Identification of Stripe Rust Resistance Genes in Wheat Genotypes Used to Differentiate North American Races of *Puccinia striiformis*

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ABSTRACT

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Crosses were made among wheat (*Triticum aestivum*) cultivars used to differentiate North American races of *Puccinia striiformis* and between those cultivars and other cultivars with known resistance genes. Seedlings of the parents and F_1, F_2, and backcross progeny from some crosses were evaluated for stripe rust resistance; eight North American races of *P. striiformis* were used. According to the data, Chinese 166, Heines VII, Fielder, Lee, Riebesel 47/51, and Moro have stripe rust resistance genes *Yr*1, *Yr*2, *Yr*6, *Yr*7, *Yr*9, and *Yr*10, respectively; and a gene in Lemhi, a gene in Tyee, and one of two genes each in Heines VII, Moro, Druchamp, Stephens, Lee, and Fielder is different from named *Yr* genes and other previously described genes. The gene in Lemhi may be allelic or closely linked to a gene in Riebesel 47/51 and one of the genes in Druchamp and Stephens. A gene in Heines VII may be allelic or closely linked with genes in Cappelle Desprez, Nord Desprez, and Stephens. Druchamp, Stephens, and Yamhill have either *Yr*3a or *Yr*4a. Druchamp has a common gene with Stephens, but not with Yamhill. Yamhill (Heines VII/Alba) has *Yr*2 from Heines VII and two genes from Alba. One of two genes of Lee, which is not *Yr*7, may be the same as or closely linked to a gene in Paha. *Yr*1, *Yr*7, *Yr*9, *Yr*10, the gene in Lemhi, and one gene in Paha were dominant. The second gene in Lee and a gene in Paha, which may be alleles, were recessive. *Yr*2, *Yr*6, *Yr*3a, or *Yr*4a, the gene in Tyee, and one of the two genes each in Stephens, Druchamp, and Fielder were dominant or recessive depending on the race and parent (genetic background) in the cross. *Yr*1 in Chinese 166, *Yr*9 in Riebesel 47/51, the gene in Lemhi, and the gene in Tyee were independent (i.e., they showed no interactions with other resistance genes). The remaining resistance genes in the North American differential cultivars showed various interactions with other resistance genes. Expression of epistasis also varied depending on race and genetic background.

Additional keywords: specific resistance, virulence, yellow rust of wheat.

Stripe rust (yellow rust), caused by *Puccinia striiformis* Westend., is an important disease of wheat (*Triticum aestivum* L.) in many parts of the world. In North America, the disease is most destructive in western North America and is sometimes destructive in the south central United States (14). Growing resistant cultivars is the most economical method of controlling stripe rust. Biffen in 1905 (6) first reported that stripe rust resistance followed Mendelian laws. Since then, stripe rust resistance has been identified and incorporated into many commercial cultivars (10,22,23). Fourteen genes for resistance at the seedling and adult stages of plant growth have been identified at the *Yr*1–*Yr*10 and *Yr*15 loci. Multiple alleles have been designated *Yr*3a, *Yr*3b, and *Yr*3c at the *Yr*3 locus and *Yr*4a and *Yr*4b at the *Yr*4 locus (16,22).

The seedling reaction of wheat cultivars is used to differentiate races of *P. striiformis*. Line and Qayoum (14) have reported on the virulence, aggressiveness, evolution, and distribution of races of *P. striiformis* in North America. By 1987, 39 races of *P. striiformis* were identified in North America on the basis of 13 North American differential cultivars. Each of the differential cultivars was resistant to at least one of the 39 races (14). Of the 13 differential cultivars, Chinese 166, Heines VII, Moro, Riebesel 47/51, and Lee have been reported to possess resistance genes *Yr*1, *Yr*2, *Yr*10, *Yr*9, and *Yr*7, respectively (16,18,20,22), but only Chinese 166 and Heines VII have been studied in diallel crosses (16,18,20). We previously reported that Heines VII, Moro, and Lee each has an additional gene; Lemhi, Chinese 166, Riebesel 47/51, and Tyee each has one gene; Druchamp, Produra, Stephens, and Fielder each has two genes; and Paha and Yamhill each has three genes for resistance to stripe rust (7). We also postulated that some of the differential cultivars may have common genes and that many of the genes were different from previously named *Yr* genes according to their interactions with different races and pedigrees. The objectives of this study were to identify the genes in the differential cultivars and determine their relationships and interactions with one another and with known genes in other cultivars.

MATERIALS AND METHODS

Fifty-four crosses among the 13 North American differential cultivars and with other cultivars (Table 1) that have specific genes for stripe rust resistance were made in a greenhouse. Also, some of the F_1 plants were backcrossed to both parents. Spring parents and F_1 plants from crosses of spring wheats and spring × winter wheats were grown in the field. Crosses of winter wheats were grown in the greenhouse in the spring of 1986. Germinated seeds of the parental winter cultivars and the F_1 progeny from crosses between winter cultivars were vernalized in petri dishes at 0–4 C for 40–56 days, or seeds treated with 100 ppm of kinetin (Sigma Chemical Co., St. Louis, MO) distilled water solution (4) were vernalized at 0–4 C for 20–30 days. The seedlings were then transplanted into plastic pots and grown in a greenhouse at 10–30 C for production of seed.

