Inheritance of Resistance to *Pseudocercosporella herpotrichoides* in Three Cultivars of Winter Wheat

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ABSTRACT

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Inheritance of foot rot resistance was studied in parental, F_1, F_2, and backcross populations of all possible crosses between the cultivars Daws (susceptible), Cappelle-Desprez (resistant), and VPM-1 (highly resistant) by using epidermal cell responses to determine the percent successful penetrations in the first-leaf sheath. One semidominant gene for resistance with a narrow-sense heritability (Hn) = 0.34 segregated in the Cappelle × Daws cross; one dominant gene for resistance with Hn = 0.35 segregated in the VPM-1 × Daws cross; and two genes for resistance exhibiting overdominance with Hn = 0.77 segregated for resistance in the VPM-1 × Cappelle cross. Based on quantitative estimates, VPM-1 contains one gene for hypersensitivity not found in Cappelle-Desprez. Maternal effects were not evident in any of the crosses. The gene action of the resistance genes in the Cappelle × Daws and VPM-1 × Daws crosses fit an additive-dominance model, while the VPM-1 × Cappelle cross was best fit by a model that included a digenic interaction. The rating system that resulted in the highest heritabilities was based on penetrations stopped by papillae or within epidermal cells in both hypersensitive and nonhypersensitive cells. Ratings derived from these epidermal cell responses are correlated with field resistance of parents, genetically associated with resistance in segregating progeny, and may be useful for screening potential cultivars.

Strawbreaker foot rot (eyespot), incited by *Pseudocercosporella herpotrichoides* (Fron) Deighton, is a widespread, chronic disease of winter wheat (*Triticum aestivum* L.) in the Pacific Northwest region of the United States and Europe (4). This disease can cause severe lodging and reductions in yield of wheat: Even resistant cultivars exhibit yield reductions when disease is severe (23). The development of cultivars with more resistance to *P. herpotrichoides* is an important goal of wheat cultivar improvement in Washington State, but selection of resistant cultivars is slow because screening for resistance is tedious and not always reliable (21). Currently, farmers in the Pacific Northwest use applications of systemic fungicides to control foot rot, but the use of resistant cultivars offers the most economical and reliable method of control.

Plant breeders have tried to improve cultivar resistance without sacrificing yield and quality, but have had only limited success. The French cultivar Cappelle-Desprez contains resistance to *P. herpotrichoides* that has remained durable for about 30 yr despite widespread exploitation (11). Investigations have also shown that an alien grass species, *Aegilops ventricosa* Tausch (syn. *Triticum ventricosum* Ces.), is highly resistant to *P. herpotrichoides* (12,28). From amphiploids obtained by Simonet (27), Maia (19) selected the hexaploid line VPM-1, which is more resistant than Cappelle-Desprez, but not as resistant as *Aegilops ventricosa* (5). Most resistant cultivars are not adapted to the Pacific Northwest and have reduced yields and grain quality in comparison to commercial cultivars. In autumn 1988, Hyak and Madsen, two winter wheat cultivars developed for the Pacific Northwest region of the United States by R. E. Allan, were released (14). These two cultivars contain the dominant resistance gene found on chromosome 7D in VPM-1 and have suitable agronomic characteristics (14). In the future, it will be important to incorporate other resistance genes into wheat cultivars while maintaining or improving grain yield and quality.

Several studies have focused on the inheritance of resistance to *P. herpotrichoides* in winter wheat. Some indicate that the inheritance of resistance is complex (1,13,16,22), while others show resistance to be simply inherited (7,9,11,15,26,32). A cytological investigation by Law et al (16) involving Chinese Spring, Cappelle-Desprez, and Mara showed that chromosomes 1A, 2B, 5D, and 7A can confer resistance to foot rot, although only 7A was associated with resistance in all of the investigations. Resistance factors in the cultivar Roazon, whose intermediate level of resistance has been acquired from VPM-1, have been assigned to chromosomes 7A, 2B, 5D, and 7D, the main effect being associated with the latter chromosome (13). The difference in the level of resistance at the seedling stage between Roazon and Cappelle is due exclusively to chromosome 7D (13). Using *Triticum turgidum* (L.) Thell. as a bridge species, Doussinault et al (7) transferred a major dominant gene for resistance from *Aegilops ventricosa* to *Triticum aestivum*. Gale et al (11) showed that VPM-1 possesses a major dominant gene for resistance on chromosome 7D using an alpha amylase isozyme marker for disease resistance. Doussinault et al (9) found one dominant gene controlling resistance in VPM-1 by using mycelial types to distinguish resistant and susceptible plants.

A quick, reliable seedling test based on epidermal responses was used to assay individual wheat plants for resistance to *P. herpotrichoides* and shown to correlate with field resistance of adult plants (24,30). In this study we used the seedling test to establish the best combination of epidermal responses for screening populations segregating for foot rot resistance, to determine the number of genes segregating for resistance in Cappelle-Desprez and VPM-1, to estimate the heritability and degree of dominance of resistance genes, and to determine the gene action associated with the resistance genes. A preliminary report has been published (29).

