Cephalosporium Stripe Resistance and Grain Yield Potential of Wheat Lines with Strawbreaker Resistance Derived from *Aegilops ventricosa*

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ABSTRACT

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Wheat (*Triticum aestivum*) having germ plasm of *Aegilops ventricosa* for resistance to strawbreaker (caused by *Pseudocercosporella herpotrichoides*) has not previously demonstrated the yield potential or the resistance to Cephalosporium stripe (caused by *Cephalosporium gramineum*) of certain adapted northwestern U.S. cultivars. Fifteen genotypes homozygous for an endopeptidase isoenzyme of *A. ventricosa* closely linked to strawbreaker resistance, as shown by starch gel electrophoresis, suffered no yield loss attributable to strawbreaker in three seasons of field testing. Yields among the 15 genotypes varied under pressure from *C. gramineum*. Reactions among genotypes to the two pathogens were expressed independently and lines with resistance to both pathogens were identified. Ten F_2-derived sibling pairs were grown under strawbreaker pressure for three seasons, with disease and control treatments. Seven pairs had contrasting reactions to strawbreaker but their yield potentials were generally similar. The incorporation into wheat of high strawbreaker resistance, derived from *A. ventricosa*, should not reduce genetic yield potential.

Strawbreaker (caused by *Pseudocercosporella herpotrichoides* (Fron) Deighton) is a serious disease of early-sown wheat (*Triticum aestivum* L.) in the northwestern United States (5) and many other parts of the world (7). The wheat line VPM-1 (VPM), obtained by the introgression of germ plasm of *Aegilops ventricosa* Tausch into hexaploid wheat, is more resistant to strawbreaker than are adapted wheats. VPM has poor agronomic potential because of late maturity, lodging susceptibility, and low yield potential (10). However, selections from a cross between VPM and cv. Moisson have good agronomic characteristics with disease resistance similar to VPM (6). This germ plasm has been used in the USDA-ARS wheat genetics program at Pullman, Washington, and in many other breeding programs.

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Response to *P. herpotrichoides* is often classified as a disease index determined by the weighted mean of the number of tillers in five disease severity lesion classes (2,9,14). This method is subjective and laborious. McMillin et al (11) developed an assay for an endopeptidase enzyme encoded by a gene that is tightly linked to the gene for strawbreaker resistance from *A. ventricosa*. Consequently, starch gel electrophoresis can be used to screen wheat lines for homozygosity for the isoenzyme of *A. ventricosa*. Heterozygosity and homozygosity for the wheat isoenzyme are not easily differentiated.

Cephalosporium stripe, caused by *Cephalosporium gramineum* Nisikado and Ikata, is also a disease of early-sown winter wheat in the northwestern United States. Cultivars resistant to the pathogen have been identified (3,12), although their level of resistance varied across seasons (3). VPM and VPM/Moisson selections are generally susceptible to *C. gramineum*. Morton and Mathre (12) defined two types of resistant response: 1) exclusion of the pathogen expressed as a reduction in frequency of diseased plants, and 2) restriction of spread of the fungus expressed as a reduction in frequency of diseased tillers within plants and as a reduction in the rate and severity of disease development. The development of wheat cultivars resistant to both pathogens is needed to facilitate early seeding to achieve maximum yield potential and to reduce soil erosion (5). Therefore, because wheat germ plasm derived

from *A. ventricosa* appeared to be resistant to strawbreaker but susceptible to Cephalosporium stripe, the first objective of this study was to compare reactions of wheat breeding lines to the two diseases. VPM/Moisson selections, used in our program to incorporate strawbreaker resistance derived from *A. ventricosa* into adapted germ plasm, have raised the concern that strawbreaker resistance could be accompanied by an inherent inability to achieve maximum yield. Based on four test years, they have yielded 22–27% less than Nugaines, an adapted cultivar in the northwestern United States with good yield potential. Substitution of chromosome 7D from VPM depressed yield by about 6% in several wheat backgrounds, according to Law et al (8). The second objective of this study was to determine whether closely related wheat lines containing germ plasm of *A. ventricosa* but differing in response to *P. herpotrichoides* had similar yield potentials in the absence of disease. If the more resistant lines yielded less than the susceptible lines, exploitation of strawbreaker resistance from *A. ventricosa* would be more difficult.

