Gene Number and Heritability of Wheat Cultivars with Durable, High-Temperature, Adult-Plant (HTAP) Resistance and Interaction of HTAP and Race-Specific Seedling Resistance to *Puccinia striiformis*

Xianming Chen and Roland F. Line

Research associate, Department of Plant Pathology, Washington State University, and plant pathologist, Agricultural Research Service, U.S. Department of Agriculture, respectively, Pullman, WA 99164-6430.
PPNS 0192, College of Agriculture and Home Economics Research Center, Washington State University, Pullman, WA 99164.
Accepted for publication 10 January 1995.

ABSTRACT

Chen, X. M., and Line, R. F. 1995. Gene number and heritability of wheat cultivars with durable, high-temperature, adult-plant (HTAP) resistance and interaction of HTAP and race-specific seedling resistance to *Puccinia striiformis*. Phytopathology 85:573-578.

Wheat cultivars Druchamp and Stephens have durable, non-race-specific, high-temperature, adult-plant (HTAP) resistance to *Puccinia striiformis*, as well as race-specific resistance expressed in both seedling and adult plants. Cultivar Paha has only race-specific seedling resistance. Cultivar Michigan Amber is susceptible to all known North American races of *P. striiformis*. To determine the gene number and heritability of HTAP resistance and the relationship of HTAP resistance to seedling resistance, diallel, reciprocal crosses were made among Druchamp, Stephens, and Paha or Michigan Amber in a greenhouse. Parents and F_1, F_2, B_1, B_2, F_3, and F_5 progeny from all crosses were tested at Pullman, WA, in a plot inoculated with race CDL-25. The same progeny from the crosses of Druchamp and Stephens with Paha were tested at Pullman in a plot inoculated with race CDL-29, and progeny from the crosses of Druchamp with Stephens were tested with naturally occurring race CDL-25 at Mount Vernon, WA. Means and variances of area under disease progress curve based on disease intensity data were used to estimate the number of genes and the heritability of resistance in Stephens and Druchamp. IT data also were analyzed to determine the number of genes in the cultivars. Two to three HTAP resistance genes were estimated for both Druchamp and Stephens. The HTAP resistance genes in Druchamp and Stephens were different from one another and different from the race-specific resistance genes in Druchamp, Stephens, and Paha. The HTAP resistances showed no specificity for races CDL-25 and CDL-29. Estimated broad- and narrow-sense heritabilities of the HTAP resistance were high. Broad-sense heritability was 96.8% for Druchamp and 95.3% for Stephens. Narrow-sense heritability was 86.1–89.1% for Druchamp and 95.4% for Stephens. When HTAP resistance and seedling resistance were combined, estimated broad-sense heritabilities remained high (85.2–98.7%), but estimated narrow-sense heritabilities became low and variable (19.8–60.2) depending on the combination of genes. HTAP genes in both Druchamp and Stephens provided high adult-plant resistance over a range of environmental conditions. Combining these genes for resistance into new commercial cultivars should provide greater stripe rust resistance.

Additional keywords: general resistance, quantitative genetics, *Triticum aestivum*, yellow rust.

In the Pacific Northwest of the United States, high-temperature, adult-plant (HTAP) resistance, which is durable and race non-specific, has been the most important method of controlling stripe rust of wheat (*Triticum aestivum* L.) caused by *Puccinia striiformis* Westend. f. sp. *tritici* (18). We (5–8) have shown that the wheat cultivars Stephens and Druchamp have both race-specific seedling resistance and non-race-specific HTAP resistance to stripe rust, and we (8) have determined the mode of inheritance and genetic components of HTAP resistance in the two cultivars and the cytoplasmic effects on HTAP resistance. The results of our study (8) indicated that the HTAP resistance in Stephens and Druchamp may be controlled by different genes.

There have been few genetic studies on the number of genes controlling HTAP resistance. Based on quantitative and qualitative analyses, Milus and Line (22) found that HTAP resistance in the winter wheat cvs. Gaines, Nugaines, and Luke was controlled by two genes, that Gaines and Nugaines have one gene in common, and that the genes in Luke are different from the genes in Gaines and Nugaines.

The objectives of this study are to determine the number of genes controlling HTAP resistance in Stephens and Druchamp and heritability of the genes and to determine the relationships between the HTAP genes and genes for race-specific, seedling resistance in the cultivars.