MATERIALS AND METHODS

Parents and progeny. On 3 October 1985, seed of Daws (CI 17419; susceptible), Cappelle-Desprez (Cappelle; PI 262223; resistant), and VPM-1124-R25-1 (VPM-1; highly resistant) was sown in a crossing block at the Palouse Conservation Field Station, Pullman, WA. In June 1986, reciprocal crosses were made in all combinations between Daws, Cappelle, and VPM-1. Parental and F_1 seed was harvested in August 1986 and planted

in the greenhouse to produce F_2 seed and make backcrosses. For backcrosses, the F_1 plants (male donor) were crossed with both of the respective parents (females).

Seedling test. Seed of the parental (P), F_1, F_2, and backcross (BC) populations was placed on moistened paper towels, cold-shocked in the refrigerator at 5 C for 3 days, and then pregerminated in the laboratory. The pregerminated seed (16/pot) was planted 1 cm deep in a potting mix (55% peat, 35% pumice, and 10% sand, w/w/w) in 15-cm-diameter plastic pots and arranged in a randomized complete block design with subsampling as previously described (24,29). Initially, the P, F_1, F_2, and BC populations for all crosses included 62–64, 76–91, 555–571, and 48–72 individuals, respectively. However, population sizes were reduced because seedlings not at the one- to two-leaf stage at inoculation or considered nonratable were discarded. The experiment was left in the greenhouse until the seedlings had reached the one- to two-leaf stage and then moved to a growth chamber at 10 C (13 C with light) with diurnal lighting (10 hr of light) of approximately 150 $\mu E/cm^2$ supplied by fluorescent light tubes (Sylvania GRO-VHO-WS and Westinghouse CW-VHO-EW [1:2 mixture]).

A virulent isolate (PH 85-9-13) of *P. herpotrichoides* originally isolated from mature wheat straw with symptoms of foot rot and maintained as a mycelial culture on Difco potato-dextrose agar, was used to produce inoculum. Conidial inoculum was produced by growing the fungus on autoclaved oat kernels and then incubating the kernels outdoors on fiberglass screen through the autumn and winter (October–March) (2,3). Conidial suspensions were prepared by washing the kernels and adjusting the concentration to 1×10^6 conidia per milliliter with a hemacytometer. Seedlings at the two-leaf stage (second leaf >1 cm) were sprayed with the suspension at the rate of 5 ml per pot. Immediately after inoculation, the pots were individually covered with a plastic bag for 24 hr as well as being placed inside a plastic tent, where they remained for the duration of the experiment. The temperature and relative humidity in the plastic tent were recorded with a hygrothermograph; relative humidity fluctuated between 85 and 100%.

Four weeks after inoculation, the first-leaf sheaths were removed and fixed in glacial acetic acid and 50% ethanol (1:17, v/v) for at least 24 hr. The sheaths were then placed in tubes containing 0.01% (w/v) trypan blue in lactophenol, heated in a water bath at 85 C for 10 min to stain and clear the specimens, mounted in lactophenol on glass slides, rubbed gently to remove mycelial mats, and observed with a microscope (24).

Disease ratings. First-leaf sheaths were considered ratable only if 20 or more infection sites (the area under a circular mycelial mat with multiple penetration attempts) were present, to ensure adequate disease development. Usually 50 infection sites were evaluated on each ratable sheath. Five rating systems based on epidermal cell responses were used to determine the percent successful penetrations (29). The epidermal cell responses used in the rating systems included attempted penetrations stopped by papillae or within epidermal cells in both hypersensitive and nonhypersensitive cells. These responses were combined to establish the following rating systems: attempted penetrations stopped by papillae or within epidermal cells in both hypersensitive and nonhypersensitive cells, attempted penetrations stopped by papillae in both hypersensitive and nonhypersensitive cells, attempted penetrations stopped by papillae or within epidermal cells in nonhypersensitive cells, attempted penetrations stopped by papillae in nonhypersensitive cells, and attempted penetrations stopped by papillae or within epidermal cells in hypersensitive cells (hypersensitive rating).

TABLE 1. Formulas used to estimate the number of genes (n) segregating for resistance to *Pseudocercosporella herpotrichoides* in the F_2 and backcross generations of winter wheat

Population tested	Formula used[a]
Backcross to the susceptible parent (BC_s)	$n = \dfrac{(\overline{P_s} - \overline{P_r})^2}{4[V_{bc} - ((V_{ps} + V_{F1})/2)]}$
F_2	$n = \dfrac{(\overline{P_s} - \overline{P_r})^2\,[1.5 - 2h(1-h)]}{8[V_{F2} - (V_{ps} + V_{pr} + 2V_{F1})/4]}$
	$h = (\overline{F_1} - \overline{P_r})/(\overline{P_s} - \overline{P_r})$

[a] $\overline{P_s}$ = the mean of the susceptible parent, $\overline{P_r}$ = the mean of the resistant parent, V_{bc} = variance of the backcross generation, V_{ps} = variance of the susceptible parent, V_{F1} = variance of the F_1 generation, V_{F2} = variance of the F_2 generation, V_{pr} = variance of the resistant parent, $\overline{F_1}$ = mean of the F_1 generation, and n = estimated number of segregating genes (32).

