Chromosomal Location of Genes for Resistance to *Puccinia striiformis* in Winter Wheat Cultivars Heines VII, Clement, Moro, Tyee, Tres, and Daws

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ABSTRACT

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The winter wheat (*Triticum aestivum*) cultivars Heines VII, Clement, Moro, Tyee, Tres, and Daws have been reported to have stripe rust resistant genes *Yr2* and *YrHVII*, *Yr9* and *YrCle*, *Yr10* and *YrMor*, *YrTye*, *YrTr1* and *YrTr2*, and *YrDa1* and *YrDa2*, respectively. To confirm the existence of the genes and determine their chromosomal location, the cultivars were crossed with the seedling susceptible cultivar Chinese Spring and a set of 21 Chinese Spring aneuploids. Monosomic F_1 plants were allowed to self-pollinate to produce F_2 seed. Seedlings of F_2 plants and their parents were inoculated with selected North American races of *Puccinia striiformis*. The results confirmed that *Yr2* is on chromosome 7B and *Yr9* and *Yr10* are on chromosome 1B, and showed the tentative location of the following genes: *YrHVII* on chromosome 4A, *YrCle* and *YrMor* on chromosome 4B, *YrTye* and *YrTr1* on chromosome 6D, *YrTr2* on chromosome 3A, *YrDa1* on chromosome 1A, and *YrDa2* on chromosome 5D.

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 in many regions of the world (25). Growing resistant cultivars is the most economical and environmentally safe method of controlling stripe rust (15,21). Information on the chromosomal location of resistance genes is useful in breeding for resistance and in identifying races of the pathogen.

The winter wheat (*Triticum aestivum* L.) cultivars Heines VII, Moro, Tyee, and Tres are used to differentiate races of *P. striiformis* in North America (16), Heines VII is used as a European differential cultivar, and Clement and Moro are used as world differential cultivars (25). Daws is a commercial cultivar grown in the United States Pacific Northwest with race-specific seedling resistance and non-race-specific, high-temperature, adult-plant (HTAP) resistance. Each of the cultivars has been reported to have one or two different genes for stripe rust resistance (3,4,5,6).

Lupton and Macer (17) reported that Heines VII has gene *Yr2* for resistance to European races of *P. striiformis*. Later, Singh and Johnson (23) determined that Heines VII has two genes for resistance. Macer (18) and McIntosh (19) reported *Yr9* in Clement and *Yr10* in Moro. Chen and Line (2,3,4,5,6) reported that Heines VII, Clement, and Moro have genes *YrHVII*, *YrCle*, and *YrMor* in addition to *Yr2*, *Yr9*, and *Yr10*, respectively. Also, Chen and Line (3,4,5) reported genes in Tyee (*YrTye*), Tres (*YrTr1* and *YrTr2*), and Daws (*YrDa1* and *YrDa2*).

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Of the 11 genes, *Yr2*, *Yr9*, and *Yr10* have been located on specific chromosomes. Labrum (14) located *Yr2* in Heines Peko on chromosome 7B using monosomic analysis. *Yr9* is present in wheat cultivars and lines that have the 1B/1R wheat-rye translocations (19,27). *Yr10* in PI 178383, one of the parents of Moro, has been reported to be on chromosome 1B based on its linkage to a gene for brown glume color (20). The objectives of this study were to confirm the chromosomal locations of *Yr2* in Heines VII, *Yr9* in Clement, and *Yr10* in Moro, and obtain information on the chromosomal locations of race-specific, seedling-resistance genes *YrHVII* in Heines VII, *YrCle* in Clement, *YrMor* in Moro, *YrTye* in Tyee, *YrTr1* and *YrTr2* in Tres, and *YrDa1* and *YrDa2* in Daws.

