yr*3 locus and/or the *Yr*4 locus. Seedlings of parents and F_1 and F_2 progeny from diallel crosses among the cultivars and of the eight cultivars crossed with Chinese 166 were tested for resistance to selected North American races of *Puccinia striiformis*. Seedlings of some BC_1 progeny also were tested. Yamhill has three resistance genes; the other cultivars have two resistance genes. Common loci were not detected in crosses of Hybrid 46 with Druchamp, Minister, or Stephens nor in crosses of Yamhill with Stephens. Minister, Cappelle Desprez, Druchamp, Nord Desprez, and Stephens have resistance genes at the *Yr*3 locus, but the gene at the *Yr*3 locus in Minister is different from the genes at the *Yr*3 locus in the other cultivars. Hybrid 46, Vilmorin 23, and Yamhill have resistance genes at the *Yr*4 locus, but the gene in Hybrid 46 is different from the genes at the *Yr*4 locus in Vilmorin 23 and Yamhill. The second resistance gene in Hybrid 46 is not at the *Yr*3 locus. Based on these results: *Yr*2 is in Yamhill; *Yr*3a is in Cappelle Desprez, Druchamp, Stephens, and Nord Desprez; *Yr*3c is in Minister; *Yr*3b is not in Hybrid 46 as previously reported; *Yr*4a is in Cappelle Desprez, Vilmorin 23, and Yamhill; and *Yr*4b is in Hybrid 46. In addition, stripe rust resistance genes not named previously were detected in Druchamp, Stephens, Nord Desprez, Vilmorin 23, Yamhill, Minister, and Hybrid 46. These genes are designated provisionally as *Yr*Dru, *Yr*Ste, *Yr*ND, *Yr*V23, *Yr*Yam, *Yr*Min, and *Yr*H46. Expression of dominance or recessiveness of resistance genes changed in many of the cultivars, depending on the race used in the test. Various epistatic interactions were observed also. Differences in resistance were observed in some reciprocal crosses, which suggests that there are maternal cytoplasmic effects on the expression of resistance by the genes.

Additional keywords: gene interaction, specific resistance, yellow rust.

Stripe rust (yellow rust), caused by *Puccinia striiformis* Westend., is an important disease of wheat (*Triticum aestivum* L.) in many regions of the world. In North America, the disease is most destructive in the western United States and is sometimes destructive in the south-central United States (15). Use of resistant cultivars is the most economical method of controlling the disease. Biffen in 1905 (4) first reported that stripe rust resistance is inherited in a Mendelian fashion. The existence of dominant and recessive genes for stripe rust resistance has been reported (25), but in most cases, the genes are not named, and the relationship of the genes to one another has not been reported. Zadoks in 1961 (27) postulated that Cappelle Desprez, Nord Desprez, and Vilmorin 23 have a common gene, "M," for stripe rust resistance. Lupton and Macer (19) designated stripe rust resistant genes in seven wheat cultivars as *Yr*1, *Yr*2, *Yr*3, and *Yr*4 and reported that three resistance alleles occur at the *Yr*3 locus, and two resistance alleles occur at the *Yr*4 locus. Specifically, they found that Cappelle Desprez has a resistance gene at *Yr*3a and *Yr*4a; Hybrid 46 has a resistance gene at *Yr*3b and *Yr*4b; and Minister has a resistance gene at *Yr*3c. Later, Macer (22) and Bayles and Thomas (3) suggested that both *Yr*3a and *Yr*4a are in Nord Desprez and Vilmorin 23. Based on race reaction and pedigree, it also has been postulated that other cultivars have resistance genes at the *Yr*3 and *Yr*4 loci (2,23,24,26). By comparing interactions of world, European, and North American differential cultivars, using races of *P. striiformis* from North America and Europe, de Vallavieille-Pope and Line (8) suggested that Stephens has a resistance gene at *Yr*3. Line et al (5,6,13,16) reported that Stephens, Druchamp, and Yamhill have resistance genes at the *Yr*3 or *Yr*4 loci. The resistance genes at the *Yr*3 and *Yr*4 loci have been reported to change in their expression of dominance and recessiveness depending on the race used in the test and/or on the parent used in the cross (5,19).

TABLE 1. Infection types[a] produced by seven North American races of *Puccinia striiformis* on wheat cultivars Cappelle Desprez (CD), Druchamp (Dru), Hybrid 46 (H46), Minister (Min), Nord Desprez (ND), Stephens (Ste), Vilmorin 23 (V23), Yamhill (Yam), and Chinese 166 (C166) and F_1 progeny of crosses among the cultivars