MATERIALS AND METHODS

To study the number of genes and the heritability of HTAP resistance in Stephens and Druchamp and their relationships to seedling resistance, F_3 and F_5 generations for each cross, obtained by single-seed descent, were planted in the same plots as the parents, F_1, F_2, and backcross generations we previously described (8). For F_3 and F_5 generations, the same 100 families for each cross were replicated three times (three blocks). For each line in each block, 10 seeds were space-planted in 1.5-m-long rows to enable recording of disease data on each plant. Plots were inoculated with *P. striiformis*; data on infection types (ITs) and rust intensity (percentage of leaf area infected) were recorded; and the intensity data were transferred into area under the disease progress curve (AUDPC) as we described in the previous study (8). Generation means and variances of AUDPC were calculated for F_3 and F_5 generations, as well as for the parental, F_1, F_2, and backcross generations of each cross, and family means and variances were calculated for F_3 and F_5 generations.

Means and variances of generations and families were used to estimate the number of genes for resistance. Ten formulae for estimating gene number (GNF) are listed in Table 1. Since each formula has its restrictions or assumptions, some formulae produce numbers that are not applicable for certain crosses. Those numbers were not used. Formula GNF 1, from Wright (30), utilizes variances of parents, F_1, and F_2 generations. Formula GNF 2, also from Wright (30), utilizes variances of F_2 and backcross populations. Formula GNF 3, from Cockerham and Zeng et al (9,31), uses the combination of variances of F_1, F_2, B_1, B_2, and

parental populations to obtain a more accurate estimate of the genetic variance and eliminate phenotypic variance of the parental population from the difference between parental means. These three formulae (GNF 1–3) use the difference between the two parental means.

For the resistant-resistant crosses, the formulae were modified according to Castle's approach (3,4) to establish formulae GNF 4–6. The modification is the substitution of the difference between phenotypic means of parental populations with the genetic range of the F_2 population. Formula GNF 7, from Bjarko and Line's (2) modification of Wright's formula (30), utilizes the F_3 population variance to estimate the genetic variance. Formula GNF 8, from Mather and Jinks (21), uses genetic variance and variance of the family variances of the F_3 population to estimate the number of genes involved. Formula GNF 9 was adapted from Jinks and Towey (11) and uses the proportion of F_3 heterozygous families to estimate the gene number directly. Formula GNF 10, also from Bjarko and Line's (2) modification of Wright's formula (30), utilizes F_5 populations. All of the formulae assume that the different genes have an equal effect, which is an oversimplification.

Based on ITs in Line and Qayoum's 0–9 scale (20), individual plants were classified as resistant (IT 0–3), intermediate (IT 5), or susceptible (IT 8). Individual families of three replications were pooled and classified as homozygous resistant (R), segregating (SEG), and homozygous susceptible (S) groups. For F_3 generations, the ratios of R:SEG:S were used to determine the number of segregating genes. For the F_5 generation, the number of R and S families were pooled to get the number of homozygous (HOM) families, and the ratios of R&S:SEG families were used to determine the number of genes segregating according to the modified formulae of Jinks and Towey (11):

$$P_{(Het)} = 1 - [(2^n - 1)/(2^n)]^m$$

and

$$P_{(Hom)} = 1 - P_{(Het)},$$

where $P_{(Het)}$ = the proportion of heterozygous families; $P_{(Hom)}$ = the proportion of homozygous resistant and susceptible families; n = number of selfing generations ($n = 4$ for F_5); and m = the number of segregating loci. Chi-square tests (26) were used to determine the goodness of fit. Generally, segregation ratios with a chi-square probability greater than 0.05 were considered acceptable.

Phenotypic variances of parents, F_1, F_2, B_1, B_2, F_3, and F_5 based on AUDPC data were used to estimate heritability of resistance using eight formulae (HF) as shown in Table 2. Formulae HF 1–3 were used to estimate broad-sense heritability. Formula HF 1 uses parental, F_1, and F_2 generations (3,27). Formulae HF 2 and 3 are adapted from Elias et al (10) and use parental, F_1, and F_3 or F_5 generations. Formula HF 4, which uses the data of F_2 and backcross generations, was used to estimate narrow-sense heritability (3,24,29).

RESULTS

Estimation of gene number. AUDPC generation means and standard deviations of parents, F_1, F_2, and backcross generations, presented in our previous paper (8), and of F_3 and F_5 generations (Table 3) were used to quantitatively estimate the number of genes for each cross (Table 4). IT data were used to qualitatively estimate the number of genes. Generally, the results from the two recordings of IT data at Pullman and the three recordings at Mount Vernon agree with each other; therefore, only the results from one recording are presented (Tables 5 and 6).

For analyses of HTAP resistance from a single parent, the number of genes controlling HTAP resistance was estimated for crosses of Stephens and Druchamp with Michigan Amber tested with race CDL-25 (virulent on Michigan Amber at all stages and on seedlings of Druchamp and Stephens). The estimated number of genes based on the AUDPC data (Table 4) was 1.4–2.0 for the Stephens/Michigan Amber cross, 1.5–2.6 for the Druchamp/Michigan cross, and 1.5–3.1 for the Michigan Amber/Druchamp cross.

