Chromosomal Location of Genes for Resistance to *Puccinia striiformis* in Seven Wheat Cultivars with Resistance Genes at the *Yr3* and *Yr4* Loci

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ABSTRACT

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Wheat cvs. Hybrid 46, Minister, Vilmorin 23, Druchamp, Stephens, Nord Desprez, and Yamhill have genes for resistance to *Puccinia striiformis* f. sp. *tritici* at the *Yr3* or *Yr4* loci as well as at other loci. To determine the chromosomal locations of the genes, the cultivars were crossed with seedling-susceptible cv. Chinese Spring and a set of 21 Chinese Spring aneuploids. F_2 seed was obtained from self-pollinated monosomic F_1 plants. Seedlings of F_2 plants and their parents were inoculated with North American races of *P. striiformis* f. sp. *tritici*. The results show that *Yr3a* in Druchamp, Stephens, and Nord Desprez and *Yr3c* in Minister are located on chromosome 1B; *Yr4a* in Vilmorin 23 and Yamhill and *Yr4b* in Hybrid 46 are on chromosome 6B; *YrMin* in Minister and *YrND* in Nord Desprez are on chromosome 4A; *YrDru* in Druchamp is on chromosome 5B; *YrSte* in Stephens is on chromosome 2B; *YrH46* in Hybrid 46 and a previously undescribed gene (*YrDru2*) in Druchamp are on chromosome 6A; a gene in Stephens previously undescribed (*YrSte2*) is on chromosome 3B; *YrV23* in Vilmorin 23 is on chromosome 2B; *Yr2* in Yamhill is on chromosome 7B; and *YrYam* in Yamhill is on chromosome 4B.

Additional keywords: cytogenetics, gene interaction, monosomic analysis, stripe rust, *Triticum aestivum*.

Wheat (*Triticum aestivum* L.) cvs. Druchamp, Stephens, and Yamhill are used to differentiate races of *Puccinia striiformis* Westend. f. sp. *tritici* in North America (10,19). Cv. Vilmorin 23 is used as a world differential cultivar, and cvs. Hybrid 46 and Nord Desprez are used as European differential cultivars (28). The seven cultivars have been used as resistance donors in breeding for stripe rust resistance.

Lupton and Macer (20) reported that Minister has a dominant or recessive gene (*Yr3c*) and Hybrid 46 has two resistance genes, designated *Yr3b* and *Yr4b* based on their allelism to *Yr3a* and *Yr4a* in cv. Cappelle Desprez. Later, Macer (21) and Bayles and Thomas (1) suggested that *Yr3a* and *Yr4a* are in Nord Desprez and Vilmorin 23. In a study of diallel crosses tested with North American races, Chen and Line (7) reported that Minister, Druchamp, Stephens, Nord Desprez, Hybrid 46, Vilmorin 23, and Yamhill have *Yr3c*, *Yr3a*, *Yr3a*, *Yr3a*, *Yr4b*, *Yr4a*, and *Yr2* and *Yr4a*, respectively, and that the seven cultivars also have *YrMin*, *YrDru*, *YrSte*, *YrND*, *YrH46*, *YrV23*, and *YrYam*, respectively. *Yr2* was reported to be on chromosome 7B (9,17). None of the other genes has been mapped to individual chromosomes.

The objective of this study was to determine the chromosomal location of the genes in the seven cultivars by monosomic analysis. Information about chromosomal location should be useful in breeding for resistance and understanding host-pathogen interactions.