Race	Parent 2	Parent 1								
		CD	Dru	H46	Min	ND	Ste	V23	Yam	C166
CDL-21	CD									
	Dru	0	0							
	H46	0	0	0						
	Min	0	0	0	0					
	ND	0	0	0	0	0				
	Ste	0	0	1/3	1/0	0	0			
	V23	0	0	0	0	0	0	0		
	Yam	0	0/−	−/0	0	0/1	1/−	0	0	
	C166	0/−	0/−	1/−	0/−	1/−	0/−	1/−	0/−	8
CDL-17	CD	1								
	Dru	1/−	1							
	H46	8	2/−	1						
	Min	1	−/1	2	1					
	ND	1/−	−/1	8/5	1	1				
	Ste	2	−	8	1	1/2	2			
	V23	8	2/1	1	2/−	2	3/2	5		
	Yam	3/−	−	8	−	−	−	8	8	
	C166	8/−	8/−	8/−	8/−	8/−	8/−	8/−	8/−	8
CDL-35	CD	2								
	Dru	0	0							
	H46	2	2	1						
	Min	2/0	0	2	0					
	ND	2	2/0	8	2	8				
	Ste	5/2	0	8/5	2/1	8	8			
	V23	5/2	1	8/5	0/3	8/−	−/8	8		
	Yam	2/−	−	−/2	2/−	−	−/2	2/5	0	
CDL-6	CD	2								
	Dru	2/−	8							
	H46	3/2	5/−	1						
	Min	2	−/8	8	1					
	ND	5	−/8	5/8	3	2				
	Ste	5/2	8	5/−	5/2	5/3	2			
	V23	8/−	−/8	2/5	8	5/8	5/3	8		
	Yam	2/−	−	−/5	3/2	−	−	5/3	0	
CDL-20	CD	2								
	Dru	5/−	8							
	H46	8/2	8/−	1						
	Min	8	−/8	8	8					
	ND	8/−	−/8	8	8/−	8				
	Ste	8	−	8	8	8	8			
	V23	8/−	−	8	8/−	8	8	8		
	Yam	2/−	−	5	−	−	−	2/8	2	
CDL-25	CD	8								
	Dru	−	8							
	H46	−/8	−	1						
	Min	8/−	−/8	−	8					
	ND	8/−	−	−	−	8				
	Ste	−/8	−	−/8	−	8/−	8			
	V23	−	−	−	−	−/8	−	8		
	Yam	−	−	−/8	−	8/−	−	8/−	8	
CDL-29	CD	2								
	Dru	−	2							
	H46	−/2	−	2						
	Min	−/1	−	−/5	2					
	ND	2/1	−	5	−	2				
	Ste	−/2	−	8	−/2	2/−	2			
	V23	−	−	−	−	2/−	−/2	5		
	Yam	−	−	−	−	8/−	−	2/−	2	

[a] Infection types (IT) are designated by a single number (0–3, 5, or 8) for the selfed parent and F_1 progeny from reciprocal crosses that had the same IT. ITs separated by a slash (/) indicate different ITs for the reciprocal crosses. In such cases, the first number is the IT when parent 1 was the female, and the second number is the IT when parent 2 was the female. − = no F_1 data. Plants with IT 0–3 are resistant, plants with IT 5 are intermediate, and plants with IT 8 are susceptible.

The purpose of this study was to determine the inheritance of stripe rust resistance in eight cultivars that have been postulated to have resistance genes at the *Yr*3 locus, the *Yr*4 locus, or both loci. The objectives were to identify the resistance genes in the cultivars; to determine their relationship to one another, their mode of inheritance, and the possible cytoplasmic effects on stripe rust resistance; to elucidate the nature of the multiple alleles at the *Yr*3 and *Yr*4 loci; and to use information on resistance genes to postulate which genes for virulence are in specific pathogenic races.

MATERIALS AND METHODS

All possible reciprocal, diallel crosses between wheat cultivars Cappelle Desprez (CD), Druchamp (Dru), Hybrid 46 (H46), Minister (Min), Nord Desprez (ND), Stephens (Ste), Vilmorin 23 (V23), and Yamhill (Yam) and all crosses between CD, Dru, H46, Min, ND, Ste, V23, and Yam and Chinese 166 (C166) were made in the greenhouse. C166, Dru, Ste, and Yam are used to differentiate races of *P. striiformis* in North America, and C166, H46, ND, and V23 are world or European differentials (15).

A single representative plant of each cultivar was used as the original parent. F_2 seeds were obtained from F_1 plants grown in the field. In addition, some backcrosses were made in the field, using F_1 plants as the female parent.

To evaluate the plants for resistance, seeds of the parents and F_1, F_2, and BC_1 progeny were planted in plastic pots filled with a potting mixture consisting of peat/perlite/sand/Palouse silt loam soil/vermiculite at a ratio of 6:2:3:3:4, with lime, Osmocote 14-14-14, and ammonium nitrate added at 1.7, 3.3, and 2.2 g/L, respectively. About 10 seeds were planted in each pot. For each cross, 10–30 parental seeds, three to seven F_1 seeds, and 121–944 F_2 seeds were used, depending on the cross and race. F_2 seeds for each test were usually from a single F_1 plant.

Seedlings were grown in a rust-free greenhouse with a diurnal temperature cycle of 10–25 C. At the two-leaf stage, seedlings were inoculated uniformly with urediospores of a specific test race, were placed in a dew chamber at 10 C for 24 hours, and were placed in a temperature controlled chamber with a diurnal temperature cycle that gradually changed from 4 C at 2 A.M. to 20 C at 2 P.M. The light period consisted of daylight supplemented with metal halide lights to extend the duration of light to 16 hours.

North American *P. striiformis* races CDL-21, CDL-17, CDL-29, CDL-35, CDL-6, CDL-20, and CDL-25 were selected for the study based on their virulence on the parental cultivars (15). The infection types produced by the seven races on CD, Dru, H46, Min, ND, Ste, V23, Yam, and C166 are shown in Table 1. C166 was used as a susceptible parent in tests with race CDL-21 because the race is avirulent on the eight cultivars (Table 1)

TABLE 2. Segregation ratios of F_2 progeny for crosses of wheat cultivars Cappelle Desprez (CD), Druchamp (Dru), Hybrid 46 (H46), Minister (Min), Nord Desprez (ND), Stephens (Ste), Vilmorin 23 (V23), Yamhill (Yam), and Chinese 166 (C166) tested with North American races of *Puccinia striiformis*