Genetic analysis. A Mendelian analysis was conducted when the parental and segregating populations could be separated into discrete groups. When the distribution of populations was continuous, quantitative estimates of the number of genes segregating for foot rot resistance were obtained using Wright's formulas (Table 1) (32). These formulas estimate the gene number by dividing the square of the genotypic range by the genotypic variance. The genotypic range was estimated from the difference of the parental means, which provides a conservative estimate of the number of genes segregating for resistance. The genotypic variance was estimated by subtracting the environmental variance from the phenotypic variance of the segregating population (either F_2 or backcross). The environmental variance was estimated from the parental and F_1 populations ($V_E = (V_{PS} + V_{PR} + 2V_{F1})/4$ for F_2 and $V_E = (V_{PS} + V_{F1})/2$ for backcross). Broad sense heritability values were calculated from a formula ($H_b = (V_{F2} - V_E)/V_{F2}$) that uses indirect estimates of environmental variation (18), although the environmental variance was estimated with the formula from Wright ($V_E = (V_{P1} + V_{P2} + 2V_{F1})/4$) (32). Narrow sense heritability values were estimated by using a backcross method ($H_n = [2V_{F2} - (V_{BC1} + V_{BC2})]/V_{F2}$) (30). A method described by Mather and Jinks (20) was used to determine the degree of dominance for each cross and was calculated as the deviation of the F_1 from the midparent (midpoint between two homozygous parents), divided by the departure of the more resistant parent from the midparent. The joint scaling tests described by Mather and Jinks (20) were used to determine the gene action of foot rot resistance in Cappelle and VPM-1. The joint scaling tests estimated the midparent, genetic components, and digenic interaction components of a cross and used these estimates to fit the data to genetic models. The parental, F_1, F_2, and backcross means and variances of each class were used in the test to determine the genetic components involved in the model tested. A cross for which parental, F_1, F_2, and backcross data are available, provides six parameters (6 df), allowing a genetic model with up to five components (5 df) to be tested. A cross with six parameters allows for testing a genetic model that includes the midparent value, the additive and dominance genetic components, and up to two interaction components. The additive (d) and dominance (h) genetic components were estimated by these tests. The additive × additive (i), additive × dominance (j), and dominance × dominance (l) interaction components were also estimated. The genetic components measured were an estimate of the net effect of all the loci at which the parents differ for the measured characteristic. Because two parents in a cross may differ at several loci, and dominance and epistasis within and among these loci may differ, genetic components were redefined as the net directional effects of all relevant loci. These net effects are symbolized as [d], [h], [i], [j], and [l]. In the joint scaling tests, the midparent value (m) is an estimate of the mean value for all homozygous individuals in the parental and segregating generations and not the same as the midparent value described in determining the degree of dominance. The data from each rating system for each cross was tested against the following genetic models: m[d][h], m[d][h][i], m[d][h][j], m[d][h][l], m[d][h][i][j], m[d][h][i][l], and m[d][h][j][l]. The simplest model that fit the data was accepted based on a chi-square test with $P \geq 0.05$, even though a more complex model may have had a lower chi-square value and/or a higher probability (20).

Components within models that fit the data were evaluated for significance with Z values.

RESULTS

Mendelian analysis. There were no significant differences between populations of the reciprocal crosses in the F_1 and F_2 generations, providing no evidence of cytoplasmic inheritance; therefore, reciprocal crosses were combined for analyses. In the Cappelle × Daws cross, the F_2 progeny segregated in a 3:1 ratio and the backcross to Daws segregated in a 1:1 ratio, suggesting one gene for resistance in Cappelle (Table 2). All disease ratings, except for the rating based on penetrations stopped by papillae in nonhypersensitive cells only, supported a one gene hypothesis. In the VPM-1 × Daws cross, the F_2 progeny segregated in a 3:1 ratio and the backcross to Daws segregated in a 1:1 ratio, suggesting one gene for resistance is present in VPM-1 (Table 2). All disease ratings, except the rating system based on penetrations stopped by papillae or within epidermal cells in both hypersensitive and nonhypersensitive cells supported a one gene hypothesis. When the VPM-1 × Cappelle cross was analyzed with the two rating systems based on penetration attempts stopped by papillae or within epidermal cells in both hypersensitive and nonhypersensitive or nonhypersensitive cells only, there was a natural break at 71–72% successful penetrations, which was also the value used to separate progeny of the Cappelle × Daws and VPM-1 × Daws crosses into resistant and susceptible classes. Based on this value, the F_2 progeny of the VPM-1 × Cappelle cross segregated in a 15:1 ratio, suggesting two genes for resistance were segregating in this cross (Table 2). However, when the VPM-1 × Cappelle cross was analyzed with the two rating systems based solely on penetrations stopped by papillae in hypersensitive and nonhypersensitive or only nonhypersensitive cells, the distribution of the F_2 progeny was continuous and Mendelian analysis was not possible. With all four rating systems, the progeny of both backcrosses (VPM-1/Cappelle-F_1 × VPM-1 and VPM-1/Cappelle-F_1 × Cappelle) were resistant. Separating the progeny from the VPM-1 × Cappelle crosses into classes other than resistant and susceptible was not possible due to variability in disease ratings.