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MATERIALS AND METHODS

Monosomic analysis was used to determine the chromosomal location of resistance genes in cvs. Minister, Druchamp, Stephens, Nord Desprez, Hybrid 46, Vilmorin 23, and Yamhill (Table 1). The 21 aneuploid cv. 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 Sears (23,24), and the seeds were provided by J. Dvořák at the University of California, Davis. Nullisomic 2A-tetrasomic 2D (NT2A2D) was used because sufficient seed of monosomic 2A was 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 seven resistant cultivars were grown in a greenhouse under conditions described previously by Chen and Line (4,5). The seven 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 for 2A. For crosses with 2A, F_1 plants with $2n = 41$, 42, or 43 were selfed to produce F_2 seed. Because Chinese Spring has gene *Vrn3* for response to vernalization on the long arm of chromosome 5D (22), 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 plants, the F_1 plants from each cross, and F_2 plants from selected crosses were made by standard Feulgen staining procedures. Monosomic F_1 plants were selected in all crosses, except crosses with NT2A2D; plants with 41, 42, and 43 chromosomes were used for crosses with NT2A2D.

Seedlings of the parents and F_2 plants were grown in the greenhouse, inoculated at the two-leaf stage with selected North

American races of *P. striiformis* f. sp. *tritici*, and grown in a growth chamber under the controlled conditions described previously by Chen and Line (4,5). Selection of a race to test each specific set of crosses was based on the results of previous studies (4–7) and on the infection types (IT) 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 on Chinese Spring (18 to 22 days after inoculation), IT were recorded according to the 0 to 9 scale described by Line and Qayoum (19). IT 0, 1, 2, 3, 5, and 8 were recorded, with IT 0 to 3 considered resistant, IT 5 considered intermediate, and IT 8 considered 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). An aneuploid cross was considered critical if F_2 segregation did not fit the theoretical ratio and was significantly different from the disomic cross. Some critical crosses were confirmed by association of rust reaction with the chromosome number of the F_2 plants.

RESULTS

The seedling reactions of parental cultivars to races of *P. striiformis* f. sp. *tritici* are shown in Table 1. Except for cv. Vilmorin 23, resistant IT ranged from 0 to 2 depending on cultivar-race interaction. An intermediate IT (IT 5) was produced on Vilmorin 23 by races CDL-1, CDL-17, and CDL-29. IT 5 was not observed in any other parents and progeny from crosses of Stephens tested with races CDL-1 and CDL-29 and crosses of Nord Desprez tested with race CDL-21. IT 5 was rare in all F_2 progeny from crosses involving Minister, Druchamp, Vilmorin 23, and Yamhill; in crosses of Stephens tested with races CDL-6 and CDL-45; and in crosses of Nord Desprez tested with races CDL-17 and CDL-45. Plants with IT 5 were slightly more frequent in F_2 populations of crosses involving Hybrid 46 and crosses of Stephens tested with race CDL-21, but they were not frequent enough to be analyzed as a distinct class. Because of these results, plants with IT 5 were analyzed both in combination with low IT plants and in combination with high IT plants. The data usually had a better fit when plants with IT 5 were combined with plants showing IT 8 and tested as a susceptible group.

Table 2 shows expected ratios, observed numbers of resistant (IT 0 to 3) and susceptible (IT 5 to 8) F_2 plants from crosses of resistant cultivars with disomic, pooled noncritical monosomic, and critical monosomic Chinese Spring tested with various races, and probabilities of chi-square tests for goodness-of-fit to theoretical ratios. Pooled noncritical monosomic crosses consisted of individual monosomic crosses that fit the disomic ratio. 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 did not agree with the chi-square tests for goodness-of-fit at the $P = 0.05$ level were included in Table 2.

Druchamp. The 15:1 ratio from the disomic cross and 19 of the 21 aneuploid crosses showed two dominant genes for resistance to race CDL-21 in Druchamp. Crosses with 1B and 5B produced fewer susceptible plants than expected, did not fit the 15:1 ratio, and were significantly different from the disomic cross. When tested with race CDL-1, the 9:7 ratio from the disomic cross and 19 of the 21 aneuploid crosses indicated there were two dominant complementary genes for resistance. In the crosses with 6A and 1B, fewer resistant plants were observed in the F_2 progeny than was expected based on the 9:7 ratio, and the crosses were significantly different from the disomic cross by chi-square tests. In tests with race CDL-45, aneuploid crosses 5A, 6B, 7B, 2D, 3D, and 5D were not available. The disomic cross and all tested aneuploid crosses, except crosses with 6A and 5B, produced a 13:3 ratio, indicating a dominant and a recessive gene for resistance. The 6A and 5B crosses did not fit the 13:3 ratio and were significantly different from the disomic cross.