Cross		Segregation ratio of F_2 progeny tested with races[a]						
Number	P1/P2	CDL-21	CDL-17	CDL-35	CDL-6	CDL-20	CDL-25	CDL-29
1	CD/Dru	1:0	1:0/–	15:1	3:1[b]/–	1:3/–	–	–
2	CD/H46	1:0	7:9	14:1:1/11:2:3	9:7	3:13	–/1:15	–/5:4:7[b]
3	CD/Min	1:0	1:0	15:1	15:1	7:3:6/1:3	1:3[b]/1:3	–/1:0
4	CD/ND	1:0	1:0/–	3:1[b]/14:1:1	7:4:5/9:3:4	1:3/–	1:15/–	1:0/–
5	CD/Ste	1:0	12:1:3/1:0	3:1	7:3:6/11:3:2	1:3	–/0:1	–/1:0
6	CD/V23	1:0	2:1:13	9:4:3/3:1	7:9	1:3	–	–
7	CD/Yam	1:0	3:1	15:1	13:3	13:3	–/1:3	–/13:2:1
8	Dru/H46	1:0/63:1	15:1/12:1:3	13:2:1	9:7	9:7/–	12:1:3/–	–
9	Dru/Min	1:0	–/1:0	15:1	–/1:3	–/1:15	–/0:1	–/1:0
10	Dru/ND	1:0	–/12:1:3	3:1	–/1:3	–/7:9	–	–
11	Dru/Ste	1:0	1:0	3:1	1:3	1:15	0:1	1:0
12	Dru/V23	1:0	13:1:2	3:1	–/1:3	–/1:15	–/0:1	–/9:1:6
13	Dru/Yam	1:0	3:1	13:3/14:1:1	3:1	3:1	3:13/–	13:2:1/–
14	H46/Min	1:0/63:1	13:3	15:1	6:1:9/3:13	1:3	–	7:4:5
15	H46/ND	255:1	21:43/9:7	7:9/9:7	4:5:7/4:1:11	1:3	1:15/–	5:4:7
16	H46/Ste	61:3	3:1:12/6:1:9	1:3/3:1	4:2:10/11:4:1	1:3	1:15/7:2:7	2:1:13/–
17	H46/V23	1:0	1:3/13:3	1:3/3:1	7:9	1:3	–	–
18	H46/Yam	1:0	1:15	13:3/15:1	14:1:1/63:1:0	9:7	–/1:3	–/15:1
19	Min/ND	1:0	1:0	3:1	15:1	1:15/–	–	–
20	Min/Ste	1:0	1:0	3:1	56:7:1	1:15	–	–/13:3
21	Min/V23	1:0	15:1	9:1:6/7:1:8	1:3	3:13	0:1/–	12:3:1
22	Min/Yam	1:0	3:1	13:1:2/14:1:1	58:3:3/59:4:1	9:7	1:1:14/–	11:2:3/–
23	ND/Ste	1:0	1:0	1:15	12:2:2/10:4:2	0:1	0:1/–	1:0
24	ND/V23	1:0	3:1	3:13/–	10:3:3/9:7	1:15	0:1	9:7
25	ND/Yam	1:0	3:1/–	3:1/3:1[b]	12:2:2/–	3:1/–	1:3/–	9:7/–
26	Ste/V23	1:0	3:1	1:15	11:2:3/9:2:5	1:15	0:1/–	7:5:4/7:9
27	Ste/Yam	1:0/61:2:1	–/1:3	3:1	6:1:9/9:4:3	–/9:1:6	–/0:1	–/7:2:7
28	V23/Yam	1:0	9:3:4[b]/7:3:6	13:1:2/7:5:4	13:2:1/11:2:3	3:1/7:9	2:1:13	9:1:6
29	CD/C166	15:1	1:3	–	–	–	–	–
30	Dru/C166	15:1	1:3	–	–	–	–	–
31	H46/C166	3:1	1:15	–	–	–	–	–
32	Min/C166	15:1	1:3	–	–	–	–	–
33	ND/C166	15:1	1:3	–	–	–	–	–
34	Ste/C166	15:1	3:13	–	–	–	–	–
35	V23/C166	15:1	0:1	–	–	–	–	–
36	Yam/C166	63:1	0:1	–	–	–	–	–

[a] Ratios of resistant (infection types [IT] 0–3), intermediate (IT 5), and susceptible (IT 8) or ratios of resistant to susceptible ITs. The size of F_2 populatons (121–944) was adequate for detecting at least three genes, and the size of nonsegregated F_2 populations (231–843, 124–475, and 228–432 for races CDL-21, CDL-17, and CDL-29, respectively) was usually adequate for detecting four genes. Except where indicated $P > 0.05$. Ratios separated by a lash (/) indicate different ratios for reciprocal crosses. In such cases, the first ratio is the ratio in which P1 (Parent 1) was the female parent, and the second ratio is the ratio in which P2 (Parent 2) was the female parent. – = no data.

[b] $P < 0.05$ but > 0.01.

and on all other North American, world, and European differential cultivars (8,15), except C166. Of the races available, CDL-21 should detect the greatest number of resistance genes in most of the cultivars. To prevent the mixing of races, the inoculum of each race was increased on plants in isolation booths and when possible, was maintained on cultivars susceptible to the specific race but resistant to the other races. Races that were virulent on common cultivars were increased in separate facilities at different times. For each test, the North American differential cultivars (15) also were inoculated to confirm the purity of the race used.

Infection-type (IT) data were recorded 18–21 days after inoculation, based on the 0–9 scale described by Line at al (14,17). Using their concept of basic and expanded scales, IT 0–3, 5, and 8 were recorded. IT 0–3 were considered resistant, IT 5 was considered intermediate, and IT 8 was considered susceptible. The intermediate group (IT 5) was analyzed both as a distinct class and in combination with either the resistant or susceptible class, depending on the cross and test race. Chi-square tests were used to determine the goodness of fit of the segregation ratios.

