Gene Action in Wheat Cultivars for Durable, High-Temperature, Adult-Plant Resistance and Interaction with Race-Specific, Seedling Resistance to *Puccinia striiformis*

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ABSTRACT

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Stephens and Druchamp wheat (*Triticum aestivum*) cultivars have both durable, high-temperature, adult-plant (HTAP) resistance and race-specific, seedling resistance to stripe rust caused by *Puccinia striiformis*. Gene action for the HTAP resistance was studied in parental and F_1, F_2, and backcross populations from reciprocal crosses among Stephens, Druchamp, Paha (a cultivar with race-specific resistance), and Michigan Amber (a susceptible cultivar) using stripe rust intensity data from the field transformed to area under the disease progress curve (AUDPC). Based on a joint scaling test, the additive component for HTAP resistance was significant for both Stephens and Druchamp. When HTAP resistance was effective and seedling resistance was ineffective in the same parent, the dominant component and the additive-additive, additive-dominant, and dominant-dominant epistatic interactions contributed significantly to HTAP resistance in Druchamp but not in Stephens. When HTAP resistance and seedling resistance were effective in the same parent, the dominant component and additive-additive and dominant-dominant epistatic interactions were significant in both Stephens and Druchamp and the additive-dominant epistatic interaction was significant in Stephens but not in Druchamp. When HTAP resistance was effective in one parent and seedling resistance was effective in the other parent, the dominant component and the additive-additive and dominant-dominant epistatic interactions were significant in the Druchamp crosses but not in the Stephens crosses. When HTAP resistance was effective in both parents, the additive and dominant components were detected at the Mount Vernon, WA, site but not the Pullman, WA, site. A constant cytoplasmic effect and a cytoplasm-dominance interaction were significant in reciprocal crosses of Stephens with Paha inoculated with race CDL-29; HTAP resistance was greater when Stephens was the female parent. A cytoplasm-additive gene interaction was significant in the reciprocal crosses of Druchamp with Paha tested with race CDL-25. Based on the gene action of HTAP resistance and its interactions with seedling resistance, it should be possible to exploit both HTAP and seedling resistances in breeding programs and in crosses; Stephens and Druchamp should be used as female parents to obtain the highest HTAP resistance.

Additional keywords: general resistance, quantitative genetics, yellow rust.

Stripe rust, caused by *Puccinia striiformis* Westend. f. sp. *tritici*, is an important disease of wheat (*Triticum aestivum* L.) in many regions of the world. In North America, the disease is highly destructive in the western United States and occasionally destructive in the south-central United States. The disease is controlled mainly by the use of resistant cultivars, of which seedling resistance and high-temperature, adult-plant (HTAP) resistances are most important (10,11). Seedling resistance to stripe rust is expressed at all stages of plant growth and is race-specific. Cultivars with seedling resistance often become susceptible within a few years after their release because of the rapid evolution of new virulent races of the pathogen that circumvent the resistance, whereas HTAP resistance is expressed as plants become older and only at higher temperatures (11,18).

Cultivars with HTAP resistance have remained resistant when exposed to many races and grown extensively in large geographic regions. Therefore, they are considered to have durable, nonspecific resistance (10,11,14,15). In the Pacific Northwest from the early 1930s to the mid-1950s, stripe rust was not considered important because most cultivars grown during that period had HTAP resistance (14,15,18). Cultivars with race-specific seedling resistance were extensively grown during the late 1950s. New virulent races appeared, and severe stripe rust epidemics occurred (11). Since 1961, stripe rust in the Pacific Northwest has been controlled primarily by HTAP resistance. Qayoum and Line (18) showed that cultivars with HTAP resistance differ in degree of resistance. HTAP resistance is expressed at high postinoculation temperatures, and flag leaves are most resistant. Cultivars with HTAP resistance are resistant in the field at the jointing and later stages of growth. Cultivars with a greater degree of HTAP resistance begin to express resistance at an earlier stage of plant growth. Milus and Line (15) studied the gene action for inheritance of HTAP resistance to stripe rust using area under disease progress curve (AUDPC). They reported that HTAP resistance in Gaines, Nugaines, and Luke wheat cultivars was partially recessive with no maternal inheritance. Using the joint scaling test of Mather and Jinks (13) to analyze the disease data, Milus and Line (15) found that there was a significant epistatic gene action for resistance in Nugaines and susceptibility in Luke and that most gene action among the loci was additive.