To evaluate the plants for resistance, we planted seeds of the parents and F_1, F_2, and BC_1 progeny in plastic pots filled with a potting mixture consisting of peat, perlite, sand, Palouse silt loam soil, and vermiculite at a ratio of 6:2:3:3:4 with lime, Osmocote 14-14-14, and ammonium nitrate added at 1.69, 3.30 and 2.20 g/L, respectively. About 10 seeds were planted in each pot.

The seedlings were grown in a rust-free greenhouse at a diurnal temperature cycle of 10–25 C. At the two-leaf stage, the seedlings were uniformly inoculated with urediospores of a specific test race, incubated in a dew chamber at 10 C for 24 h, and then

placed in a growth chamber within the greenhouse at a diurnal temperature cycle that gradually changed between 4 C at 2 a.m. and 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 h. Seedlings of the parental cultivars and progeny were tested with the same race at the same time. When possible, 33–44 seeds of parents, four to seven F_1 seeds, 159–625 F_2 seeds, and 24–209 BC_1 seeds were used for each cross depending on the cross and race.

The eight North American races of *P. striiformis* listed in Table 1 were selected on the basis of their avirulence on the specific parents. The race used to evaluate the parents and progeny of a specific cross was avirulent on both parents, except for a few tests. The initial source of inoculum was urediospores that had been stored in liquid nitrogen. Inoculum of each race was originally purified by single-spore isolation or by transfer of single pustules for several generations. Freshly collected spores or spores that had been stored at 4 C for less than 1 mo were used for all tests. To prevent mixing of races, the inoculum of each race was increased on plants in isolation booths and, when possible, maintained on cultivars that were susceptible to that specific race but resistant to the other races. Races virulent on common cultivars were increased in separate facilities at different periods of time. For each test, a set of the 13 differential cultivars were also inoculated to confirm the purity of the race used.

Because infection types produced by some races on some cultivars can vary depending on the duration of time after inoculation (14), data on infection types were recorded twice. The first recording was when uredia were fully developed on the susceptible cultivars (13–18 days after inoculation). The second recording was 7–10 days later (21–25 days after inoculation). Infection types were recorded according to the 0–9 infection type scale described by Line et al (13). Using the concept of basic and expanded scales (13,15), we recorded infection types 0–3, 5, and 8. Infection types 0–3 were considered resistant, infection type 5 was intermediate, and infection type 8 was susceptible. The intermediate class (infection type 5) was analyzed as a distinct class and in combination with the resistant or susceptible classes. Chi-square tests were used to determine the goodness of fit of the segregation ratios.

RESULTS

Data from the F_2 generations are presented in Table 2. In some crosses, more than one ratio was significant; however, only ratios with the best fit are shown. Best fit always occurred either when the intermediate infection types were analyzed separately or when they were combined with resistant infection types, but they never occurred when intermediate infection types were combined with the susceptible infection type. Combining the intermediate with the resistant infection types did not change the number of genes but did change the gene interactions. Corroborating F_1 and backcross data were obtained for some crosses and are discussed in the text. First and last recordings were identical in all tests (cross-race combinations).

Gene identification. F_2 progeny in tests 4-21, 5-21, 6-21, 9-21, 10-21, 25-21, 26-21, 27-21, 28-21, 31-21, 32-21, 33-21, 35-21, 36-21, 43-21, and 48-11 (crosses of Lemhi with Druchamp, Stephens, and Riebesel 47/51; Heines VII with Cappelle Desprez and Nord Desprez; Druchamp with Vilmorin 23, Cappelle Desprez, Nord Desprez, and Hybrid 46; Yamhill with Heines VII, Alba, Nord Desprez, and Vilmorin 23; Stephens with Vilmorin 23; and Fielder with Heines Kolben) did not segregate. These results indicated that the two parents in each cross may have the same gene, alleles, or closely linked genes for resistance to race CDL-21 or race CDL-11.

In tests 17-20, 29-21, 34-11, 44-21, 45-21, 49-11, and 50-11, no susceptible plants were observed in the F_2 progeny, but segregation ratios were obtained between infection types within the resistant groups or between the resistant and intermediate groups. These results indicated that the parents in the crosses of Paha with Lee; Stephens with Druchamp, Nord Desprez, and Heines VII; Yamhill with Heines Peko; Fielder with Heines Peko; and Heines Peko with Heines Kolben may have the same gene, alleles, or closely linked genes as well as different genes for resistance to race CDL-20, CDL-21, or CDL-11.

