Chromosomal Location of Genes for Stripe Rust Resistance in Spring Wheat Cultivars Compair, Fielder, Lee, and Lemhi and Interactions of Aneuploid Wheats with Races of *Puccinia striiformis*

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ABSTRACT

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The spring wheat (*Triticum aestivum*) cultivar Lemhi has one gene, and cultivars Lee, Compair, and Fielder have two genes each for resistance to stripe rust, caused by *Puccinia striiformis*. To determine the chromosomal locations of the genes, the cultivars were crossed with susceptible disomic Chinese Spring and a set of 21 Chinese Spring aneuploids. Monosomic F_1 plants were cytologically determined, grown in a greenhouse, and self pollinated to produce F_2 seed. F_2 seedlings and their parents were inoculated with selected North American races of *P. striiformis*. The results confirmed that *Yr6* in Fielder is on chromosome 7B and *Yr8* in Compair is on chromosome 2D; and they show that *YrLem* in Lemhi is on chromosome 1B, *YrLe1* in Lee is on chromosome 4D, *YrLe2* in Lee is on chromosome 6D, *YrCom* in Compair is on chromosome 5B, and *YrFie* in Fielder is on chromosome 6D. None of the Lee genes that we detected with North American races is *Yr7*. We propose official gene designations *Yr19* for *YrCom*, *Yr20* for *YrFie*, *Yr21* for *YrLem*, *Yr22* for *YrLe1*, and *Yr23* for *YrLe2*.

Additional keywords: cytogenetics, gene interaction, host–pathogen interaction, monosomic analysis, 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 (34). In North America, the disease is most destructive in the western United States and sometimes destructive in the south central United States. Growing resistant cultivars is the most economical method of controlling the disease (18).

The spring wheat cultivars Lemhi, Lee, and Fielder are used to differentiate North American races of *P. striiformis* (19); Compair is used to differentiate European races; and Lee is used to differentiate world races of *P. striiformis* (34). An understanding of the genetics of the differential cultivars improves their usefulness in identifying races and breeding wheats for resistance. Macer (22) designated *Yr7* as the resistance gene in Lee I; however, he pointed out that the designation was not proved by a complete set of diallel crosses. Later, McIntosh et al (25) reported that *Yr7* was located on chromosome 2B because of its linkage with *Sr9g*, which confers resistance to stem rust (*Puccinia graminis* f. sp. *tritici*). Compair was developed by Riley et al (27,28) by crossing the hexaploid wheat cultivar Chinese Spring with *Aegilops comosa*. They reported that Compair had a dominant gene for resistance to the tested European races and demonstrated that the gene was transferred to wheat chromosome 2D by genetically induced homoeologous recombination with *A. comosa* chromosome 2M. The gene was later designated by Macer (22) as *Yr8*.

In inheritance studies of resistance to stripe rust, Chen and Line (3–6,8) reported that Lemhi has one gene and Lee, Compair, and Fielder have two genes each for resistance to North American races of *P. striiformis*. Based on the results of diallel crosses and race reactions, they identified one of the Fielder genes as *Yr6*, which has been reported in Heines Kolben, Heines Peko, and other cultivars (13,22,29). Gene *Yr6* in Heines Kolben and Heines Koga II was subsequently reported to be located on chromosome 7B (11,17). Because Lee is used to differentiate both North American and European races and its resistance to European races has been attributed to *Yr7*, Chen and Line (5,6) suggested that one of the genes they detected in Lee should be *Yr7*. Similarly, Chen and Line (3,5,6,8,18) suggested that one of the genes in Compair should be *Yr8*. The Lemhi gene and the additional genes in Lee, Compair, and Fielder were shown to be different from other reported genes and were therefore provisionally designated

YrLem, YrLee, YrCom, and *YrFie* (5,6,8,18). Information on chromosomal location of these genes in the four cultivars would be useful for breeding improved cultivars, identifying stripe rust races, and understanding interactions between host and pathogen.

The objectives of this study were to determine the chromosomal locations of the provisionally designated genes and to clarify whether *Yr6, Yr7,* and *Yr8* were the genes detected in Fielder, Lee, and Compair, respectively. An additional objective was to determine interactions among resistance genes, stripe rust races, and host chromosomal conditions.

MATERIALS AND METHODS

Monosomic analysis was used to determine the chromosomal location of the resistance genes in Compair, Fielder, Lee, and Lemhi (Table 1). The 21 monotelosomic, monosomic, and nullisomic tetrasomic Chinese Spring lines were originally developed by E. R. Sears (30,31), and the seeds were provided by J. Dvorák at the University of California, Davis. Sufficient seed of monosomic 2A was not available, so nullisomic 2A-tetrasomic 2D (NT2A2D) was used instead. Plants confirmed cytologically to be monotelosomic or monosomic and the four stripe rust resistant cultivars were grown in a greenhouse under conditions described by Chen and Line (4,5). The four resistant cultivars were crossed with disomic Chinese Spring and the 21 aneuploids. In all crosses, Chinese Spring and the aneuploid lines were used as the female parent. Cytologically confirmed monosomic F_1 plants were grown in the greenhouse to obtain F_2 seed.