Stephens has seedling resistance genes *Yr3a* and *YrSte* and Druchamp has seedling resistance genes *Yr3a* and *YrDru* (1,2,3). Both cultivars also have high HTAP resistance. Since its release in 1978 (7), Stephens has been grown extensively in the Pacific Northwest and never has been severely damaged by stripe rust. Druchamp, an older French cultivar introduced into the Pacific Northwest, has remained resistant but is no longer grown because other cultivars have higher yields. The objectives of this study were to determine the gene action of HTAP resistance in Stephens and Druchamp, to determine the relationships of HTAP resistance to seedling resistance and to determine the effect of cytoplasm on resistance.

MATERIALS AND METHODS

Four wheat (*T. aestivum*) cultivars were used in this study; Stephens (CI017596) and Druchamp (CI013723), which have both seedling and HTAP resistance, Paha (CI014485), which has seedling resistance (1,2,3) but not HTAP resistance, and Michigan Amber (PI315203), which has no seedling or HTAP resistance to North American races of *P. striiformis*. The initial seed of each cultivar came from an individual plant.

The effect of temperature and stage of plant growth on the resistance of the four cultivars to races CDL-25 and CDL-29 of *P. striiformis* was studied in the greenhouse and field. Race CDL-25 was selected because it is virulent on seedlings of Stephens and Druchamp, avirulent on adult plants of Stephens and Druchamp, avirulent on both seedlings and adult plants of Paha, and virulent on Michigan Amber. Race CDL-29 was selected because it is virulent on both seedlings and adult plants of Paha and Michigan Amber and avirulent on both seedling and adult plants of Stephens and Druchamp. Race CDL-25 is common wherever Stephens is grown extensively. Race CDL-29 is common where club wheat cultivars are grown in the Pacific Northwest.

Conditions and methods for growing and inoculating plants in the greenhouse were as described by Chen and Line (1,2,3). Seedlings were inoculated at the two-leaf stage, and adult-plants were inoculated at the heading stage. Plants inoculated with each race were placed in a dew chamber at 10 C for 24 h and then placed in two separate greenhouse chambers for rust development: one at a low diurnal temperature cycle (4–20 C) and the other at a high diurnal temperature cycle (10–35 C). Infection types based on a 0–9 scale (12) were recorded for each plant 20–25 days after inoculation.

Reciprocal, diallel crosses between Stephens and Druchamp and of Stephens and Druchamp with Paha or Michigan Amber were made in a greenhouse. F_1 plants were grown in the field to obtain F_2 seed. Backcrosses to both parents were made in the field using F_1 plants as the female parent. Parents and F_1, F_2, and backcross generations of all crosses were planted in late September to mid-October 1989. Parents and progeny from crosses of Stephens and Druchamp with Michigan Amber were planted at Pullman, WA, and inoculated with race CDL-25. Two separate plots at Pullman were planted with parents and progeny from crosses of Stephens and Druchamp with Paha: one plot inoculated with race CDL-25 and the other plot inoculated with race CDL-29. Parents and progeny from reciprocal crosses of Stephens with Druchamp were planted at Pullman and inoculated with race CDL-25 and at Mount Vernon, WA, were exposed to naturally occurring race CDL-25. Each plot was a randomized complete-block design with three replications. Each replication consisted of one row of each parent, one row of F_1, three to 10 rows of each backcross, and 20 rows of F_2. Five seeds were planted in each F_1 row, and 10 seeds were planted in each of the other rows. The seeds were planted 15 cm apart in 1.5 m long rows, with a 30 cm space between rows. Plants of each row in the Pullman plots were inoculated with urediospores mixed with talc when the plants were at the three- to five-leaf stage (mid-April). Seedlings of Druchamp and Stephens that had been inoculated in a greenhouse were uniformly transplanted into the plots to further ensure adequate inoculation.

Infection types and rust intensity (percentage of leaf area infected) based on the modified Cobb's scale for measuring rust severity (17) were recorded during the spring and summer of 1990, twice at Pullman and three times at Mount Vernon. The first recording was at the stem-elongation stage (jointing) at Mount Vernon (14–15 April) and heading to anthesis stages at Pullman (5–8 June for the plot inoculated with race CDL-29, 13–14 June for the crosses with Michigan Amber inoculated with race CDL-25, and 19–20 June for the crosses with Paha inoculated with race CDL-25). The second and the third records were made 2–3 wk after the previous recording.