Two resistance genes were indicated in tests 7-27, 7-29, 8-20, 29-14, 29-22, 37-20, 38-20, 51-20, 52-20, 52-21, 26-22, 43-14, 27-22, 40-1, and 43-22. Thus, in the crosses of Riebesel 47/51 with Chinese 166; Tyee with Chinese 166; Stephens with Druchamp

TABLE 1. Wheat genotypes used in crosses, previously named genes, and the reaction of the cultivars to North American races of *Puccinia striiformis*

Genotype[a]		Named	Differential number[c]			Reaction to North American CDL races[d]							
ID number	Name	genes[b]	NA	World	Europe	1	11	14	17	20	21	22	27
CI011415	Lemhi	Not named	1			S	S	S	S	S	R	S	S
CI011765	Chinese 166	*Yr*1	2	1		S	R	R	S	R	S	R	R
PI201195	Heines VII	*Yr*2	3		8	R	R	R	S	R	R	S	S
CI013740	Moro	*Yr*10	4	5		R	R	R	R	R	R	R	R
CI014485	Paha	Not named	5			R	R	R	R	R	R	R	R
CI013723	Druchamp	Not named	6			R	R	R	R	S	R	R	R
PI295999	Riebesel 47/51	*Yr*9	7	9		R	R	R	R	R	R	R	R
CI017406	Produra	Not named	8			R	R	S	R	S	R	R	R
CI014563	Yamhill	Not named	9			R	R	R	S	R	R	R	R
CI017596	Stephens	Not named	10			R	R	R	R	S	R	R	R
CI012488	Lee	*Yr*7	11	2		R	R	R	S	R	R	R	R
CI017268	Fielder	Not named	12			R	R	S	R	S	R	S	S
CI017773	Tyee	Not named	13			R	R	R	R	R	R	R	S
PI180619	Heines Kolben	*Yr*6		3		R	R	S	R	S	R	S	S
CI011773	Vilmorin 23	*Yr*3a and *Yr*4a		4		R	R	R	S	S	R	R	R
WA007716	Clement	*Yr*9		9		R	R	R	R	R	R	R	R
PI174655	Hybrid 46	*Yr*3b and *Yr*4b			1	R	R	R	R	R	R	R	R
PI180620	Heines Peko	*Yr*6 and *Yr*2			3	R	R	R	R	R	R	S	S
PI167419	Nord Desprez	*Yr*3a and *Yr*4a			4	R	R	R	R	S	R	R	R
PI325842	Compair	*Yr*8			5	R	R	R	R	R	R	R	R
PI192448	Spaldings Prolific	Not named			7	R	R	R	R	R	R	R	R
WA005768	*Triticum spelta album*	*Yr*5				R	R	R	R	R	R	R	R
PI262223	Cappelle Desprez	*Yr*3a and *Yr*4a				R	R	R	R	R	R	R	R
PI191303	Alba	Not named				R	R	R	S	S	R	R	R
CI017419	Daws	Not named				S	R	S	S	S	R	S	S

[a] Lemhi, Produra, Lee, Heines Kolben, Heines Peko, and Compair are spring wheats. All others are winter wheats.
[b] Sources of information on named *Yr* genes are from Bayles and Thomas (6), Johnson et al (10), and McIntosh (24).
[c] North American (NA) differential numbers are from Line and Qayoum (14). World and European numbers are from Stubbs (25).
[d] R = resistant and S = susceptible; CDL = Cereal Rust Laboratory designations.

and Vilmorin 23; Yamhill with Clement, Lee, and Fielder; Tyee with Clement and Hybrid 46; Druchamp with Cappelle Desprez and Nord Desprez; and Yamhill with Fielder, each parent has a different gene for resistance to the specific races.

Three genes were indicated in tests 1-21, 2-21, 15-20, 19-21, 30-21, 53-20, 3-21, 46-20, 21-14, 14-20, 34-11, 39-20, 23-1, 54-1, and 12-14 (crosses of Lemhi with Lee and Fielder; Tyee with Moro, Compair, Lee, Druchamp, and Cappelle Desprez; Druchamp with *Triticum spelta album*, Compair, and Moro; Riebesel 47/51 with Compair; and Yamhill with Moro, Heines Peko, and Compair). The results indicated that each parent has one or two genes, and the two parents do not have common genes for resistance to the specific races.

Four genes were indicated in tests 11-21, 47-1, 42-21, 20-11, 22-1, 41-11, 18-21, 20-21, 13-20, 24-1, 41-21, and 16-11 (crosses of Moro with Daws and Compair; Fielder with Compair, Stephens, and Druchamp; Stephens with Compair; Druchamp with Clement and Yamhill; and Paha with Spaldings Prolific and Heines Kolben). Thus, each parent has one, two, or three genes, and the parents do not have a common gene, alleles, or closely linked genes for resistance to the races.

A single recessive gene for resistance was indicated in tests 33-20, 49-14, and 50-14, which involve crosses of resistant and susceptible parents. The results show that Yamhill has a gene for resistance to race CDL-20 that is not in Alba, and Heines Peko has a recessive gene for resistance to race CDL-14 that is not in Fielder and Heines Kolben. F_1 and backcross data corroborate the results from test 49-14. Progeny from a backcross to Fielder were all susceptible, and a backcross to Heines Peko segregated at 51:57 for resistant/susceptible plants, which best fits 1:1 ($\chi^2 = 0.23$ and $P = 0.63$). Two recessive genes for resistance were detected in tests 31-27 and 32-27. These crosses also were between resistant and susceptible parents. Those results showed that both Yamhill and Alba have two recessive genes for resistance to race CDL-27, and Heines VII has no resistance to the race. In test 49-27, both parents (Fielder and Heines Peko) were susceptible, and all F_2 plants were susceptible. These data indicated that Fielder and Heines Peko have common loci for susceptibility.

