Effects of Host Resistance to *Pseudocercosporella herpotrichoides* and Foot Rot Severity on Yield and Yield Components in Winter Wheat

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ABSTRACT

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Yield of winter wheat in plots inoculated with *Pseudocercosporella herpotrichoides* was inversely related to disease severity. In plots where disease was severe, lodging was greater and yield, tillers per square meter, and 1,000-kernel weight were less than in plots where disease was mild. When disease was relatively mild, yield deficits resulted primarily from fewer tillers per square meter. Host resistance to *P. herpotrichoides* resulted in less disease and was equivalent to delayed inoculation. With severe disease, resistant cultivars had smaller reductions in tillers per square meter and 1,000-kernel weight than susceptible cultivars under similar conditions. Distribution of tillers in lesion classes revealed that even at severe levels of disease, some healthy tillers remained. Compensation by healthy tillers on plants with diseased tillers may result from reduced competition among tillers or adjacent plants.

Additional key words: disease resistance, eyespot, strawbreaker foot rot

Pseudocercosporella herpotrichoides (Fron) Deighton causes strawbreaker (eyespot) foot rot of wheat (*Triticum aestivum* L. em. Thell.), barley (*Hordeum vulgare* L.), oats (*Avena sativa* L.), and rye (*Secale cereale* L.). This fungus survives on infested host debris between crops. Conidia are produced on debris in the autumn and spring and are splashed to nearby plants. Infection occurs on leaf sheaths, and the fungus colonizes successive leaf sheaths and eventually the stem. Extracellular enzymes of the fungus degrade stem cell-wall materials, weakening the stem and resulting in lodging or dead stems that remain standing and form whiteheads. Yields may be less than 50% of that attainable without the disease; late autumn or early winter infection results in lower yields than infection in the spring (2,4).

Several reports concur that minor strawbreaker foot rot lesions have little or no effect on yield and yield components of wheat (3,5–8,12–15). In contrast, severe lesions may result in a lower 1,000-kernel weight, fewer kernels per head and tillers per square meter, and more lodging. Different authors have attributed lower yields as a result of severe lesions to effects on different yield components. Glynne et al (7) found that 1,000-kernel weight was reduced by severe lesions but that the number of kernels per head was not affected, whereas Doussinault (6) found reductions in number of kernels per head but not in 1,000-kernel weight. Clarkson (3) and Jorgensen (8) reported reductions in both 1,000-kernel weight and kernels per head for plants with severe lesions. Scott and Hollins (13) also found that 1,000-kernel weight and number of kernels per head were reduced on plants with severe lesions but that 1,000-kernel weight was more likely to be reduced than was number of kernels per head on plants with moderate lesions. Defosse and Rixhon (5) reported a progressively lower 1,000-kernel weight with increasing disease severity. Scott and Hollins (13,14) reported that yield was indirectly related to the amount of lodging caused by strawbreaker foot rot and that cultivars with resistance to *P. herpotrichoides* were unlikely to yield more than susceptible cultivars unless they also were resistant to lodging. They showed that lodging suppressed yield, number of kernels per head, and 1,000-kernel weight beyond that attributable to severity of foot rot lesions alone.

A few reports include evidence of compensation among plants or among infected and uninfected tillers on the same plant. For example, Glynne et al (7) found that plants with slight to moderate lesions yielded more than uninfected plants. They speculated that the greater yield may be due to less competition among the surviving plants as a result of death of more severely diseased plants. Scott and Hollins (13) found no difference in yield between inoculated and uninoculated treatments despite early counts that revealed an 11% reduction in tillers per square meter in the inoculated treatment. Ponchet (12) showed that compensation by individual tillers increased as the ratio of uninfected to infected tillers on a plant decreased. Jorgensen (8) found no evidence of compensation.

The purpose of this study was to determine how yield and yield components are affected by host resistance and disease severity.

MATERIALS AND METHODS

Cultivars and locations of experiments. Six cultivars of wheat were selected for this study (11): VPM-1124R251 (VPM), highly resistant; Cappelle-Desprez (Cappelle) (PI 262223), resistant; Viking (PI 316424), moderately resistant; and Sprague (CI 15376), Daws (CI 17419), and Selection 101 (Sel 101) (CI 13438), all susceptible. These cultivars were grown in the 1980–1981, 1981–1982, and 1983–1984 crop years (hereafter referred to as 1981, 1982 and 1984, respectively) at the Palouse Conservation Field Station, Pullman, WA, and in 1982 (1982 Lind) at the Washington State University Dryland Research Unit, Lind, WA. In addition, Nugaines wheat (CI 13968), susceptible, was used in a study of the effect of disease severity on yield and yield components at Lind during 1981–1982.