The rust intensity data were used to calculate AUDPC for each plant using the formula

$$Y = \Sigma[(\chi_i + \chi_{i+1})/2](t_{i+1} - t_i)$$

where Y = AUDPC, χ_i = the rust intensity of the ith note, χ_{i+1} = the rust intensity of the $i+1$th note, and $(t_{i+1} - t_i)$ = the number of days between the ith note and the $i + 1$th note. Means and variances of AUDPC were calculated for the parental, F_1, and segregation generations of each cross and used to determine gene action.

The method described by Falconer (5) was used to determine the degree of dominance for each cross and was calculated as the deviation of the F_1 from the midparent (h) divided by the departure of the more susceptible parent from the midparent (d). The joint scaling test described by Mather and Jinks (6,13) was used to determine the resistance gene action. The joint scaling test estimates nuclear genetic components and nonallelic interactions of a cross and uses the estimates to fit different genetic models to the data. The theoretical basis of the joint scaling test is a linear model. For the stripe rust data, the mean AUDPC (X) of a generation can be described by the following linear equation:

$$X = m + d + h + i + j + l$$

where m = mean of all possible homozygotes, d = additive component, h = dominance component, i = additive-additive interaction, j = additive-dominance interaction, and l = dominance-dominance interaction. The coefficients of these components for different generations as described by Mather and Jinks (13) are shown in Table 1. Means and variances of the parental, F_1, F_2, and backcross generations of each cross were used in the test to determine the genetic components involved in the tested models. Because six generations (the two parents, F_1, F_2, and two backcrosses) for each cross were utilized in this study, models involving two to five parameters can be tested.

A chi-square test was used to determine the goodness of fit of each genetic model. For those genetic models with a 0.05 or greater chi-square probability, individual genetic components were tested for significance using a t test. Those genetic components significantly different from zero ($P \leq 0.05$) were considered to contribute to the genetic models. We accepted the models that fit the data and had components that were all significant.

The t test for differences in reciprocal generation means and the method of Mosjidis et al (16) were used to test for the occurrence of cytoplasmic inheritance. In the t test, mean AUDPC values of F_1, F_2, and backcross generations of reciprocal crosses were compared. A pair of reciprocal generations were considered different if $P < 0.05$. The method of Mosjidis et al (16) combines cytoplasmic effects with the additive-dominance model described by Mather and Jinks (13). Similar to the joint scaling tests for nuclear genetic components, cytoplasmic components were incorporated into the additive-dominance model. Two models for cytoplasmic effects as well as nuclear genetic components were used. The first model was used to determine a constant cytoplasmic effect c, which remains constant through successive generations. The cytoplasm from one parent increases AUDPC by [c], while the cytoplasm of the other parent decreases AUDPC by [c],

TABLE 1. Coefficients for genetic components as described by Mather and Jinks (13)

Coefficient for genetic component	Generation[a]					
	P_1	P_2	F_2	F_2	B_1	B_2
Mean of homozygous generations (m)	1	1	1	1	1	1
Additive component (d)	1	−1	0	0	0.5	−0.5
Dominant component (h)	0	0	1	0.5	0.5	0.5
Additive-additive (i)	1	1	0	0	0.25	0.25
Additive-dominant (j)	0	0	0	0	0.25	−0.25
Dominant-dominant (l)	0	0	1	0.25	0.25	0.25

[a] P_1 = parent 1; P_2 = parent 2; B_1 = backcross to P_1; and B_2 = backcross to P_2.

regardless of genotype. The second model was used to determine whether there is an interaction between cytoplasm and genotype. This model assumes that the effect of cytoplasm on resistance is not equally present in all generations. When there is a cytoplasm-additive interaction (c_a), the cytoplasm from one parent increases AUDPC of a homozygous susceptible plant by $[c_a]$, while the cytoplasm of the other parent decreases AUDPC by the same amount. When there is a cytoplasm-dominance interaction (c_d), the cytoplasm of one parent increases AUDPC of a heterozygous plant by $[c_d]$, while the cytoplasm of the other parent decreases AUDPC of a heterozygous plant by $[c_d]$. Since data of 10 generations (two parents, two F_1, two F_2, and four backcross populations) are available for a pair of reciprocal crosses, up to nine parameters can be estimated. In this analysis, tests of all possible combinations were unnecessary, because joint scaling tests for genetic components of additive, dominance, and nonallelic interactions were already made. As in the tests of nuclear genetic components, a chi-square test was used to determine the fitness of a model involving cytoplasmic effects. If the model fit the data, according to the chi-square tests, a t test also was used to determine if the cytoplasmic component was significant. Similarly, we accepted the models that fit the data and consisted of components that were significantly different from zero.