RESULTS

Number and expression of resistance genes. All parents, except C166 (IT 8), were resistant (IT 0) to race CDL-21 (Table 1). Resistance was completely dominant in the crosses of CD, Dru, Min, Ste, and Yam with susceptible C166. In the crosses of H46, ND, and V23 with C166, ITs of F_1 progeny were slightly higher (IT 1, compared to IT 0 for resistant parents). F_2 segregation ratios (Table 2) of crosses 29–36 tested with the same race indicated that H46 has one dominant gene; CD, Dru, Min, ND, Ste, and V23 have two dominant genes; and Yam has three dominant genes.

Race CDL-17 produced IT 1 on CD, Dru, H46, Min, and ND, produced IT 2 on Ste, and produced IT 5 on V23 (Table 1). Yam and C166 were susceptible to CDL-17. F_1 plants from the cross of CD with Yam were resistant, and F_1 plants from the crosses of CD with C166, Dru with C166, H46 with Yam and C166, Min with C166, ND with C166, Ste with C166, and V23 with Yam and C166 were susceptible (Table 1). In the F_2 generation (Table 2) of crosses of resistant parents CD (cross 7), Dru (cross 13), Min (cross 22), and ND (cross 25) with Yam, resistance in each parent was conferred by one dominant gene; however, resistance was inherited in a recessive manner in crosses with C166 (crosses 29, 30, 32, and 33). Resistance in H46 when crossed with either Yam or C166 (crosses 18 and 31) was the result of two recessive genes. One recessive gene was detected in Ste when crossed with Yam (cross 27), but the 3:13 ratio of Ste crossed with C166 (cross 34) indicated either one recessive and one dominant resistance gene in Ste or one recessive gene in Ste with two epistatic susceptible genes in C166. Although the ratios of the cross of V23 with Yam (cross 28) indicated two genes and that of V23 with C166 (cross 35) indicated no gene, the moderate resistance of V23 was probably conferred by one gene, indicated by the digenic ratios of the crosses with CD, Dru, and Min (crosses 6, 12, and 21).

Race CDL-35 was avirulent on CD, Dru, H46, Min, and Yam and was virulent on ND, Ste, and V23 (Table 1). Based on F_2 (Table 2) and backcross segregation ratios (data not shown) of resistant × susceptible crosses, one dominant gene for resistance was detected in Dru (crosses 10–12), Min (crosses 19 and 20), Yam (crosses 25 and 27), and CD (crosses 4–6). In crosses of H46 with the susceptible cultivars (crosses 16 and 17), resistance conferred by one gene in H46 was dominant when H46 was the male parent and was recessive when H46 was the female parent.

Race CDL-6 produced IT 0 on Yam, produced IT 1 on H46 and Min, produced IT 2 on CD, ND, and Ste, and produced IT 8 on Dru and V23 (Table 1). Based on F_1 data (Table 1), resistance in H46 was dominant (H46 × V23) or partially dominant (Dru × H46 and V23 × H46), and resistance in ND and Ste was recessive (ND × Dru, V23 × ND, and Ste × Dru) or partially recessive (V23 × ND and Ste × V23). Based on F_2 data (Table 2), one resistance gene was detected in CD (cross 1), Min (crosses 9 and 21), ND (cross 10), Ste (cross 11), and Yam (cross 13). Two dominant resistance genes were detected in H46 when crossed with Dru (cross 6), but two recessive resistance genes were detected in H46 when crossed with V23 (cross 17). When crossed with Dru, one dominant gene was detected in Yam (cross 13). One recessive gene was detected in Min when crossed with either Dru (cross 9) or V23 (cross 21). A dominant gene was detected in CD when crossed with Dru (cross 1), and one recessive gene was detected when CD was crossed with V23 (cross 6). In contrast, one recessive gene was detected in ND (cross 10) and Ste (cross 11) when crossed with Dru, and one dominant gene was detected in ND (cross 24) and Ste (cross 26) when crossed with V23.

Race CDL-20 produced IT 1 on H46, produced IT 2 on CD and Yam, and produced IT 8 on Dru, Min, ND, Ste, and V23 (Table 1). Based on F_1 data (Table 1), the resistance in Yam was either dominant or recessive, depending on the female parent. In crosses with the susceptible cultivars (Table 2), one resistance gene was detected in CD (crosses 1 and 4–6), H46 (crosses 14–17), and Yam (crosses 13, 25, and 28). The gene in Yam was dominant (crosses 13 and 18 and cross 28 with V23 as the female parent), and the genes in CD and H46 were recessive (crosses 1 and 4–6 and cross 3 with Min as the female parent).

H46 was resistant to (IT 1) and the other cultivars were susceptible (IT 8) to race CDL-25 (Table 1). When crossed with CD (cross 2), ND (cross 15), Ste (cross 16), and Yam (cross 18), two recessive resistance genes were detected in H46 (Table 2). In the cross of Dru with H46 (cross 8), one dominant and one recessive gene were detected.

Gene interactions and relationships. When F_2 data were tested with race CDL-21 (Table 2), common, allelic, or closely linked genes were detected in all crosses, except the crosses of H46 with Dru (cross 8), Min (cross 14), ND (cross 15), and Ste (cross 16), and the cross of Yam with Ste (cross 27). IT 0 and 1 segregated at 15:1 in the crosses of CD with ND (data not shown). Thus, CD and ND have a common gene for resistance that was expressed as IT 1 when alone and as IT 0 in combination with the other genes. A second, different resistance gene also was detected in CD and ND.