Cultural practices and experimental design. At Pullman, the plots were located on Naff silt loam in 1981 and 1984 and on Thatuna silt loam in 1982; plots were on Wheeler loam at Lind in 1982. Seeding dates were 16 September 1980 and 1983 and 25 September 1981 at Pullman and 2 September 1981 at Lind. All cultivars were seeded in four-row plots with a row spacing of 40.6 cm. Plot size was 1.8 × 3.0 m in 1981, 1982, and 1982 (Lind) and 1.8 × 6.1 m in 1984. Plots within experiments were arranged in a randomized complete block, split-plot design (main plot = cultivar, subplot = inoculation) in 1981 and 1984 and in randomized complete block, split-block design in 1982, with the inoculation treatment applied across cultivars within blocks. Six replicates were used in all years except 1981, when there were four replicates. Seeding rate was constant among cultivars within experiments at 40 seeds per meter in 1981, 1982, and 1982 (Lind) and 80 seeds per meter in 1984.

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Plots were inoculated in late November each year by spraying a freshly prepared conidial suspension ($2–3 \times 10^5$/ml) near the crown until visible runoff (2). At inoculation, plants in 1981, 1982 (Lind), and 1984 were in the four- to 10-tiller stage, and plants in 1982 had one tiller. All uninoculated plots were sprayed with benomyl (1 lb a.i./acre) in the spring to prevent disease development (1).

Manipulation of disease severity. Different levels of disease severity were obtained by inoculating plots at different times. Nugaines wheat was seeded on 2 September 1981 at Lind in four-row plots arranged in a completely random design with five replicates. Plants were inoculated with a conidial suspension on four dates: 3 November, 3 December, 19 February, and 16 March 1982. Plant growth stage at inoculation ranged from four to 10 tillers per plant to jointing.

Determination of yield components and yield. Numbers of tillers per square meter and spikelets per head were determined about 1 mo before harvest. The number of kernels per head was calculated from the number of tillers per square meter, yield, and 1,000-kernel weight, where number of kernels per head = [(yield per square meter)/kernel weight]/(tillers per square meter). The 1,000-kernel weight was determined by randomly selecting 1,000 kernels by hand from cleaned samples of grain.

Yield in 1981 and 1982 (Pullman and Lind) was determined by cutting one or two of the center rows from each plot, bundling the stems, and threshing with a Vogel stationary bundle thresher. In 1984, entire plots were harvested with a Wintersteiger plot combine. Length of row harvested was measured and used to calculate yields. One-meter sections of row were harvested to determine yield for the date of inoculation study in 1982 at Lind.

Determination of disease severity. Individual tillers were rated for lesion severity on a scale of 0–4 (11), where 0 = no lesions (healthy) and 4 = severe lesions girdling the tiller or dead tiller. A mean was calculated and used as the disease index. Disease severity was determined at the boot to heading growth stage on 50–100 tillers per plot.

Statistical analysis. Each experiment was analyzed separately because of differences in experimental design and number of replicates. Experiments were analyzed according to their design (listed before), using plot means to determine significance of cultivar and inoculation main effects and if significant interactions occurred. Yield and yield components expressed as a percentage of the uninoculated control ([inoculated/uninoculated] × 100) were analyzed as a randomized complete block design to determine differences among cultivars. The protected least significant difference (LSD) was used to test for significant differences between inoculated and uninoculated treatments within cultivars and differences among cultivars for yield components expressed as a percentage of the uninoculated control.