RESULTS

Effect of temperature and plant growth stage on resistance. At all temperatures and stages in the greenhouse and fields, Michigan Amber was susceptible to races CDL-25 and CDL-29, Paha was resistant to race CDL-25 and susceptible to race CDL-29, and Stephens and Druchamp were resistant to race CDL-29 (Table 2). Seedlings of Stephens and Druchamp were susceptible to race CDL-25 at all temperatures. Adult plants of the two cultivars were susceptible at the low temperature cycle but resistant at the high temperature cycle. Fewer pustules developed on adult plants of Druchamp than on Stephens, suggesting the HTAP resistance in Druchamp was greater than that in Stephens.

Degree of dominance. In crosses with Michigan Amber inoculated with race CDL-25, HTAP resistances of Stephens and Druchamp were partially recessive or partially dominant, depending on the reciprocal crosses (Table 3). The degree of dominance was greater than 1.0 for reciprocal crosses at Mount Vernon and when Stephens was the female parent at Pullman, indicating that the heterozygotes were more susceptible than either parent.

In all of the tests in which HTAP and seedling resistances were both present, the degree of dominance for susceptibility was negative (the heterozygote was closer to the resistant parent). Resistance varied from partially dominant in Paha/Stephens inoculated with race CDL-29 to almost completely dominant in Paha/Druchamp inoculated with either CDL-25 or CDL-29. There were no significant differences in the degree of dominance between the reciprocal crosses of Druchamp with Paha, but the degree of dominance for resistance was always higher when Stephens was the female parent.

Genetic components of resistance. The generation means and variances of AUDPC (Table 4) were used to estimate genetic components based on the joint scaling test. Those models that fit the data, as indicated by chi-square test at $P \geq 0.05$, and

TABLE 2. Effect of low (4–20 C) and high (10–35 C) diurnal temperature cycles on infection types produced by races CDL-25 and CDL-29 of *Puccinia striiformis* on wheat cultivars inoculated at seedling and heading stages of plant growth in the greenhouse and infection types and rust intensities on flag leaves of wheat cultivars at the dough stage of plant growth in plots at Pullman and Mount Vernon, WA

		Infection type[a]						Rust intensity at dough stage	
		Seedling stage		Heading stage		Dough stage			
Cultivar	CDL race	4–20 C	10–35 C	4–20 C	10–35 C	Pullman	Mt. Vernon	Pullman	Mt. Vernon
Michigan Amber	25	8	8	8	8	8	8	90	95
	29	8	8	8	8	8	...[b]	90	...
Paha	25	2	2	1	0–1	0	0	0	0
	29	8	8	8	8	8	...	95	...
Druchamp	25	8	7	8[c]	0–3	0–2	0–3	<1	<1
	29	2	1–2	0	0	0	...	0	...
Stephens	25	8	8	8	0–3	0–2	0–3	<1	2
	29	2	2	0	0	0	...	0	...

[a]Infection types are based on a 0–9 scale as described by Line and Qayoum (12); 0 = no rust; 1–3 = highly resistant to moderately resistant; 7 = moderately susceptible; and 8 = susceptible.
[b]Race CDL-29 did not occur at Mt. Vernon.
[c]Fewer uredia developed on Druchamp at the low temperature.

TABLE 3. Values as measured by area under disease progress curve (AUDPC) for midparent (m), the departure of the more susceptible parent from the midparent (d), the deviation of the F_1 from the midparent (h), and degree of dominance (h/d) for progeny of reciprocal wheat crosses inoculated with races CDL-25 and CDL-29 of *Puccinia striiformis* at Pullman, WA, and tested with naturally occurring race CDL-25 at Mount Vernon, WA