Quantitative analysis. Quantitative estimates of the numbers of genes segregating for foot rot resistance (excluding the rating system based only on hypersensitivity) ranged from one to three genes for Cappelle and one to two genes for VPM-1 (Table 3). Quantitative estimates based on the hypersensitive response alone estimated 0.1–0.3 genes in Cappelle, and 1.1–1.3 genes in VPM-1, suggesting that VPM-1 has a gene for hypersensitivity not found in Cappelle.

Heritability and dominance. All four rating systems (excluding the rating based solely on hypersensitivity) gave fairly uniform results and therefore were considered together in establishing heritability and dominance. On average, the broad and narrow sense heritability estimates for the VPM-1 × Daws cross were larger than those for the Cappelle × Daws cross, but the VPM-1 × Cappelle cross had the largest estimates of narrow sense heritability (Table 4). Heritability estimates based on the rating system using penetrations stopped by papillae or within epidermal cells in both hypersensitive and nonhypersensitive cells was greater than or equal to the heritabilities for the other rating systems in all three crosses. For the rating based only on hypersensitivity, the broad and narrow sense heritability estimates were 0.44 and 0.13 for the Cappelle × Daws cross and 0.23 and 0.13 for the VPM-1 × Daws cross, respectively. In the VPM-1 × Cappelle cross the narrow sense heritability estimate for the hypersensitive rating was 0.69.

Resistance (excluding the hypersensitive rating) was

TABLE 2. Segregation of progeny resistant and susceptible to *Pseudocercosporella herpotrichoides* from crosses between Daws, Cappelle-Desprez, and VPM-1

Generation[a]	No. rated	Expected ratio (R:S)[b]	Rating system[c]			
			HSR + NHSR		NHSR	
			PAP + CELL	PAP	PAP + CELL	PAP
Cappelle × Daws						
Cappelle	47	1:0	47:0[d]	47:0	47:0	47:0
Daws	54	0:1	0:54	0:54	0:54	0:54
F1	69	1:0	61:8	60:9	57:12	57:12
F2	433	3:1	338:95	311:122	310:123	288:145
			(2.16)	(2.33)	(2.67)	(16.66*)
Cappelle × F1	31	1:0	29:2	28:3	27:4	26:5
Daws × F1	27	1:1	16:11	14:13	13:14	14:13
			(0.92)	(0.04)	(0.04)	(0.04)
VPM-1 × Daws						
VPM-1	51	1:0	51:0	51:0	51:0	51:0
Daws	54	0:1	0:54	0:54	0:54	0:54
F1	60	1:0	60:0	59:1	60:0	58:2
F2	411	3:1	335:76	308:103	322:89	298:113
			(9.28*)	(0.00)	(2.45)	(1.36)
VPM-1 × F1	30	1:0	30:0	28:2	30:0	25:5
Daws × F1	38	1:1	24:14	20:18	22:16	20:18
			(2.64)	(0.11)	(0.95)	(0.06)
VPM-1 × Cappelle						
VPM-1	51	1:0	51:0	...[e]	51:0	...
Cappelle	47	1:0	47:0	...	47:0	...
F1	55	1:0	55:0	...	55:0	...
F2	375	15:1	358:17	...	351:24	...
			(1.87)	...	(0.01)	...
VPM-1 × F1	23	1:0	23:0	...	23:0	...
Cappelle × F1	19	1:0	19:0	...	19:0	...

[a]Daws (susceptible), Cappelle (resistant), and VPM-1 (highly resistant).
[b]R = resistant; S = susceptible.
[c]HSR = hypersensitive cell, NHSR = nonhypersensitive cell, PAP = penetration stopped by papilla, and CELL = penetration stopped within an epidermal cell.
[d]Figures represent ratio of resistant:susceptible progeny; figures in parentheses are chi-square values for ratio above; * = chi-square value with $P < 0.05$.
[e]Mendelian analysis not possible due to continuous distribution of progeny.

semidominant in the Cappelle × Daws cross, nearly dominant in the VPM-1 × Daws cross, and overdominant in the VPM-1 × Cappelle cross (Table 5). With the rating based solely on hypersensitivity, the degree of dominance for resistance was 0 for Cappelle, 1.00 for VPM-1, and 0.34 for the VPM-1 × Cappelle cross.

Gene action. For the Cappelle × Daws cross, the data predominantly fit the additive-dominance model (m[d][h]) with both genetic components contributing significantly to the model (Table 6). However, the additive × additive interaction component ([i]) contributed significantly to the model with two of the ratings for the Cappelle × Daws cross. With the ratings based only on the hypersensitive response, the Cappelle × Daws cross fit a model containing significant additive ([d]), dominance ([h]), and additive × additive components ([i]).