MATERIALS AND METHODS

Monosomic analysis was used to determine the chromosomal locations of the resistance genes in Heines VII, Clement, Moro, Tyee, Tres, and Daws (Table 1). The 21 aneuploid Chinese Spring lines (monotelosomic 1A, 3A, 4A, 5A, 6A, 7A, 1B, 3B, 5B, 6B, 7B, 1D, 3D, 4D, 5D, 6D, and 7D; monosomic 2B, 4B, and 2D; and nullisomic 2A-tetrasomic 2D for 2A) were originally developed by E. R. Sears (22), and the seed were provided by J. Dvořák, University of California, Davis. Nullisomic 2A-tetrasomic 2D (NT2A2D) was used because sufficient seed of monosomic 2A were not available. Monotelosomic or monosomic plants of the Chinese Spring lines that were confirmed cytologically, disomic plants of Chinese Spring, and vernalized plants of the six resistant cultivars were grown in a greenhouse under conditions described by Chen and Line (3,4). The six 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 for all crosses except with 2A. For crosses with 2A, F_1 plants with $2n = 41$, $2n = 42$, or $2n = 43$ were selfed to produce F_2 seed. Since Chinese Spring has gene *Vrn3* for response to vernalization on the long arm of chromosome 5D (19), monosomic F_1 plants from crosses of the resistant winter wheat cultivars with monotelosomic 5DL Chinese Spring were vernalized at 4°C for 30 to 60 days prior to being moved to the greenhouse.

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

All seedlings of parental and F_2 plants were grown in the greenhouse, inoculated at the two-leaf stage with selected North American races of *P. striiformis*, and grown in a growth chamber under the same controlled conditions as described by Chen and Line (3,4). Selection of the race to test a specific set of crosses was based on the results of previous studies (3,4,5,6) and on the reactions of the resistant cultivars (Table 1). Races that were avirulent on the resistant parent were used to test the progeny from crosses with that parent. When uredia were fully developed (18 to 22 days after inoculation) on Chinese Spring, infection types (IT) were recorded according to the 0 to 9 scale described by Line and Qayoum (16). We recorded ITs 0, 1, 2, 3, 5, and 8. ITs 0 to 3 were considered resistant, IT 5 was intermediate, and IT 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 ratios. 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 (13).

RESULTS

The reactions of parental cultivars to the races of *P. striiformis* are shown in Table 1. Resistant ITs ranged from 0 to 2 depending upon cultivar-race interaction. Intermediate ITs (IT 5) were observed in F_2 plants of some crosses. F_2 plants with IT 5 were not observed in crosses with Heines VII tested with race CDL-6, Moro tested with race CDL-20, and Tres tested with race CDL-35. Also, F_2 plants with IT 5 were very rare in all crosses involving Clement, Tyee, and Daws and in crosses with Heines VII tested with races CDL-21 and CDL-35 and Moro tested with races CDL-25 and CDL-45. Plants with IT 5 were slightly more frequent in F_2 populations of crosses involving Moro and Tres tested with race CDL-21 and Tres tested with race CDL-20, but were too infrequent to be analyzed as a distinct class. Therefore, intermediate IT (IT 5) was analyzed in combination with low ITs and in combination with high IT. Data usually had a better fit when the intermediate IT was combined with the high IT (IT 8) and were treated as susceptible reactions. The numbers of observed resistant (IT 0 to 3) and susceptible plants (IT 5 to 8) and probabilities of chi-square tests for goodness of fit are presented in Table 2. In general, the results of chi-square tests for association agreed with those of chi-square tests for goodness of fit. Only the probabilities for chi-square tests for association that do not agree with the chi-square tests for goodness of fit at the $P = 0.05$ level are included in the table.

Heines VII. When Heines VII crosses were tested with race CDL-21 (Table 2), F_2 progeny from the disomic cross and 19 of the aneuploid crosses segregated in a 15 resistance/1 susceptible ratio, indicating that Heines VII had two dominant genes for resistance to race CDL-21, which was consistent with previous reports that Heines VII has two genes for resistance. Although it did not fit the 15:1 ratio ($P = 0.04$), the cross with the aneuploid for 5B (228 resistant and seven susceptible plants) was not significantly different from the disomic cross ($P = 0.15$). Thus, the cross with the aneuploid for 5B was not a critical cross. Crosses with aneuploids for 4A and 7B did not fit the expected ratio. The 4A cross produced fewer susceptible plants than expected based on the 15:1 ratio, indicating that it carried a resistance gene. The F_2 population of the cross with the aneuploid for 7B was significantly different from that of the disomic cross, and produced more susceptible plants than expected based on the 15:1 ratio. Similar results were obtained when the same crosses were tested with race CDL-6, except that the cross with the aneuploid for 5B clearly fit the 15:1 ratio. Also, in the test with race CDL-6, the cross with the aneuploid for 4A produced fewer susceptible F_2 plants, further indicating that chromosome 4A carried a resistance gene. The cross with the aneuploid for 7B produced more susceptible F_2 plants than expected based on the 15:1 ratio. The segregation of the 7B cross fit a 13:3 ratio ($P = 0.18$) when tested with race CDL-21, but the probability of the F_2 segregation fitting a 13:3 ratio was low ($P = 0.01$) when tested with race CDL-6. The tests with races CDL-21 and CDL-6, therefore, indicated that chromosome 7B may have had a dominant gene that was ineffective when hemizygous.