Gene expression and interaction. Two independent, dominant genes were indicated in tests 7-27, 7-29, 8-20, 29-14, 29-22, 37-20, 38-20, 51-20, 52-20, and 52-21; three independent, dominant genes were detected in tests 1-21, 2-21, 15-20, 19-21, 30-21, and 53-20. Two dominant genes and one recessive gene were inherited independently in tests 3-21, 46-20, and 21-14. F_2 ratios in tests 26-22 and 43-14 indicated two dominant genes and gene interactions. Backcross data in test 26-22 agreed with the F_2 data. In the backcross to Cappelle Desprez, all 154 progeny were resistant; in the backcross to Druchamp, 60 plants were resistant and 21 were susceptible, which fit 3:1 ($\chi^2 = 0.00$ and $P = 1.0$). Two partially recessive genes were indicated in tests 27-22 and 40-1. The results also were supported by the intermediate reaction of the F_1 plants and the backcross ratios. In the backcross to Druchamp, 87 plants were resistant (infection types 2–3), and 84 were intermediate to susceptible (infection types 5–8); in the backcross to Nord Desprez, 82 plants were resistant, and 85 were intermediate to susceptible, which best fit 2:2 ($\chi^2 = 0.02$ and $P = 0.88$). In tests 43-22 and 44-22, two dominant genes for resistance and susceptible epistasis were detected.

TABLE 2. Observed numbers of plants showing different infection types (IT) and expected phenotypic ratios of resistant (IT 0–3), intermediate (IT 5), and susceptible (IT 7–8) plants for parents and F_2 progeny inoculated with races of *Puccinia striiformis* at the seedling stage, and chi-square (χ^2) tests with probabilities (P) for goodness of fit

Test (cross-race)	Parents (female/male)	IT[a]		Number of F_2 plants[b]			Expected ratio[c]	χ^2	P[d]
		FP	MP	R(IT)	I(IT)	S(IT)			
1-21	Lemhi/Lee	0	0	439(0–2)	...	9(8)	63:1	0.33	0.57
2-21	Lee/Lemhi	0	0	463(0–2)	...	10(8)	63:1	0.61	0.43
3-21	Lemhi/Fielder	0	1	310(0–1)	...	14(8)	61:3	0.03	0.86
4-21	Druchamp/Lemhi	0	0	340(0–1)	...	...	1:0		
5-21	Stephens/Lemhi	1	0	356(0–1)	...	...	1:0		
6-21	Riebesel 47/51/Lemhi	0	0	222(0–1)	...	...	1:0		
7-27	Riebesel 47/51/Chinese 166	0	1	213(0–1)	...	16(8)	15:1	0.11	0.75
7-29	Riebesel 47/51/Chinese 166	1	2	228(0–2)	...	14(8)	15:1	0.03	0.87
8-20	Tyee/Chinese 166	1	1	588(1–2)	...	37(8)	15:1	0.07	0.80
9-21	Heines VII/Cappelle Desprez	0	0	424(0)	...	...	1:0		
10-21	Heines VII/Nord Desprez	0	0	376(0)	...	...	1:0		
11-21	Daws/Moro	2–3	1	391(1–3)	13(5)	44(8)	229:27[e]	0.18	0.67
12-14	Druchamp/Moro	3	2	341(2–3)	22(5)	57(8)	52:3:9	0.34	0.84
13-20	Moro/Compair	2	2	376(2–3)	...	10(8)	249:7	0.00	1.00
14-20	Yamhill/Moro	2	2	376(2)	...	39(8)	57:7	0.86	0.35
15-20	Tyee/Moro	1	2	343(1–2)	...	9(8)	63:1	1.66	0.20
16-11	Paha/Heines Kolben	1	1	323(1)	52(5)	89(8)	180:27:49	0.23	0.89
17-20	Paha/Lee	2	2	240(2) 5(3)	...	...	63:1	0.12	0.73
18-21	Paha/Spaldings Prolific	2	0	313(0–2)	...	5(8)	253:3	0.16	0.69
19-21	Druchamp/*T. spelta album*	1	0	351(0–3)	...	8(8)	63:1	0.65	0.42
20-21	Druchamp/Fielder	1	2–3	459(1–3)	...	8(8)	253:3	0.76	0.38
20-11	Druchamp/Fielder	2	1	302(1–2)	...	98(8)	193:63	0.00	1.00
21-14	Druchamp/Compair	3	2	434(2–3)	41(5)	23(8)	55:6:3	0.78	0.68
22-1	Druchamp/Clement	2–3	1	185(1–3)	...	59(8)	193:63	0.01	0.94
23-1	Tyee/Druchamp	2	2–3	305(1–3)	...	48(8)	55:9	0.03	0.86
24-1	Yamhill/Druchamp	2	2–3	192(2–3)	47(5)	58(8)	207:49[e]	0.01	0.92
25-21	Druchamp/Vilmorin 23	0	0	249(0–1)	...	...	1:0		
26-21	Druchamp/Cappelle Desprez	0	0	284(0)	...	...	1:0		
26-22	Druchamp/Cappelle Desprez	0	0	240(0–3)	44(5)	23(8)	13:2:1	1.94	0.38
27-21	Druchamp/Nord Desprez	0	0	288(0–1)	...	...	1:0		

continued on next page

[a] IT = infection type; FP = female parent and MP = male parent.
[b] Number of F_2 seedlings with either resistant (R), intermediate (I), or susceptible (S) reactions to *Puccinia striiformis*. Numbers in parentheses represent infection types.
[c] Ratio of resistant to susceptible or ratio of resistant to intermediate to susceptible plants.
[d] P = probability of a greater value due to chance alone.
[e] Expected ratio when intermediate infection types are included with resistant infection type.