With race CDL-17 (Table 2), common, allelic, or closely linked genes were detected in crosses of CD with Dru (cross 1), Min (cross 3), ND (cross 4), and Ste (cross 5), in crosses of Dru with Min (cross 9) and Ste (cross 11), in crosses of Min with ND (cross 19) and Ste (cross 20), and in crosses of ND with Ste (cross 23). No common resistance genes were detected in crosses of H46 with CD (cross 2), Dru (cross 8), Min (cross 14), ND (cross 15), or Ste (cross 16). The F_2 data of the tests with race CDL-29 (Table 2) provided further evidence that H46 does not have a gene in common with Min, ND, or Ste.

No common genes for resistance were detected with races CDL-35, CDL-6, CDL-20, or CDL-25 (Table 2). When tested with race CDL-35, F_2 progeny of all crosses segregated, indicating that the genes effective against race CDL-35 in CD, Dru, H46, Min, and Yam are different from each other (crosses 1–3, 7–9, 13, 14, 18, and 22). In susceptible × susceptible crosses (Table 2), two recessive genes for resistance were detected in crosses of ND with Ste (cross 23) and V23 (cross 24) and of Ste with V23 (cross 26). When F_2 plants were tested with race CDL-6 (Table 2), each resistant cultivar had at least one different gene. In resistant × resistant crosses (Table 1), F_1 plants from crosses of CD with ND and Ste; H46 with ND, Ste, and Yam; Min with ND, Ste, and Yam; and ND with Ste had higher infection types than those of their parents, indicating that the genes interacted with each other. F_1 plants tested with race CDL-20 from CD × Yam and H46 × CD crosses were resistant, from the H46 × CD cross were susceptible, and from H46 × Yam crosses were intermediate, indicating that the effective gene in H46 is different from the genes in CD and Yam (Table 1). Complementary genes for resistance were detected in F_2 progeny (Table 2) from crosses of Yam with Min (cross 22) and Ste (cross 27). All susceptible × susceptible crosses, except crosses of ND with Ste (cross 23), produced resistant progeny and a segregation

ratio indicating one recessive resistance gene in each parent (crosses 9–12, 19–21, 24, and 26). These results indicate that ND and Ste share common loci for susceptibility. F_2 progeny (Table 2) from crosses of Dru with Min (cross 9), Ste (cross 11), and V23 (cross 12); of Ste with CD (cross 5), ND (cross 23), V23 (cross 26), and Yam (cross 27); and of V23 with Min (cross 21) and ND (cross 24) were all susceptible to race CDL-25. Therefore, those parents have common loci for susceptibility and do not have complementary alleles for resistance to race CDL-25. The ratio from cross 4 (CD × ND) indicated each parent has a recessive gene for resistance, and the two genes are hypostatic.

Comparison of reciprocal crosses. Differences in infection types were observed in the F_1 progeny of reciprocal crosses tested with all the races, except CDL-25 (Table 1). For example, in the H46 × Ste crosses tested with race CDL-21, IT 1 was produced when H46 was the female, but IT 3 was produced when H46 was the male parent. When tested with race CDL-35, F_1 progeny from the crosses of Ste with CD produced IT 2 when Ste was the female parent but produced IT 5 when Ste was the male parent. When tested with race CDL-20, F_1 plants in the crosses of H46 with CD produced IT 2 when H46 was the female parent and produced IT 8 when H46 was the male parent.

Reciprocal differences in expression of dominance in F_2 tests also were observed (Table 2). For example, in progeny of Dru × H46 (cross 8) tested with race CDL-17, one of the two resistance genes was dominant when Dru was the female parent but was recessive when Dru was the male parent. In reciprocal crosses of CD with ND (cross 4) and of Ste with Yam (cross 27) tested with race CDL-6, two recessive genes for resistance were detected when CD or Ste were the female parents, and two dominant genes were detected when ND or Yam were the female parents.

Reciprocal differences in nonallelic gene interactions were observed in tests with races CDL-17, CDL-6, and CDL-25 (Table 2). In addition, differences in the number of resistance genes were observed in a few reciprocal crosses (crosses 15 and 17 tested with race CDL-17, crosses 4 and 6 tested with race CDL-35, and crosses 3 and 28 tested with race CDL-20).

DISCUSSION

Each cultivar used in this study was represented by a single plant to minimize genetic variation, to test the same progeny with different races, and to compare reciprocal crosses. Because the cultivars may not consist of only one genotype, these results may not reflect the genetic background of the whole cultivar population. However, the plants were representative of each cultivar, based on morphology and race interactions.

Identification of resistance genes. Twelve resistance genes were identified at 10 loci in the eight cultivars postulated to have resistance genes at the *Yr*3 and *Yr*4 loci. Of the 12 genes, four have not been reported previously. Using European races of *P. striiformis*, Lupton and Macer (19) reported that CD had the genes *Yr*3a and *Yr*4a, H46 had genes *Yr*3b and *Yr*4b, and Min had gene *Yr*3c. They also concluded that *Yr*3a, *Yr*3b, and *Yr*3c were alleles, and *Yr*4a and *Yr*4b were alleles. We detected two genes in CD that we considered to be *Yr*3a and *Yr*4a. Two genes were also detected in H46 and Min. One of the genes in H46 is at the *Yr*4 locus; it should be *Yr*4b because it is allelic to *Yr*4a in CD. However, the other gene in H46 is not *Yr*3b because it is not at the same locus as *Yr*3a in CD. One of the genes in Min should be *Yr*3c because it is at the same locus as *Yr*3a in CD. The other gene in Min has not been reported previously. The two genes in Min are not allelic to either of the two genes in H46, providing further evidence that H46 does not have a resistance gene at the *Yr*3 locus, as interpreted by Lupton and Macer (19).