F_1 plants from the crosses of Druchamp and Stephens with Michigan Amber had the same IT as Michigan Amber (Table 5), indicating that the overall effects of the HTAP resistance in Druchamp and Stephens was recessive. Models for one, two, or three genes fit the F_2 data, depending on how the ITs were grouped. When IT 5 was combined with IT 0–3, a single recessive gene was indicated for both Druchamp and Stephens. When IT 5 was analyzed separately, two genes were detected for Stephens, and three genes were detected for Druchamp. Backcross data (B_1 and B_2) support the two- and three-gene models. The results of both analyses indicate that two to three genes control HTAP resistance in Stephens and Druchamp.

For analyses of HTAP resistance from both parents, the reciprocal crosses of Stephens with Druchamp tested with race CDL-25 at Pullman and Mount Vernon were used to measure the combined HTAP resistances from both Stephens and Druchamp and to eliminate any effect of race-specific seedling resistance. By analyses of AUDPC data of earlier generations (F_1, F_2, B_1, and B_2) (Table 4 of the previous paper [8]), four to nine genes

TABLE 1. Formulae for estimating number of genes for resistance to *Puccinia striiformis* in wheat cultivars

GNF[a]	Formula[b]
Formulae using P_1, P_2, F_1, F_2, B_1, and B_2 data	
GNF 1	$n = (P_1 - P_2)^2[1.5 - 2h(1-h)]/8(V_{F2} - V_E)$, where n = number of genes; $h = (F_1 - P_1)/(P_2 - P_1)$, and $V_E = 0.25(V_{P1} + V_{P2} + 2V_{F1})$
GNF 2	$n = (P_1 - P_2)^2/8(2V_{F2} - V_{B1} - V_{B2})$, where n = number of genes
GNF 3	$m_p = [(P_h - P_l)^2 - (V_h + V_l)/n]/8V_s$, where m_p = number of genes, n = number of $(P_1 + P_2)$, and $V_s = 0.2(4V_{F2} + V_{B1} + V_{B2}) - 0.4(V_{P1} + V_{P2} + V_{F1})$
GNF 4	$n = (F_{2max} - F_{2min})^2[1.5 - 2h(1-h)]/8V_s$, where n = number of genes, and $V_s = [V_{F2} - (V_{P1} + V_{P2} + 2V_{F1})/4]$
GNF 5	$n = (F_{2max} - F_{2min})^2/8(2V_{F2} - V_{B1} - V_{B2})$, where n = number of genes
GNF 6	$m_p = [(F_{2h} - F_{2l})^2 - (V_h + V_l)/n]/8V_s$, where m_p = number of genes, n = number of $(P_1 + P_2)$, and $V_s = 0.2(4V_{F2} + V_{B1} + V_{B2}) - 0.4(V_{P1} + V_{P2} + V_{F1})$
Formulae using F_3 data	
GNF 7	$n = (GR)^2/5.33[V_{F3} - (V_{P1} + V_{P2})/2]$, where n = number of genes and $(GR) = (max - min)$ of F_3 family means
GNF 8	$n = {}_HV_3^2/{}_HV(V_3)$, where n = number of genes, ${}_HV_3 = F_3$ genetic variance $= [V_{F3} - (V_{P1} + V_{P2} + V_{F1})/3]$, and ${}_HV(V_3)$ = variance of genetic variances of F_3 family
GNF 9	$P_{max} = 1 - [1 - 5/2^{n+2}]^k$, where P_{max} = maximum proportion of heterozygous families in F_3 population, $n = 3$ for F_3 generation, and k = number of gene
Formula using F_5 data	
GNF 10	$n = (GR)^2/4.27[V_{F5} - (V_{P1} + V_{P2})/2]$, where n = number of genes and $(GR) = (max - min)$ of F_5 family means

[a] GNF = gene number formula.

[b] In the formulae, P_1, P_2, F_{2max}, F_{2min}, and so on are means of respective generations or families. GNF 1–2 and 4–5 were referred to Wright (30); GNF 3 and 6 were referred to Zeng et al (31); GNF 6 also was referred to Castle (4); GNF 7 and 10 were referred to Bjarko and Line (2); GNF 8 was referred to Mather and Jinks (26); and GNF 9 was referred to Jinks and Towey (11).

were estimated based on Pullman data, and seven to nine genes were estimated based on Mount Vernon data (Table 4). Based on the F_3 generation, two to seven genes were estimated using Pullman data, and one to 10 genes were estimated using Mount Vernon data. Based on the F_5 generation, six to eight genes were estimated using Pullman data, and five to six genes were estimated using Mount Vernon data. The estimates based on F_5 generation at Mount Vernon were close for the reciprocal crosses and were the most reliable results, because at Mount Vernon rust developed fast and the intensities allowed the effects of individual genes to be separated. The estimated five to six genes were close to the total number (four to five) of genes in both Stephens and Druchamp using F_1, F_2, and backcross generations of the crosses with Michigan Amber at Pullman.