When tested with race CDL-35, the F_2 populations of the disomic cross and 19 of the aneuploid crosses segregated in a 13 resistant/3 susceptible ratio, indicating one dominant gene and one recessive gene for resistance. The F_2 populations from crosses with aneuploids for 4A and 7B did not fit the 13:3 ratio and were significantly different from the disomic cross. The cross with the aneuploid for 4A again produced fewer susceptible plants than expected based on the 13:3 ratio, indicating a resistance gene on chromosome 4A. The chromosomal location of the second gene for resistance to race CDL-35 was inconclusive because the 7B cross produced more susceptible plants, but no crosses other than those with the aneuploids for 4A and 7B indicated genes for resistance.

Clement. When tested with race CDL-21 (Table 2), the F_2 populations from the disomic cross and 19 of the aneuploid crosses segregated in a 15 resistant/1 susceptible ratio, indicating

TABLE 1. Wheat cultivars, their respective genes for resistance, and infection types produced by North American races of *Puccinia striiformis*

Identification number[a]	Cultivar	*Yr* gene[b]	Infection type by North American CDL race[c]							
			1	6	20	21	25	29	35	45
CI 014108	Chinese Spring		8	8	8	8	8	8	8	8
CI 017773	Tyee	*YrTye*	1	1	0	2	2	2	2	8
PI 201195	Heines VII	*Yr2, YrHVII*	0	0	1	0	8	8	1	8
WA 007716	Clement	*Yr9, YrCle*	0	0	1	0	1	1	0	0
CI 013740	Moro	*Yr10, YrMor*	0	0	0	0	1	8	0	1
CI 017917	Tres	*YrTr1, YrTr2*	2	2	2	2	2	2	2	2
CI 017419	Daws	*YrDa1, YrDa2*	8	8	8	2	8	8	8	8

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

[b] *Yr* genes designated by number (e.g., *Yr2*) were previously named (2,3,4,5,6,17,18,19) and *Yr* genes designated by letters (e.g., *YrTye*) were provisionally named (2,3,4,5,6).

[c] Infection types 0, 1, and 2 were considered resistant and infection type 8 was considered susceptible (16).

that Clement had two dominant genes for resistance. The F_2 populations from crosses with aneuploids for 1B and 4B did not fit the ratio and were significantly different from the disomic cross. Fewer susceptible plants were observed than expected based on a 15:1 ratio for the 1B cross tested with all three races and for the 4B cross tested with race CDL-21. The results showed that one of the genes was on chromosome 1B and the other on chromosome 4B. Seed from crosses with aneuploids for 3A and 2D were not available for testing with race CDL-45 and seed from nine aneuploid crosses (1A, 3A, 6A, 7A, 3B, 7B, 2D, 3D,

TABLE 2. Expected ratios, observed numbers of resistant (R) and susceptible (S) F_2 plants from crosses of resistant cultivars with disomic, pooled noncritical monosomic, and critical monosomic Chinese Spring lines inoculated with North American races of *Puccinia striiformis*, and probabilities of chi-square test for goodness of fit to theoretical ratios