Segregation of three dominant genes was indicated from the data of tests 14-20, 34-11, and 39-20, whereas segregation of four dominant genes was indicated in tests 13-20, 24-1, 41-21, and 16-11. One dominant gene and two recessive genes were indicated in tests 23-1, 54-1, and 12-14. One dominant gene and three recessive genes were indicated in tests 11-21 and 47-1. Two dominant and two recessive genes were indicated in tests 42-21, 20-11, 22-1, and 41-11. Three dominant genes and one recessive gene were indicated in tests 18-21 and 20-21. Intermediate plants were observed in tests 12-14, 16-11, 21-14, and 39-20, indicating gene interactions. Susceptible epistases were indicated at two of the loci in tests 13-20, 14-20, 20-11, 22-1, 34-11, 39-20, and 41-11 and at four loci in tests 24-1, 41-21, and 16-11.

The F_2 data of tests 33-20, 49-14, and 50-14 indicated that the single gene in Yamhill for resistance to race CDL-20 and the single gene in Heines Peko for resistance to race CDL-14 are recessive. The data of tests 31-27 and 32-27 indicate that the two genes in Yamhill and Alba for resistance to race CDL-27 are also recessive. These results were supported by the F_1 data from tests 31-27, 32-27, and 49-14; the F_1 plants had an infection type of 8, which was identical to that of the susceptible parent.

All the F_1 results supported the F_2 data. F_1 progeny in tests 7-29, 9-21, 26-22, 28-21, 29-22, 31-21, 32-21, 36-21, 51-20, and 52-21 had infection types identical to those of at least one resistant parent, indicating that the two parents in the crosses may have the same genes or that genes in one parent may be dominant. F_1 progeny in tests 29-21, 30-21, and 35-21 exhibited an infection type of 1, which was slightly higher than that of either parent (infection type 0). Thus, the two parents in the crosses may not have the same genes. Only F_1 plants in test 27-22 had an intermediate infection type (type 5), although both parents were resistant, indicating that Druchamp and Nord Desprez have different genes for resistance to race CDL-22.

DISCUSSION

Figure 1 shows the cultivars with the same gene, alleles, or closely linked genes for resistance to *P. striiformis*, and Table 3 lists the genes identified in the North American differential cultivars. *Yr* genes designated by numbers are genes previously named, and *Yr* genes designated by letters are new, provisional names.

Lemhi was once considered universally susceptible (23). The detection of a race that was avirulent on Lemhi (CDL-21) suggested that Lemhi has a gene or genes for resistance (14). Subsequently, we reported that Lemhi had a single dominant gene for resistance to race CDL-21 (7). In the present study, one single resistance gene in Lemhi (*Yr*Lem) was confirmed. The gene is not linked to any gene in Lee and Fielder. No segregation was detected in F_2 progeny from crosses of Lemhi with Riebesel 47/51, Druchamp, and Stephens when tested with race CDL-21. Therefore, the gene in Lemhi is probably the same, allelic, or closely linked to genes in those cultivars. Because Lemhi is resistant only to race CDL-21 and susceptible to all other North American

