Inheritance of Stripe Rust Resistance in Wheat Cultivars Used to Differentiate Races of *Puccinia striiformis* in North America

Xianming Chen and Roland F. Line

Research associate, Department of Plant Pathology, Washington State University, and plant pathologist, Agricultural Research Service, U.S. Department of Agriculture, respectively, Pullman, WA 99164-6430.

ABSTRACT

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Inheritance of stripe rust resistance in 13 wheat (*Triticum aestivum*) cultivars used to differentiate races of *Puccinia striiformis* in North America was determined using nine North American races of the pathogen. The differential cultivars, which were resistant to specific races, were crossed with cultivars susceptible to the specific races. We found that Lemhi, Chinese 166, Riebesel 47/51, and Tyee each have a single resistance gene; Heines VII, Moro, Druchamp, Produra, Stephens, Lee, and Fielder each have two resistance genes; and Paha and Yamhill each have three resistance genes. Of the 24 resistance genes, each differential cultivar has at least one gene that is different from the genes in the other differential cultivars. Some of the cultivars may also have common genes. The genes

in Lemhi, Chinese 166, Heines VII, Moro, and Riebesel 47/51, and one of the genes in Paha and in Lee were dominant. The two genes in Produra, one of the genes in Lee, and at least one of the genes in Paha were recessive. The single gene in Tyee and both genes in Druchamp, Fielder, and Stephens were dominant or recessive depending on the race, parent in the cross (genetic background), interaction of race and parent, and duration of infection. Various epistatic interactions were observed. This information should be useful in evaluating germ plasm for resistance, monitoring races of the pathogen, determining the relationships of races to one another, and developing new cultivars with more effective stripe rust resistance.

Additional keywords: specific resistance, virulence, yellow rust.

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 the western regions and is sometimes destructive in the south central United States (13). Growing resistant cultivars is the most economical method of controlling the disease. Biffen (6) first demonstrated that stripe rust resistance followed Mendelian laws. Since then, stripe rust resistance has been identified and incorporated into many commercial cultivars (1,9,15,19,21,23). Nineteen genes at 16 loci have been named as *Yr*1 to *Yr*16. Multiple alleles at the *Yr*3 locus have been designated *Yr*3a, *Yr*3b, and *Yr*3c, and at the *Yr*4 locus they have been designated *Yr*4a and *Yr*4b (15,19). Genes *Yr*11, *Yr*12, *Yr*13, *Yr*14, and *Yr*16 are race-specific genes that are expressed only when the plants are in the adult stages of plant growth. All other *Yr* genes are race-specific and expressed in seedlings as well as adult plants.

Seedlings of wheat cultivars with different race-specific resistance genes are used to differentiate races of *P. striiformis*. However, the number of genes and the inheritance of the genes in most of the stripe rust differential cultivars are not known. Line and Qayoum (13) have reported on the virulence, aggressiveness, evolution, and distribution of races of *P. striiformis* in North America. By 1987, 39 races of *P. striiformis*, based on 13 North American differential cultivars, were identified in North America. Each of the North American differential cultivars is resistant to at least one of the 39 races (11,13). Of the 13 differential cultivars, Chinese 166, Heines VII, Moro, Riebesel 47/51, and Lee have resistance genes *Yr*1, *Yr*2, *Yr*10, *Yr*9, and *Yr*7, respectively (15,17-19). Of these five differentials, only Chinese 166 and Heines VII have been studied in diallelic crosses. Therefore, the number of stripe rust resistance genes in Moro, Riebesel 47/51, and Lee is not known. The genetics of stripe rust resistance

has not been studied in differential cultivars Lemhi, Druchamp, Produra, Stephens, Yamhill, Fielder, and Tyee. De Vallavieille-Pope and Line (7) compared the virulences of North American and European races of *P. striiformis* on North American, world, and European differential wheat cultivars and postulated which resistance genes may be in some of the North American differential cultivars by using the pedigree of the cultivars and their host-pathogen interaction. The objective of this study was to determine the number and inheritance of genes for stripe rust resistance in the 13 North American differential cultivars.