Resistant parent	Race	Expected R:S ratio	Cross	Observed ratio[a]		
				R	S	P^b
Heines VII	CDL-21	15:1	Disomic	250	15	0.692
			Pooled noncritical monosomic	3829	236	0.242
			4A	357	10	0.005**(0.062)
			7B	164	29	<0.001***
	CDL-6	15:1	Disomic	123	8	0.946
			Pooled noncritical monosomic	3024	192	0.512
			4A	151	3	0.027*(0.069)
			7B	190	25	0.001***(0.090)
	CDL-35	13:3	Disomic	139	28	0.511
			Pooled noncritical monosomic	3035	655	0.120
			4A	134	12	0.001***
			7B	166	68	<0.001***
Clement	CDL-21	15:1	Disomic	360	23	0.843
			Pooled noncritical monosomic	5183	358	0.517
			1B	362	7	<0.001***
			4B	231	5	0.009**
	CDL-45	15:1	Disomic	144	7	0.413
			Pooled noncritical monosomic	2387	163	0.767
			1B	154	0	0.001***
			4B	173	23	0.002**
	CDL-29	15:1	Disomic	171	15	0.307
			Pooled noncritical monosomic	1571	123	0.086
			1B	203	2	0.002**
			4B	181	37	<0.001***
Moro	CDL-21	13:3	Disomic	196	45	0.975
			Pooled noncritical monosomic	3278	605	<0.001***(0.202)
			1B	138	0	<0.001***
			4B	183	15	<0.001***
	CDL-20	15:1	Disomic	142	8	0.643
			Pooled noncritical monosomic	3303	214	0.686
			1B	200	2	0.002**
			4B	77	22	<0.001***
	CDL-45	3:1	Disomic	180	59	0.911
			Pooled noncritical monosomic	3077	960	0.073
			1B	140	3	<0.001***
	CDL-25	3:1	Disomic	114	29	0.192
			Pooled noncritical monosomic	2566	832	0.488
			1B	195	7	<0.001***
Tyee	CDL-21	1:3	Disomic	44	139	0.765
			Pooled noncritical monosomic	600	1992	0.029*(0.782)
			6D	5	52	0.004**
	CDL-6	3:1	Disomic	89	35	0.407
			Pooled noncritical monosomic	1754	628	0.124
			6D	90	60	<0.001***
	CDL-20	3:1	Disomic	90	31	0.875
			Pooled noncritical monosomic	1562	551	0.253
			6D	99	55	0.002**
Tres	CDL-21	9:7	Disomic	130	106	0.718
			Pooled noncritical monosomic	1711	1398	0.172
			3A	46	132	<0.001***
			6D	41	180	<0.001***
	CDL-35	15:1	Disomic	204	13	0.875
			Pooled noncritical monosomic	1735	95	0.061
			3A	156	4	0.050*(0.107)
			6D	179	4	0.023*(0.060)
Daws	CDL-21	3:13	Disomic	57	229	0.609
			Pooled noncritical monosomic	795	3714	0.054
			1A	23	253	<0.001***
			5D	183	55	<0.001***

[a] R = resistant (infection types 0, 1, 2, and 3) and S = susceptible (infection type 8 for crosses of Heines VII tested with race CDL-6, Moro tested with race CDL-20, and Tres tested with race CDL-35; and infection types 5 and 8 for crosses of Heines VII tested with races CDL-21 and CDL-35, Clement tested with races CDL-21, CDL-45, and CDL-29, Moro tested with races CDL-21, CDL-25, and CDL-45, Tyee tested with races CDL-21, CDL-6, and CDL-20, Tres tested with race CDL-21, and Daws tested with race CDL-21).

[b] Probabilities of chi-square test for goodness of fit to the expected resistant/susceptible ratios. Probabilities of the chi-square test for association are not shown when they agree with the test for goodness of fit at $P = 0.05$. When the test for association does not agree with the test for goodness of fit, the probability is shown in parentheses.

and 7D) were not available for testing with race CDL-29. More susceptible plants were observed when the 4B cross was tested with races CDL-29 and CDL-45, rather than the expected excess of resistant plants. The F_2 segregation of the 4B cross fit a 13:3 ratio when tested with race CDL-29 ($P = 0.50$) and did not fit the ratio well when tested with race CDL-45 ($P = 0.01$). These results indicated that the resistance gene on chromosome 4B, when in the hemizygous state, may have been effective for race CDL-21 but ineffective for races CDL-29 and CDL-45. This was partially supported by a test for correlation of rust reaction with chromosome number. When 12 F_2 plants were inoculated with race CDL-45 after their chromosomes were counted, four $2n = 42$ plants were resistant, one $2n = 40$ plant was susceptible, five $2n = 41$ plants were resistant, and two $2n = 41$ plants were susceptible. Although the number of plants tested were relatively small, the results agreed with the hypothesis that the gene on chromosome 4B was ineffective when in the hemizygous state. Since there were two genes involved, plants monosomic for 4B could be either resistant or susceptible, depending upon whether or not they carried the second gene.