Mitotic chromosomes of all parental and F_1 plants of all crosses and F_2 plants of selected crosses were analyzed using standard Feulgen staining procedures. Monosomic F_1 plants were selected in all crosses except those with NT2A2D, for which plants with 41, 42, and 43 chromosomes were used.

Seedlings of parents and F_2 plants were inoculated with North American races of *P. striiformis* and grown in the greenhouse under the conditions and procedures described by Chen and Line (4,5). Selection of a specific race to inoculate a set of crosses was based on previous studies and the reactions of the resistant cultivars (Table 1). When uredia were fully developed (18–22 days after inoculation) on Chinese Spring , infection types (IT) were recorded according to the 0–9 scale described by Line and Qayoum (19). Infection types 0–3 were considered resistant, infection type 5 was intermediate, and infection type 8 was susceptible. Chi-square tests for goodness of fit were used to determine whether the data fit a theoretical ratio and whether the pooled data of monosomic crosses excluding the critical cross(es) fit the theoretical ratio. Chi-square tests for association (contingency chi-square) were used to test for homogeneity of each aneuploid cross and the pooled aneuploid crosses excluding the critical cross(es) with the disomic cross (16).

TABLE 1. Wheat cultivars and their respective genes for resistance and reactions to North American races of *Puccinia striiformis*

ID number[a]	Cultivar	*Yr* gene[b]	Reaction to CDL race[c]						
			1	17	20	21	29	35	45
CI014108	Chinese Spring		S	S	S	S	S	S	S
CI011415	Lemhi	*YrLem*	S	S	S	R	S	S	S
CI012488	Lee	*YrLe1, YrLe2*	R	S	R	R	R	R	R
PI325842	Compair	*Yr8, YrCom*	R	R	R	R	R	R	R
CI017268	Fielder	*Yr6, YrFie*	R	R	S	R	R	R	S

[a] PI = plant identification number (formerly plant introduction number), and CI = cereal investigation number.

[b] *Yr* genes designated by number (e.g., *Yr8*) were previously named (3,4,5,6,8,22,23). *Yr* genes designated by letters (e.g., *YrCom*) are genes with provisional designations (3,4,5,6,8). *YrLe1* was previously considered to be *Yr7* by Chen and Line (4,5).

[c] R = resistant (IT 0, IT 1, and IT 2) and S = susceptible (IT 8). Resistance in Lemhi to race CDL-21 was IT 1; in Lee resistance to races CDL-20, CDL-21, and CDL-29 was IT 2, to race CDL-35 was IT 0, and to race CDL-45 was IT 1; in Compair resistance to races CDL-1 and CDL-21 was IT 0 and to race CDL-45 was IT 1; and in Fielder resistance to races CDL-1, CDL-17, CDL-21, and CDL-35 was IT 2.

RESULTS

Resistant infection types of the parent cultivars ranged from IT 0 to IT 2, depending upon cultivar–race interaction (Table 1). Intermediate infection types (IT 5) were recorded for the F_2 population of some crosses. In crosses involving Lemhi and Compair, intermediate infection types were very rare (tests of F_2 plants from crosses involving Lemhi and Compair with race CDL-21) or absent (tests of F_2 plants from crosses involving Compair with races CDL-1 and CDL-45). In crosses involving Fielder and Lee, intermediate infection types were sometimes more frequent but too infrequent to be analyzed as a distinct class. Therefore, intermediate infection types were combined with the high infection types and treated as susceptible reactions. The numbers of observed plants that were resistant (IT 0–IT 3) and susceptible (IT 5–IT 8) and probabilities of chi-square tests for goodness of fit are presented in Tables 2–7. When an aneuploid cross or the pooled noncritical aneuploid crosses fit the same F_2 ratio as the disomic cross using the chi-square test for goodness of fit and they are not significantly different from the disomic cross using the chi-square test for association, the results of the two tests can be considered to agree. In general, the results of chi-square tests for association agree with the results of chi-square tests for goodness of fit. Therefore, the probabilities for the association tests are presented in the tables only when they are different.

Compair. When tested with race CDL-21 (Table 2), the F_2 plants from the cross of Compair with disomic Chinese Spring and 18 of the 21 monotelosomic and monosomic aneuploids segregated 15 resistant to one susceptible, indicating that Compair has two dominant genes for resistance to race CDL-21 (Table 2). The F_2 segregation from the crosses with 2A, 5B, and 2D were significantly different from a 15:1 ratio. The results of a chi-square test for association show that the F_2 segregation of crosses with 2A, 5B, and 2D was significantly different from that of the disomic cross, while each of the noncritical aneuploid crosses and the crosses pooled were not significantly different from the disomic cross. Because the 2A chromosomes in the 2A line were replaced by 2D chromosomes and one of the resistance genes appeared to be on 2D, the deviating segregation ratio for the 2A cross was probably caused by the extra 2D. Therefore, the two genes in Compair are on chromosomes 5B and 2D. When tested with race CDL-45, the F_2 plants of the disomic cross segregated in a 15:1 ratio. F_2 segregation of all aneuploid crosses except 2A, 5B, and 2D fit the ratio. These results indicate that the two dominant resistance genes in Compair are located on chromosomes 2D and 5B. When tested with race CDL-1, F_2 plants of all crosses except 2D segregated in a 3:1 ratio. Unlike the other aneuploid crosses, the cross with 2D was significantly different from the disomic cross when the numbers of resistant and susceptible F_2 plants of the two crosses were compared. The results show that the gene for resistance to race CDL-1 is located on chromosome 2D and that the resistance gene on chromosome 5B is not effective against race CDL-1.