When IT data were analyzed (Tables 5 and 6), four to five genes were detected with the early generation data, three to four genes with the F_3 generation, and four to five genes with the F_5 generation. The IT data from these crosses agree with IT and AUDPC data from the analyses of crosses with Michigan Amber. The results of AUDPC and IT analyses indicate that the genes for HTAP resistance in Druchamp are different from the genes in Stephens.

For analyses of HTAP resistance from one parent and seedling resistance from Paha, crosses of Stephens and Druchamp with Paha tested with race CDL-25 were studied. Using AUDPC data (Table 4), two to five genes based on the earlier generations (F_1, F_2, and backcrosses), two to nine based on F_3, and three to five based on F_5 were estimated for the crosses of Stephens with Paha. For the crosses of Druchamp with Paha, two to six genes were estimated based on the earlier generations, two to 10 genes were estimated based on the F_3 generation, and three genes were estimated based on the F_5 generation.

When IT data were analyzed (Tables 5 and 6), three genes were detected in early and F_3 generations, and three to four genes were detected in the F_5 generation for the crosses of Stephens with Paha; for the crosses of Druchamp with Paha, three to five genes in the early and F_3 generations, and four to five genes in the F_5 generation were detected.

For analyses of HTAP resistance and seedling resistance in the same parent, crosses of Stephens and Druchamp tested with race CDL-29 were used. In these crosses, both HTAP and seedling resistance were effective in Stephens and Druchamp. For the crosses of Stephens with Paha, two to four genes based on AUDPC analyses of F_1, F_2, and backcrosses, two to seven genes based on F_3, and three to four genes based on F_5 generations were estimated (Table 4). For the crosses of Druchamp with Paha, two to nine genes based on F_1, F_2, and backcrosses, two to eight genes based on F_3, and three genes based on F_5 generations were estimated.

Segregation of ITs based on the early generation data (Tables 5) indicate three genes for resistance in the crosses of Stephens and Druchamp with Paha tested with race CDL-29. The F_3 data also indicate three genes for all crosses but one; four genes were detected in the Paha/Druchamp cross. The F_5 data suggest five genes in the crosses of Stephens with Paha and three or six genes in the crosses of Druchamp with Paha.

Heritability. The estimated heritabilities for each cross-race

TABLE 2. Formulae used to estimate broad- and narrow-sense heritabilities of resistance in wheat cultivar crosses to *Puccinia striiformis*

HF[a]	Formula[b]
Broad-sense heritability	
HF 1	$h^2 = [V_{F2} - (V_{P1} + V_{P2} + V_{F1})/3]/V_{F2}$, where V = generation variance
HF 2	$h^2 = [V_{F3} - (V_{P1} + V_{P2} + V_{F1})/3]/V_{F3}$
HF 3	$h^2 = [V_{F5} - (V_{P1} + V_{P2} + V_{F1})/3]/V_{F5}$
Narrow-sense heritability	
HF 4	$h^2 = (2V_{F2} - V_{B1} - V_{B2})/V_{F2}$

[a] HF = heritability formula.
[b] Formula HF 1 was referred to Burnette and White (3) and Van Ginkel and Scharen (27), HF 2 and 3 were referred to Elias et al (10), and HF 4 was referred to Burnette and White (3), Nyquist (24), and Warner (29).

TABLE 3. Generation means and standard deviations of area under disease progress curve (AUDPC) for wheat cultivar crosses tested with races CDL-25 and -29 of *Puccinia striiformis* in the field at Pullman and Mount Vernon, WA

	Generation mean and standard deviation of AUDPC			
	F_3		F_5	
Crosses[a] P_1/P_2 (P_2/P_1)	P_1/P_2	P_2/P_1	P_1/P_2	P_2/P_1
Race CDL-25, Pullman				
STE/DRU (DRU/STE)	60 ± 149	17 ± 73	54 ± 142	25 ± 69
STE/Paha (Paha/STE)	51 ± 166	173 ± 311	115 ± 300	268 ± 428
DRU/Paha (Paha/DRU)	93 ± 207	132 ± 250	164 ± 270	126 ± 256
Race CDL-25, Mt. Vernon				
STE/DRU (DRU/STE)	555 ± 453	325 ± 192	676 ± 573	454 ± 328
Race CDL-29, Pullman				
STE/Paha (Paha/STE)	119 ± 256	335 ± 376	215 ± 352	440 ± 419
DRU/Paha (Paha/DRU)	129 ± 357	92 ± 198	149 ± 256	96 ± 231

[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 cross P_2/P_1. STE = cv. Stephens and DRU = cv. Druchamp.