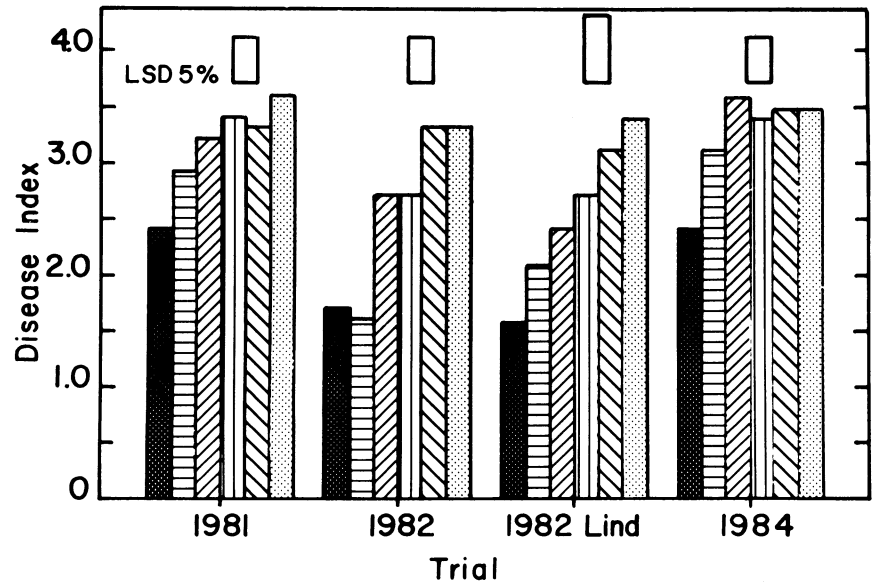

Fig. 1. Strawbreaker foot rot disease indexes of six winter wheat cultivars inoculated with *Pseudocercosporella herpotrichoides* in four trials. The disease index is based on a scale of 0–4, where 0 = healthy tiller with no lesions and 4 = severe lesion girdling the tiller or a dead tiller. The protected least significant difference is for comparison of means within trials ($P = 0.05$). For each year or location, bars from left to right represent VPM, Cappelle-Desprez, Viking, Sprague, Daws, and Sel 101.

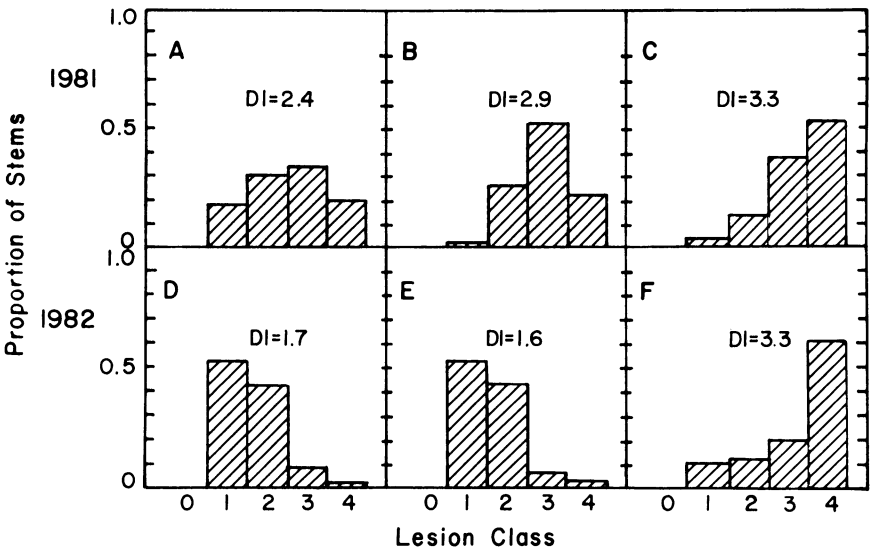

Fig. 2. Distribution of tillers in lesion severity classes of three wheat cultivars inoculated with *Pseudocercosporella herpotrichoides* at Pullman, WA, 1981 and 1982. Disease index is based on a scale of 0–4, where 0 = healthy tiller and 4 = dead tiller. A and D = VPM, B and E = Cappelle-Desprez, and C and F = Daws.

RESULTS

Cultivar trial. The most resistant cultivar, VPM, had the lowest disease index in all years, followed by the resistant Cappelle, the moderately resistant Viking, and the susceptible Sprague, Daws, and Sel 101 (Fig. 1). The lowest average disease index occurred in 1982 at Pullman and Lind (disease indexes = 2.5 and 2.6, respectively) and the highest in 1984 and 1981 (disease indexes = 3.2 and 3.1, respectively); the cultivars were ranked about the same each year. Disease was absent or less than disease index = 0.2 in uninoculated plots sprayed with benomyl in all years. In general, as disease severity increased, the frequency of tillers with severe lesions increased (Fig. 2). Even cultivars with a low disease index had some tillers with severe lesions (Fig. 2A,D,E). Conversely, at high disease indexes, some tillers remained relatively healthy (Fig. 2B,C,F).