MATERIALS AND METHODS

Association of resistance to strawbreaker and Cephalosporium stripe. Fifteen wheat genotypes were selected from F_5-derived F_6 lines or F_6-derived F_7 lines, for variation in their reactions to the strawbreaker and Cephalosporium stripe pathogens. Genotypes included five VPM/Moisson 951//2*Hill 81 (VPM/M951//2*H81) lines, two VPM/Moisson 951//Hill 81 (VPM/M951//H81) lines, two VPM/Moisson 951//2*Barbee (VPM/M951//2*BRB) lines, four VPM/Moisson 421//Raeder (VPM/M421//RDR) lines, and two VPM/Moisson 421//2*Raeder (VPM/M421//2*RDR) lines. VPM, Raeder, Hill 81, Barbee, and Stephens were grown as checks. Among these cultivars, Hill 81 expresses the most resistance to *C. gramineum*.

Over 3 yr (1985–1987), the 20 genotypes were assessed for their response to *P. herpotrichoides* and to *C. gramineum* in separate trials, each grown in a randomized complete block design with four replications. The strawbreaker tests included diseased and control treatments as subplots. Plots in the strawbreaker trials all consisted of four rows 0.30 m apart and 2.58 m long. Cephalosporium stripe plots consisted of two rows 1.52 m long in 1985 and four rows 2.13 m and 2.58 m long in 1986 and 1987, respectively. All trials were grown near Pullman, and planting occurred each year during the second half of September. Seeding rates were 2.1 g of seed per meter. Trials were grown on Palouse silt loam (Pachic Ultic Haploxerolls, fine-silty, mixed, mesic) following a season of summer fallow. Superphosphate fertilizer was applied at 44.8 kg of P/ha and urea at 67.2 kg of N/ha. Bromoxynil (39.4% by weight) was applied in the spring at 2.24 kg/ha in 187 L of water for weed control.

In 1985, disease subplots of the strawbreaker trial were inoculated in mid-November with a suspension of *P. herpotrichoides* containing 5×10^5 spores per milliliter of water with the method of Murray and Bruehl (13). The 1986 trial was inoculated with 2.5×10^5 spores per milliliter during February because an early snowfall prevented inoculation in November. Dry oat inoculum (4) was applied to the 1987 trial late in October at 53.8 kg/ha, followed by a spray application of 5×10^5 spores per milliliter in mid-November. Each year, the control subplots were sprayed with benomyl fungicide (50% a.i. at 2.24 kg/ha in 75.6 L of water) during the first half of April. In 1985 and 1986, reaction to *C. gramineum* within the strawbreaker trial was estimated visually on a scale of 0–100, based on plant stunting and the severity and extent of chlorotic streaking of leaves and stems. The trials were harvested each year in August. The center two rows in 1985 and 1987 and one row in 1986 of four-row plots were mechanically harvested and threshed with a stationary plot thresher. Harvested plots were 1.60 m^2, 0.74 m^2, and 1.49 m^2 in 1985, 1986, and 1987, respectively. The D/C ratio was calculated as the grain yield in diseased (D) plots divided by the grain yield in control (C) plots. Following harvest, the stubble from 0.5 m (10–15 plants) of each harvested row was removed. For both treatments of the 1985 trial, two stems per plant were scored for strawbreaker reaction, and in the disease treatment of the 1987 trial, three stems per plant were used. The 1986 trial was not scored because disease incidence was very low. Disease incidence was scored on a scale from one to five, according to McMillin et al (11). Yield data (excluding D/C) were analyzed for a split-plot design with genotype as the main plot and treatment (disease or control) as the subplot. Disease response data were analyzed for a randomized complete block design.