When tested with race CDL-35, the 3:1 ratio from the disomic cross and 20 aneuploid crosses indicated a dominant gene for resistance. The cross with 6A did not fit the 3:1 ratio and was significantly different from the disomic cross, but there was an excess of susceptible plants. Because of this excess, 12 F_2 plants from the cross with 6A were inoculated with race CDL-35 after their chromosomes were counted. Six plants were $2n = 42$ and resistant and six plants were $2n = 41$ and susceptible. The results indicate that chromosome 6A carries a dominant gene for resistance to race CDL-35 and that the resistance gene is ineffective in the hemizygous state. Six of the aneuploid crosses were not available for tests with race CDL-29. When tested with race CDL-29, the disomic cross and all tested aneuploid crosses, except the cross with 1B, fit a 3:1 ratio. The cross with 1B had fewer susceptible F_2 plants than expected based on the 3:1 ratio. Based on these crosses, Druchamp has a gene on chromosome 1B that provides resistance to races CDL-21, CDL-1, and CDL-29; a gene on chromosome 5B that provides resistance to races CDL-21 and CDL-45; and a gene on chromosome 6A that provides resistance to races CDL-1, CDL-35, and CDL-45. The genes on chromosomes 1B and 5B are dominant, and the gene on chromosome 6A may be either dominant or recessive.

Stephens. When Stephens crosses were tested with race CDL-21, the 13:3 ratio from the disomic cross and 19 aneuploid crosses indicated a dominant and a recessive gene for resistance. Crosses with 1B and 2B did not fit the ratio and were significantly different from the disomic cross. When tested with race CDL-1, none of the crosses fit the genetic ratios for one or two genes. The disomic cross and 19 aneuploid crosses segregated in 14 resistant:2 susceptible. Therefore, the 14:2 ratio was used to make comparisons among the crosses. Crosses with 1B and 3B did not fit the ratio and

TABLE 1. Wheat cultivars, their respective genes for resistance, and infection types (IT) produced on their seedlings by North American races of *Puccinia striiformis* f. sp. *tritici*

Identification number[a]	Cultivar	*Yr* gene[b]	IT produced by CDL race[c]						
			1	6	17	21	29	35	45
CI014108	Chinese Spring		8	8	8	8	8	8	8
CI013723	Druchamp	*Yr3a, YrDru*	2	8	1	0	2	0	0
CI017596	Stephens	*Yr3a, YrSte*	2	2	2	0	2	8	2
PI167419	Nord Desprez	*Yr3a, YrND*	1	2	1	0	2	8	1
PI201196	Minister	*Yr3c, YrMin*	0	1	1	0	2	0	1
PI125093	Vilmorin 23	*Yr4a, YrV23*	5	8	5	0	5	8	8
CI014563	Yamhill	*Yr2, Yr4a, YrYam*	0	0	8	0	2	0	2
PI164755	Hybrid 46	*Yr4b, YrH46*	2	1	1	0	2	1	2

[a] CI = cereal investigation number, and PI = plant identification number (formerly plant introduction number).
[b] The *Yr* genes were named in publications 5–8, 13, 18, 20–22.
[c] IT 0, 1, and 2 were considered resistant; IT 8 was considered susceptible; and IT 5 was considered intermediate.