Zadoks (27) postulated that CD, ND, and V23 have a common resistance gene, and Bayles and Thomas (3) suggested that ND and V23 have the same genes as CD (*Yr*3a and *Yr*4a). Our results show that ND has *Yr*3a in common with CD, but CD does not have a resistance gene at the *Yr*4 locus. Also, V23 has *Yr*4a in common with CD, which is at the same locus as *Yr*4b in H46, but V23 does not have a resistance gene at the *Yr*3 locus. Crosses of ND with V23 tested with races CDL-17 and CDL-29 further indicated that V23 does not have a resistance gene at the *Yr*3 locus. However, ND and V23 may have resistance genes at a common locus or have closely linked genes, because no segregation was found in crosses of ND with V23 (cross 24) when tested with race CDL-21 (Table 2). The additional genes in ND and V23 are designated provisionally as *Yr*ND and *Yr*V23. If these additional genes are at a common locus, they are either different alleles or the same gene. If *Yr*ND and *Yr*V23 are the same, they must be modified by another gene or genes, because when tested with CDL-6, ND was resistant, and V23 was susceptible (Table 1).

Yam has three genes for resistance. We previously identified one of the genes as *Yr*2 (5,6,16). One of the other Yam genes is *Yr*4a, which is also in V23 and CD and is at the same locus as *Yr*4b in H46. The third gene has been designated provisionally as *Yr*Yam (5).

CD, Dru, ND, and Ste have a common ancestor, Vilmorin 27, which may be their source of *Yr*3a. CD and ND were both derived from Hybrid de Joncquois × Vilmorin 27 (18). Dru was derived from Vilmorin 27 × Fleche d'or and Ste from ND × Selection 101 (12,18). V23, the female parent of Hybrid de Joncquois (18), was probably the source of *Yr*4a in CD. Yam was derived from Heines VII × Alba (28). Alba and CD share some ancestors in their pedigree, such as Gros Bleu and Squarehead (28). *Yr*4a in CD and Yam may be from those common ancestors. H46 was derived from Hatif Inversable × Teverson, and Min was derived from Benoist 40 (selected from Hatif Inversable) × Professeur Delos (K_3 × Teverson) (28). Although H46 and Min have Teverson and Hatif Inversable as common ancestors, they do not have a common resistance gene.

From these results and our previous studies (5,6,13,16), at least 32 genes at 30 loci have been identified in 24 cultivars. Some of the genes previously had been postulated, based on race-cultivar interaction and parentage. The *Yr*3 and *Yr*4 loci remain the only loci with confirmed multiple alleles for stripe rust resistance (19). There is a possibility that a gene in ND and a gene in V23 may be allelic at a locus other than the *Yr*3 or *Yr*4, but current data are inconclusive. Of the allelic genes reported by Lupton and Macer (19), we could not detect *Yr*3b in H46.

If we use CD as the starting point for identifying the genes at the *Yr*3 and *Yr*4 loci, Dru, ND, and Ste have *Yr*3a; Min has *Yr*3c; V23 and Yam have *Yr*4a; and H46 has *Yr*4b. In addition, Dru, H46, Min, ND, Ste, V23, and Yam have resistance genes that are not at the *Yr*3 or *Yr*4 loci and that are different from each other. These genes are designated provisionally as *Yr*Dru, *Yr*H46, *Yr*Min, *Yr*ND, *Yr*Ste, *Yr*V23, and *Yr*Yam. All of the genes, except *Yr*H46, are resistant to race CDL-21.

Expression of resistance genes and gene interactions. For many of the genes, expression of dominance and epistasis varied with the test race and parents. For example, with race CDL-21, all of the genes were dominant, except *Yr*H46, which is not resistant to race CDL-21 (Table 2). *Yr*3a expressed dominance in all crosses when tested with race CDL-29. However, when tested with race CDL-17, *Yr*3a was dominant in crosses with Yam (crosses 7, 13, and 25) and recessive in crosses with C166 (crosses 29, 30, 33, and 34) and was recessive in the crosses of Ste with Yam (cross 27).

Reversal of dominance has been reported for resistance to wheat leaf rust (*Puccinia recondita*) (1,9) and stem rust (*Puccinia graminis* f. sp. *tritici*) (11). Both dominant and recessive expressions of *Yr*3a, *Yr*3c, and *Yr*4a were reported by Lupton and Macer (19). The effects of race and parent on expression of dominance for *Yr*2 and *Yr*6 (6,7,10,20,21) as well as other resistance genes (5–7) also have been reported. Environmental factors, such as light and temperature, could alter the expression of resistance. However, such factors could not influence our results because the tests with specific races were conducted at the same time under controlled environmental conditions. Therefore, the reversal of dominance must be the result of gene interactions. Several hypotheses explaining the reversal of dominance (19,25), such

as a dosage effect, closely linked genes, and a heterozygosity effect of pathogen races, have been proposed, but the mechanisms remain unclear. Our results suggest that genes in different cultivars and/or genes in different races may influence the inheritance of the resistance genes.