Moro. When progeny from crosses with Moro were tested with race CDL-21 (Table 2), F_2 populations from the disomic cross and 17 of the aneuploid crosses segregated in a 13 resistant/3 susceptible ratio, indicating that Moro had a dominant gene and a recessive gene for resistance to race CDL-21. Segregation in the 5B cross (191 resistant and 30 susceptible plants) was different from the 13:3 ratio ($P = 0.05$), but it was not different from segregation in the disomic cross ($P = 0.14$), indicating that the 5B cross was probably not a critical cross. The segregation ratio (203 resistant and 28 susceptible plants) in the cross with the aneuploid for 2A (NT2A2D) differed from the 13:3 ratio ($P = 0.01$) and from segregation in the disomic cross ($P = 0.05$), but the significance levels were not as high as those for the crosses with 1B and 4B. Also, the 2A cross produced more susceptible plants than the 1B and 4B crosses. The F_2 populations from the 1B and 4B crosses produced fewer susceptible plants than expected, did not fit the 13:3 ratio, and were significantly different from the disomic cross. Those results indicated that one gene was on chromosome 1B and one on chromosome 4B.

When tested with race CDL-20, F_2 populations of the disomic cross and 19 of the aneuploid crosses segregated in a 15 resistant/1 susceptible ratio, indicating two dominant genes for resistance in Moro. The F_2 segregation of the 1B and 4B crosses did not fit a 15:1 ratio and were significantly different from segregation in the disomic cross. The segregation of the 4B cross fit a 13:3 ratio ($P = 0.38$), indicating that the gene on chromosome 4B may have been ineffective when hemizygous.

When tested with race CDL-45, the F_2 populations from all crosses, except the cross with the aneuploid for 1B, segregated in a 3:1 ratio, indicating a dominant gene for resistance to race CDL-45. The F_2 population from the 1B cross produced fewer susceptible plants, did not fit the ratio, and was significantly different from the disomic cross. Thus, the gene for resistance to race CDL-45 was on chromosome 1B. The cross with the aneuploid for 2A, when tested with race CDL-25, was not available, but the results from the other crosses agreed with the results with race CDL-45.

Tyee. When progeny from the Tyee crosses were tested with race CDL-21 (Table 2), the F_2 populations of all crosses, except the cross with the aneuploid for 6D, segregated in a 1 resistant/3 susceptible ratio. The F_2 population from the cross with 6D did not fit the 1:3 ratio and was significantly different from those of the disomic cross. Only five out of 57 F_2 plants in the cross with 6D were resistant. The results with race CDL-21 suggested that the recessive gene may have been located on chromosome 6D and ineffective in the hemizygous state.

When tested with races CDL-6 and CDL-20, the F_2 populations from all crosses, except the cross with the aneuploid for 6D, segregated in a 3 resistant/1 susceptible ratio. Again, the cross with 6D did not fit the 3:1 ratio and was significantly different from the disomic cross. Because the 6D cross produced excess susceptible plants with both races, chromosomes of 12 F_2 plants from the same F_2 population that were used in the tests with races CDL-6 and CDL-20 were counted and then inoculated with race CDL-20. The results showed that the F_2 plants from the same F_1 plant tested with race CDL-6 and race CDL-20 were from a selfed $2n = 41 + t$ plant rather than a monosomic plant. Of the 12 F_2 plants, two $2n = 42$, three $2n = 41$, one $2n = 40 + 2t$, and one of four $2n = 40 + t$ plants were resistant; one $2n = 40$, three of four $2n = 40 + t$, and one $39 + 2t$ plants were susceptible. The segregation of resistant and susceptible plants in confirming the correlation test did not differ from the ratio of the 2D cross tested with race CDL-20 ($P = 0.68$) or race CDL-6 ($P = 0.91$). None of $2n = 42$ and $2n = 41$ were susceptible, indicating that chromosome 6D carried the resistance gene. The resistant reactions of the $2n = 40 + 2t$ plant and the one $2n = 40 + t$ plant could have been because the telosome or one of the two telosomes was a long arm. The results indicated that the resistance gene may have been on the long arm of chromosome 6D.