TABLE 4. Estimated number of genes for resistance in wheat cultivar crosses based on area under disease progress curve produced by *Puccinia striiformis* race CDL-25 at Pullman and Mount Vernon, WA, and CDL-29 at Pullman using the GNF 1–10 formulae from Table 1

	Number of genes[b]									
	Based on F_1, F_2, and backcrosses						Based on F_3			Based on F_5
Cross[a] P_1/P_2 (P_2/P_1)	GNF 1	GNF 2	GNF 3	GNF 4	GNF 5	GNF 6	GNF 7	GNF 8	GNF 9	GNF 10
Race CDL-25, Pullman										
STE/MA	1.5	1.4	1.5	2.0	1.9	2.0	...[c]	...	...	...
DRU/MA (MA/DRU)	1.9 (2.1)	1.5	1.8 (1.9)	2.6 (3.1)	2.1 (2.2)	2.5 (2.7)	...	...	...	...
STE/DRU (DRU/STE)	–[d]	–	–	7.7 (–)	4.4 (–)	9.0 (–)	2.1 (–)	–	6.9 (–)	7.6 (6.1)
STE/Paha (Paha/STE)	–	–	–	– (2.5)	– (1.8)	5.1 (2.9)	9.9 (3.1)	– (2.7)	2.2 (9.5)	5.2 (2.5)
DRU/Paha (Paha/DRU)	–	–	–	6.4 (4.8)	6.6 (1.8)	4.1 (4.0)	1.6 (–)	1.5 (1.7)	7.5 (9.8)	2.6 (3.4)
Race CDL-25, Mt. Vernon										
STE/DRU (DRU/STE)	–	–	–	8.8 (6.9)	9.5	8.8 (6.9)	1.0 (4.0)	–	5.2 (9.2)	4.8 (6.1)
Race CDL-29, Pullman										
STE/Paha (Paha/STE)	2.8 (1.4)	3.4 (1.2)	2.0 (1.4)	3.3 (2.3)	4.1 (1.9)	2.4 (2.2)	4.6 (2.4)	2.3 (6.9)	4.2 (–)	3.8 (2.8)
DRU/Paha (Paha/DRU)	–	–	3.9 (3.5)	5.8 (9.0)	–	1.8 (2.7)	2.5 (1.5)	8.4 (1.2)	4.8 (6.5)	2.5 (3.1)

[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 cross P_2/P_1. STE = cv. Stephens; DRU = cv. Druchamp; and MA = cv. Michigan Amber. The reciprocal cross (MA/STE) was not available.
[b] GNF = gene number formula. A single value indicates no difference between the reciprocal crosses. When values were different for the reciprocal crosses, the value in parentheses is for the reciprocal cross in parentheses.
[c] No data.
[d] A dash means that the estimated number was abnormal and discarded because the formula was not appropriate for the data.

combination using different formulae are shown in Table 7. The results from crosses of Stephens and Druchamp with Michigan Amber indicated that broad-sense heritability of HTAP resistance in both Stephens and Druchamp was very high and close (93% for Stephens and 95% for Druchamp). The narrow-sense heritability of HTAP resistance was higher in Stephens (95%) than in Druchamp (86–89%).

When the HTAP resistances from different parents were combined (crosses of Druchamp with Stephens tested with race CDL-25), the broad-sense heritability was greater than 90%, except an estimate of 78% was obtained from HF 3 using F_3 data for the cross when Druchamp was the female parent at Mount Vernon. The narrow-sense heritability was between 65 and 85% for the reciprocal crosses at Mount Vernon.

TABLE 5. Infection types (IT) of parent and F_1 and number of plants, segregation ratios of resistant (R), intermediate (I), and susceptible (S) plants, and probability (*P*) of chi-square test for goodness of fit for F_2 and backcross wheat cultivar progeny inoculated with CDL-25 and -29 races of *Puccinia striiformis* in the fields at Pullman and Mount Vernon, WA