	Value					
			h		h/d	
Cross[a] P_1/P_2 (P_2/P_1)	m	d	P_1/P_2	P_2/P_1	P_1/P_2	P_2/P_1
Race CDL-25 at Pullman						
STE/MA (MA/STE)	522	493	59	−24	0.12	−0.05
DRU/MA (MA/DRU)	501	496	117	−99	0.24	−0.19
STE/Paha (Paha/STE)	77	73	−70	−53	−0.96	−0.73
DRU/Paha (Paha/DRU)	109	109	−105	−106	−0.97	−0.97
DRU/STE (STE/DRU)	10	5	−6	34	−1.10	6.40
Race CDL-25 at Mt. Vernon						
DRU/STE (STE/DRU)	270	67	292	193	4.35	2.87
Race CDL-29 at Pullman						
STE/Paha (Paha/STE)	708	707	−554	−233	−0.78	−0.33
DRU/Paha (Paha/DRU)	626	626	−605	−608	−0.97	−0.97

[a]P_1 = parent 1 and P_2 = parent 2. P_1 is the female parent in cross P_1/P_2 and P_2 is the female parent in the cross P_2/P_1, within parentheses. STE = cv. Stephens; DRU = cv. Druchamp; and MA = cv. Michigan Amber.

that had all components with significant differences from zero at $P < 0.05$ of the t test are shown in Table 5.

Data from the crosses of Stephens and Druchamp with Michigan Amber inoculated with race CDL-25 were used to study HTAP resistance in Stephens or Druchamp. The data from Stephens/Michigan Amber fit 15 models using the joint scaling tests, but only one model, $m[d]$, was acceptable according to the t test, indicating that only the additive component contributed significantly to HTAP resistance from Stephens. In crosses Druchamp/Michigan Amber, additive [d] and dominance [h] components and additive-additive [i], additive-dominance [j], and dominance-dominance [l] interactions contributed significantly to HTAP resistance from Druchamp, but the additive-additive component was not significant in the reciprocal cross, Michigan Amber/Druchamp. The additive component was significant in all of the acceptable models, indicating that it was a major contributor to HTAP resistance in Druchamp.

Reciprocal crosses of Stephens with Druchamp tested with race CDL-25 were used to study HTAP resistance from both parents. None of the components were significant at Pullman. At Mount Vernon, the dominant component [h] was significant when Druchamp was the female parent, and the additive component

TABLE 4. Generation means and standard deviations of area under disease progress curve (AUDPC) for crosses tested with races CDL-25 and CDL-29 of *Puccinia striiformis* in the field at Pullman and Mount Vernon, WA, and comparisons of reciprocal generation means by t tests

Cross[a] P_1/P_2 (P_2/P_1)	Mean and standard deviation of AUDPC and significance of t test for comparison of reciprocal crosses[b]					
	P_1	P_2	F_1	F_2	B_1	B_2
Race CDL-25, Pullman						
STE/MA (MA/STE)	29 ± 30	1,015 ± 71	581 ± 117 (499 ± 114)*	462 ± 303 (...)[c]	220 ± 189 (...)	864 ± 246 (...)
DRU/MA (MA/DRU)	5 ± 11	997 ± 78	618 ± 82 (401 ± 60)**	314 ± 281 (286 ± 252)	26 ± 51 (19 ± 51)	777 ± 248 (797 ± 215)
STE/DRU (DRU/STE)	16 ± 8	5 ± 11	45 ± 25 (4 ± 9)**	43 ± 109 (18 ± 82)**	11 ± 45 (15 ± 31)	14 ± 37 (10 ± 70)
STE/Paha (Paha/STE)	4 ± 7	149 ± 141	7 ± 7 (24 ± 56)**	50 ± 168 (168 ± 301)**	65 ± 212 (66 ± 150)	259 ± 241 (144 ± 190)**
DRU/Paha (Paha/DRU)	0 ± 2	218 ± 78	4 ± 10 (3 ± 6)	104 ± 238 (138 ± 259)*	28 ± 79 (9 ± 36)**	202 ± 265 (83 ± 108)**
Race CDL-25, Mt. Vernon						
STE/DRU (DRU/STE)	337 ± 84	203 ± 62	462 ± 149 (564 ± 115)*	526 ± 356 (499 ± 335)	667 ± 324 (543 ± 314)**	439 ± 258 (343 ± 175)**
Race CDL-29, Pullman						
STE/Paha (Paha/STE)	1 ± 5	1,415 ± 127	154 ± 64 (476 ± 73)**	239 ± 352 (463 ± 438)**	17 ± 46 (46 ± 167)*	620 ± 416 (765 ± 378)*
DRU/Paha (Paha/DRU)	0 ± 2	1,252 ± 73	22 ± 3 (18 ± 8)*	50 ± 143 (62 ± 162)	407 ± 3 (542 ± 9)**	468 ± 419 (407 ± 434)

[a] P_1 = parent 1 and P_2 = parent 2. P_1 was the female parent in cross P_1/P_2 and P_2 was the female parent in the cross P_2/P_1, within parentheses. STE = cv. Stephens; MA = cv. Michigan Amber; and DRU = cv. Druchamp.