MATERIALS AND METHODS

Crosses among the 13 North American differential cultivars (Table 1) were made in a greenhouse in the winter of 1985. Some of the differential cultivars were used as susceptible parents. Crosses of Moro with Daws, Tyee with Heines Kolben, and Paha, Yamhill, and Tyee with Michigan Amber were also made. Daws, Heines Kolben, and Michigan Amber were used as susceptible parents. Backcrosses to both parents were made for some crosses by using the F_1 plants as the female parents. Spring parents and F_1 plants from crosses of spring wheats with spring wheats and spring wheats with winter wheats were grown in the field. Crosses of winter wheats with 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 the 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 (5) were vernalized at 0–4 C for 20–30 days. The seedlings were then transplanted into plastic pots and grown in a greenhouse.

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 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, placed in a dew chamber at 10 C for 24 h, and then placed in a growth chamber at a diurnal temperature cycle that gradually changed from 4 C at 2 a.m. to 20 C at 2 p.m. The light period was daylight supplemented with metal halide lights, which extended 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 parental seeds, 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 nine North American races listed in Table 1 were selected according to their avirulence on the specific parents. In all crosses except cross 2-1 (Tyee × Chinese 166), which was inoculated with race CDL-20, the race used to evaluate the parents and progeny of a specific cross was avirulent on one of the parents and virulent on the other parent. The initial source of inoculum was urediospores that had been stored in liquid nitrogen. Each race was originally purified by single spore isolation or by transfer of single pustules for several generations. Either 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, we increased the inoculum of each race on plants in isolation booths and, when possible, maintained it on cultivars that were susceptible to that specific race but resistant to the other races. Races that were virulent on common cultivars were increased in separate facilities at different periods of time. For each test, we also inoculated a set of the 13 differential cultivars to confirm the purity of the race that was used.

Because infection types produced by some races on some cultivars can vary with the duration of time after inoculation (13), 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 (12). Using the concept of basic and expanded scales (12,14), we recorded infection types 4, 5, and 6 as infection type (IT) 5, and infection types 7, 8, and 9 were recorded as IT 8. Infection types 0, 1, 2, and 3 were classified as resistant reactions, IT 5 was classified as an intermediate reaction, and IT 8 was classified as a susceptible reaction. The intermediate class (IT 5) was analyzed both as a distinct class and in combination with the resistant and susceptible classes. Chi-square tests were used to determine the goodness of fit of the segregation ratios.

TABLE 1. Resistance and susceptibility of the North American differential cultivars to test races of *Puccinia striiformis* [a]

USDA accession number	Differential cultivar [b]	Differential number	North American CDL race [c]								
			1	11	14	17	20	21	22	27	29
CI011415	Lemhi	1	S	S	S	S	S	R	S	S	S
CI011765	Chinese 166	2	S	R	R	S	R	S	R	R	R
PI201195	Heines VII	3	R	R	R	S	R	R	S	S	S
CI013740	Moro	4	R	R	R	R	R	R	R	R	S
CI014485	Paha	5	R	R	R	R	R	R	R	R	S
CI013723	Druchamp	6	R	R	R	R	S	R	R	R	R
PI295999	Riebesel 47/51	7	R	R	R	R	R	R	R	R	R
CI017406	Produra	8	R	R	S	R	S	R	R	R	R
CI014563	Yamhill	9	R	R	S	R	S	R	R	R	R
CI017596	Stephens	10	R	R	R	S	R	R	R	R	R
CI012488	Lee	11	R	R	R	S	R	R	R	R	R
CI017268	Fielder	12	R	R	S	R	S	R	S	S	R
CI017773	Tyee	13	R	R	R	R	R	R	R	S	R

[a] S designates susceptibility of the cultivar and virulence of the race, and R designates resistance of the cultivar and avirulence of the race.

[b] Lemhi, Produra, Lee, and Fielder are spring wheats and all other cultivars are winter wheats.

[c] CDL = Cereal Disease Laboratory race designations.

RESULTS

Data from the F_2 generation of all crosses are presented in Table 2. For some crosses, corroborating F_1 and backcross data were collected but are not presented. First and last recordings were identical in all crosses except for 6-3, 10-2, and 11-2, which had some progeny that were resistant at the first and susceptible at the last recording date.

Crosses 1-1 and 3-1 segregated in ratios that indicated that Lemhi has a single dominant gene and Heines VII has two dominant genes for resistance to race CDL-21, respectively. Two dominant genes for resistance to race CDL-20 were identified in cross 2-1, one from Chinese 166 and one from Tyee. Crosses 4-1 and 4-2 indicated that Moro has two dominant genes for resistance to race CDL-1 and one dominant gene for resistance to race CDL-27. Segregation ratios of progeny from crosses 5-1 and 5-2 showed that Paha has three genes for resistance to race CDL-21 (one dominant, one recessive, and one partially recessive) and two genes for resistance to race CDL-17 (one dominant and one recessive).