Cross[a] (P_1/P_2)	Note[b]	IT[c]			F_2			B_1			B_2			No. of genes
		P_1	P_2	F_1	No. of plants	Exp. ratio[d]	*P*	No. of plants	Exp. ratio[d]	*P*	No. of plants	Exp. ratio[d]	*P*	
Race CDL-25, Pullman														
STE/MA	1st	0–3	8	8	348	2:1:13	0.16	221	1:1:2	0.04	78	0:1	1.00	2
DRU/MA	2nd	0	8	8	468	9:6:49	0.86	132	7:1	0.19	112	1:7	0.89	3
MA/DRU	1st	8	0	8	521	12:4:48	0.95	97	0:1	1.00	223	7:1	1.00	3
STE/Paha	1st	0–2	0	0	440	55:5:4	0.79	134	6:1:1	0.33	57	1:1	0.43	3
Paha/STE	1st	8	0	0–5	414	45:3:16	0.62	77	7:1	0.76	133	5:2:1	0.47	3
DRU/Paha	1st	0	0	0	376	48:2:14	0.79	105	7:1	0.48	68	3:1	1.00	3
Paha/DRU	1st	0	0	0	374	46:4:14	0.95	100	7:1	1.00	160	15:1	0.87	4
STE/DRU	2nd	0–2	0	2–3	439	202:27:27	0.93	269	31:1	0.75	216	28:2:2	0.71	4
DRU/STE	2nd	0	0–2	0	415	238:9:9	0.81	268	31:1	0.51	251	30:1:1	0.13	4
Race CDL-25, Mt. Vernon														
STE/DRU	2nd	2–3	2–3	2–5	411	140:89:27	0.71	179	9:6:1	0.24	59	9:6:1	0.45	4
DRU/STE	2nd	2–3	2–3	2–5	362	153:76:27	0.72	50	9:6:1	0.93	91	7:8:1	0.60	4
Race CDL-29, Pullman														
STE/Paha	1st	0	8	3–5	512	37:3:24	0.59	141	7:1	0.29	57	1:3	0.82	3
Paha/STE	1st	8	0	5–8	536	23:4:37	0.83	85	1:7	0.49	145	7:1	0.10	3
DRU/Paha	1st	0	8	0	458	57:1:6	0.72	111	1:0	1.00	59	4:4	0.79	3
Paha/DRU	1st	8	0	0	508	57:1:6	0.51	110	3:1:4	0.53	176	1:0	1.00	3

[a] P_1 = parent 1, used as female parent; and P_2 = parent 2, used as male parent. STE = cv. Stephens; MA = cv. Michigan Amber; and DRU = cv. Druchamp.

[b] The first recording was at the stem-elongation stage (jointing) for the Mount Vernon plot (14–15 April) and at the heading to anthesis stage for the Pullman plots (5–8 June for the plot inoculated with race CDL-29, 13–14 June for the plot of crosses with Michigan Amber inoculated with race CDL-25, and 19–20 June for the plot of crosses with Paha inoculated with race CDL-25). The second recording was 1 mo after the previous one at Mount Vernon. At Pullman, the second recording was 2 wk after the first.

[c] IT were based on the 0–9 scale (19,20). IT 0–3 were considered resistant, 4–6 intermediate, and 7–8 susceptible.

[d] The expected segregation ratios are R:I:S or R:S.

TABLE 6. Expected segregation ratios, probabilities (*P*) of chi-square test for goodness of fit, and number of genes based on infection type data of F_3 and F_5 generations of wheat cultivar crosses tested with CDL-25 and -29 races of *Puccinia striiformis* in the field plots at Pullman and Mount Vernon, WA

Cross (P_1/P_2)[a]	Note[b]	F_3			F_5		
		Exp. ratio (R:SEG:S)[c]	*P*	No. of genes	Exp. ratio (R&S:SEG)[d]	*P*	No. of genes
Race CDL-25, Pullman							
STE/Paha	1st	26:37:1	0.14	3	67.0:33.0	0.46	3
Paha/STE	1st	10:52:2	0.98	3	58.6:41.4	0.21	4
DRU/Paha	1st	80:175:1	0.61	4			
Paha/DRU	1st	9:54:1	0.65	3			
STE/DRU	2nd	26:37:1	0.29	3	58.6:41.4	0.56	4
DRU/STE	2nd	26:37:1	0.76	3	51.3:48.7	0.58	5
Race CDL-25, Mt. Vernon							
STE/DRU	2nd	5:56:3	0.39	3	58.6:41.4	0.43	4
DRU/STE	2nd	80:175:1	0.43	4	51.3:48.7	0.58	5
Race CDL-29, Pullman							
STE/Paha	2nd	26:37:1	0.30	3	51.3:48.7	0.58	5
Paha/STE	2nd	3:56:5	0.99	3	51.3:48.7	0.87	5
DRU/Paha	1st	26:37:1	0.41	3	44.9:55.1	0.38	6
Paha/DRU	1st	80:175:1	0.50	4	67.0:33.0	0.46	3

[a] P_1 = parent 1, used as female parent; and P_2 = parent 2, used as male parent. STE = cv. Stephens; MA = cv. Michigan Amber; and DRU = cv. Druchamp.

[b] The first recording was at the stem-elongation stage (jointing) for the Mount Vernon plot (14–15 April) and at the heading to anthesis stage for the Pullman plots (5–8 June for the plot inoculated with race CDL-29, 13–14 June for the plot of crosses with Michigan Amber inoculated with race CDL-25, and 19–20 June for the plot of crosses with Paha inoculated with race CDL-25). The second recording was 1 mo after the previous one at Mount Vernon. At Pullman, the second recording was 2 wk after the first.