In 1987, grain samples from each genotype were collected after harvest, combined over replications, and grown in the greenhouse. Seedlings at the two-leaf stage were assayed for endopeptidase isoenzymes of wheat (encoded by *EP-D1*) and of *A. ventricosa* (encoded by *EP-D1b*; 16), following the starch gel electrophoresis method of McMillin et al (11) with the modifications of Roberts (15). Assuming that lines were homozygous, leaves from about 20 seedlings per genotype were homogenized together and assayed as one sample. Genotypes homozygous for the isoenzyme from *A. ventricosa* were readily distinguished from those that were heterozygous or homozygous for the wheat isoenzyme.

The Cephalosporium stripe trials were grown each year in fields naturally infested with *C. gramineum*. Reaction to the fungus was determined early in July, about 1 mo after anthesis. In 1985, the disease index was defined as the percentage of tillers in 1 m of row affected by the disease. A visual estimate of disease was also made, as described for the strawbreaker trial. In 1986 and 1987, 0.5 m of row was cut from each plot and each tiller was scored on a scale from one (resistant) to five (susceptible). A disease index was calculated from the weighted mean of individual scores (1). The scores were as follows: 1) infection up to and including the third leaf below the flag leaf, 2) infection up to and including the second leaf below the flag leaf, 3) infection up to and including the penultimate leaf, 4) infection up to and including the flag leaf, and 5) infection up to and including the spike. The trials were harvested during August by the same method used for the strawbreaker trials. Harvested plots were 0.93 m^2, 0.65 m^2, and 0.74 m^2 in 1985, 1986, and 1987, respectively. Grain yield data were collected and analyzed as a randomized complete block design.

Strawbreaker resistance and yield potential. In 1985, F_3 derivatives of VPM/Moisson were grown in three replications, with disease and control treatments, at Spillman Farm near Pullman. Fungal inoculation and benomyl application were the same as for the 1985 strawbreaker trial. A visual estimate of response to strawbreaker, based on plant stunting and whiteheads, was made on each plot. The plots, each one row 1.01 m long (0.31 m^2), were harvested by hand and threshed with a stationary plot thresher. Paired sibling selections were made on the basis of their reaction to strawbreaker, with the expectation that each sib represented differential resistance to the pathogen. One pair of sibs was selected from each family of VPM/Moisson 951//Cerco///Daws (VPM/M951//CER///DAWS), VPM/Moisson 951/2*Barbee//Daws (VPM/M951/2*BRB//DAWS), VPM/Moisson 421//2*Tyee (VPM/M421//2*TYEE), VPM/Moisson 421/WA 6241//Daws (VPM/M421/WA6241//DAWS), Tyee//Roazon/Tres (TYEE//RZN/TRES), and Daws*2/Roazon (DAWS*2/RZN). Two pairs of sibs were selected from families of VPM/Moisson 421/VH 66354/WA 5827/WA 6241//Nugaines (VPM/M421/VH66354/WA5827/WA6241//NGN) and VPM/Moisson 951//Peck/Stephens/Daws (VPM/M951//PECK/SPN/DAWS), making a total of 10 pairs of sibs.

In 1986 and 1987, the F_4 and F_5 sibs, respectively, were grown in four replications with disease and control treatments. Trial conditions, procedures, treatments, data collection, and endo-

peptidase assays were the same as for the strawbreaker trials. Data were analyzed as a factorial arrangement of family and sibling in a randomized complete block design, with treatment (disease or control) as a subplot effect.