The results of crosses 6-1 to 6-8, tested with seven different races, indicated that Druchamp has two resistance genes and that expression of dominance and epistatic interactions differed depending on genetic background of the susceptible parent, the specific race used to test the cross, and the duration of infection. In cross 6-1 tested with races CDL-1 and CDL-11, the two genes were recessive. In cross 6-4 tested with race CDL-21, the two genes were dominant and independent. In crosses 6-2 tested with race CDL-17, 6-5 tested with race CDL-22, 6-6 tested with race CDL-27, and 6-7 tested with race CDL-29, the two genes were dominant and complementary. When the progeny of cross 6-5 were tested with race CDL-27, the two genes were partially recessive and complementary. In cross 6-3 tested with race CDL-17, the two genes were dominant when notes were first recorded (15 days after inoculation), but recessive when the data were recorded 25 days after inoculation.

The data for crosses 7-1 to 7-4 indicated that Riebesel 47/51 has a single dominant gene for resistance to races CDL-1, CDL-11, CDL-14, CDL-17, and CDL-21. Cross 8-1 showed that Produra may have two recessive genes for resistance to race CDL-17. Data for cross 9-1 indicated that Yamhill has three dominant or partially dominant genes for resistance to race CDL-21.

Segregation ratios for crosses 10-1 tested with races CDL-1 and CDL-11, 10-2 tested with race CDL-14, 10-5 tested with race CDL-29, and 10-2 tested with race CDL-22 at 21 days after inoculation indicated that Stephens has two recessive genes for resistance to those races, but when data for cross 10-2 tested with race CDL-22 were recorded 17 days after inoculation, the ratio indicated that one of the genes is only partially recessive. The ratios for the reciprocal crosses of Stephens with Tyee (crosses 10-3 and 10-4) indicated that Stephens has one recessive gene for resistance to race CDL-27. In contrast, for reciprocal crosses with Paha (crosses 10-5 and 10-6) tested with race CDL-29, two recessive genes were found when Stephens was the female parent, and two dominant genes were found with complementary interaction when Paha was the female parent.

The ratios for crosses 11-1, 11-2, and 11-3 indicated that Lee has two genes for resistance. In crosses 11-1 and 11-2, when data were recorded 18 days after inoculation, one gene was dominant and one gene was recessive. However, when data for cross 11-2 were recorded earlier (13 days after inoculation), the two genes were both dominant. In cross 11-3, one gene was dominant and one gene was partially dominant for resistance to race CDL-21. One of the genes was expressed as IT 3, the other gene was expressed as IT 5, and the two genes together were expressed as IT 0-1. The homozygous, recessive genes at the two loci were expressed as IT 8.

In cross 12-1 tested with race CDL-1, two completely recessive genes with complementary interaction were found. When the same cross was tested with race CDL-17, the heterozygotes had an intermediate reaction (IT 5), indicating that two partially dominant genes with complementary interaction were involved.

Results of crosses 13-1 and 13-2 tested with race CDL-21 indicated that Tyee has a single recessive gene for resistance. In contrast, the results of crosses 13-3 tested with race CDL-14, 13-4 tested with race CDL-17, and 2-1 tested with race CDL-20 indicated that Tyee has a single dominant gene for resistance.

DISCUSSION

Because of the number of crosses and of tests required for the study, the results were based primarily on F_2 data. The F_2 data indicated the number of genes in each of the 13 differential cultivars used in North America and their mode of inheritance. In all cases in which they were made, F_1 and backcross data corroborated the findings of the F_2 generation. The study included 35 crosses involving the 13 differential cultivars and 42 tests with

specific races of *P. striiformis*, which add support to the interpretation. Data on the pedigree of the cultivars and cultivar-race interactions (7,13) provided further evidence for the conclusions.