[b] The first mean and standard deviation are for cross P_1/P_2, and the second mean and standard deviation, within parentheses, are for cross P_2/P_1. Reciprocal generation means were compared using a t test; one asterisk indicates a significant difference at $P = 0.05$ and two asterisks indicate a significant difference at $P = 0.01$.

[c] Data from the reciprocal cross were not available.

TABLE 5. Acceptable genetic models, their chi-square values and probabilities and components of genetic variation and their significance for crosses between wheat cultivars tested with races CDL-25 and CDL-29 of *Puccinia striiformis* at Pullman and Mount Vernon, WA

Cross[a]	CDL race	Location	Joint scaling test of the model		
			Model[b]	χ^2	P
HTAP resistance in one parent					
STE/MA	25	Pullman	$m[d]$**	3.59	0.46
DRU/MA	25	Pullman	$m[d]$**[j]*	2.78	0.43
			$m[d]$**[h]*[i]*	1.74	0.42
			$m[d]$**[h]*[l]*	0.81	0.67
MA/DRU	25	Pullman	$m[d]$**[j]*	2.41	0.49
			$m[d]$**[h]*[l]*	1.81	0.40
HTAP resistance in both parents					
DRU/STE	25	Pullman	None	...[c]	...
STE/DRU	25	Pullman	None	...	...
DRU/STE	25	Mt. Vernon	$m[h]$*	1.06	0.90
STE/DRU	25	Mt. Vernon	$m[d]$*	3.11	0.54
HTAP and seedling resistance in different parents					
STE/Paha	25	Pullman	None	...	...
Paha/STE	25	Pullman	None	...	...
DRU/Paha	25	Pullman	$m[d]$*[h]*	0.26	0.97
			$m[d]$*[i]*	0.79	0.85
			$m[d]$*[l]*	0.06	1.00
Paha/DRU	25	Pullman	$m[d]$*[h]*	0.21	0.98
			$m[d]$*[i]*	1.11	0.77
			$m[d]$*[l]*	0.49	0.98
HTAP and seedling resistance in the same parent					
STE/Paha	29	Pullman	$m[d]$**[h]**	1.52	0.68
			$m[d]$**[i]**	1.31	0.73
			$m[d]$**[j]*[l]**	2.15	0.34
Paha/STE	29	Pullman	$m[d]$**	8.31	0.08
			$m[d]$**[h]*	1.54	0.67
			$m[d]$**[i]*	0.67	0.88
			$m[d]$**[l]*	2.71	0.44
DRU/Paha	29	Pullman	$m[d]$**[h]**[i]*	3.18	0.20
			$m[d]$**[h]**[l]*	3.45	0.18
Paha/DRU	29	Pullman	$m[d]$**[h]**	3.44	0.33

[a] DRU = cv. Druchamp; MA = cv. Michigan Amber; and STE = cv. Stephens. HTAP = high-temperature, adult-plant resistance.

[b] In the models, m = the mean of all possible homozygotes in the six populations; d = additive component; h = dominance component; i = additive-additive epistatic component; j = additive-dominance epistatic component; and l = dominance-dominance component (13). The components with two asterisks were significant at $P = 0.01$, and those with one asterisk were significant at $P = 0.05$. None = none of the components in the model was significant at $P = 0.05$.

[c] Chi-square values and probabilities are not listed because none of the models are acceptable.

[*d*] was significant when Stephens was the female parent.

In reciprocal crosses of Stephens and Druchamp with Paha tested with race CDL-25, HTAP and seedling resistance were from different parents. None of the genetic components were significant in the crosses of Stephens with Paha. In crosses of Druchamp with Paha, additive [*d*] component, dominance [*h*] component, additive-additive [*i*] interaction, and dominance-dominance [*l*] interaction were significant in the reciprocal crosses. The additive component was significant in all of the acceptable models, indicating that the additive component was a major contributor when HTAP resistance from Druchamp was combined with seedling resistance from Paha.