Nonallelic gene interactions, including both epistasis and complementary interactions, were also common (Table 2). In crosses 2, 15, and 16, the combination of two recessive genes in H46 controlled the resistance to race CDL-25 (Table 2). The 1:15 F_2 ratio of susceptible × susceptible crosses (crosses 9, 11, 12, 19, 20, and 24 when tested with race CDL-20 and cross 4 when tested with race CDL-25) indicates that the combination of two recessive genes conferred resistance to the races. Each parent had a recessive gene for resistance that was not expressed because of epistasis of susceptible genes at another locus. Resistance genes were generally epistatic as indicated by the ratios of 15:1 (crosses 29, 30, 32–35 tested with race CDL-21), 63:1 (cross 36 tested with CDL-21), 13:3 (cross 7 tested with races CDL-6 and CDL-20), 7:9 (cross 6 tested with race CDL-6), and others (Table 2). However, resistance genes in some cases were hypostatic, indicated by ratios of 9:7 (cross 2 tested with race CDL-6) and 3:13 (cross 2 tested with race CDL-20), as well as others (Table 2).

In a few cases, the detected number of resistant genes in a cultivar were different when crosses involved different susceptible cultivars inoculated with the same race. For example, when tested with race CDL-17, Ste was resistant, and both C166 and Yam were susceptible. The F_2 generation (Table 2) segregated at 3:13 when Ste was crossed with C166 (cross 34) but segregated at 1:3 when crossed with Yam (cross 27). The 1:3 ratio, which indicates one recessive gene for resistance in Ste, was consistent with other crosses. The 3:13 ratio could be the result of chance and may in reality be a 1:3 ratio. If both ratios are correct, C166 must have two epistatic genes for susceptibility to the race.

Maternal cytoplasmic effects. The reciprocal differences observed in this study and in our previous studies (6,7) show that there are maternal cytoplasmic effects on the expression of stripe rust resistance. The majority of reciprocal differences were associated with races CDL-6, CDL-17, and CDL-35. Therefore, the reciprocal differences observed may be the result of cytoplasm-genotype interactions. In this study, cytoplasmic genes did not determine stripe rust resistance directly, but they may have modified expressions of nuclear resistance genes. The cytoplasm of H46 and V23 appeared to interact with resistance genes more often than did those of other cultivars. In reciprocal crosses of H46 with CD (cross 2), ND (cross 15), Ste (cross 16), V23 (cross 17), and Yam (cross 18 tested with race CDL-35), the resistance gene (*Yr*H46) was recessive when H46 was the female parent but was dominant when CD, ND, Ste, V23, or Yam were the female parents (Table 2). However, reciprocal crosses of H46 with Dru (cross 8) and Min (cross 14) did not show such differences (Table 2). Thus, the maternal or cytoplasmic effect of H46 is also specific to genetic background. In conclusion, the reciprocal differences suggest there are modifying maternal cytoplasmic factors that affect the expression of stripe rust resistance.

Virulence genes in stripe rust races and use of the resistance genes. The information on genes for stripe rust resistance in the eight wheat cultivars, their mode of inheritance, and maternal cytoplasmic effects are important in studying virulences in the pathogen, in understanding host-pathogen relationships, and in breeding for stripe rust resistance. Because the fungus does not have a pycnidial and aecial stage, virulence genes in *P. striiformis* cannot be determined by crossing different races. However, virulence genes can be postulated, based on the interactions of races of the pathogen and host cultivars with known resistance genes. Based on this study, virulence genes in the seven races corresponding to the resistance genes in the eight cultivars can be postulated. Table 3 shows that a specific race has virulence genes that overcome specific resistance genes. This information should be useful for more accurately monitoring and identifying the virulences of races and for determining the relationships among pathogen populations and the evolution of races. The genes identified in this study can be useful in developing resistant cultivars and multilines, and the information on the inheritance and expression of the resistance genes and the effects of maternal cytoplasm can aid in parental selection and screening procedures.

LITERATURE CITED

1. Anderson, R. G. 1966. Studies on the inheritance of resistance to leaf rust of wheat. Hereditas Suppl. 2:144-155.
2. Bartos, P., Johnson, R., and Stubbs, R. W. 1987. Postulated genes for resistance to yellow rust in Czechoslovakian wheat cultivars. Cereal Rusts Bull. 15:79-84.
3. Bayles, R. A., and Thomas, J. E. 1984. Yellow rust of wheat. U. K. Cereal Pathog. Virul. Surv. Ann. Rep. 1984, 18-25.
4. Biffen, R. H. 1905. Mendel's law of inheritance and wheat breeding. J. Agric. Sci. 1:4-48.
5. Chen, X. M., and Line, R. F. 1992. Identification of stripe rust resistance genes in wheat genotypes used to differentiate North American races of *Puccinia striiformis*. Phytopathology 82:1428-1434.
6. Chen, X. M., and Line, R. F. 1992. Inheritance of stripe rust resistance in wheat cultivars used to differentiate races of *Puccinia striiformis* in North America. Phytopathology 82:633-637.
7. Chen, X. M., and Line, R. F. 1992. Genes for resistance to stripe rust in 'Tres' wheat. Crop Sci. 32:692-696.
8. de Vallavieille-Pope, C., and Line, R. F. 1990. Virulences of North American and European races of *Puccinia striiformis* on North American, world, and European differential wheat cultivars. Plant Dis. 74:739-743.