TABLE 2. Continued from preceding page

Test (cross-race)	Parents (female/male)	IT[a]		Number of F_2 plants[b]			Expected ratio[c]	χ^2	P[d]
		FP	MP	R(IT)	I(IT)	S(IT)			
27-22	Druchamp/Nord Desprez	0	2	169(2-3)	135(5)	78(8)	7:6:3	1.06	0.59
28-21	Druchamp/Hybrid 46	0	1	431(0-1)	...	...	1:0		
29-21	Stephens/Druchamp	0	0	21(0) 367(1)	...	...	1:15	0.33	0.56
29-14	Stephens/Druchamp	2	3	239(2-3)	19(5)	22(8)	15:1[e]	0.98	0.32
29-22	Stephens/Druchamp	2	0	262(0-3)	...	20(8)	15:1	0.21	0.64
30-21	Riebesel 47/51/Compair	0	0	377(0-2)	...	4(8)	63:1	0.36	0.55
31-21	Yamhill/Heines VII	0	0	303(0)	...	...	1:0		
31-27	Yamhill/Heines VII	2-3	8	72(2-3)	59(5)	177(8)	4:3:9	0.43	0.81
32-21	Alba/Heines VII	0	0	87(0)	...	...	1:0		
32-27	Alba/Heines VII	2	8	25(2-3)	6(5)	56(8)	6:1:9	2.86	0.24
33-21	Alba/Yamhill	0	0	205(0)	...	...	1:0		
33-20	Alba/Yamhill	8	2	55(2-3)	...	145(8)	1:3	0.54	0.46
34-11	Yamhill/Heines Peko	1	1	329(1-3)	37(5)	...	57:7	0.18	0.67
35-21	Yamhill/Nord Desprez	0	0	214(0-1)	...	...	1:0		
36-21	Yamhill/Vilmorin 23	0	0	336(0-1)	...	...	1:0		
37-20	Yamhill/Clement	2	0	53(0-2)	...	4(8)	15:1	0.00	1.00
38-20	Yamhill/Lee	2	2	262(2-3)	...	20(8)	15:1	0.21	0.64
39-20	Yamhill/Compair	2	2	330(2-3)	29(5)	40(8)	52:5:7	0.56	0.76
40-1	Yamhill/Fielder	2	2	200(2-3)	111(5)	145(8)	7:4:5	0.12	0.94
41-11	Stephens/Fielder	2	1	274(1-2)	...	87(8)	193:63	0.03	0.87
41-21	Stephens/Fielder	0	0	339(0-3)	...	80(8)	207:49	0.00	1.00
42-21	Stephens/Compair	0	0	471(0-3)	...	23(8)	247:9	1.94	0.16
43-14	Stephens/Vilmorin 23	2	2	221(2-3)	139(5)	26(8)	9:6:1	0.44	0.80
43-21	Stephens/Vilmorin 23	0	0	403(0-1)	...	...	1:0		
43-22	Stephens/Vilmorin 23	2	2	242(0-3)	85(5)	119(8)	9:3:4	0.84	0.66
44-21	Stephens/Nord Desprez	0	0	257(0) 58(1)	...	...	13:3	0.01	0.94
44-22	Stephens/Nord Desprez	2	2	211(0-3)	78(5)	102(8)	9:3:4	0.85	0.66
45-21	Stephens/Heines VII	0	0	138(0) 25(1)	...	...	13:3	1.03	0.31
46-20	Tyee/Lee	1	2	331(1-3)	...	14(8)	61:3	0.18	0.67
47-1	Compair/Fielder	1	2	516(1-3)	11(5)	61(8)	229:27[e]	0.01	0.94
48-11	Fielder/Heines Kolben	2	2	420(2-3)	...	...	1:0		
49-11	Fielder/Heines Peko	2	1	430(1-3)	31(5)	...	15:1	0.11	0.75
49-14	Fielder/Heines Peko	8	3-5	92(1-3)	20(5)	312(8)	1:3[e]	0.38	0.54
49-27	Fielder/Heines Peko	8	8	...	...	403(8)	0:1		
50-11	Heines Peko/Heines Kolben	1	1	379(1)	28(5)	...	15:1	0.18	0.67
50-14	Heines Peko/Heines Kolben	3-5	8	69(2-3)	17(5)	265(8)	1:3[e]	0.02	0.88
51-20	Tyee/Clement	1	0	171(0-1)	...	7(8)	15:1	1.26	0.26
52-20	Tyee/Hybrid 46	1	0	257(1-3)	...	15(8)	15:1	0.14	0.71
52-21	Tyee/Hybrid 46	1	0	487(0-2)	...	18(8)	15:1	5.77	0.02
53-20	Tyee/Compair	1	2	389(1-3)	...	6(8)	63:1	0.00	1.00
54-1	Tyee/Cappelle Desprez	2	2	483(2-3)	...	68(8)	55:9	1.21	0.27

races, including races that are avirulent on those cultivars, *Yr*Lem must be different from the gene in Riebesel 47/51 (*Yr*9) and other previously reported genes. Unlike Lemhi, Riebesel 47/51, which was derived from a rye-wheat cross, is resistant to all stripe rust races in North America (12,14). The gene in Riebesel 47/51 (*Yr*9) is located on a segment of the 1R chromosome from rye, which was translocated to chromosome 1B of wheat (22,25,27). Luthra et al (17) and Stubbs and Yang (26) reported that Riebesel 47/51 may have two or more genes. On the basis of the results with five North American races, we detected only one gene for resistance in Riebesel 47/51 (7). In the present study, the results with two additional races as well as race CDL-21 also indicated one gene for resistance in Riebesel 47/51. Clement was reported to have *Yr*9 from Riebesel 47/51 (20,22) and was postulated to possess *Yr*2 from Heines VII (25). Although we did not cross Clement with Riebesel 47/51, the presence of *Yr*9 in Clement is supported by its resistance to all North American races of *P. striiformis*. Our unpublished data from crosses of Clement with susceptible cultivars and the present data from resistant with resistant crosses also indicated that Clement has two genes for resistance. One of the genes is probably *Yr*9. Yamhill has a common gene for resistance with Heines VII and Heines Peko, which should be *Yr*2. According to these results, cultivar-race interactions, and the cross between Clement and Yamhill tested with race CDL-20, the second gene in Clement is not *Yr*2 and is probably different from other known genes.

Lupton and Macer (11,16) reported that Chinese 166 has *Yr*1 and that Heines VII has *Yr*2. Heines VII also has been reported to have a second gene (7,24). These results also indicated that Chinese 166 has *Yr*1 and Heines VII has *Yr*2, and a second gene in Heines VII (*Yr*HVII) is allelic or closely linked to genes in Cappelle Desprez, Nord Desprez, and Stephens. Cappelle Desprez and Nord Desprez have been reported or postulated to have *Yr*3a and *Yr*4a (5,16,22). According to Lupton and Macer's research (16), Heines VII does not have *Yr*3a or *Yr*4a.