In crosses of Stephens and Druchamp with Paha inoculated with race CDL-29, HTAP resistance and seedling resistance were in the same parent. In these crosses, the additive component was significant in all of the acceptable models. In reciprocal crosses of Stephens with Paha, the dominance component [*h*] and additive-additive [*i*] interaction, and dominance-dominance [*l*] interaction were significant. The additive-dominance [*j*] interaction was significant when Stephens was the female parent but was not significant when Paha was the female parent. The additive [*d*] and dominance [*h*] components were significant in reciprocal crosses of Druchamp with Paha. The components of additive-additive [*i*] and dominance-dominance [*l*] interactions were significant when Druchamp was the female parent but were not significant when Paha was the female parent. The dominance component [*h*] was significant in all of the acceptable models for the Druchamp crosses but was only significant in some of the acceptable models for the Stephens crosses. These results indicate that the effect of the dominance component [*h*], probably from the seedling resistance, was greater in Druchamp than in Stephens.

Cytoplasmic effects. The results of comparing reciprocal generations using the *t* test are presented in Table 4. Significant differences were detected in the F_1 generations of reciprocal crosses of Stephens and Druchamp with Michigan Amber; the F_1, F_2, and the backcross to Paha of the crosses of Stephens with Paha; the F_2 and the two backcrosses of the crosses of Druchamp with Paha; the F_1 and F_2 generations of the crosses of Druchamp with Stephens at Pullman; the F_1 and the two backcrosses of the crosses of Druchamp with Stephens at Mount Vernon; all progeny from crosses of Stephens with Paha; and the F_1 and the backcross to Druchamp of the crosses of Druchamp with Paha tested with race CDL-29. These results suggest that there are maternal or cytoplasmic factors that affect stripe rust resistance.

The results of using the joint scaling tests for the models containing cytoplasmic components and the *t* test for the significance of genetic components are shown in Table 6. The component of cytoplasm-additive interaction [c_a] was significant in the reciprocal crosses of Druchamp with Paha tested with race CDL-25. The components of constant cytoplasmic effect (*c*) and the cytoplasm-dominant interaction [c_d] were significant in the reciprocal crosses of Stephens with Paha tested with race CDL-29. The results indicate that the Paha cytoplasm increases and the Stephens cytoplasm decreases susceptibility.

DISCUSSION

The partially recessive inheritance of HTAP resistance in Stephens and Druchamp agree with studies by Milus and Line (15) on inheritance of HTAP resistance in cvs. Gaines, Nugaines, and Luke. Based on both studies, partially recessive inheritance appears to be a general characteristic of HTAP resistance.

Based on joint scaling tests of crosses with Michigan Amber (Table 5), the additive component is a major contributor to HTAP resistance in Druchamp and the only significant contributor to HTAP resistance in Stephens. The gene action of Druchamp is more complex because it includes a dominant component and nonallelic interactions. None of the genetic components were significant in the crosses of Druchamp with Stephens when tested at Pullman. At Mount Vernon, however, the additive component was detected when Stephens was the female parent, and the dominant component was detected when Druchamp was the female parent. The difference at the two locations could be due to the different climatic conditions. At Mount Vernon, where the environment was most favorable for stripe rust, the earlier occurrence of the disease and greater number of cycles of infection during the growing season enabled a greater differentiation between the two parents and the progeny. At Pullman, where the rust occurred later and progressed slower, the differences among the generations may not have been sufficient to detect significant genetic effects. When both HTAP and seedling resistance were effective, significant additive and dominance components were detected in most tests. Most additive effects were apparently from HTAP resistance genes, and most dominant effects were apparently from seedling resistance genes.

Cytoplasmic effects are usually studied by comparing the phenotypic expressions of reciprocal F_1 or F_2 generations (15), but the method does not measure cytoplasmic effects that are not constant in other generations. By analyses of backcrosses, as well as the F_1 and F_2 generations, we detected significant differences for at least one pair of reciprocal generations in all crosses (Table 4). Significant differences in all progeny (F_1, F_2, B_1, and B_2) were detected only in reciprocal crosses of Stephens with Paha tested with race CDL-29 and in the segregating generations (F_2, B_1, and B_2) in reciprocal crosses of Druchamp with Paha tested with race CDL-25. In some cross-race combinations, reciprocal differences were not in the same direction for different generations (Table 4). For example, in crosses of Stephens with Paha tested with race CDL-25, the mean AUDPC was higher for the F_1 and F_2 generations when Paha was the female parent, but in the backcross to Paha, the mean AUDPC was higher when Stephens was the female parent in the initial cross. This inconsistency cannot be explained.