For the VPM-1 × Daws cross, the data predominantly fit the additive-dominance model with both genetic components contributing significantly to the model. With the rating system based on attempted penetrations stopped by papillae or within epidermal cells in both hypersensitive and nonhypersensitive cells; however, there was also a significant dominance × dominance interaction ([l]). With the hypersensitive rating alone, this cross fit the additive-dominance model with both components contributing significantly to the model.

In the VPM-1 × Cappelle cross, the two rating systems based only on papilla formation fit a model that included significant additive, dominance, and additive × additive components. The two rating systems based on penetrations stopped by papillae or within epidermal cells, fit a model that included significant additive, dominance, additive × additive, and dominance × dominance components. With the hypersensitive rating, the data fit the additive-dominance model, but only the dominance component contributed significantly to the model.

DISCUSSION

Results of both Mendelian and quantitative analyses indicate that there is one semidominant gene for resistance in Cappelle, one dominant gene for resistance in VPM-1, and two genes for resistance exhibiting overdominance in the VPM-1 × Cappelle cross. Finding one semidominant gene for resistance in Cappelle is consistent with work by Law et al (16), who showed that only chromosome 7A conferred resistance in all experiments, and that dominance was towards resistance. The presence of one dominant gene for resistance in VPM-1 is consistent with earlier reports indicating one dominant gene on chromosome 7D (9,13,15,26,31). When VPM-1 and Cappelle were crossed, two genes were found to segregate in a 15:1 ratio for the two rating systems based on penetrations stopped by papillae and within epidermal cells. This two gene hypothesis is consistent with cytological studies (13,16) that show the primary resistance genes in VPM-1 and Cappelle are found on different chromosomes. Although our data indicate that two different genes are associated with resistance in VPM-1 and Cappelle, we can not rule out the possibility that these so-called genes are actually linkage groups or large pieces of DNA that may involve many genes at the molecular level, or that the susceptible parent may have contributed to resistance. Also, since these assays were done at the seedling stage, we can not eliminate the possibility that other genes might be involved in resistance at a later stage of plant development.

In the Cappelle × Daws cross, penetrations stopped by hypersensitive cells seem to be controlled by a different gene(s) than penetrations stopped by papillae or within epidermal cells. Evidence to support this hypothesis comes from estimates of the degree of dominance for resistance (0 for the hypersensitive rating and 0.52 for the other ratings) and quantitative estimates of the number of genes segregating for resistance (0.1–0.3 for the hypersensitive rating and 1.4–3.0 for the other ratings). Also, the distribution of the data for the progeny when the ratings were based only on hypersensitivity was continuous, while the distributions for the other rating systems were bimodal. Based on quantitative estimates of the number of genes segregating for resistance in both the Cappelle × Daws and VPM-1 × Daws crosses, VPM-1 was found to contain one gene for resistance not present in Cappelle. Other investigations have shown that VPM-1 possesses a higher percentage of hypersensitive responses to penetration attempts by *P. herpotrichoides* than Cappelle (24,29), but a genetic basis for the difference has never been established.

Because no significant differences between reciprocal populations were found in the crosses among Daws, Cappelle, and VPM-1, there was no evidence of cytoplasmic inheritance. In crosses between resistant and susceptible cultivars, Roberts (25) found

TABLE 3. Estimates of the number of genes segregating for resistance to *Pseudocercosporella herpotrichoides* in winter wheat measured by epidermal responses to attempted penetration of the first-leaf sheath

		Rating system[b]				
		HSR + NHSR		NHSR		HSR
Cross[a]	Population tested	PAP + CELL	PAP	PAP + CELL	PAP	PAP + CELL
Cappelle	BC_s[c]	1.6[d]	1.6	1.4	1.6	0.1
× Daws	F_2	2.0	2.2	3.0	2.3	0.3
VPM-1	BC_s	1.4	1.2	1.2	1.2	1.3
× Daws	F_2	1.5	1.3	1.8	1.4	1.1

[a]Resistant parent is listed first.
[b]HSR = hypersensitive cell, NHSR = nonhypersensitive cell, PAP = penetration stopped by papilla, and CELL = penetration stopped within an epidermal cell.
[c]BC_s = backcross to susceptible parent (Daws).
[d]Formulas to estimate gene number came from: Wright, pp. 381-403 (33); also see Table 1.

TABLE 4. Estimates of heritability for resistance to *Pseudocercosporella herpotrichoides* as measured by epidermal responses in the first-leaf sheath

		Rating system[a]				
		HSR + NHSR		NHSR		
Cross	Heritability	PAP + CELL	PAP	PAP + CELL	PAP	Avg.
Cappelle	Broad[b]	0.48	0.45	0.33	0.40	0.42
× Daws	Narrow	0.43	0.34	0.30	0.31	0.34
VPM-1	Broad	0.76	0.65	0.59	0.61	0.65
× Daws	Narrow	0.66	0.51	...	0.23	0.47
VPM-1	Broad	...[c]	...	...	...	...
× Cappelle	Narrow	0.78	0.78	0.75	0.78	0.77

[a]HSR = hypersensitive cell, NHSR = nonhypersensitive cell, PAP = penetration stopped by papilla, and CELL = penetration stopped within an epidermal cell.
[b]Broad = broad sense heritability estimate = $(V_{F2} - V_E)/V_{F2}$, where $V_E = (V_{P1} + V_{P2} + 2V_{F1})/4$; narrow = narrow sense heritability estimate = $[2V_{F2} - (V_{BC1} + V_{BC2})]/V_{F2}$.
[c]Too much variability was present in the data to estimate reliably heritability.