were significantly different from the disomic cross. When tested with race CDL-29, a 1:3 ratio from the disomic cross and 20 aneuploid crosses indicated a recessive gene for resistance. The cross with 1B was critical. When tested with race CDL-6, a dominant gene (3:1 ratio) for resistance was detected from the disomic cross and 19 aneuploid crosses. Segregation of the cross with 3B was different from the 3:1 ratio ($P = 0.042$) but was not significantly different from the disomic cross ($P = 0.129$). Segregation of the cross with 2B was different from the 3:1 ratio and significantly different from the disomic cross. Thus, the cross with 2B was the critical cross. When tested with race CDL-45, the disomic cross and 20 aneuploid crosses produced a 3:1 ratio, indicating a dominant gene for resistance. Only the cross with 3B produced fewer susceptible plants than expected based on the 3:1 ratio and, thus, was critical. These results indicate that Stephens has a gene on chromosome 1B for resistance to races CDL-21, CDL-1, and CDL-29; a gene on chromosome 2B for resistance to races CDL-21 and CDL-6; and a gene on chromosome 3B for resistance to races CDL-1 and CDL-45. The gene on chromosome 1B is apparently recessive, and the genes on 2B and 3B are apparently dominant.

Nord Desprez. When Nord Desprez crosses were tested with races CDL-21 and CDL-45, the disomic cross and 19 of the 21 aneuploid crosses segregated in a 15:1 ratio. Crosses with 4A and 1B did not fit a 15:1 ratio and were significantly different from the disomic cross. The cross with 4A had fewer susceptible plants than expected, and the cross with 1B had more susceptible plants than expected based on the 15:1 ratio. Crosses with 4A and 1B were significantly different from the disomic cross. The results indicate that the two dominant genes are on chromosomes 4A and 1B.

Minister. When tested with race CDL-21, the 15:1 ratio from the disomic cross and 17 aneuploid crosses indicated two dominant genes for resistance. Crosses with 7B and 2D did not fit the 15:1 ratio ($P = 0.024$ for 7B and $P = 0.040$ for 2D), but they were not significantly different from the disomic cross ($P = 0.702$ for 7B and $P = 0.484$ for 2D). Crosses with 4A and 1B, which were significantly different from the 15:1 ratio, also were significantly different from the disomic cross and, therefore, were critical. These results were confirmed by tests with race CDL-45; crosses with 4A and 1B did not segregate in a 15:1 ratio and were significantly different from the disomic cross.

When tested with race CDL-1, the disomic cross and 20 aneuploid crosses fit a 3:1 ratio. The cross with 1B did not fit the ratio and was significantly different from the disomic cross. The 1B cross produced excessive susceptible plants, indicating the gene may be ineffective in the hemizygous state. To further confirm the results, chromosomes of 11 F_2 plants from the 1B cross were counted before they were inoculated with race CDL-1. Of the 11 plants, 4 were $2n = 42$ and resistant; 2 were $2n = 40$ and susceptible; and 5 were $2n = 41$ and susceptible. Therefore, the single dominant gene for resistance to race CDL-1 was on chromosome 1B. For tests with race CDL-17, only the disomic cross and crosses with 1A, 4A, 1B, 4B, 7B, 2D, 4D, and 5D were available. The disomic cross and seven of the eight aneuploid crosses produced a 1:3 ratio, indicating a recessive gene for resistance. The cross with 4A did not fit the 1:3 ratio and was significantly different from the