Because the method of Mosjidis et al (16) utilizes all possible generations, it should provide not only a more accurate estimate of the total effect of the cytoplasm but also should indicate the type of effect. Using this method, the constant cytoplasmic effect and cytoplasm-dominance interaction were evident in crosses of Stephens with Paha when tested with race CDL-29, and the cytoplasm-additive interaction was significant in the crosses of Druchamp with Paha tested with race CDL-25. For the two crosses, significant reciprocal differences also were observed for

TABLE 6. The joint scaling tests for the models involving cytoplasmic effects in the reciprocal wheat crosses tested for resistance to races of *Puccinia striiformis* at Pullman and Mount Vernon, WA

Reciprocal crosses[a]	Joint scaling test for the model		
	Model[b]	χ^2	*P*
Race CDL-25, Pullman			
DRU/MA & MA/DRU	None[c]	...[d]	...
STE/Paha & Paha/STE	None	...	...
DRU/Paha & Paha/DRU	*m*[*d*]**[*h*]**	0.48	1.00
	m[*h*]*[c_a]*	4.72	0.69
DRU/STE & STE/DRU	None	...	...
Race CDL-25, Mt. Vernon			
DRU/STE & STE/DRU	None	...	...
Race CDL-29, Pullman			
STE/Paha & Paha/STE	*m*[*d*]**[*h*]**[*c*]**	6.89	0.33
	m[*d*]**[*h*]**[c_d]**	4.33	0.63
DRU/Paha & Paha/DRU	*m*[*d*]**[*h*]**	14.63	0.04

[a] DRU = cv. Druchamp; MA = cv. Michigan Amber; and STE = cv. Stephens.

[b] In the models, *m* = the average of possible homozygotes of 10 generations from the reciprocal crosses between the two cultivars; *d* = the additive component; *h* = the dominance components; *c* = the constant cytoplasmic effect from the female parent; c_a = the cytoplasm-additive interaction; and c_d = the cytoplasm-dominance interaction (13,16). The components with two asterisks were significant at $P = 0.01$, and those with one asterisk were significant at $P = 0.05$.

[c] None of the model fit the data, and therefore, *t* tests for components were not conducted.

[d] Chi-square values and probabilities are not listed because none of the models with cytoplasmic components are acceptable.

the F_3 and F_5 generations (data not shown), further indicating the presence of specific cytoplasmic effects. The joint scaling method did not detect significant cytoplasmic effects in the other cross-race combinations.

There are few reports of the effects of host cytoplasm on stripe rust resistance. Krupinsky and Sharp (8) in their report on identification of additive genes for resistance to stripe rust attributed some resistance to cytoplasmic effects, but Röbbelen and Sharp (19) in their review of the genetics of stripe rust resistance concluded that extrachromosomal factors had not been shown for stripe rust resistance. Milus and Line (15) in their study of HTAP resistance to stripe rust in cvs. Gaines, Nugaines, and Luke reported that there were no significant differences in the reciprocal F_1 and F_2 generations. Labrum (9) reported that wheat lines with *Aegilops ovata* cytoplasm had better adult-plant resistance to stripe rust than similar lines with *T. aestivum* cytoplasm. We previously presented evidence of specific interactions of cytoplasm and race-specific nuclear genes (3). In this study, constant cytoplasmic effects and a cytoplasm-dominance interaction were clearly evident.

The different phenotypic expressions of HTAP resistance in Druchamp and Stephens (Table 2), the segregation of the progeny from the crosses of Druchamp and Stephens (Table 4), and the different genetic components of HTAP resistance (Tables 5 and 6) indicate that the genes for HTAP resistance in Druchamp are different from the genes for HTAP resistance in Stephens and that genotypes with greater resistance can be selected from crosses of Druchamp with Stephens. This also is supported by our studies on gene number of HTAP resistance in Druchamp and Stephens (4). The information on the degree of dominance, genetic components, interaction of HTAP and seedling resistances, and cytoplasmic effects should be useful in developing new cultivars with superior HTAP and seedling resistance. It should be possible to use advanced generations from crosses of Stephens and Druchamp to develop cultivars with more resistance. Since the commonly grown club wheats do not have HTAP resistance, progeny from crosses of Stephens and Druchamp with Paha should be especially valuable in developing club wheat cultivars with durable, HTAP resistance.

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