Because there was an excessive number of susceptible plants observed for the cross with 2D when tested with race CDL-1 (Table 2), a further experiment was conducted to determine the association of rust reaction with chromosomal status. The chromosome numbers of 11 F_2 plants were checked before inoculation with race CDL-1. The reactions to race CDL-1 of disomic, monosomic, and nullisomic plants were as expected for a critical cross. The four $2n = 41$ and five $2n = 42$ plants were resistant, and the two $2n = 40$ plants were susceptible. Although the number of tested plants was too small to confirm the 80:60 segregation ratio in Table 2, the results showed that the gene is on chromosome 2D.

Fielder. The F_2 segregation ratio of 13 resistant to three susceptible for disomic Chinese Spring/Fielder (Table 3) indicates that Fielder has one dominant gene and one recessive gene for

TABLE 2. Observed numbers of resistant and susceptible F_2 plants from crosses of Compair with Chinese Spring and 21 monotelosomic or monosomic aneuploids inoculated with races CDL-21, CDL-45, and CDL-1 of *Puccinia striiformis* and probabilities of chi-square test for goodness of fit to theoretical ratios

	CDL-21		CDL-45		CDL-1	
Female parent[a]	Obs. R:S[b]	$P_{15R:1S}$[c]	Obs. R:S[b]	$P_{15R:1S}$[c]	Obs. R:S[b]	$P_{3R:1S}$[c]
Disomic	242:12	0.315	134:7	0.528	114:40	0.780
1A	243:18	0.666	106:8	0.735	81:35	0.198
2A	212:34	<0.001***	134:21	<0.001***	116:40	0.853
3A	233:13	0.532	143:3	0.036	112:38	0.925
4A	170:12	0.848	92:5	0.656	92:27	0.560
5A	188:14	0.689	117:6	0.530	120:28	0.088
6A	213:20	0.141	110:5	0.399	88:32	0.673
7A	201:15	0.673	110:6	0.632	190:36	0.355
1B	159:14	0.317	114:9	0.625	77:33	0.226
2B	168:17	0.100	125:8	0.911	97:33	0.919
3B	193:18	0.171	121:6	0.478	95:24	0.223
4B	177:16	0.242	143:8	0.629	114:37	0.888
5B	171:26	<0.001***	118:16	0.007**	110:25	0.082
6B	211:18	0.314	142:11	0.631	104:45	0.143
7B	216:20	0.158	127:11	0.404	99:37	0.552
1D	136:14	0.119	101:7	0.921	70:25	0.767
2D	96:38	<0.001***	138:1	0.007**	80:60	<0.001***
3D	207:14	0.958	162:9	0.594	136:38	0.336
4D	183:11	0.739	117:12	0.152	94:30	0.836
5D	137:6	0.310	106:10	0.292	57:22	0.559
6D	214:19	0.230	130:11	0.447	106:38	0.700
7D	144:15	0.097	168:7	0.219	126:47	0.510
Total-monosomic (exc. critical)	3,393:274[d]	0.002**	2,234:142	0.582	1,984:670	0.771

[a] 2A was nullisomic 2A tetrasomic 2D (2n = 42) Chinese Spring and the others were either monotelosomic (2n = 40 + t) or monosomic (2n = 41) Chinese Spring used in crossing. Except for 2A, F_2 plants of the aneuploid crosses were produced by selfing monosomic F_1 plants.
[b] R = resistant (infection types 0, 1, 2, and 3) and S = susceptible (infection type 8).
[c] Probabilities of chi-square test for the resistant:susceptible ratio.
[d] $P = 0.104$ when the ratio was compared with that of the disomic using chi-square test for association.

resistance to race CDL-21. The segregation of crosses of Fielder with 19 of the 21 aneuploids also fit a 13:3 ratio for resistant to susceptible, further supporting the hypothesis of one dominant and one recessive gene for resistance. Segregation of the F_2 plants from crosses of Fielder with 7B and 6D did not fit a 13:3 ratio, indicating that chromosomes 7B and 6D carry the genes for resistance to race CDL-21. When tested with race CDL-35, F_2 plants of the disomic cross and all aneuploid crosses except 7B and 6D also segregated in a 13:3 ratio. Segregation of the crosses with 7B and 6D differed significantly from the 13:3 ratio. The results indicate that chromosomes 7B and 6D carry the resistance genes. Compared to the test with race CDL-21, few susceptible plants were obtained when tested with race CDL-35. However, the results of the tests with both races indicate that chromosomes 7B and 6D carry the resistance genes. When tested with race CDL-1 (Table 4), all F_2 populations except those from crosses with 7B and 6D segregated in a three resistant to 13 susceptible ratio, indicating that a dominant gene and a recessive gene confer resistance and that the two genes are complementary. Since segregation of the crosses with 7B and 6D did not fit the 3:13 ratio, chromosomes 7B and 6D presumably carry the genes for resistance to race CDL-1. When the same crosses were tested with race CDL-17 (Table 4), the F_2 population of the crosses of Fielder with disomic Chinese Spring and with all aneuploids segregated in a 3:1 ratio, except the cross with 6D. Because the F_2 population had an excess of susceptible plants, further testing was conducted. Available seed was limited, but the chromosomal constitution and rust reaction of 12 F_2 plants were correlated. Four 2n = 42 plants were resistant, but seven 2n = 41 plants and one 2n = 40 plant were susceptible. Based on a chi-square test for association, the 4:8 segregation ratio was not significantly different from the 60:74 ratio in Table 4 ($P = 0.44$). These results indicate that Fielder has a dominant gene for resistance to race CDL-17, the gene is located on chromosome 6D, and the dominant gene is ineffective against race CDL-17 in the hemizygous condition.