RESULTS AND DISCUSSION

Association of resistance to strawbreaker and Cephalosporium stripe. Assay of the populations for the endopeptidase isoenzyme encoded by *EP-D1b*, contributed by *A. ventricosa*, revealed that VPM and the 15 test genotypes were homozygous for this isoenzyme. Raeder, Hill 81, Barbee, and Stephens have the wheat isoenzyme, and their pedigrees did not include any genotypes derived from *Aegilops*, so they were omitted from data analyses. VPM was also omitted from the analyses because it is unadapted to the Pacific Northwest region. Inclusion of these genotypes would have biased the statistical significance of the results. Analyses of the data from trials grown under exposure to strawbreaker for three consecutive years showed that mean grain yields between disease and control treatments never differed significantly. Because all 15 selections were homozygous for the *EP-D1b* marker gene and were highly likely to have the gene for strawbreaker resistance from *A. ventricosa*, it was reasonable to expect that yield loss would not be significant across all genotypes. For each year, regardless of the disease severity within the trial, Raeder, Hill 81, Barbee, and Stephens consistently had D/C ratios <1, and in 1987 they were as low as 0.5. VPM had a D/C ratio of 0.9 in 1985, 1.1 in 1986, and 1.0 in 1987. The lesion indices obtained for VPM were 3.3 and 3.8 in the disease plots of 1985 and 1987, respectively. Lesion indices in disease plots were 3.4–3.9 for the susceptible checks in 1985 and 4.5–4.6 in 1987. Hence, VPM expresses a very high level of resistance to strawbreaker, even under severe infection levels. The possibility also existed that yield was depressed only when the vascular system of the plant was severely damaged by *P. herpotrichoides* (class five lesion score).

Genotypes differed for grain yield (means in Table 1) in the control plots ($P < 0.05$) and in the strawbreaker infected plots ($P < 0.01$) in 1987. There were significant ($P < 0.01$) yield differences among genotypes in the disease treatments of the 1985 and 1986 strawbreaker trials; however, natural infection by *C. gramineum* confounded these data and means are not presented. Significant ($P < 0.05$) genotypic differences (means in Table 2) occurred for strawbreaker index in 1987.

There were significant ($P < 0.05$, $P < 0.01$, and $P < 0.01$ for 1985, 1986, and 1987, respectively) genotypic differences in grain yield for the trials naturally infested with *C. gramineum* (Table 1). Low (≤18%) coefficients of variation for seed yield (Table 1) and disease response (Table 2) showed that infestation was uniform. No significant genotypic differences for response to *C. gramineum* were observed in 1987 when grain yields were highest, so this trial was not included in Table 2.

Mean grain yields (Table 1) revealed considerable variation among genotypes for response to *P. herpotrichoides* and *C. gramineum*. Occurrence of low levels of Cephalosporium stripe caused confounding in the 1985 and 1986 strawbreaker trials (mean visual estimate of 42% and 33%, respectively) so their severities for strawbreaker are not presented. No Cephalosporium stripe was observed in the 1987 strawbreaker trial, and no strawbreaker was detected in any of the Cephalosporium stripe trials.

Comparisons among selections confirmed that resistance to the two pathogens could be combined. Among the four northwestern U.S. cultivars (Raeder, Barbee, Hill 81, and Stephens), Hill 81 expressed the highest level of resistance to Cephalosporium stripe in each of the three tests (Table 2). Four selections had Cephalosporium stripe reactions comparable ($P > 0.05$) to Hill 81 in each trial, yet they had strawbreaker indices indicating they were more ($P < 0.05$) resistant than Hill 81 and the other three northwestern U.S. cultivars, Raeder, Barbee, and Stephens. Among these four selections, VPM/M951//2*H81 selection D, VPM/M951//2*H81 selection E and VPM/M421//RDR selection A had mean grain yields (Table 1) equal ($P > 0.05$) to Hill 81 in each of the Cephalosporium stripe trials. These three lines also had higher ($P < 0.05$) mean grain yields in the diseased portion of the strawbreaker trial than did Hill 81. They had D/C ratios of 0.95 to 1.05 vs. 0.55 for Hill 81. Although the data for strawbreaker index and yield under strawbreaker pressure were from a single replicated trial, there is good assurance that

Table 1. Mean grain yields (g/m^2), within each trial for which there were genotypic differences, of 15 selections from five wheat populations grown under pressure from *Cephalosporium gramineum* and *Pseudocerosporella herpotrichoides* for three successive years