TABLE 5. Degree of dominance for resistance to *Pseudocercosporella herpotrichoides* in crosses between Daws, Cappelle-Desprez, and VPM-1 winter wheat

	Rating system[a]				
	HSR + NHSR		NHSR		
Cross	PAP + CELL	PAP	PAP + CELL	PAP	Mean
Cappelle × Daws	0.38[b]	0.58	0.48	0.64	0.52
VPM-1 × Daws	1.07	0.78	1.10	0.92	0.97
VPM-1 × Cappelle	2.03	1.95	4.70	3.12	2.95

[a]HSR = hypersensitive cell, NHSR = nonhypersensitive cell, PAP = penetration stopped by papilla, and CELL = penetration stopped within cell.
[b]Degree of dominance is calculated by dividing the deviation of the F_1 from the midparent by the the deviation of the resistant parent from the midparent; 0 = no dominance, 1 = complete dominance, and >1 = overdominance.

reciprocal effects were not significant, but data from others indicates that some cytoplasms may contribute to resistance (6,7,10).

The data on gene action from the Cappelle × Daws and VPM-1 × Daws crosses best fit a simple additive-dominance model (m[d][h]). This dominance effect is consistent with the degree of dominance estimates that showed resistance in Cappelle to be semidominant and that in VPM-1 to be dominant (Table 5). In the VPM-1 × Cappelle cross, two of the rating systems fit a model assuming additive × additive interaction (m[d][h][i]), while the two other rating systems fit a model that also included a dominance × dominance interaction (m[d][h][i][l]). These additive and dominance effects are consistent with the earlier results, since the VPM-1 × Cappelle cross was found to exhibit extreme overdominance. If the additive gene action is due to interaction between alleles at different loci, as implied from cytological data (13,16), then transgressive segregation would be expected, but may be difficult to identify due to the variability associated with the disease ratings.

As would be expected, the broad sense heritability estimates (Table 4) were higher than the narrow sense estimates. The largest narrow sense heritability was obtained from the VPM-1 × Cappelle cross, while the heritability estimates for VPM-1 were higher than those for Cappelle. The heritability estimates indicate that it may be possible to select for individuals with more resistance genes and increase resistance to *P. herpotrichoides* in winter wheat.

A possible breeding strategy for improving resistance to *P. herpotrichoides* would be to use single seed descent (25). In this procedure, F_2 plants of a population are grown and one F_3 seed per plant is harvested and bulked. The procedure of selecting one seed per plant, bulking, and planting can be repeated until the desired level of inbreeding (homozygosity) is achieved. The generations can be readily created, since unlike pedigree and mass selection methods, plants do not have to be grown in environments in which genetic differences would be expressed for the characters under selection. Thus, the F_6 generation can be attained in 2–3 yr, using both the greenhouse and field. This method also has the advantage of requiring little space and ensures the maintenance of variability for genes not under selection pressure. Selection for resistance in the advanced generations could be accomplished by exposing plants to intense pressure from *P. herpotrichoides*. These resistant types could then be screened by evaluating epidermal cell responses to attempted penetration at the seedling stage (24,29). This seedling test provides a nondestructive method to identify resistant plants quickly and accurately. Also, disease ratings do not have to be done immediately since the harvested sheaths can be left in the fixative or the staining solution for extended periods of time (at least 6 mo).

In the past, the natural or mass selection breeding methods used in breeding for resistance to *P. herpotrichoides* have been of questionable value, especially in early generations (25). In analyzing genetic material for resistance to *P. herpotrichoides* at the seedling stage, previous workers (6,8,9,13,15,16) have predominantly used the technique of Macer (17), but low heritabilities normally were obtained (16). By evaluating for resistance to *P. herpotrichoides* using epidermal cell responses at the seedling stage (24,29), higher heritabilities were obtained.

Epidermal responses (papilla formation, penetrations stopped within epidermal cells, and hypersensitive responses) in the first-leaf sheaths of wheat inoculated with *P. herpotrichoides* were useful tools for separating resistant and susceptible plants; however, some combinations of epidermal responses were more reliable. The rating system that resulted in the highest heritability was based on penetrations stopped by papillae or within epidermal cells in both hypersensitive and nonhypersensitive cells (PAP + CELL in HSR and NHSR cells). However, the rating system based on penetrations stopped by papillae or within epidermal cells in only nonhypersensitive cells (PAP + CELL in only HSR) fit the genetic hypotheses more consistently than the other rating systems. The basis of the observed differences among these disease rating systems is not known. Variation in the ratings themselves was of concern and the causes are also unknown. We attempted, insofar as possible, to reduce environmental variation by selecting plants at the same stage of growth for inoculation, using the same inoculum concentration and rates, and growing plants in different experiments in conditions as similar as possible. One potential source of variation could be background effects from either the resistant or susceptible parent. Previous work (13,16) has shown that three chromosomes, in addition to chromosome 7A where the major gene is located, are involved with resistance. Also, the environment (especially relative humidity) had large effects on disease development: Differences in disease development between experiments could also account for variation in disease ratings. Even with the observed differences, all of the ratings based on epidermal cell responses in the first-leaf sheath are correlated with field resistance (24), genetically associated with resistance in segregating progeny, and useful for screening potential cultivars.