Lee. The F_2 segregation of Lee crossed with Chinese Spring fit a ratio of 13 resistant to three susceptible in tests with race

TABLE 3. Observed numbers of resistant and susceptible F_2 plants from crosses of Fielder with Chinese Spring and 21 monotelosomic or monosomic aneuploids inoculated with races CDL-21 and CDL-35 of *Puccinia striiformis*, and probabilities of chi-square test for goodness of fit to theoretical ratios

	CDL-21		CDL-35	
Chromosome[a]	Obs. R:S[b]	$P_{13R:3S}$[c]	Obs. R:S[b]	$P_{13R:3S}$[c]
Disomic	156:40	0.552	75:15	0.613
1A	135:37	0.353	96:21	0.824
2A	134:26	0.418	102:15	0.100
3A	125:26	0.630	113:21	0.361
4A	111:36	0.075	107:26	0.813
5A	120:30	0.695	108:22	0.594
6A	137:32	0.951	90:20	0.879
7A	143:37	0.535	130:27	0.618
1B	126:29	0.990	125:28	0.887
2B	105:22	0.680	70:17	0.850
3B	128:26	0.553	66:14	0.775
4B	114:31	0.417	63:16	0.732
5B	162:39	0.813	88:20	0.951
6B	124:37	0.169	80:15	0.460
7B	112:77	<0.001***	95:3	<0.001***
1D	96:26	0.469	74:18	0.841
2D	110:26	0.913	60:15	0.782
3D	140:34	0.789	39:8	0.761
4D	112:26	0.978	139:31	0.863
5D	125:24	0.409	93:18	0.494
6D	66:67	<0.001***	142:8	<0.001***
7D	115:30	0.550	73:14	0.525
Total-monosomic (exc. critical)	2,362:574	0.267	1,716:366	0.171

[a] 2A was nullisomic 2A tetrasomic 2D (2n = 42) Chinese Spring and the others were either monotelosomic (2n = 40 + t) or monosomic (2n = 41) Chinese Spring used in crossing. Except for 2A, F_2 plants of the aneuploid crosses were produced by selfing monosomic F_1 plants.
[b] R = resistant (infection types 0, 1, 2, and 3) and S = susceptible (infection type 8).
[c] Probabilities of chi-square test for the resistant:susceptible ratio.

TABLE 4. Observed numbers of resistant and susceptible F_2 plants from crosses of Fielder with Chinese Spring and 21 monotelosomic or monosomic aneuploids inoculated with races CDL-1 and CDL-17 of *Puccinia striiformis*, and probabilities of chi-square test for goodness of fit to theoretical ratios

	CDL-1		CDL-17	
Chromosome[a]	Obs. R:S[b]	$P_{3R:13S}$[c]	Obs. R:S[b]	$P_{3R:1S}$[c]
Disomic	36:150	0.833	143:53	0.509
1A	39:170	0.973	126:46	0.597
2A	33:148	0.858	116:34	0.509
3A	33:171	0.346	107:35	0.923
4A	32:161	0.440	110:34	0.700
5A	31:134	0.990	117:37	0.780
6A	32:154	0.589	125:38	0.619
7A	31:159	0.390	132:42	0.793
1B	36:144	0.667	121:36	0.549
2B	31:132	0.930	96:31	0.878
3B	36:186	0.333	110:33	0.595
4B	36:121	0.180	113:33	0.504
5B	39:167	0.947	126:41	0.893
6B	35:210	0.073	120:39	0.891
7B	74:103	<0.001***	138:46	1.000
1D	28:129	0.769	92:32	0.836
2D	27:131	0.593	104:33	0.805
3D	31:156	0.447	128:44	0.860
4D	48:213	0.882	97:36	0.582
5D	51:214	0.836	114:34	0.569
6D	80:177	<0.001***	60:74	<0.001***
7D	50:214	0.937	104:40	0.441
Total-monosomic (exc. critical)	679:3,114	0.181	2,296:744	0.503

[a] 2A was nullisomic 2A tetrasomic 2D (2n = 42) Chinese Spring and the others were either monotelosomic (2n = 40 + t) or monosomic (2n = 41) Chinese Spring used in crossing. Except for 2A, F_2 plants of the aneuploid crosses were produced by selfing monosomic F_1 plants.
[b] R = resistant (infection types 0, 1, 2, and 3) and S = susceptible (infection type 8).
[c] Probabilities of chi-square test for the resistant:susceptible ratio.