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

Resistant parent	Race	Expected R:S ratio	Cross	Observed F_2 plants[a]		P[b]
				R	S	
Druchamp	CDL-21	15:1	Disomic	160	10	0.843
			Pooled noncritical monosomic	3,087	221	0.306
			1B	197	3	0.006**
			5B	215	2	0.001***
	CDL-1	9:7	Disomic	91	74	0.776
			Pooled noncritical monosomic	1,372	1,133	0.136
			6A	66	92	<0.001***
			1B	67	102	<0.001***
	CDL-45	13:3	Disomic	181	43	0.864
			Pooled noncritical monosomic	1,451	333	0.928
			6A	124	64	<0.001***
			5B	134	17	0.018*
	CDL-35	3:1	Disomic	123	42	0.893
			Pooled noncritical monosomic	2,552	822	0.393
			6A	134	93	<0.001***
	CDL-29	3:1	Disomic	143	46	0.834
			Pooled noncritical monosomic	1,694	544	0.449
			1B	89	17	0.033* (0.095)
Stephens	CDL-21	13:3	Disomic	186	43	0.992
			Pooled noncritical monosomic	3,095	704	0.730
			1B	164	69	<0.001***
			2B	149	80	<0.001***
	CDL-1	14:2	Disomic	190	30	0.610
			Pooled noncritical monosomic	3,153	410	0.073
			1B	175	39	0.011*
			3B	148	3	<0.001***
	CDL-29	1:3	Disomic	53	162	0.906
			Pooled noncritical monosomic	596	1,926	0.113
			1B	22	185	<0.001***
	CDL-6	3:1	Disomic	173	57	0.939
			Pooled noncritical monosomic	3,055	990	0.440
			2B	150	81	<0.001***
	CDL-45	3:1	Disomic	155	44	0.347
			Pooled noncritical monosomic	1,870	573	0.078
			3B	114	20	0.007** (0.103)

(*continued on the next page*)

[a] R = resistant (infection types [IT] 0, 1, 2, and 3); S = susceptible (IT 8 for crosses of Stephens tested with races CDL-1 and CDL-29 and Nord Desprez tested with race CDL-21 and IT 5 and 8 for crosses of Minister tested with races CDL-21, CDL-45, CDL-1, and CDL-17; Druchamp tested with races CDL-21, CDL-1, CDL-45, CDL-35, and CDL-29; Stephens tested with races CDL-21, CDL-6, and CDL-25; Nord Desprez tested with races CDL-45 and CDL-17; Hybrid 46 tested with races CDL-21, CDL-1, and CDL-45; Vilmorin 27 tested with race CDL-21; and Yamhill tested with races CDL-1 and CDL-35).

[b] Probabilities of the chi-square test for goodness-of-fit to the expected resistant:susceptible ratios; probabilities of the chi-square test for association are shown in parentheses when the test for association does not agree with the test for goodness-of-fit at $P = 0.05$. * = significant at $P = 0.05$; ** = significant at $P = 0.01$; and *** = significant at $P = 0.001$.

disomic cross and, therefore, was critical. These results indicate that Minister has a gene on chromosome 1B for resistance to races CDL-21, CDL-45, and CDL-1 and a gene on chromosome 4A for resistance to races CDL-21, CDL-45, and CDL-17. The gene on chromosome 1B is dominant, and the gene on chromosome 4A is either dominant or recessive depending on the race.

Vilmorin 23. For Vilmorin 23 crosses tested with race CDL-21, the 15:1 ratio from the disomic cross and 19 aneuploid crosses indicated two dominant genes for resistance. Crosses with 2B and 6B did not fit the 15:1 ratio and were significantly different from the disomic cross and, thus, were critical. To confirm the results, chromosomes of 12 F_2 plants from each of the two crosses were counted before inoculation with race CDL-21. Of the 12 plants from the 2B cross, 4 $2n = 42$ and 6 $2n = 41$ plants were resistant, and 2 $2n = 40$ plants were susceptible. Of the 12 plants from the 6B cross, 3 $2n = 42$ plants were resistant, 2 $2n = 40$ plants were susceptible, 5 $2n = 41$ plants were resistant, and 2 $2n = 41$ plants were susceptible. These results support the conclusion that Vilmorin 23 has a gene on chromosome 2B and a gene on chromosome 6B and indicate the gene on 2B is effective in the hemizygous state and the gene on 6B is ineffective in the hemizygous state.