Inoculated plots averaged 29–42% more lodging than uninoculated plots in all Pullman trials (Table 1); lodging was most severe in 1981 and 1984 with many uninoculated plots lodging extensively. Lodging did not occur at Lind (1982).

Cultivar × inoculation interactions were significant where lodging occurred; in 1981 and 1984, when the most lodging occurred, differences between inoculated and uninoculated treatments were least for Sprague and VPM, both of which have a propensity for lodging in the absence of disease.

Yield was consistently lower in inoculated than in uninoculated plots (Table 2), and significant cultivar × inoculation interactions occurred at all locations except Lind 1982. The difference in yield between inoculated and uninoculated plots was greatest in 1981 (2,849 kg/ha), followed by 1984 (1,950 kg/ha), 1982 (911 kg/ha), and Lind 1982 (314 kg/ha). Yields of inoculated plots were 61, 73, 84, and 88% of the yields of uninoculated plots, respectively. At Pullman, yields of Sprague, Daws, and Sel 101 (all susceptible) in inoculated plots were significantly lower than uninoculated plots in all trials and varied from a low of 42% of the uninoculated control to a high of 88% of the control. Yields of inoculated Cappelle (resistant) and Viking (susceptible) were significantly less than those of the uninoculated controls only in 1981 and 1984, yielding between 72 and 78% of the control, respectively. Yield of inoculated VPM (resistant) in 1984 was 82% of the uninoculated control; this was the only year in which the yield of inoculated plots of VPM was significantly less than those of uninoculated plots. In 1981, yield expressed as percentage of the control was highest in VPM followed by Cappelle, Viking, and the susceptible cultivars Sprague, Daws, and Sel 101. This same trend was apparent in 1984, except Sprague had the highest percent yield. This apparent anomaly is probably due to the severe lodging of Sprague in inoculated and uninoculated plots. Differences among cultivars for the percentage of the uninoculated control were not significant although there was a trend toward a higher percentage in the resistant cultivars in 1982 at Pullman. At Lind in 1982, average yield of all cultivars was less in inoculated than in uninoculated plots, and all cultivars were affected about the same, except Sel 101, which had a yield of only 72% of the control compared with 89–97% for the other cultivars.

Significantly fewer tillers per square meter were present in inoculated plots in all trials, except Lind 1982 (Table 3). The largest differences occurred in 1981 and 1984, with 259 and 239 fewer tillers per square meter, respectively, representing 69 and 73% of the control. A cultivar × inoculation interaction was present only in 1981; neither VPM nor Cappelle had significantly fewer tillers per square meter in inoculated treatments, but all of the susceptible cultivars had fewer tillers per square meter in inoculated treatments. When the data were expressed as a percentage of the uninoculated control, however, VPM and Cappelle differed significantly only from Sel 101. In 1982 and 1984 at Pullman, the effect of inoculation on tillers per square meter was similar for all cultivars, ranging from 89 to 99% and 65 to 83% of the control, respectively. At Lind in 1982, significant differences in tillers per square meter existed among cultivars, but they were not related to resistance.

The 1,000-kernel weight was significantly reduced in inoculated plots in 1981 and 1984 but not in 1982 at either Pullman or Lind (Table 4). A significant cultivar × inoculation interaction occurred in 1981 and 1984, but it was not completely related to host resistance. In 1981, 1,000-kernel weight, as a percentage of the control, was generally higher in resistant than susceptible cultivars. In 1984, differences among resistant and susceptible cultivars did not appear to be related to resistance.