CDL-21, indicating that Lee has one dominant and one recessive gene for resistance to this race (Table 5). The F_2 segregation from crosses of Lee with 19 aneuploids also fit the 13:3 ratio; the crosses of Lee with 4D and 6D, however, were significantly different from that ratio, indicating that chromosomes 4D and 6D carry the two resistance genes in Lee to race CDL-21. To confirm that chromosomes 4D and 6D carry the resistance genes, chromosomes of F_2 plants of crosses of Lee with 4D and 6D were counted and the plants were inoculated with race CDL-21. Among nine F_2 plants of the cross with 4D, two 2n = 42 plants and five 2n = 41 were resistant; one of the two 2n = 40 plants was resistant and the other was susceptible. Among eight F_2 plants of the cross with 6D, three 2n = 42 and four 2n = 41 plants were resistant and one 2n = 40 + t plant was susceptible. Even though the numbers of plants used in chromosome counting and rust inoculation were too small to confirm the segregation ratios in Table 5, the results indicate that chromosomes 4D and 6D carry the resistance genes. In both cases, none of the disomic and monosomic plants was susceptible. When tested with races CDL-45 and CDL-29 (Table 5), the F_2 generations of all crosses except for 4D and 6D segregated in a 15:1 ratio. The results indicate that Lee has two dominant genes for resistance to races CDL-45 and CDL-29 located on chromosomes 4D and 6D. When tested with race CDL-20 (Table 6), the F_2 population of the disomic cross segregated in a three resistant to one susceptible ratio, indicating one dominant gene for resistance to race CDL-20. The F_2 segregation of the aneuploid crosses, except the cross with 6D, fit a 3:1 ratio. The cross with 6D was also significantly different from the disomic cross when the numbers of resistant and susceptible classes were compared. The results show that the gene for resistance to race CDL-20 in Lee is located on chromosome 6D. When tested with race CDL-35 (Table 6), the F_2 populations of the disomic cross and 18 monosomic crosses segregated in a 3:1 ratio, indicating the presence in Lee of a dominant gene for resistance to race CDL-35. Crosses with 2A and 5D were not available. The cross with 4D had fewer susceptible F_2 plants than expected for a 3:1 ratio and was significantly different from

TABLE 5. Observed numbers of resistant and susceptible F_2 plants from crosses of Lee with Chinese Spring and 21 monotelosomic or monosomic aneuploids inoculated with races CDL-21, CDL-45, and CDL-29 of *Puccinia striiformis* and probabilities of chi-square test for goodness of fit to theoretical ratios

	CDL-21		CDL-45		CDL-29	
Chromosome[a]	Obs. R:S[b]	$P_{13R:3S}$[c]	Obs. R:S[b]	$P_{15R:1S}$[c]	Obs. R:S[b]	$P_{15R:1S}$[c]
Disomic	253:51	0.378	149:10	0.984	147:12	0.499
1A	216:49	0.914	159:9	0.633	123:7	0.684
2A	246:49	0.346	161:13	0.506	140:8	0.671
3A	221:46	0.524	140:8	0.671	139:8	0.686
4A	157:36	0.972	193:8	0.184	134:10	0.731
5A	162:39	0.813	124:8	0.928	139:7	0.468
6A	186:31	0.092	154:13	0.413	167:9	0.533
7A	153:29	0.330	166:13	0.576	154:10	0.936
1B	202:46	0.935	143:14	0.167	145:10	0.917
2B	266:60	0.873	142:11	0.631	141:7	0.445
3B	203:53	0.423	161:8	0.415	137:8	0.716
4B	166:35	0.627	156:11	0.857	157:10	0.889
5B	130:41	0.080	120:9	0.733	149:10	0.984
6B	182:57	0.043*	157:17	0.055	127:10	0.612
7B	211:39	0.202	151:16	0.075	116:9	0.661
1D	201:43	0.652	156:13	0.439	140:9	0.916
2D	178:49	0.274	144:9	0.851	123:7	0.684
3D	199:37	0.227	182:7	0.148	141:7	0.445
4D	185:83	<0.001***	165:24	<0.001***	183:4	0.020*
5D	220:43	0.319	170:12	0.848	136:10	0.765
6D	135:110	<0.001***	158:22	<0.001***	148:2	0.013*
7D	210:50	0.843	164:17	0.081	146:9	0.820
Total-monosomic (exc. critical)	3,709:832	0.460	2,943:216	0.172	2,654:165	0.384

[a] 2A was nullisomic 2A tetrasomic 2D (2n = 42) Chinese Spring and the others were either monotelosomic (2n = 40 + t) or monosomic (2n = 41) Chinese Spring used in crossing. Except for 2A, F_2 plants of the aneuploid crosses were produced by selfing monosomic F_1 plants.
[b] R = resistant (infection types 0, 1, 2, and 3) and S = susceptible (infection type 8).
[c] Probabilities of chi-square test for the resistant:susceptible ratio.

the disomic cross. The results indicate that the gene for resistance to race CDL-35 is located on chromosome 4D. Based on the results from the tests of Lee crosses with the five races, the genes on chromosomes 4D and 6D confer resistance to races CDL-21, CDL-45, and CDL-29, the gene on chromosome 4D confers resistance to race CDL-35, and the gene on chromosome 6D confers resistance to race CDL-20.