Tres. When progeny from the Tres crosses were tested with race CDL-21 (Table 2), F_2 populations of the disomic cross and 17 of the aneuploid crosses segregated in a 9 resistant/7 susceptible ratio, indicating that Tres had two complementary dominant genes for resistance to race CDL-21. Crosses with 4A (77 resistant and 85 susceptible plants) and 6A (89 resistant and 93 susceptible plants) were different from the 9:7 ratio ($P = 0.03$ for the 4A cross and $P = 0.05$ for the 6A cross), but were not significantly different from the disomic cross ($P = 0.14$ for the 4A cross and $P = 0.21$ for the 6A cross). Crosses with 3A and 6D were different from the 9:7 ratio, different from the disomic cross at the $P = 0.01$ level, and produced fewer resistant plants than expected based on the 9:7 ratio. The segregation of the two crosses was not significantly different from a 19.5:80.5 ratio ($P = 0.03$ for the 3A cross and $P = 0.72$ for the 6D cross) based on the hypothesis that the two genes were complementary and may have been ineffective in the hemizygous state. These results indicated that the two genes may have been on chromosomes 3A and 6D.

When tested with race CDL-35, crosses with aneuploids for 3B, 6B, and 2D were not available. The F_2 populations of the disomic cross and 16 of the aneuploid crosses segregated in a 15 resistant/1 susceptible ratio, indicating that Tres had two dominant genes for resistance to race CDL-35. The 3A and 6D crosses produced fewer susceptible plants than expected based on the 15:1 ratio. The results with race CDL-35 further supported the conclusion that the resistance genes were on chromosomes 3A and 6D.

Daws. When progeny from Daws crosses were tested with race CDL-21 (Table 2), F_2 populations from the disomic cross and 19 of the aneuploid crosses segregated in a 3 resistant/13 susceptible ratio, indicating that there were two genes (one dominant and one recessive) for resistance with complementary interaction. Crosses with 1A and 5D did not fit the 3:13 ratio and were significantly different from the disomic cross. The results indicated that one of the genes was on chromosome 1A and the other on chromosome 5D. Resistant and susceptible F_2 plants from the 1A cross were not different from the expected rates of 6% (24% × 25%) and 94%, respectively ($P = 0.10$). The rates were based on the assumptions that a dominant gene on chromosome 1A and a recessive gene on chromosome 5D were complementary and that the frequency of disomic plants in the F_2 population of the 1A cross was 24%. Similarly, resistant and susceptible F_2 plants from the 5D cross were not different from the rates of 72.75% (75% × 97%) and 27.25%, respectively ($P = 0.15$). The rates were also based on the assumptions that a dominant gene on chromosome 1A and a recessive gene on chromosome 5D were complementary and that the total rate of disomic and monosomic plants in the F_2

population of the 5D cross was 97%. The results suggested that the dominant gene was on chromosome 1A and ineffective when hemizygous, and that the recessive gene was on chromosome 5D and effective when hemizygous.

DISCUSSION

The results of this study provided further evidence that there are 11 genes for stripe rust resistance in the six winter wheat cultivars (two genes in Heines VII, Clement, Moro, Tres, and Daws and one gene in Tyee) and confirmed by monosomic analyses that *Yr2* in Heines VII is on chromosome 7B, and *Yr9* in Clement and *Yr10* in Moro are on chromosome 1B. Chromosomal locations of these genes and *YrHVII*, *YrCle*, *YrMor*, *YrTye*, *YrTr1*, *YrTr2*, *YrDa1*, and *YrDa2*, which were previously reported by Chen and Line (2,3,4,5), are listed in Table 3.