		Cephalosporium stripe plots[w]			Strawbreaker plots (1987)	
Population	Sel[x]	1985	1986	1987	Disease	Control
VPM/M421//	A	472 a–f[y]	641 ab	713 bc	555 e	464 d
2*RDR	B	531 a–d	639 ab	594 cde	684 b–e	674 abc
VPM/M951//	A	433 c–f	520 c	669 bcd	719 a–d	746 abc
2*H81	B	491 a–f	595 abc	706 bc	761 abc	815 ab
	C	430 def	632 abc	699 bcd	741 a–d	708 abc
	D	496 a–f	648 ab	716 bc	865 a	827 a
	E	547 abc	645 ab	659 bcd	831 a	827 a
VPM/M951//	A	390 f	556 bc	578 de	718 a–d	740 abc
H81	B	493 a–f	632 abc	741 b	793 ab	689 abc
VPM/M421//	A	574 a	679 a	628 bcd	596 de	626 cd
RDR	B	406 ef	401 d	503 e	634 cde	702 abc
	C	514 a–e	599 abc	886 a	632 cde	614 cd
	D	470 a–f	523 c	640 bcd	755 abc	830 a
VPM/M951//	A	560 ab	615 abc	673 bcd	607 de	631 cd
BRB	B	445 b–f	638 ab	645 bcd	641 cde	636 bcd
VPM[z]		324	432	508	499	520
Raeder		480	566	722	257	451
Hill 81		601	733	665	399	720
Barbee		581	663	511	270	543
Stephens		333	456	502	420	804
CV (%)		17	13	13	15	18
LSD ($P = 0.05$)		110	116	138	152	184

[w] Treatments for Cephalosporium stripe trials were disease only.
[x] Sel = selection.
[y] Means within a column not followed by the same letter differ significantly ($P < 0.05$) by an LSD test.
[z] Means of check cultivars presented for comparison with test lines.

these lines have resistance to the strawbreaker pathogen because they possess the *EP-D1b* gene that is tightly linked to the strawbreaker resistance gene derived from *A. ventricosa*.

Correlation coefficients (Table 3) among the Cephalosporium stripe trials showed that grain yields for the three seasons were significantly correlated with each other, so the data was not confounded by year. Similarly, all estimates of genotypic response to *C. gramineum* were correlated. The visual estimate of Cephalosporium stripe (1985) had the highest association with yield under pressure from *C. gramineum*. The disease index from 1987 was also correlated with yields from each season, but the coefficients were lower than those obtained with the 1985 visual estimate.

The optimal method of evaluating response to *C. gramineum* was a subjective, visual estimate of chlorosis and plant stunting within a plot. This estimate could be made very quickly and correlated consistently with grain yields of the wheat genotypes in diseased conditions. The success of the visual method was probably because it estimated both resistant reactions described by Morton and Mathre (12).

The 1987 strawbreaker index was not correlated with grain yield means nor with disease score means from the trials infested by *C. gramineum*. Grain yield from the strawbreaker trial also did not correlate with grain yields from the Cephalosporium trials. Therefore, responses to *C. gramineum* and *P. herpotrichoides* were independent in 15 genotypes derived from *A. ventricosa*. The genotypes tested were all homozygous

Table 2. Mean disease indices, within disease treatments of each trial for which there were genotypic differences, of 15 selections from five wheat populations grown under pressure from *Cephalosporium gramineum* and *Pseudocercosporella herpotrichoides* for three successive years

		Cephalosporium stripe plots			Strawbreaker
		1985		1986	1987
Population	Sel[t]	Visual[u] score (%)	Infected[v] tillers (%)	Disease[w] index	Disease[x] index
VPM/M421//	A	65 abc[y]	91 a	2.6 a	3.8 ab
2*RDR	B	65 abc	85 abc	2.7 a	4.0 ab
VPM/M951//	A	58 bcd	69 de	1.7 de	4.1 a
2*H81	B	57 cd	88 ab	1.9 b–e	3.8 ab
	C	58 bcd	74 b–e	1.6 e	3.9 ab
	D	53 cd	73 b–e	1.8 cde	3.8 ab
	E	48 d	62 de	1.7 de	4.1 a
VPM/M951//	A	70 ab	87 ab	2.0 bcd	3.7 ab
H81	B	57 cd	85 abc	1.8 b–e	3.7 ab
VPM/M421//	A	53 cd	62 e	2.0 bcd	3.9 ab
RDR	B	78 a	86 ab	2.7 a	3.5 bc
	C	58 bcd	78 a–d	2.1 bc	3.5 bc
	D	60 bcd	71 cde	1.8 cde	3.1 c
VPM/M951//	A	58 bcd	88 ab	2.2 b	3.7 ab
BRB	B	60 bcd	77 a–e	1.9 b–e	3.6 ab
VPM[z]		65	76	1.9	3.8
Raeder		60	81	2.6	4.5
Hill 81		50	63	1.5	4.5
Barbee		52	85	2.2	4.6
Stephens		73	89	2.4	4.5
CV (%)		15	14	12	9
LSD ($P = 0.05$)		13	16	0.4	0.5