Lemhi. The F_2 population from the cross of Lemhi with disomic Chinese Spring fit a ratio of three resistant to one susceptible, indicating that one dominant gene in Lemhi confers resistance to race CDL-21 (Table 7). The F_2 segregation of all aneuploid crosses, except for 1B, fit a 3:1 ratio. An excess of F_2 plants in the cross with 1B were susceptible. Because of the excess of susceptible plants, three monosomic F_1 plants of the cross were inoculated with race CDL-21. All three were susceptible, suggesting that the dominant resistance gene is not effective when in the hemizygous condition. To further confirm these results, the chromosomes of 58 F_2 plants from the three monosomic F_1 plants were counted before testing with race CDL-21. Of the 58 F_2 plants, 20 (34.5%) were 2n = 42 and resistant, four (6.9%) were 2n = 40 and susceptible, 32 (55.2%) were 2n = 41 and susceptible, one plant with 43 chromosomes was susceptible, and one plant with 41 chromosomes was resistant. The 21:37 ratio was not significantly different from the 32:41 ratio in Table 7 ($P = 0.377$), based on chi-square test for association. The results show that chromosome 1B of Lemhi carries the resistance gene.

DISCUSSION

Among the 12 *Yr* genes for resistance to *P. striiformis* that have been previously located on wheat chromosomes (1,16,23, 24,32), few were located by using monosomic analysis (1,11,17,21). Using monosomic analysis, we have determined the locations of seven genes in four spring wheat cultivars. The location of five of the genes had not been previously determined. Except for Lemhi, which is resistant to only one race (CDL-21), three or more races were used to locate the resistance genes. Use of several races provided strong confirmation of the conclusions.

Compair is resistant to all races of *P. striiformis* in North America. Based on crosses of Compair with susceptible cultivars tested with North American races, X. M. Chen and R. F. Line (unpublished data) found that Compair had two resistance genes. Their results were confirmed in other studies on identification of genes for stripe rust resistance in North American differential cultivars (5,6,8). They used *Yr8* to designate one of the genes and *YrCom* to designate the other. These results further confirm that Compair has two resistance genes and show that one of the genes is on chromosome 2D and the other on chromosome 5B (Table 2). The gene on chromosome 2D should be *Yr8,* and the gene on chromosome 5B should be *YrCom.* Because *Aegilops speltoides* was used to disrupt the diploid pairing of wheat during the transfer of *Yr8* from *A. comosa* chromosome 2M to the wheat chromosomes 2D (27,28), it is possible that *YrCom* is derived from *A. speltoides.* If this is true, the transfer of *YrCom* could be similar to that of *Lr28,* a gene for leaf rust resistance. McIntosh et al (26) demonstrated that *Lr28* in some wheat lines derived from the cross of a wheat and *A. comosa* was from *A. speltoides.*

The results of this study confirmed previous reports that Fielder has two genes for resistance (4–6). Based on reactions to North American and European races, de Vallavieille-Pope and Line (10) postulated that Fielder has the gene *Yr6.* The results of diallel crosses among Fielder, Heines Kolben, and Heines Peko showed that Fielder has one gene in common with Heines Peko and at least one gene (probably two genes) in common with Heines Kolben (5); therefore, Chen and Line (5) postulated that Fielder has *Yr6* plus an additional gene provisionally named *YrFie.* Using European race 104E137, Labrum (17) found that Heines Kolben and Heines Peko had a common gene and that *Yr6* in Heines

TABLE 6. Observed numbers of resistant and susceptible F_2 plants from crosses of Lee with Chinese Spring and 21 monotelosomic or monosomic aneuploids inoculated with races CDL-20 and CDL-35 of *Puccinia striiformis,* and probabilities of chi-square test for goodness of fit to theoretical ratios

Chromosome[a]	CDL-20		CDL-35	
	Obs. R:S[b]	$P_{3R:1S}$[c]	Obs. R:S[b]	$P_{3R:1S}$[c]
Disomic	123:39	0.785	94:27	0.495
1A	125:46	0.566	118:31	0.237
2A	133:40	0.568	...[d]	...
3A	119:41	0.855	40:14	0.875
4A	142:43	0.581	113:29	0.208
5A	121:29	0.109	147:44	0.531
6A	141:36	0.152	41:21	0.107
7A	124:54	0.100	114:41	0.676
1B	126:33	0.216	116:37	0.816
2B	123:33	0.267	39:11	0.624
3B	144:35	0.092	53:16	0.728
4B	98:36	0.618	136:40	0.486
5B	102:34	1.000	142:52	0.562
6B	119:42	0.750	45:17	0.660
7B	101:40	0.356	50:18	0.779
1D	113:38	0.963	100:31	0.724
2D	102:43	0.195	37:11	0.739
3D	112:34	0.633	79:23	0.568
4D	128:49	0.410	109:14	<0.001***
5D	61:25	0.383	...	...
6D	91:60	<0.001***	108:28	0.235
7D	109:40	0.603	73:23	0.814
Total-monosomic (exc. critical)	2,343:771	0.756	1,551:487	0.250

[a]2A was nullisomic 2A tetrasomic 2D (2n = 42) Chinese Spring and the others were either monotelosomic (2n = 40 + t) or monosomic (2n = 41) Chinese Spring used in crossing. Except for 2A, F_2 plants of the aneuploid crosses were produced by selfing monosomic F_1 plants.
[b]R = resistant (infection types 0, 1, 2, and 3) and S = susceptible (infection type 8).
[c]Probabilities of chi-square test for the resistant:susceptible ratio.
[d]... = No data.