These results agreed with the previous report that there are two genes in Heines VII (3,4,5,23). *Yr2* has been reported to be either dominant or recessive depending upon the race used in the test (3,4,14,17,23). The results of this study showed that *Yr2* was dominant when tested with races CDL-21 and CDL-6, but recessive when tested with race CDL-35. Using monosomic analysis, Labrum (14) reported that *Yr2* in Heines Peko is on chromosome 7B and ineffective at the hemizygous state. Our results with races CDL-21 and CDL-6 also indicated that *Yr2* in Heines VII was located on chromosome 7B and ineffective when hemizygous. If we assumed that *Yr2* was recessive and ineffective at the hemizygous state when tested with race CDL-35, the critical cross for *Yr2* should have been close to the 13:3 ratio that was typical for the disomic cross and noncritical monosomic crosses. However, because two genes were involved, it was difficult using the race CDL-35 data to explain the abnormal segregation in the 7B cross and prove conclusively that *Yr2*, which is effective against race CDL-35, was on chromosome 7B.

The chromosomal location of *YrHVII* in Heines VII had not been previously determined. These results showed that *YrHVII* was on chromosome 4A. *YrMin* in Minister, *YrND* in Nord Desprez (7), and an unnamed gene in Cometa Klein (24) have also been reported to be on chromosome 4A. *YrHVII* is different from *YrMin* and *YrND* based on reaction to North American races of *P. striiformis* (7,15,16). *YrHVII* is probably not the same as the unnamed gene in Cometa Klein, because Heines VII shares no common genotypes with Cometa Klein in its pedigree (28).

Clement has been reported to have *Yr9*, a gene from rye on the 1B/1R translocation (19). Clement is also postulated by Stubbs (25) and Johnson (10) to have *Yr2* that was reported in Heines VII (17), one of the parents of Clement (28). X. M. Chen and R. F. Line (*unpublished data*) crossed Clement with susceptible *Triticum spelta saharense*, tested the F_2 population with several North American races, and found that Clement had two genes for resistance. Based on diallel crosses with other cultivars, they reported that the additional gene in Clement was not *Yr2*, was different from other reported genes, and designated it *YrCle* (2,4,5,6). The results of this study also showed that Clement had two genes for resistance and that neither gene was *Yr2*. Using monosomic analysis, we confirmed that *Yr9* is on chromosome 1B. Tests with race CDL-21 clearly showed that *YrCle* was on chromosome 4B and effective when hemizygous. Tests with races CDL-45 and CDL-29 were less conclusive, but were in agreement with the tests with race CDL-21. The segregation ratios of the 4B cross inoculated with races CDL-45 and CDL-29 suggested that *YrCle* on chromosome 4B may have been ineffective when hemizygous. The results indicated that interactions between the resistance gene and virulence genes may have been different with the different races.

TABLE 3. Genes for resistance to *Puccinia striiformis* and associated chromosomes in six wheat cultivars

Cultivar	*Yr* gene[a]	Chromosome[b]
Heines VII	*Yr2*	7B
	YrHVII	4A
Clement	*Yr9*	1B
	YrCle	4B
Moro	*Yr10*	1B
	YrMor	4B
Tyee	*YrTye*	6D
Tres	*YrTr1*	6D
	YrTr2	3A
Daws	*YrDa1*	1A
	YrDa2	5D

[a] The chromosomal locations of *Yr2*, *Yr9*, and *Yr10* have been previously reported (13,16,17,18,19,20,26). The remaining *Yr* genes have been provisionally designated by letters (3,4,5,6,15).
[b] Confirmed and tentative chromosomal locations.

In 1970, when Metzger and Silbaugh (20) reported the location of the gene for stripe rust resistance in PI 178383, one of the cultivars used to develop Moro based on its association with a gene on chromosome 1B conferring brown glume color (20). They did not designate the gene. In 1975, Macer (18) first designated a gene in Moro as *Yr10* without complete diallel crosses. Later, *Yr10* was generally accepted to be in both Moro and PI 178383 and, therefore, on chromosome 1B (19). In a series of studies, Chen and Line (3,4,5) showed that Moro has two genes for resistance to North American races of *P. striiformis*. Because Moro also has the brown glume color, they postulated that one of the genes should be *Yr10*. The additional gene designated as *YrMor* is different from the other reported genes. The results of this study confirmed by monosomic analysis that Moro has two resistance genes, that *Yr10* is on chromosome 1B, and that *YrMor* is either dominant or recessive when Moro is crossed with various cultivars and tested with different races. The results with both races CDL-21 and CDL-20 indicated that *YrMor* may have been on chromosome 4B. There are at least three genes for stripe rust resistance (*YrMor*, *YrCle*, and *YrYam*) that are on chromosome 4B (8). *YrMor* and *YrCle* confer resistance to different races. Also, *YrMor* is different from *YrYam* based on race reaction (4,16). The linkage relationships among *YrMor*, *YrCle*, and *YrYam* has not been determined.