[c] The segregation ratios are of resistant (R):segregating (SEG):susceptible (S) families for F_3 generations.

[d] The segregation ratios for F_5 generations are of resistant and susceptible (R&S):segregating (SEG), based on the following equations (11): $P_{(Het)} = 1 - [(2^n - 1)/(2^n)]^m$, where $P_{(Het)}$ = proportion of heterozygote, n = generation (observed F_5 heterozygotes were actual F_4s, thus $n = 4$), m = number of genes; and $P_{(Hom)} = 1 - P_{(Het)}$.

When HTAP resistance from Stephens or Druchamp and seedling resistance from Paha were effective (crosses of Stephens and Druchamp with Paha tested with race CDL-25), the broad-sense heritability was 76–96% for the Stephens crosses and 95–98% for the Druchamp crosses. The narrow-sense heritability was not estimated because the formula was not applicable to the data. When both HTAP and seedling resistances of Stephens or Druchamp (crosses of Stephens and Druchamp with Paha tested with race CDL-29) were effective, the broad-sense heritability was 90–96% for the Stephens crosses and 91–99% for the Druchamp crosses.

DISCUSSION

Based on AUDPC data, two HTAP resistance genes were estimated in Stephens and two to three HTAP resistance genes were estimated in Druchamp. The estimated number of genes based on the crosses of Stephens and Druchamp with Michigan Amber should indicate the number of genes for HTAP resistance in each of those resistant parents, since Michigan Amber does not have HTAP resistance (8). The estimated number of genes (five to six) for the crosses of Stephens with Druchamp based on the F_5 generation at Mount Vernon supports the estimate of two to three HTAP resistance genes in Stephens and two to three HTAP resistance genes in Druchamp. The results indicate that the genes for HTAP resistance in Stephens and Druchamp are different. The number of HTAP resistance genes in Stephens and Druchamp based on crosses with Paha agree with the results from crosses of Druchamp or Stephens with Michigan Amber. Paha has one gene for resistance to race CDL-25, and Druchamp and Stephens each have one gene (*Yr3a*) for race-specific resistance to CDL-29 (data not shown).

The estimated number of three to five genes based on F_5 AUDPC dada (Table 4) for the crosses with Paha were the HTAP resistance genes plus the seedling resistance gene in Paha tested with race CDL-25 or in Druchamp or Stephens tested with race CDL-29. Therefore, the genes conferring HTAP resistance in Druchamp and Stephens are not linked to race-specific seedling resistance in Druchamp, Stephens, or Paha. The results also indicate that the seedling resistance genes from Stephens, Druchamp, or Paha do not suppress the HTAP resistance genes. In breeding for resistance, this could be important when seedling and HTAP resistances are combined in a single cultivar. Based on the results from various cross-race combinations, we can conclude that Stephens and Druchamp each have two or three genes for HTAP resistance, that the HTAP resistance genes in Stephens are different from the HTAP resistance genes in Druchamp, that the genes for HTAP resistance are different from the race-specific seedling resistance genes in each cultivar, and that HTAP resistance genes show no specificity for race CDL-25 or CDL-29.

The estimated number of genes for HTAP resistance in Stephens and Druchamp was similar to the estimated number that Milus and Line (22) reported for cvs. Nugaines and Luke. We do not have data on the relationship of HTAP resistance genes in Druchamp or Stephens to HTAP resistance genes in Nugaines and Luke. Based on pedigrees, Stephens could have a gene in common with Luke, because both cultivars have Pullman Selection 101 (CI13438) in their parentage (16,32). The results of this study and the study by Milus and Line (22) indicate that HTAP resistance is conferred by relatively few genes (two to three in each cultivar) rather than many genes as suggested by Johnson (12). On the other hand, the resistance genes could be linked and could segregate as a group or "effective factor" (21,22). In this case, the formulae would estimate the number of effective factors, and the number of individual genes would be greater. Milus and Line (22) suggested that since an effective factor consists of linked genes, the estimated number of genes must be expected to increase as generations advance, because linkage groups will continue to be broken in later generations. We did not observe obvious increases in the estimated gene number as generation advanced. Therefore, the numbers that we estimated are for genes, not for effective factors.

In general, the results from analyzing IT data agree with the results from analyzing AUDPC data. Similar analyses were used by Milus and Line (22) to determine the number of genes for HTAP resistance in Nugaines, Luke, and Gaines, but they used AUDPC data for both qualitative and quantitative analyses. In this study, we used AUDPC data for quantitative analyses and IT data for qualitative analyses. There is a high correlation between IT and disease-intensity data. The genes that control IT have a strong effect on disease intensity. When IT data were used to analyze the number of genes, usually more than one ratio fit the data, but our conclusions are based on correlations of the results from different generations and crosses, not on a specific segregation ratio.