[t] Sel = selection.
[u] Estimated from plant stunting and chlorosis.
[v] Percentage of diseased tillers in 0.5 m of row.
[w] Weighted mean of tiller disease score.
[x] Weighted mean of tiller disease score.
[y] Means not followed by the same letter differ significantly ($P < 0.05$) by an LSD test.
[z] Means of check cultivars presented for comparison with test lines.

Table 3. Correlation coefficients among mean grain yields and disease indices of 15 wheat genotypes grown under pressure from *Cephalosporium gramineum* (Cg) for 3 yr and under pressure from *Pseudocercosporella herpotrichoides* (Ph) for 1 yr

	Yield (Cg)			Yield (Ph)		Visual (Cg)[a]	Index (Cg)			Index
	1985	1986	1987	(Disease) 1987	(Control) 1987	score 1985	1985[b]	1986[c]	1987[c]	(Ph)[c] 1987
Yield (Cg) 1985										
Yield (Cg) 1986	−0.82**[d]									
Yield (Cg) 1987	0.38*	0.47*								
Disease Yield (Ph) 1987	−0.05 NS[e]	0.05 NS	0.32 NS							
Control Yield (Ph) 1987	−0.06 NS	−0.06 NS	−0.04 NS	0.65**						
Visual Score (Cg) 1985	−0.74**	−0.76**	−0.47*	−0.11 NS	−0.10 NS					
Index (Cg) 1985	−0.32 NS	−0.31 NS	−0.16 NS	−0.17 NS	−0.26 NS	0.63**				
Index (Cg) 1986	−0.12 NS	−0.30 NS	−0.25 NS	−0.39*	−0.48*	0.64**	0.67**			
Index (Cg) 1987	−0.52*	−0.50*	−0.48*	0.32 NS	−0.04 NS	0.64**	0.49*	0.52*		
Index (Ph) 1987	−0.12 NS	0.23 NS	−0.24 NS	−0.66**	−0.20 NS	−0.23 NS	−0.05 NS	0.07 NS	0.32 NS	

[a] Visual disease score based on stunting, chlorosis.
[b] Percentage of infected tillers.
[c] Weighted mean of tillers scores.
[d] * = Significant at $P = 0.1$ and ** = significant at $P = 0.05$.
[e] Not significant.

Table 4. Means for disease response, grain yield potential, and ratio of disease to control yield (D/C) of sibling pairs selected from each of seven winter wheat populations and exposed to strawbreaker for three seasons under disease and control treatments

Population	Sib[w]	Visual score 1985	Disease index 1987	D/C ratio 1985	D/C ratio 1987	Grain yield potential (g/m^2)[v] 1985	1986	1987
VPM/M951//	S	4.3 a[x]	4.7 a	0.65	0.56*[y]	546[z]	438[z]	603 a
CER///DAWS	R	2.7 b	4.0 b	0.96	0.84	544	553	732 a
VPM/M421/VH66354/	S	4.0 a	4.2 a	0.86	0.47*	601	532	781 a
WA5827/WA6241//NGN	R	2.5 b	3.7 b	1.03	0.90	624	471	729 a
VPM/M421/VH66354/	S	4.0 a	4.2 a	0.85	0.65*	635	511	826 a
WA5827/WA6241//NGN	R	2.8 b	3.7 b	0.92	0.89	733	588	718 a
VPM/M951//PECK/	S	5.7 a	4.8 a	0.76	0.60*	743	481	466 b
SPN/DAWS	R	2.6 n	4.2 b	1.11	0.85	696	546	806 a
VPM/M951//PECK/	S	4.3 a	4.2 a	0.81	0.66*	742	584	771 a
SPN/DAWS	R	2.3 b	3.8 b	0.98	0.84	668	509	859 a
VPM/M421//	S	3.8 a	4.7 a	0.84	0.64*	657	604	688 a
2* TYEE	R	2.8 b	3.7 b	0.99	0.93	589	535	675 a
TYEE//RZN/TRES	S	4.3 a	4.5 a	1.11	0.87	600	505	469 b
	R	2.8 b	4.0 b	0.96	0.89	758	594	697 a