Chen and Line (3,4,5) showed that Tyee has one gene for resistance (*YrTye*), the gene is different from all other reported genes, and the resistance gene can be either dominant or recessive depending upon the parent in the cross and the race in the test. These results confirmed the reports on the effect of race on expression of dominance and suggested that *YrTye* may have been located on chromosome 6D.

Chen and Line (5) reported that Tres has two genes designated as *YrTr1* and *YrTr2*. These results showed that one gene may have been on chromosome 3A and the other may have been on chromosome 6D. The gene on chromosome 6D should have been *YrTr1* and the gene on chromosome 3A should have been *YrTr2*. Based on these and previous studies (9), *Yr23* (*YrLe2*) in Lee, *Yr20* (*YrFie*) in Fielder, *YrTye* in Tyee, and *YrTr1* in Tres are on chromosome 6D. The four genes are different based on diallel crosses (5).

Using crosses of Daws with susceptible cultivars, X. M. Chen and R. F. Line (*unpublished data*) detected two resistance genes. They confirmed the two genes using diallel crosses and designated them *YrDa1* and *YrDa2* (4,5). The results of this study agreed with those results and indicated that *YrDa1* may have been on chromosome 1A and *YrDa2* may have been on chromosome 5D. No other genes have been reported to be on chromosomes 1A or 5D. The origins of the two genes in Daws are not clear. Daws was developed from a three-way cross, CI 14484//CI 13645/PI 178383 (28). Daws and Moro have PI 178383 in their parentage, but they do not have common genes and Daws has white glumes. The Daws genes could have been from CI 14484, CI 13645, and/or Washington Selection 101, which was used to develop CI 14484.

An excess of susceptible plants was observed in the cross of Heines VII with monosomic 7B when tested with races CDL-21, CDL-6, and CDL-35; the cross of Clement with monosomic 4B when tested with races CDL-29 and CDL-45; and the cross of Moro with monosomic 4B when tested with race CDL-20. In general, the aberrant ratios were consistent when tested with different races, indicating that in those critical crosses the excess of susceptible plants was not by chance. For example, *Yr2* was previously shown to be on chromosome 7B (14). This study also showed that *Yr2* was on chromosome 7B, even though the cross with monosomic 7B produced an excess of susceptible plants. The segregation ratios indicated that the excess of susceptible plants could have been due to ineffectiveness of genes in the hemizygous state. We (9) previously reported excessive susceptible plants in critical crosses and showed by association analyses of rust reactions and chromosome numbers that some dominant genes are ineffective in the hemizygous condition. Bhowal (1) and Kerber and Dyck (11) reported high rates of nullisomic progeny from monosomic wheats. High rates of nullisomic plants from selfed monosomic oats have also been reported (12). High rates of nullisomic plants could have caused the excess of susceptible plants in some crosses in our study. Therefore, it was clearly evident that this phenomenon was not uncommon. Further cytological work is needed to confirm if these genes are hemizygous ineffective.

These results showed that some resistance genes were dominant when tested with one race and recessive when tested with another race. This phenomenon was consistent with results in previous studies of these genes (3,4,5), but still not clearly understood. More studies are needed to understand the complexity of these interactions between wheat cultivars and stripe rust races. Because the objective of this study was to find the chromosomes and because the abnormal ratios were observed in the tests with different races, ditelosomic analysis should be used to confirm the chromosomes that carry *YrHVII*, *YrCle*, *YrMor*, *YrTye*, *YrTr1*, *YrTr2*, *YrDa1*, and *YrDa2* and to determine the chromosomal arms of these genes.

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