Transgressive segregation for enhanced resistance has frequently been observed among progeny derived from crosses between cultivars (1,17,22,23,25,28). In this study, transgressive segregation would be difficult to detect because both Stephens and Druchamp are highly resistant. In the F_2 population from the crosses of Stephens with Druchamp tested at Mount Vernon, some F_2 plants had AUDPC values lower than those of either resistant parent, but the differences could not be statistically tested. In F_3 and F_5 generations, some individual plants had AUDPC values lower than that of their resistant parent(s), but none of the families had mean AUDPC values statistically lower than the values of their resistant parent(s). At Pullman, both Stephens and Druchamp only occasionally had IT 3 or lower and an intensity of 5% or lower. Therefore, transgressive segregation could not be detected.

Druchamp and Stephens have gene *Yr3a*, which is found in Cappelle Desprez (7); the three cultivars are related (7). Cappelle Desprez has been reported to have adult-plant resistance (12–14), which gene has been named *Yr16* and has been reported to be located on the chromosome 2DS (15). It is possible that a gene for adult-plant resistance in Druchamp and Stephens could be *Yr16*. However, in other studies (data not shown), crosses of Stephens and Druchamp with Cappelle Desprez tested with race CDL-25 in the field showed that Stephens and Druchamp had no common adult-plant resistance genes with Cappelle Desprez. Therefore, the HTAP resistance genes in Stephens and Druchamp must be different from *Yr16*.

Both broad- and narrow-sense heritabilities of HTAP resistances in Stephens and Druchamp were very high, indicating that the effects of their HTAP resistance genes are mostly additive. These results agree with our conclusions from studies of HTAP

TABLE 7. Estimated heritabilities (percent) of resistance in wheat cultivar crosses to stripe rust based on area under the disease progress curve of parental, F_1, F_2, B_1, B_2, F_3, and F_5 generations tested with races of *Puccinia striiformis* at Pullman and Mount Vernon, WA, using the formulae (HF) in Table 2

Cross (P_1/P_2)[a]	Broad-sense heritability[b]			Narrow-sense heritability[b]
	HF 1	HF 2	HF 3	H 4
Race CDL-24, Pullman				
STE/MA	93 (...[c])	...	...	95 (...)
DRU/MA	95	...	...	89 (86)
STE/Paha	76 (91)	76 (84)	93 (96)	NA
DRU/Paha	96 (97)	95 (97)	97	NA
STE/DRU	98 (99)	99 (98)	98 (99)	NA
Race CDL-25, Mt. Vernon				
STE/DRU	92 (93)	95 (78)	97 (93)	85 (65)
Race CDL-29, Pullman				
STE/Paha	95 (96)	90 (95)	95 (96)	NA
DRU/Paha	91 (93)	99 (95)	97	NA

[a] P_1 = parent 1 and P_2 = parent 2. STE = cv. Stephens, MA = cv. Michigan Amber, and DRU = cv. Druchamp.

[b] Values for reciprocal crosses are in parentheses; the first value indicates when P_1 was the female parent; and the second value, in parentheses, indicates when P_2 was the female parent. NA means that the formula was not applicable to the data.

[c] No data.

gene action (8) and suggest that this resistances can be introduced into other cultivars. The results of heritability studies for HTAP resistance agree with our report that only the additive component contributes significantly to the HTAP resistance in Stephens, and dominance and nonallelic gene interactions also contribute significantly to the HTAP resistance in Druchamp (8). The estimates of broad-sense heritability based on different generations were similar (Table 7), suggesting that selections can be made in the early generations. The narrow-sense heritability was not estimated for the crosses involving both HTAP and seedling resistance because of nonallelic interactions and the strong dominance effects of seedling resistance. As Nyquist (24) pointed out, narrow-sense heritability has no useful meaning per se in inbred populations, except in the fully inbred population in the absence of additive-additive types of epistasis. In the cross of Stephens with Michigan Amber, the additive-additive interaction was absent. In the crosses of Druchamp with Michigan Amber, the additive-additive interaction was absent when Michigan Amber was the female parent (8). Therefore, the estimates of narrow-sense heritability should be useful for breeding for HTAP resistance.

Since HTAP resistance in Stephens and Druchamp is controlled by different genes, their resistances could be combined to develop cultivars with HTAP resistance that have a broader genetic basis and a high degree of resistance, which may be more durable. The club wheat cultivars currently grown in the Pacific Northwest do not have HTAP resistance (18). A worthy goal would be to incorporate HTAP resistance genes into club wheats. The results on gene number and inheritability of HTAP resistance, gene interactions of HTAP and seedling resistance, and inheritance of HTAP resistance in the Paha background based on this study and our previous study (8) show that incorporating HTAP resistance into club wheats should be possible.

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