Yamhill. In tests with race CDL-1, segregation of the disomic cross and 17 aneuploid crosses fit a 55:9 ratio, which indicated one dominant gene and two recessive genes for resistance. Crosses with 4B, 6B, and 7B did not fit the 55:9 ratio and were significantly different from the disomic cross and, therefore, were critical. The cross with 2D was not available for tests with races CDL-1 and CDL-35. When tested with race CDL-35, segregation of the disomic cross and 18 of the 21 aneuploid crosses fit a 13:3 ratio, indicating a dominant and a recessive gene for resistance. Crosses with 4B and 7B were critical. These results indicate the genes on chromosome 4B and 7B are effective against races CDL-1 and CDL-35, and the gene on chromosome 6B is effective against race CDL-1 but not race CDL-35.

Hybrid 46. When tested with race CDL-21, a 3:1 ratio was produced by the disomic and 20 aneuploid crosses. Segregation of the cross with 6B did not fit the 3:1 ratio and was significantly different from the disomic cross. Because of excessive susceptible plants, chromosomes of 12 F_2 plants from the cross with 6B were counted before inoculation with race CDL-21. Three plants were $2n = 42$ and resistant, six plants were $2n = 41$ and resistant, and three plants were $2n = 40$ and susceptible. The results indicate that the gene is on chromosome 6B and that the 6B cross produces a higher rate of nullisomic plants. When tested with race CDL-45, segregation of the disomic cross and 20 monosomic crosses fit a 1:3 ratio. Only the cross with 6B did not fit the 1:3 ratio and was significantly different from the disomic cross. When tested with race CDL-1, the disomic cross and 17 of the 21 aneuploid crosses segregated in a 3:13 ratio, indicating a dominant and a recessive gene for resistance with complementary interactions. Crosses with 1A and 7B did not fit the 3:13 ratio ($P = 0.036$ for 1A and $P = 0.018$ for 7B) but were not significantly different from the disomic cross ($P = 0.153$ for 1A and $P = 0.108$ for 7B). Crosses with 6A and 6B, which did not fit the 3:13 ratio, also were significantly different from the disomic cross and, therefore, were critical. Based on these results, Hybrid 46 has a gene on chromosome 6B for resistance to

TABLE 2. (*continued from the preceding page*)

Resistant parent	Race	Expected R:S ratio	Cross	Observed F_2 plants[a]		P^b
				R	S	
Nord Desprez	CDL-21	15:1	Disomic	156	15	0.173
			Pooled noncritical monosomic	3,790	277	0.139
			4A	121	2	0.034
			1B	318	65	<0.001***
	CDL-45	15:1	Disomic	157	13	0.452
			Pooled noncritical monosomic	2,954	206	0.532
			4A	120	2	0.035*
			1B	162	19	0.018*
Minister	CDL-21	15:1	Disomic	231	16	0.882
			Pooled noncritical monosomic	3,431	256	0.082
			4A	224	3	0.002**
			1B	191	66	<0.001***
	CDL-45	15:1	Disomic	181	17	0.175
			Pooled noncritical monosomic	3,438	226	0.838
			4A	175	4	0.026*
			1B	150	2	0.012*
	CDL-1	3:1	Disomic	151	47	0.682
			Pooled noncritical monosomic	3,016	1,000	0.884
			1B	80	59	<0.001***
	CDL-17	1:3	Disomic	64	185	0.798
			Pooled noncritical monosomic	264	826	0.518
			4A	39	184	0.010*
Vilmorin 23	CDL-21	15:1	Disomic	216	13	0.720
			Pooled noncritical monosomic	3,692	235	0.491
			2B	192	3	0.007**
			6B	225	32	<0.001***
Yamhill	CDL-1	55:9	Disomic	161	26	0.950
			Pooled noncritical monosomic	2,705	410	0.148
			4B	177	15	0.013*
			6B	186	15	0.007**
			7B	178	10	<0.001***
	CDL-35	13:3	Disomic	150	33	0.804
			Pooled noncritical monosomic	2,394	505	0.067
			4B	109	12	0.013*
			7B	158	13	<0.001***
Hybrid 46	CDL-21	3:1	Disomic	212	65	0.555
			Pooled noncritical monosomic	3,185	999	0.093
			6B	145	87	<0.001***
	CDL-1	3:13	Disomic	48	217	0.791
			Pooled noncritical monosomic	866	3,841	0.536
			6A	27	321	<0.001***
			6B	37	450	<0.001***
	CDL-45	1:3	Disomic	68	195	0.749
			Pooled noncritical monosomic	1,153	3,581	0.306
			6B	16	239	<0.001***

races CDL-1, CDL-21, and CDL-45 and a gene on chromosome 6A for resistance to race CDL-1. The gene on chromosome 6B is dominant or recessive depending on the race with which it is tested.