Line and co-workers (8,14) suggested that Stephens should have a gene in common with Druchamp, and that Druchamp and Stephens should differ at least by one other resistance gene. The present results confirm their hypothesis. Stephens has two resistance genes and shares a gene (*Yr*3a or *Yr*4a) with Druchamp and Nord Desprez. The second gene in Stephens (*Yr*Ste) is different from the second gene in Druchamp (*Yr*Dru) and is the same as or closely linked to a gene in Heines VII. According to race reactions (14), the second gene in Stephens should not be *Yr*2. Stephens and Heines VII could have the same gene, because they share ancestors in their pedigree (28). One of the genes in Druchamp may be *Yr*3a or *Yr*4a. Druchamp has a resistance gene at the same locus as resistance genes in Cappelle Desprez and Nord Desprez. All three cultivars have Vilmorin 27 in their pedigree (28).

We previously reported that Yamhill has three genes (7), and Line and Qayoum (14) postulated that Yamhill should have *Yr*2 from one of its parents, Heines VII. The present results show that one of the genes in Yamhill is *Yr*2, whereas the other two genes are from Alba. Because Nord Desprez and Vilmorin 23 have been reported to have *Yr*3a and *Yr*4a (5) and no segregation was detected in the F_2 populations from crosses of Yamhill with Nord Desprez and Yamhill with Vilmorin 23 when tested with race CDL-21, one of the two genes of Yamhill from Alba should be *Yr*3a or *Yr*4a. The other gene is provisionally designated *Yr*Yam.

Cappelle Desprez, Nord Desprez, and Vilmorin 23 have been postulated to have the same resistance genes (5,22). We did not make crosses among these cultivars. Data from crosses of these cultivars with North American differential cultivars indicated that each of these cultivars has two resistance genes. However, segregation ratios for the three cultivars were different when crossed with the same cultivars, suggesting that the three cultivars may have different and common genes.

*Yr*6 has been reported to be in Heines Kolben and Heines Peko (9,18,19). On the basis of the reaction of Heines Kolben, Heines Peko, and Fielder to North American stripe rust races, Fielder was postulated to have *Yr*6 (7,14). The present results indicated that Fielder, Heines Kolben, and Heines Peko have a gene in common that should be *Yr*6. Each of the three cultivars also has a second gene for resistance. Data from the cross of Yamhill with Heines Peko and host-pathogen interactions (8,14) support the report by Johnson et al (9) that Heines Peko has *Yr*2. Race CDL-14 differentiates Heines Peko and Heines VII from Heines Kolben and Fielder. Heines Peko and Heines VII are resistant to race CDL-14, whereas Fielder and Heines Kolben are highly susceptible to race CDL-14. Therefore, Fielder and Heines Kolben do not have *Yr*2. Because the reaction of Fielder and Heines Kolben to races in North America and Europe are identical (8,14), it is possible that the second gene in Fielder (*Yr*Fie) and the second gene in Heines Kolben (*Yr*HK) are the same.

Macer (20) reported that Moro has *Yr*10, but did not make diallel crosses to check the relationship of *Yr*10 to genes in other resistant cultivars. We reported that Moro has two genes for resistance to race CDL-1 and one gene for resistance to race CDL-27 (7). The present data indicated that there are four genes from the cross of Moro with Daws (test 11-21) and Compair (test 13-20), and three genes were detected from the crosses of

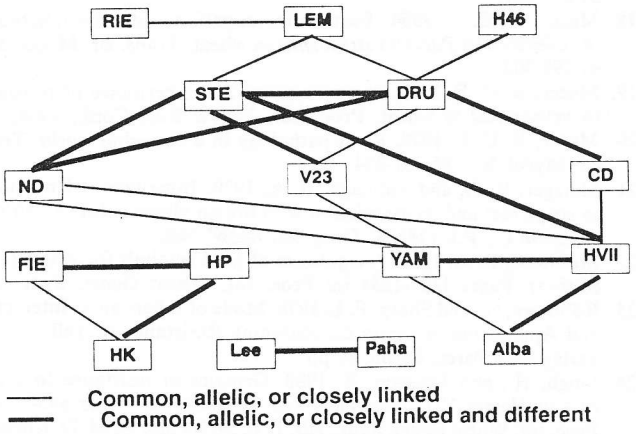

Fig. 1. Cultivars with a common gene, alleles, or closely linked genes. RIE = Riebesel 47/51; LEM = Lemhi; H46 = Hybrid 46; STE = Stephens; DRU = Druchamp; ND = Nord Desprez; V23 = Vilmorin 23; CD = Cappelle Desprez; FIE = Fielder; HP = Heines Peko; YAM = Yamhill; HVII = Heines VII; and HK = Heines Kolben.