TABLE 6. Gene action of genes for resistance to *Pseudocercosporella herpotrichoides* in crosses between Daws, Cappelle-Desprez, and VPM-1 winter wheat

Rating system[a]	Model fit[b]	X^2 value	P[c]	Component fit[d]
Cappelle × Daws				
PAP + CELL in HSR + NHSR	m [d] [h] [i]	6.0	0.05	[d] [i]
PAP in HSR + NHSR	m [d] [h]	3.3	0.34	[d] [h]
PAP + CELL in NHSR	m [d] [h]	7.8	0.05	[d] [h]
PAP in NHSR	m [d] [h]	2.4	0.50	[d] [h]
PAP + CELL in HSR	m [d] [h] [i]	1.2	0.56	[d] [h] [i]
VPM-1 × Daws				
PAP + CELL in HSR + NHSR	m [d] [h] [l]	2.9	0.24	[d] [h] [l]
PAP in HSR + NHSR	m [d] [h]	6.0	0.11	[d] [h]
PAP + CELL in NHSR	m [d] [h]	6.0	0.11	[d] [h]
PAP in NHSR	m [d] [h]	6.2	0.10	[d] [h]
PAP + CELL in HSR	m [d] [h]	5.2	0.16	[d] [h]
VPM-1 × Cappelle				
PAP + CELL in HSR + NHSR	m [d] [h] [i] [l]	0.0	0.99	[d] [h] [i] [l]
PAP in HSR + NHSR	m [d] [h] [i]	2.4	0.30	[d] [h] [i]
PAP + CELL in NHSR	m [d] [h] [i] [l]	1.6	0.21	[d] [h] [i] [l]
PAP in NHSR	m [d] [h] [i]	3.4	0.18	[d] [h] [i]
PAP + CELL in HSR	m [d] [h]	6.4	0.09	[d]

[a]HSR = hypersensitive cells, NHSR = nonhypersensitive cells, PAP = penetration stopped by papilla, CELL = penetration stopped within an epidermal cell.

[b]m = estimated mean of all homozygous individuals, [d] = additive component, [h] = dominance component, [i] = additive × additive interaction component, [j] = additive × dominance interaction component, and [l] = dominance × dominance interaction component.

[c]Model was considered to fit if the chi-square value had a $P \geq 0.05$.

[d]The individual components listed differed significantly from zero at $P = 0.05$ and thus contributed significantly to the model.