We found at least 24 genes for resistance to stripe rust in the 13 differential cultivars. Lemhi, Chinese 166, Riebesel 47/51, and Tyee each have one gene; Heines VII, Moro, Druchamp, Produra, Stephens, Lee, and Fielder each have two genes; and Paha and Yamhill each have three genes. These cultivars could also have resistance genes that can not be identified with this group of North American races. As previously postulated (7,13), some cultivars could have a common gene, and some of the genes may be the same as previously reported *Yr* genes. However, of the 24 genes, many appear to be different from previously named genes. Crosses of these cultivars with cultivars that have known

TABLE 2. Reaction of F_2 plants from crosses of wheat stripe rust differentials and susceptible parents

Cross[b]	Race	Parents[c]	Number of F_2 plants[a]			Expected ratio[d]	χ^2	P[e]
			R	I	S			
1-1	CDL-21	Lemhi/Chinese 166	161 (1-2)	...	52 (8)	3:1	0.014	0.91
2-1	CDL-20	Tyee/Chinese 166[f]	588 (1-2)	...	37 (8)	15:1	0.067	0.80
3-1	CDL-21	Heines VII/Chinese 166	371 (0-1)	...	25 (8)	15:1	0.000	1.00
4-1	CDL-1	Daws/Moro	486 (1-3)	...	35 (8)	15:1	0.123	0.73
4-2	CDL-27	Tyee/Moro	423 (2)	...	130 (8)	3:1	0.579	0.45
5-1	CDL-21	Paha/Michigan Amber	270 (2-3)	85 (5)	55 (8)	43:12:9	1.090	0.59
5-2	CDL-17	Paha/Lee	330 (2-3)	...	78 (8)	13:3	0.016	0.90
6-1	CDL-1	Druchamp/Lemhi	242 (1-5)	...	346 (8)	7:9	1.504	0.22
6-1	CDL-11	Druchamp/Lemhi	204 (2)	...	256 (8)	7:9	0.045	0.83
6-2	CDL-17	Druchamp/Lee	248 (1)	...	200 (8)	9:7	0.111	0.74
6-3	CDL-17	Lee/Druchamp						
		first notes	236 (1)	...	187 (8)	9:7	0.020	0.89
		last notes	176 (1)	...	247 (8)	7:9	0.704	0.40
6-4	CDL-21	Druchamp/Chinese 166	536 (1-3)	...	37 (8)	15:1	0.014	0.91
6-5	CDL-22	Druchamp/Fielder	156 (0-5)	...	116 (8)	9:7	0.093	0.76
6-5	CDL-27	Druchamp/Fielder	25 (2-3)	104 (5)	274 (8)	1:4:11	0.140	0.93
6-6	CDL-27	Druchamp/Tyee	131 (0)	...	119 (8)	9:7	1.353	0.24
6-7	CDL-29	Druchamp/Paha	189 (0-2)	...	139 (8)	9:7	0.198	0.66
6-8	CDL-29	Paha/Druchamp	158 (0-2)	...	177 (8)	7:9	1.451	0.23
7-1	CDL-1	Riebesel 47/51/Lemhi	188 (0)	...	62 (8)	3:1	0.000	1.00
7-1	CDL-11	Riebesel 47/51/Lemhi	303 (0)	...	104 (8)	3:1	0.040	0.84
7-2	CDL-14	Riebesel 47/51/Fielder	119 (0-2)	...	40 (8)	3:1	0.000	1.00
7-3	CDL-17	Riebesel 47/51/Lee	280 (0-1)	...	101 (8)	3:1	0.386	0.53
7-4	CDL-17	Riebesel 47/51/Chinese 166	236 (0-2)	...	83 (8)	3:1	0.126	0.72
7-4	CDL-21	Riebesel 47/51/Chinese 166	310 (0-1)	...	103 (8)	3:1	0.000	1.00
8-1	CDL-17	Lemhi/Produra	122 (1-2)	...	155 (8)	7:9	0.000	1.00
9-1	CDL-21	Yamhill/Michigan Amber	391 (1-3)	57 (5)	59 (8)	50:7:7	0.336	0.85
10-1	CDL-1	Stephens/Lemhi	211 (2-3)	38 (5)	314 (8)	6:1:9	0.248	0.88
10-1	CDL-11	Stephens/Lemhi	138 (2-3)	63 (5)	270 (8)	5:2:9	0.959	0.63
10-2	CDL-14	Stephens/Fielder	170 (2-5)	...	190 (8)	7:9	1.625	0.20
10-2	CDL-22	Stephens/Fielder						
		first notes	119 (1-3)	83 (5)	252 (8)	4:3:9	0.364	0.83
		last notes	23 (2-3)	...	431 (8)	1:15	0.893	0.34
10-3	CDL-27	Stephens/Tyee	67 (2-3)	...	198 (8)	1:3	0.000	1.00
10-4	CDL-27	Tyee/Stephens	74 (2-3)	...	215 (8)	1:3	0.029	0.87
10-5	CDL-29	Paha/Stephens	172 (0-2)	...	200 (8)	7:9	0.836	0.36
10-6	CDL-29	Stephens/Paha	234 (0-2)	...	166 (8)	9:7	0.734	0.39
11-1	CDL-1	Lee/Lemhi	458 (2-3)	...	103 (8)	13:3	0.033	0.86
11-2	CDL-1	Lemhi/Lee						
		first notes	237 (2-3)	...	23 (8)	15:1	2.564	0.11
		last notes	217 (2-3)	...	43 (8)	13:3	0.696	0.40
11-3	CDL-21	Lee/Chinese 166	235 (0-1), 84 (3)	82 (5)	31 (8)	9:3:3:1	0.979	0.81
12-1	CDL-1	Fielder/Lemhi	27 (2-5)	...	355 (8)	1:15	0.308	0.58
12-1	CDL-17	Fielder/Lemhi	142 (1-3)	104 (5)	186 (8)	5:4:7	0.559	0.76
13-1	CDL-21	Tyee/Michigan Amber	132 (1-3)	...	383 (8)	1:3	0.078	0.78
13-2	CDL-21	Tyee/Chinese 166	126 (1-3)	...	383 (8)	1:3	0.006	0.94
13-3	CDL-14	Tyee/Heines Kolben	343 (1)	...	115 (8)	3:1	0.000	1.00
13-4	CDL-17	Tyee/Lee	292 (1)	...	96 (8)	3:1	0.003	0.95