TABLE 3. Genes for resistance to *Puccinia striiformis* in North American differential cultivars

Cultivar number	Cultivar name	Total genes	Gene designation and expression[a]
1	Lemhi	1	*Yr*Lem(D)
2	Chinese 166	1	*Yr*1(D)
3	Heines VII	2	*Yr*2(DR), *Yr*HVII(DR)
4	Moro	2	*Yr*10(D), *Yr*Mor(DR)
5	Paha	3	*Yr*Pal(D), *Yr*Pa2(R), *Yr*Pa3(DR)
6	Druchamp	2	*Yr*3a or *Yr*4a(DR), *Yr*Dru(DR)
7	Riebesel 47/51	1	*Yr*9(D)
8	Produra	2	*Yr*Pr1(R), *Yr*Pr2(R)
9	Yamhill	3	*Yr*2(DR), *Yr*3a or *Yr*4a(DR), *Yr*Yam(DR)
10	Stephens	2	*Yr*3a or *Yr*4a(DR), *Yr*Ste(DR)
11	Lee	2	*Yr*7(D), *Yr*Lee(R)
12	Fielder	2	*Yr*6(DR), *Yr*Fie(DR)
13	Tyee	1	*Yr*Tye(DR)

[a] *Yr* genes designated by a number (i.e., *Yr*1 and *Yr*3a) were previously named (24) and *Yr* genes designated by letters (i.e, *Yr*Lem and *Yr*Pal) are provisional names. Genes *Yr*HVII and *Yr*Ste and genes *Yr*Pa3 and *Yr*Lee may be the same genes. D = dominant, R = recessive, and DR = either dominant or recessive, depending on race and/or genetic background.

Moro with Druchamp, Yamhill, and Tyee (tests 12-14, 14-20, and 15-20). According to these results, previous studies (7), and unpublished data, Daws has two genes for resistance to race CDL-21; Compair has two genes (*Yr*8 and a second gene) for resistance to race CDL-20; Druchamp has one gene for resistance to race CDL-14; both Yamhill and Tyee have one gene for resistance to race CDL-20; and Moro has two genes for resistance to races CDL-14, CDL-20, and CDL-21. These data show that the two genes in Moro differ from the genes in Daws, Compair, Druchamp, Yamhill, and Tyee. Moro was developed from the cross of PI 178383 with Omar (28). PI 178383 has a dominant resistance gene (*Yr*10) located on chromosome 1B (1,10,21,22). The second gene in Moro (*Yr*Mor) could be from Omar (the other parent) or from a different plant of PI 178383.

We reported that Lee has two genes and Paha has three genes for resistance to North American races of *P. striiformis* (7). According to Macer (20), Lee has a single gene (*Yr*7). The present results also indicated that one of the two genes in Lee should be *Yr*7 and that the second gene (*Yr*Lee) is the same as or closely linked to a gene in Paha (*Yr*Pa2). Paha is a sister cultivar of Suwon 92-Omar (CI 013749) (3), and the two wheat lines have identical reactions to North American and European stripe rust races (8,14). Suwon 92 has been reported to have a single dominant gene for resistance (2). Therefore, the dominant gene in Paha (*Yr*Pa1) should be the gene from Suwon 92. The third gene in Paha (*Yr*Pa3) is different from other genes described.

As of 1987, Tyee was resistant to all North American races except CDL-27 and CDL-33 (14). We reported that Tyee has one gene for resistance (7). The present results show that the gene in Tyee (*Yr*Tye) is different from the other reported genes. Tyee has a common parent (Omar) with Paha and Moro but does not have their resistance genes.

On the basis of responses to North American races, Line and Qayoum (14) postulated that Produra, a durum wheat, may have a gene in common with Fielder. We detected two resistance genes in Produra (7). According to cultivar-race interactions, at least one of those genes is different from the other genes. Because of limited crosses, we could not compare genes in Produra with genes in other cultivars. Because Produra is a tetraploid wheat, the two genes in Produra (*Yr*Pr1 and *Yr*Pr2) must be located on the A or B set of chromosomes.

Expression of dominance, recessiveness, or variation in dominance of the genes is shown in Table 3. *Yr*1, *Yr*7, *Yr*9, *Yr*10, *Yr*Lem, and *Yr*Pa1 were dominant, and *Yr*Lee, *Yr*Pa2, *Yr*Pr1, and *Yr*Pr2 were recessive. The remaining genes were either dominant or recessive depending on the race in the test and/or the parent in the cross. Similar shifts in dominance have been previously reported and discussed (7,16,23).

*Yr*1 in Chinese 166, *Yr*9 in Riebesel 47/51, *Yr*Lem in Lemhi, and *Yr*Tye in Tyee were independent from other resistance genes (i.e., they had no effects or were not influenced by other genes). All other genes showed various epistatic interactions. In most interactions, alleles for resistance at one locus were epistatic to alleles for susceptibility at other loci. However, resistance was hypostatic in some crosses. In some crosses, interactions were different when crossed with different parents or when tested with different races.

There are 24 genes for resistance to North American races of *P. striiformis* in the 13 North American differential cultivars and several genes in the other cultivars used in the crosses that have not been previously identified. Among the 24 genes, at least 19 are uniquely different from one another, and at least nine have not been previously identified or named. The resistance genes were dominant, recessive, or shifted from being dominant to being recessive depending on the race used in the test and parent in the cross (genetic background). Among the genes, some were inherited independently, whereas others had various epistatic interactions with one another. This information on the genes in differential cultivars, their inheritance, and their relationships and interactions with one another should aid in monitoring races, identifying and differentiating new races of *P. striiformis*, and determining the genes for virulence or avirulence in the pathogen. The information should also be useful for developing stripe rust resistant cultivars and for managing the genes for resistance so that maximum benefits can be obtained from them.

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