TABLE 7. Observed numbers of resistant and susceptible F_2 plants from crosses of Lemhi with disomic Chinese Spring and 21 monotelosomic or monosomic aneuploids inoculated with race CDL-21 of *Puccinia striiformis,* and probabilities of chi-square test for goodness of fit to the theoretical ratio

Chromosome[a]	Obs. (R:S)[b]	$P_{3R:1S}$[c]
Disomic	162:52	0.813
1A	88:27	0.706
2A	99:36	0.655
3A	122:46	0.476
4A	114:51	0.080
5A	110:47	0.153
6A	101:32	0.802
7A	86:30	0.830
1B	32:41	<0.001***
2B	103:41	0.478
3B	109:39	0.704
4B	121:40	0.964
5B	76:30	0.432
6B	120:43	0.684
7B	138:44	0.797
1D	124:39	0.752
2D	92:35	0.505
3D	113:38	0.963
4D	111:45	0.267
5D	95:29	0.678
6D	105:40	0.472
7D	126:42	1.000
Total-monosomic (exc. 1B)	2,200:717	0.600

[a]2A was nullisomic 2A tetrasomic 2D (2n = 42) Chinese Spring and the others were either monotelosomic (2n = 40 + t) or monosomic (2n = 41) Chinese Spring used in crossing. Except for 2A, F_2 plants of the aneuploid crosses were produced by selfing monosomic F_1 plants.
[b]R = resistant (infection types 0, 1, 2, and 3) and S = susceptible (infection type 8).
[c]Probabilities of chi-square test for the resistant:susceptible ratio.

Kolben was located on chromosome 7B. Using European races 33E32 and 32E128, El-Bedewy and Röbbelen (11) also reported that *Yr6* in Heines Kolben was on chromosome 7B. They found that resistance conferred by the gene to race 33E32 was dominant and to race 32E128 was recessive. Later, Heines Kolben was reported to have *Yr2* in addition to *Yr6* (12,13). However, Chen and Line (5) failed to show the presence of *Yr2* in Heines Kolben and Fielder when diallel crosses were tested with North American races. Using both races CDL-1 and CDL-21, we located one of the genes in Fielder (probably *Yr6*) on chromosome 7B and the other (*YrFie*) on chromosome 6D (Tables 3 and 4). *YrFie* on chromosome 6D was further confirmed in the test with race CDL-17 (Table 4). Our results agree with the previous reports that *Yr6* is on chromosome 7B. Thus, both *YrLee* and *YrFie* are located on chromosome 6D. However, the two genes must be different because *YrFie* is effective and *YrLee* is ineffective against race CDL-17. At this point, however, we do not know if they are allelic, linked, or genetically independent.

Macer (22) designated a resistance gene in Lee as *Yr7*. Chen and Line (4,5) reported that Lee has two genes for resistance to North American races of *P. striiformis,* one dominant and the other either dominant or recessive depending upon the test race, parent, and duration of infection. The dominant Lee gene was provisionally designated *Yr7,* and the second gene was designated *YrLee*. Our results confirm that Lee has two genes for resistance but do not confirm that one of the genes is *Yr7*. *Yr7* was reported to be on the long arm of chromosome 2B based on linkage with *Sr9g,* which confers resistance to stem rust caused by *P. g. tritici* (23,25). Thus, neither of the two genes that we detected in Lee is *Yr7*.

Lemhi was considered to be susceptible to all known races of *P. striiformis* (27). However, race CDL-21 from California, which was identified in 1978, is avirulent on Lemhi, indicating that Lemhi has at least one gene for resistance. From a study of inheritance of stripe rust resistance in North American differential cultivars, Chen and Line (4,5) reported that Lemhi had a dominant gene for resistance to race CDL-21, which was confirmed in this study (Table 7). The Lemhi gene was provisionally designated *YrLem* (5). Based on nonsegregation of the F_2 plants from the cross of Lemhi with Riebesel 47/51, Chen and Line (5) postulated that *YrLem* should be on chromosome 1B, because *Yr9* in Riebesel 47/51 is on a translocation chromosome involving 1RS of rye and 1BL of wheat (23,35). The results of the present study demonstrate that *YrLem* is on chromosome 1BL. Thus, *YrLem* may be at a homoeolocus of *Yr9* on the 1BL/1RS translocation.