[a] Number of F_2 seedlings with either resistant (R), intermediate (I), or susceptible (S) reactions to *Puccinia striiformis*. Numbers in parentheses represent infection types (12).
[b] The first number in each cross is the differential number of the resistant parent, and the second number represents the cross number.
[c] The female parent in the cross is always listed first.
[d] Ratio of resistant to susceptible or ratio of resistant to intermediate to susceptible plants.
[e] Probability of a greater value due to chance alone.
[f] Both parents were resistant to race CDL-20.

Yr genes should provide further information about their relationships. Even without those crosses, some interpretations and speculations can be made about the nature of the genes in each cultivar.

The single dominant gene in Chinese 166 is probably *Yr1*, and one of the two dominant genes in Heines VII is probably *Yr2*. *Yr1* and *Yr2* have been reported by Lupton and Macer (15). Our results confirm the report by Singh and Johnson (22) that Heines VII has a gene in addition to *Yr2*. The second gene in Heines VII has not been named. Yamhill, which was developed from a cross of Heines VII × Alba, has three resistance genes. Because all races that are virulent on Yamhill are also virulent on Heines VII and some races that are virulent on Heines VII are not virulent on Yamhill (13), one of the genes in Yamhill is probably *Yr2* from Heines VII (8,15). The other genes in Yamhill are probably from Alba.

Two genes were identified in Druchamp and Stephens, as suggested by Line and co-workers (7,13). Using cultivar-race interactions, Line and co-workers postulated that Druchamp and Stephens may have a common gene and that each of the cultivars must have at least one different gene. De Vallavieille-Pope and Line (7) postulated that Druchamp and Stephens may have *Yr3* or *Yr4* because of the relationships of host-race interactions for these cultivars to the host-race interactions for Cappelle Desprez and Nord Desprez, which have *Yr3* and *Yr4* (15,19,21). The genes in Druchamp and Stephens were either dominant or recessive depending on the race used in the test, the parent in the cross (genetic background), and the duration of infection.

Fielder had two recessive resistance genes. Line and associates (7,13) postulated that Fielder could have *Yr6*, because Fielder and Heines Kolben (which has *Yr6*) (8,10,18,19) have the same reactions to North American and European races. The other gene in Fielder has not been previously reported.

Lee has two genes for resistance to North American races. One of the genes in Lee is probably *Yr7*, which was reported by Macer (18). The second gene in Lee has not been previously reported or postulated.

We identified a single dominant gene in Riebesel 47/51, which is probably *Yr9* (18,19). Riebesel 47/51, derived from a rye × wheat cross (25), has a segment of a rye chromosome (25). It is resistant to all isolates of *P. striiformis* that have been collected in North America (13), but it is susceptible to some races in Europe (7,23). Using comparisons of the resistances of Riebesel 47/51 and Clement to rust collections from different regions of the world, Stubbs and Yang (24) postulated that Clement has *Yr9* and Riebesel 47/51 has *Yr9* and at least one additional gene for resistance. Luthra et al (16) reported that Riebesel 47/51 has four genes for resistance to Indian races. If Riebesel 47/51 has additional genes, they can not be detected with North American races.