LITERATURE CITED

1. Bruehl, G. W. 1983. Nonspecific genetic resistance to soilborne fungi. Phytopathology 73:948-951.
2. Bruehl, G. W., and Machtmes, R. 1985. Production of *Pseudocercosporella herpotrichoides* spores. Plant Dis. 69:862-863.
3. Bruehl, G. W., and Nelson, W. L. 1964. Technique for mass inoculations of winter wheat in the field with *Cercosporella herpotrichoides*. Plant Dis. Rep. 48:863-865.
4. Bruehl, G. W., Nelson, W. L., Koehler, F., and Vogel, O. A. 1968. Experiments with *Cercosporella* foot rot (strawbreaker) disease of winter wheat. Wash. Exp. Stn. Bull. 694. 14 pp.
5. Dosba, F., and Doussinault, G. 1973. Resistance to eyespot (*Cercosporella herpotrichoides*) introduced to bread wheat from *Aegilops ventricosa*. Pages 409-413 in: Proc. 4th Int. Wheat Genetics Symp. Mo. Agric. Exp. Stn., Columbia, MO.
6. Dosba, F., and Doussinault, G. 1981. Les lignées d'addition blé — *Aegilops ventricosa*. I.—Etude du comportement vis-à-vis du piétin-verse des différentes lignées obtenues. Agronomie 1:503-511.
7. Doussinault, G., Delibes, A., Sanchez-Monge, R., and Garcia-Olmedo, F. 1983. Transfer of a dominant gene for resistance to eyespot disease from a wild grass to hexaploid wheat. Nature (London) 303:698-700.
8. Doussinault, G., and Dosba, F. 1977. An investigation into increasing the variability for resistance to eyespot in wheat. Z. Pflanzenzuchtz. 79:122-133.
9. Doussinault, G., Dosba, F., and Jahier, J. 1983. New results on the improvement of the level of resistance to eyespot in wheat. Pages 193-198 in: Proc. 6th Int. Wheat Genet. Symp., Kyoto, Japan.
10. Doussinault, G., and Douaire, G. 1978. Analyse d'un croisement diallèle chez le Blé tendre pour l'étude de la résistance au Piétin-verse (*Cercosporella herpotrichoides* Fron.). Ann. Amélior. Plantes 28:479-491.
11. Gale, M. D., Scott, P. R., Law, C. N., Ainsworth, C. C., Hollins, T. W., and Worland, A. J. 1984. An alpha-amylase gene from *Aegilops ventricosa* transferred to bread wheat together with a factor for eyespot resistance. Heredity 52:431-435.
12. Groll, U., Frauenstein, K., and Hammer, K. 1985. Prüfung von *Aegilops* - Arten auf Resistenz gegen *Pseudocercosporella herpotrichoides* (Fron) Deighton. Kulturpflanze 33:165-172.
13. Jahier, J., Doussinault, G., Dosba, F., and Bourgeois, F. 1978. Monosomic analysis of resistance to eyespot in the variety "Roazon". Pages 437-440 in: Proc. 5th Int. Wheat Genetics Symp., New Delhi.
14. Kephart, K. D., and Allan, R. E. 1988. Madsen and Hyak soft white winter wheats. Univ. of Idaho College of Agric., Curr. Info. Ser. No. 823. 4 pp.
15. Kimber, G. 1967. The incorporation of the resistance of *Aegilops ventricosa* to *Cercosporella herpotrichoides* into *Triticum aestivum*. J. Agric. Sci. Camb. 68:373-376.
16. Law, C. N., Scott, P. R., Worland, A. J., and Hollins, T. W. 1976. The inheritance of resistance to eyespot (*Cercosporella herpotrichoides*) in wheat. Genet. Res. Camb. 25:73-79.
17. Macer, R. C. F. 1966. Resistance to eyespot disease (*Cercosporella herpotrichoides* Fron) determined by a seedling test in some forms of *Triticum*, *Aegilops*, *Secale*, and *Hordeum*. J. Agric. Sci. Camb. 67:389-396.
18. Mahmud, I., and Kramer, H. H. 1951. Segregation for yield, height, and maturity following a soybean cross. Agron. J. 43:605-609.
19. Maia, N. 1967. Obtention de blés tendres résistants au piétin-verse par croisements interspécifiques blés × *Aegliops*. C. R. Acad. Agric. Fr. 53:149-154.
20. Mather, K., and Jinks, J. L. 1982. Biometrical Genetics: The Study of Continuous Variation. 3rd ed. Chapman and Hall, New York and London. 396 pp.
21. McMillin, D. E., Allan, R. E., and Roberts, D. E. 1976. Association of an isozyme locus and strawbreaker foot rot resistance derived from *Aegilops ventricosa* in wheat. Theor. Appl. Genet. 72:743-747.
22. Murray, T. D. 1983. Resistance in winter wheat to *Pseudocercosporella herpotrichoides*. Ph.D. dissertation. Washington State University, Pullman.
23. Murray, T. D., and Bruehl, G. W. 1986. Effects of host resistance to *Pseudocercosporella herpotrichoides* and foot rot severity on yield components in winter wheat. Plant Dis. 70:851-856.
24. Murray, T. D., and Ye, H. 1986. Papilla formation and hypersensitivity at penetration sites and resistance to *Pseudocercosporella herpotrichoides* in winter wheat. Phytopathology 76:737-744.
25. Roberts, D. E. 1988. Disease resistance and other factors in wheat lines that contain *Aegilops ventricosa* germplasm. Ph.D. dissertation. Washington State University, Pullman.
26. Saragoussi, M. 1986. Study of the genetic composition of the resistance to *Pseudocercosporella herpotrichoides* (Fron) Deighton in four varieties of bread wheat (*Triticum aestivum* L.) by a diallelic cross. Rev. Bras. Genet. 9:625-636.
27. Simonet, M. 1957. Hybrids interspécifiques et intergénériques. Ann. Amélioration Plantes 4:395-411.
28. Sprague, R. 1936. Relative susceptibility of certain species of Gramineae to *Cercosporella herpotrichoides*. J. Agric. Res. 53:659-670.
29. Strausbaugh, C. A., and Murray, T. D. 1988. Inheritance of resistance to foot rot (*Pseudocercosporella herpotrichoides*) in three cultivars of winter wheat. (Abstr.) Phytopathology 78:1542.
30. Strausbaugh, C. A., and Murray, T. D. 1989. Use of epidermal cell responses to evaluate resistance of winter wheat cultivars to *Pseudocercosporella herpotrichoides*. Phytopathology 79:1043-1047.
31. Warner, J. N. 1952. A method for estimating heritability. Agron. J. 44:427-430.
32. Worland, A. J., and Law, C. N. 1987. Resistance to eyespot. (Abstr.) Rev. Plant Pathol. 66:246.
33. Wright, S. 1968. Evolution and Genetics of Populations. Vol. I. Genetic and Biometric Foundations. University of Chicago Press, Chicago. 469 pp.