DISCUSSION

The results of this study corroborate earlier results indicating the existence of specific genes, expression of dominance or recessiveness, and gene interactions reported by Chen and Line (7). Using monosomic analyses, we also detected a third gene in Druchamp (*YrDru2*) and a third gene in Stephens (*YrSte2*) and determined the chromosomal locations of 14 genes (Table 3). The chromosomal locations of 13 of the genes have not been reported previously.

The genes on chromosome 1B should be the genes reported for the *Yr3* locus by Chen and Line (5,7). The gene on chromosome 1B in Druchamp, Stephens, and Nord Desprez should be *Yr3a*, and the gene in Minister should be *Yr3c*. Based on diallel studies, Chen and Line (5) postulated that *Yr3a* is on chromosome 1B. They showed that *Yr3a* in Druchamp and Stephens is either allelic or closely linked to *Yr21* (*YrLem*) in cv. Lemhi and that *Yr21* is either allelic or closely linked to *Yr9* in cv. Riebesel 47/51, which was translocated from the rye chromosome 1RS to wheat chromosome 1BL (29). They further postulated that *Yr3a* in Druchamp, Stephens, and Nord Desprez should be on the long arm of chromosome 1B. Later, Chen et al. (3,9) showed by monosomic analyses that *Yr21* in cv. Lemhi and *Yr9* in cv. Clement are on chromosome 1B. The location of *Yr3c* in Minister on chromosome 1B provides further evidence that the *Yr3* locus is on chromosome 1B. The results of monosomic analyses in this study provide further evidence that Vilmorin 23 does not have *Yr3a* and Hybrid 46 does not have *Yr3b* on chromosome 1B.

Chen and Line (5,7) reported that Vilmorin 23, Yamhill, and Hybrid 46 have a common locus for resistance and, based on diallel crosses and race interactions, determined that Vilmorin 23 and Yamhill have *Yr4a* and Hybrid 46 has *Yr4b*. These results show that the *Yr4* locus is on chromosome 6B and that the gene at that locus in Vilmorin 23 and Yamhill should be *Yr4a* and in Hybrid 46 should be *Yr4b*.

YrDru in Druchamp and *YrSte* in Stephens were reported previously by Chen and Line (4,5,7). The third gene in Druchamp and the third gene in Stephens were not reported because the avirulent races used in those tests did not detect all three genes in each cultivar. Based on IT data produced by different races, the gene on chromosome 5B in Druchamp should be *YrDru*, and the gene on chromosome 2B in Stephens should be *YrSte*. The third gene on chromosome 6A in Druchamp is designated provisionally as *YrDru2*, and the third gene on chromosome 3B in Stephens is designated provisionally as *YrSte2*. The location of *YrSte* on chromosome 2B is less conclusive because there was an excess of susceptible plants. However, the location of that gene on chromosome 2B is supported by previous evidence that a resistance gene in Stephens is linked with *YrV23* in Vilmorin 23 (7), which in this monosomic study was detected on chromosome 2B. Based on studies by Chen and Line (5,7), *YrSte* and *YrV23* are allelic or closely linked when tested with race CDL-21. They are not the same gene, however, because *YrSte* provides resistance to race CDL-6 and *YrV23* is overcome by race CDL-6.