We detected two dominant resistance genes in Moro by using race CDL-1 and one gene by using race CDL-27. According to host-race interaction (13), one of the two genes in Moro is probably *Yr10*, which was named by Macer (18). Because Moro was developed from the cross PI178383/2*Omar, it is thought to have a major gene from PI178383. Metzger and Silbaugh (20) reported that PI178383 has a dominant resistance gene (probably *Yr10*), which is on chromosome 1B. Allan and Purdy (2) and Sharp and co-workers (21) also identified one dominant gene in PI178383. The second gene that we identified in Moro may not be from PI178383, or it may not be a gene that can be identified with the races used by those workers. Also, PI178383 is not a pure line, thus, the parent plant of PI178383 used in their crosses may be different from the PI178383 parent used to develop Moro. Luthra et al (16) reported that Moro has at least two dominant genes and one recessive gene for resistance to Indian races. Using the North American races, we did not identify a recessive gene.

The segregation ratios indicated that Paha has three genes for resistance to race CDL-21 and two genes for resistance to the other races in the tests. The best evidence for three genes is the data from cross 5-1 inoculated with race CDL-21. The ratio suggests one dominant gene and two recessive genes. One of the recessive genes is for partial resistance and is epistatic. When the intermediate and the low infection types were combined and considered as resistant, the ratio indicated one dominant and two recessive genes with no epistasis. If populations with intermediate and high infection types are pooled, the F_2 data from cross 5-1 would fit a 11:5 ratio, indicating that there are two, not three, genes. However, it would be inappropriate to combine the intermediate infection type with either the low infection type or the high infection type, because IT 5 was clearly defined and evident. The dominant gene in Paha is probably the single dominant gene from Suwon 92 reported by Allan and associates (2,3) for Sel. 63301. The pedigree of Sel. 63301 (Suwon 92/3*Omar) is similar to the pedigree of Paha (Suwon 92/4*Omar) (2,4), and the two selections differentiate the same races (13).

Lemhi was first included in the North American set of differential cultivars, because it was susceptible to all known races of *P. striiformis* in the world (14). In 1978, however, a new race (CDL-21) collected from triticale (*Triticosecale* Witt.) in California was found to be avirulent on all of the North American differential cultivars except Chinese 166, indicating that Lemhi should have a gene or genes for stripe rust resistance (13). Our results show that Lemhi has a single dominant gene for resistance.

Until 1983, when race CDL-27 first appeared, Tyee was resistant to all North American races of *P. striiformis* (13). According to cultivar-race interactions, Tyee should have a new gene or a new combination of genes for resistance. These results show that Tyee has a single gene for resistance, which is either dominant or recessive depending on race and genetic background. This gene is different from the previously identified *Yr* genes and genes identified in the other differential cultivars.

According to its reaction to North American races, Produra should have at least one gene for resistance that is different from the resistance genes in the other differential cultivars. These data suggest that Produra has two recessive genes for resistance, but the results are relatively inconclusive. Because Produra is a tetraploid that is difficult to cross with common hexaploid wheats, only two F_1 seeds were obtained from a cross of Produra with Lemhi. Aneuploid progeny could be produced from the cross, which may affect segregation.

Resistance in the North American differential cultivars was dominant, recessive, or either dominant or recessive depending on the race in the test, genetic background, and duration of the infection. Various epistatic interactions were observed. The variability in gene expression was especially evident for Druchamp, Stephens, Fielder, and Tyee. The effects of race and genetic background on expression of resistance have also been previously reported for the rusts. Lupton and Macer (15) were the first to report changes of dominance that depended on races of *P. striiformis*. Röbbelen and Sharp (21) have discussed the "reversal of dominance". Hypotheses such as "dosage effect", "heterozygosity of races", and "linkage genes" were proposed. However, the mechanisms of the phenomenon are not clear, and our data do not provide a conclusive explanation.

This information on the number of genes for resistance in each of the North American differential cultivars and on their mode of inheritance should be useful for monitoring races of *P. striiformis*, identifying and differentiating new races of the pathogen, and breeding for stripe rust resistance.

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