[v] Control plots.
[w] S = susceptible, R = resistant.
[x] Means for siblings within a population that are not followed by the same letter differ significantly ($P < 0.05$) by an LSD test.
[y] * Denotes D/C ratio for sibling S is significantly ($P < 0.05$) less than that for sibling R by an LSD test.
[z] There were no differences between siblings for grain yield potential in 1985 and in 1986.

for the endopeptidase allele from *A. ventricosa*, *EP-D1b*, and were more resistant to strawbreaker than the susceptible checks. Because of the close linkage between *EP-D1b* and strawbreaker resistance (11,16), this trend could be expected to hold in other seasons.

Independence between resistance to *C. gramineum* and *P. herpotrichoides* has the important implication that breeding VPM/Moisson derivatives for resistance to one pathogen would not necessarily result in vulnerability to the other. On the other hand, the potential usefulness of a strong positive correlation between the two diseases was also lacking. Resistance to *C. gramineum* would have to be incorporated into wheat lines independently of resistance to *P. herpotrichoides*. This study showed conclusively that it is possible to develop lines with resistance to both pathogens.

Strawbreaker resistance and yield potential. Analyses of variance for the 1985 data revealed significant ($P < 0.01$) differences between siblings for strawbreaker response. There were significant ($P < 0.05$) differences among populations and between siblings for grain yield in disease plots. Nonsignificance between siblings for D/C ratio (disease yield/control yield) may have been attributable to the small plot size. The sibling × treatment interaction for grain yield was significant ($P < 0.05$).

Analysis of variance for grain yield for the 1986 trial showed nonsignificance for all effects. Early crop growth was retarded because germination conditions were poor. Disease severity was very low so the stubble was not scored for infection by *P. herpotrichoides*.

Significant differences ($P < 0.01$) in 1987 appeared among population and sibling main effects and between treatments (disease and control). The population × sibling, population × treatment, and sibling × treatment interactions were also significant ($P < 0.01$), showing that changes in grain yield were inconsistent among populations and siblings. The second order interaction among these factors was not significant.

From the 10 sibling pairs included in the 1985 trials, seven were retained that showed differential response to strawbreaker in both 1985 and in 1987 (Table 4). The same sibling from each population was more susceptible to *P. herpotrichoides* in both years. Therefore, there was potential for differential yield loss between siblings under disease pressure. In 1987, the susceptible siblings from each population, except TYEE//RZN/TRES, had significant yield reduction in disease plots, whereas the resistant siblings did not. Starch gel electrophoresis revealed that only the siblings of the TYEE//RZN/TRES population were both homogeneous for the *EP-D1b* gene from *A. ventricosa*, causing its lack of response to the pathogen. There were no yield potential (control plots) differences between siblings in 1985 and in 1986. In 1987, only the susceptible siblings from populations VPM/M951//PECK/DAWS and TYEE//RZN/TRES had lower yield potentials than the resistant siblings. The anomalous result from the TYEE//RZN/TRES population was caused by severe lodging in control plots of the susceptible sibling and was probably a function of straw strength rather than disease response.

Siblings from five of the seven populations had similar yield potentials in three seasons but showed contrasting disease responses in two seasons. Therefore, it is possible for closely related lines, which differ in strawbreaker resistance, to have similar yield potentials. Resistance to the strawbreaker pathogen may be exploited in wheat breeding without causing a corresponding reduction in yield.

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