TABLE 3. Expression of resistance genes in wheat and virulence genes in North American races of *Puccinia striiformis*, based on F_2 segregation ratios in Table 2

Resistance gene	Cultivar[a]	Infection types[b] produced by North American races						
		CDL-21	CDL-17	CDL-35	CDL-6	CDL-20	CDL-25	CDL-29
*Yr*1	C166	H	H	L	L	L	L	L
*Yr*2	Yam	L	H	L	L	L	H	H
*Yr*3a	CD, Dru, ND, Ste	L	L	H	H	H	H	L
*Yr*3c	Min	L	L	L	L	H	H	L
*Yr*4a	CD, V23, Yam	L	H	L	L	L	H	H
*Yr*4b	H46	L	L	H	H	L	L	L
*Yr*ND	ND	L	H	H	H	H	H	H
*Yr*Dru	Dru	L	H	L	H	H	H	H
*Yr*Ste	Ste	L	H	H	L	H	H	b
*Yr*Min	Min	L	H	H	H	H	H	H
*Yr*V23	V23	L	I	H	H	H	H	I
*Yr*Yam	Yam	L	H	H	H	H	H	L
*Yr*H46	H46	H	L	L	L	H	L	H

[a] Cultivars that have corresponding resistance genes include: C166 = Chinese 166, CD = Cappelle Desprez, Dru = Druchamp, H46 = Hybrid 46, Min = Minister, ND = Nord Desprez, Ste = Stephens, V23 = Vilmorin 23, and Yam = Yamhill.

[b] H = high-infection type (IT 8), the resistance gene is ineffective and the race is virulent; I = intermediate-infection type (IT 5), the race resistance and virulence are intermediate; and L = low-infection type (IT 0–3), the resistance gene is effective and the race is avirulent.

9. Dyck, P. L., and Samboski, D. S. 1974. Inheritance of virulence in *Puccinia recondita* on alleles at the *Lr*2 locus for resistance in wheat. Can. J. Genet. Cytol. 12:689-694.
10. Johnson, R., Taylor, A. J., and Smith, G. M. B. 1986. Resistance to British races of *Puccinia striiformis* in the differential wheat cultivars Heines Kolben and Heines Peko. Cereal Rusts Bull. 14:20-23.
11. Knott, D. R., and Anderson, R. G. 1956. The inheritance of rust resistance. I. The inheritance of stem rust resistance in ten varieties of common wheat. Can. J. Agric. Sci. 36:174-195.
12. Kronstad, W. E., Rohde, C. R., Kolding, M. F., and Metzger, R. J. 1978. Registration of Stephens wheat. Crop Sci. 18:1097.
13. Line, R. F., and Chen, X. M. 1988. Inheritance of resistance to *Puccinia striiformis* in North American, European and World differential wheat cultivars. (Abstr.) Pap. Int. Cong. Plant Pathol. 5th, 275.
14. Line, R. F., Konzak, C. F., and Allan, R. E. 1974. Evaluating resistance to *Puccinia striiformis* in wheat. Induced Mutations for Disease Resistance in Crop Plants. Int. At. Energy Agency (Vienna) 180:125-132.
15. Line, R. F., and Qayoum, A. 1992. Virulence, aggressiveness, evolution, and distribution of races of *Puccinia striiformis* (the cause of stripe rust of wheat) in North America, 1968–1987. USDA Tech. Bull. 1788, 44 pp.
16. Line, R. F., Qayoum, A., and Chen, X. M. 1988. Virulence, evolution, and distribution of *Puccinia striiformis* in North America and inheritance of stripe rust resistance in differential cultivars. Proc. Eur. and Mediterr. Cereal Rusts Conf. 7th, 107-109.
17. Line, R. F., Sharp, E. L., and Powelson, R. L. 1970. A system for differentiating races of *Puccinia striiformis* in the United States. Plant Dis. Rep. 54:992-993.
18. Lupton, F. G. H. 1987. Wheat Breeding: Its Scientific Basis. Chapman & Hall (UK), London. 566 pp.
19. Lupton, F. G. H., and Macer, R. C. F. 1962. Inheritance of resistance to yellow rust (*Puccinia glumarum* Erikss. & Henn.) in seven varieties of wheat. Tran. Br. Mycol. Soc. 45:21-45.
20. Macer, R. C. F. 1963. The formal and monosomic genetic analysis of stripe rust (*Puccinia striiformis*) resistance in wheat. Pages 127-143 in: Proc. 2nd Int. Wheat Genet. Symp. Vol. 2. M. James, ed. Univ. Lund, Sweden.
21. Macer, R. C. F. 1966. Pages 19-26 in: The inheritance of resistance to yellow rust in wheat. Proc. Eur. Yellow Rust Conf., 1964. 3rd.
22. Macer, R. C. F. 1975. Plant pathology in a changing world. Trans. Br. Mycol. Soc. 65:351-374.
23. McIntosh, R. A. 1983. A catalogue of gene symbols for wheat (1983 edition). Pages 1197-1254 in: Proc. Int. Wheat Genet. Sym., 6th. S. Sakamoto, ed.
24. McIntosh, R. A. 1986. Catalogue of gene symbols for wheat. 1986 supplement. Cereal Res. Commun. 14:105-115.
25. Röbbelen, G. H. C., and Sharp, E. L. 1978. Mode of Inheritance, Interaction and Application of Genes Conditioning Resistance to Yellow Rust. Verlag Paul Parey, Berlin. 88 pp.
26. Worland, A. J., and Law, C. N. 1987. Seedling resistance to yellow rust. Ann. Rep. Plant Breeding Inst., Cambridge. 1986, 74-75.
27. Zadoks, J. C. 1961. Yellow rust on wheat: Studies in epidemiology and physiologic specialization. Tijdschr. Plantenziekten 67:69-256.
28. Zeven, A. C., and Zeven-Hissink, N. C. 1976. Genealogies of 14,000 wheat varieties. The Netherlands Cereals Centre-NGC, Wageningen, and The International Maize and Wheat Improvement Center, Mexico. 119 pp.