Generally, when a dominant gene for resistance is involved, only the nullisomic plants in the critical cross are expected to be susceptible. Sears (30) reported that the transmission rate of n-1 gametes was 75% through the female and 4% through the male. Therefore, the proportion of nullisomic plants in the progeny of a selfed monosomic plant was about 3%. Later, Sears (31) reported 2.4% nullisomic plants from selfed monosomic 1B. According to this rate, we would expect that only two of 73 F_2 plants would be susceptible in the cross of Lemhi with 1B (Table 7). However, in this cross, we obtained a greater number of susceptible F_2 plants than expected. The correlations of the monosomic condition with susceptibility based on three monosomic F_1 plants and 58 F_2 plants support the hypothesis that *YrLem* is ineffective in the hemizygous state. Similar results were obtained from the cross of Fielder with 6D tested with race CDL-17. There have been several reports of genes that are ineffective in the hemizygous state. Singh and McIntosh (33) found that *Sr8b,* which confers resistance to *P. g. tritici,* was hemizygous-ineffective. Labrum (17) reported that *Yr2* in Heines Peko was not well-expressed in hemizygotes. El-Bedewy and Röbbelen (11) reported that *Yr6* in Heines Kolben and Heines Koga II was ineffective in hemizygotes. Because the genes that they studied were recessive, El-Bedewy and Röbbelen (11) attributed the ineffectiveness of the resistance gene in the hemizygous state to a dosage effect. Because *YrLem* and *YrFie* were dominant in this study, dosage effect could not be used to explain observations that these genes were not expressed in the hemizygous state but were expressed in the heterozygous state. The full expression of the dominant resistance may require both alleles. Thus, in the absence of an allele, plants are susceptible. High frequencies of nullisomic plants in the progeny of selfed monosomic plants have been reported in wheat (2,14) and in oats (15). Therefore, it is possible that high rates of nullisomic plants may contribute to the excessive number of susceptible plants in some crosses.

In some cases, the proportion of susceptible plants in the critical crosses varied in tests with different races. Although the mechanism of this phenomenon cannot be fully explained at this point, it might be because of interactions of the resistance genes in the host with the virulence genes in the different races, different F_1 plants (although they were all $2n = 41$), and variation in rate of nullisomic plants in the progeny of selfed monosomic F_1 plants. However, the detection of the same critical chromosomes using different races shows that those chromosomes carry the genes.

Chen and Line (4,5) reported that *Yr6* and *YrFie* in Fielder and *YrLee* in Lee were either dominant or recessive, depending upon the race used, and that gene interactions were also dependent upon the race. In this study of crosses of Lee with Chinese Spring, one dominant gene and one recessive gene were detected with race CDL-21, but two dominant genes were detected with races CDL-29 and CDL-45 (Table 5). In the Fielder crosses, one dominant gene and one recessive gene were detected with races CDL-21, CDL-35, and CDL-1. However, the two genes were not complementary when tested with races CDL-21 and CDL-35 (Table 3), but were complementary when tested with race CDL-1 (Table 4). Others (4–8,13,20,21) have also reported reversals of dominance and changes of gene interaction when different stripe rust races were used.

Among the seven resistance genes that were located on chromosomes in the four cultivars in this study, only *Yr8* was previously located in Compair. *Yr6* was also previously located in other cultivars. *YrLem* in Lemhi was previously postulated to be on chromosome 1B (5). Chen and Line (5) reported that *YrLem* was allelic or closely linked to *Yr3a* in Druchamp and Stephens. We have also reported that the *Yr3* locus is on chromosome 1B by using monosomic analysis (9). The results of this study confirmed that *Yr6* is in Fielder. The location of *Yr6* on chromosome 7B of Fielder is in agreement with previous reports of *Yr6* in Heines Kolben, Heines Peko, and Heines Koga II (11,17). Based on this study and the earlier studies by Chen and Line (3–6,8), we propose permanent names for the resistance genes in the four spring wheat cultivars. The previously used names and proposed names for the genes in the four cultivars and the chromosomal locations of the genes are listed in Table 8. The information on chromosomal location and race–cultivar interactions should contribute to a better understanding of the genetics of resistance to stripe rust and may be useful in tagging resistance genes and transferring the genes into commercial cultivars through chromosomal manipulation.

TABLE 8. Proposed designations for the resistance genes and associated chromosomes in Compair, Fielder, Lemhi, and Lee

Cultivar	Previous *Yr* name	Proposed *Yr* name	Chromosome
Compair	*Yr8*[a]	*Yr8*	2D
	YrCom	*Yr19*	5B
Fielder	*Yr6*[b]	*Yr6*	7B
	YrFie	*Yr20*	6D
Lemhi	*YrLem*	*Yr21*	1B
Lee	*YrLe1*[c]	*Yr22*	4D
	YrLe2	*Yr23*	6D

[a] *Yr8* was previously located on chromosome 2D (27,28). The results of this study confirmed *Yr8* to be on 2D.

[b] *Yr6* was previously reported to be on chromosome 7B (11,17). The results of this study confirmed *Yr6* to be on 7B using a different cultivar, Fielder.

[c] *Yr7* was reported to be on chromosome 2B (25). We detected two genes in Lee with North American races and provisionally used *Yr7* for one of the genes (*YrLe1*) (4,5). This study shows that none of the genes are on chromosome 2B. Therefore, we give new designations for the two genes in Lee.

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