Chen and Line (5,7) named the second gene in Nord Desprez *YrND* and the second gene in Minister *YrMin*. The two genes may be the same. Both genes are on chromosome 4A, both genes are effective against the same races (CDL-21 and CDL-45), and both cultivars have cvs. Hatif Inversable and Squarehead in their pedigrees (30). In contrast, *YrH46* in Hybrid 46 and *YrDru2* in Druchamp are both located on chromosome 6A, but they are not the same gene. *YrDru2* is effective against race CDL-21, whereas *YrH46* is not.

Yamhill was previously reported as having *Yr2*, *Yr4a*, and *YrYam* (4–8). Gene *Yr2* was reported in Heines VII and Heines Peko (12, 20,26) and was located on chromosome 7B in the two cultivars by monosomic analysis (9,17). The gene on chromosome 7B in Yamhill (Heines VII/Alba) should be *Yr2* (4,5,7). Because *Yr4a* is on chromosome 6B and *Yr2* is on chromosome 7B, the gene on chromosome 4B should be *YrYam* (4,5,7). Based on IT data produced by different races, *YrYam* is different from *YrMor* and *YrCle*, which are also on chromosome 4B (9).

The excess of susceptible plants in some crosses (Table 2) made interpretation of the results more difficult. However, the results of studies with multiple races and studies of the association of rust reaction with the chromosomal number of the plants provide confirmation of the locations of the genes. For example, in the cross of Minister with monosomic 1B tested with race CDL-1, the cross of Druchamp with 6A tested with race CDL-35, and the cross of Vilmorin 23 with 6B tested with race CDL-21, we showed that the resistance genes were ineffective when in the hemizygous state. The phenomenon of hemizygous ineffectiveness has been reported in several previous studies (3,9,11,17,27). Shen et al. (25) reported that a recessive gene in wheat cv. C39 in the hemizygous state was effective against a race of *P. striiformis*. Others have reported that recessive genes in the hemizygous state were ineffective (11, 17,27). We clearly demonstrated that the dominant gene (*Yr21*) in cv. Lemhi was ineffective when in the hemizygous state (3). High rates of nullisomic plants could have caused an excess of susceptible plants in some of the crosses. In some tests, nullisomic plants were in excess of the 3% average reported by Sears (23,24). Our data showed that the frequency of nullisomic plants for chromosome 6B in Hybrid 46 could be higher than 3%. High frequencies of nullisomic plants in progeny of selfed monosomic plants have been reported in studies with both wheat and oat (2,14,15). Additional cytological studies are needed to determine conclusively that the excess of susceptible plants is due to hemizygous ineffectiveness or high rates of nullisomic plants.

These results add significant information to our knowledge of the chromosomal location of genes for stripe rust resistance. Genes for stripe rust resistance have been found on all chromosomes, except 5A, 7A, and 1D (3,9,11,17,22,25). The distribution of stripe rust resistance genes on wheat chromosomes suggests that gene pyramiding for wheat resistance is more feasible than in some crop-disease systems in which resistance genes are clustered or occur as multiple alleles. The information on chromosomal location should contribute to a better understanding of the genetics of resistance to stripe rust in wheat and may be useful in tagging resistance genes and transferring the genes to commercial cultivars.

TABLE 3. Genes for resistance to *Puccinia striiformis* f. sp. *tritici* and associated chromosomes in seven wheat cultivars

Yr gene[a]	Cultivar	Chromosome
Yr2	Yamhill	7B
Yr3a	Druchamp	1B
	Stephens	1B
	Nord Desprez	1B
Yr3c	Minister	1B
Yr4a	Vilmorin 23	6B
	Yamhill	6B
Yr4b	Hybrid 46	6B
YrDru	Druchamp	5B
YrDru2	Druchamp	6A
YrSte	Stephens	2B
YrSte2	Stephens	3B
YrND	Nord Desprez	4A
YrMin	Minister	4A
YrV23	Vilmorin 23	2B
YrYam	Yamhill	4B
YrH46	Hybrid 46	6A

[a] The *Yr* genes were named in publications 5–8, 13, 18, 20–22.

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