Test weight (volumetric weight) was significantly less in inoculated than

Table 1. Percent lodging of six wheat cultivars inoculated and not inoculated with *Pseudocercosporella herpotrichoides* in three years at Pullman, WA

Cultivar[a]	Treatment[b]	Lodging[c] (%) 1981	1982	1984
VPM	I	95	3	82
	NI	88	3	63*
Cappelle	I	96	1	84
	NI	54*	0	45*
Viking	I	94	3	78
	NI	19*	1	42*
Sprague	I	100	80	100
	NI	97	8*	100
Daws	I	100	31	96
	NI	33*	7*	43*
Sel 101	I	100	78	100
	NI	48*	3*	62*
Mean	I	98	33	90
	NI	56	4	59
LSD[d]		23	8	18

[a] Cultivars listed in decreasing order of resistance to *P. herpotrichoides*.
[b] Treatments: I = inoculated and NI = not inoculated.
[c] Lodging was estimated visually.
[d] Protected least significant difference for comparing inoculated and uninoculated treatments within cultivars. * = Significant difference ($P = 0.05$).

Table 2. Yield of six winter wheat cultivars inoculated and not inoculated with *Pseudocercosporella herpotrichoides* and the percentage of the uninoculated treatment in four trials

Cultivar[a]	Treatment[b]	Yield (kg/ha and % NI)[c] Pullman (1981)	Pullman (1982)	Lind (1982)	Pullman (1984)
VPM	I	4,622 (98)	4,041 (94)	2,163 (89)	4,603 (82)
	NI	4,759	4,313	2,458	5,698*
Cappelle	I	5,093 (74)	4,248 (106)	2,234 (90)	4,817 (78)
	NI	6,872*	4,603	2,438	6,232*
Viking	I	4,974 (73)	5,458 (96)	2,596 (97)	5,351 (72)
	NI	6,813*	5,670	2,731	7,447*
Sprague	I	2,892 (42)	4,604 (79)	2,658 (92)	5,736 (88)
	NI	6,948*	5,882*	2,906	6,613*
Daws	I	5,567 (58)	5,615 (84)	2,499 (95)	6,441 (70)
	NI	9,633*	6,678*	2,673	9,296*
Sel 101	I	3,685 (42)	3,933 (64)	2,036 (72)	5,050 (60)
	NI	8,904*	6,220*	2,865	8,410*
Mean[d]	I	4,472	4,650	2,364	5,333
	NI	7,321	5,561	2,678**	7,283
LSD[e]		50	1,012		659
LSD[f]		17	ns	ns	13

[a] Cultivars listed in decreasing order of resistance to *P. herpotrichoides*.
[b] Treatments: I = inoculated and NI = not inoculated.
[c] Percentage of control given in parentheses: %NI = (N/NI) × 100. The figures presented were not calculated from the I and NI means and may differ because of rounding-off.
[d] ** = Significant difference ($P = 0.05$) between inoculated and uninoculated treatment means based on F tests. Nonsignificant cultivar × inoculation interaction allows comparison of main effect treatment means; ns = not significant according to F tests.
[e] Protected least significant difference. Values given are for comparing inoculated and uninoculated treatments among cultivars. * = Significant difference ($P = 0.05$).
[f] Protected least significant difference for comparing the percentage of NI among cultivars ($P = 0.05$).

uninoculated plots in 1981 and 1984 but not in 1982 (Table 5). Differences among cultivars were not significant, and there was no interaction between cultivar and inoculation in any trial. When the data were expressed as a percentage of the control, however, there was a trend toward higher test weight among resistant cultivars in both 1981 and 1982 at Pullman. This trend was not present in 1984 at Pullman or in 1982 at Lind.

The number of kernels per head was not significantly reduced by inoculation in any trial (Table 6); in 1984, however, there were more kernels per head in the inoculated plots. Differences occurred among cultivars but were not significant in any trial. The number of spikelets per head was not affected by inoculation and differences among cultivars were independent of resistance to *P. herpotrichoides*.

Effect of disease severity. Earlier inoculations resulted in more severe disease (Table 7), ranging from a disease index of 3.7 with the November inoculation to 1.2 with the February and March inoculations. At Lind, tillers were distributed predominately in either mild or severe lesion classes, even for intermediate disease indexes (Fig. 3); for a given disease index, tillers were either relatively healthy or nearly dead.

Yield, tillers per square meter, and number of kernels per head followed similar patterns, i.e., the more severe disease the lower the yield and the fewer the tillers per square meter and kernels per head. Yields ranged from 48% of the uninoculated control with the November inoculation to 94% of the control with the March inoculation. Tillers per square meter ranged from 65 to 86% of the control and number of kernels per head ranged from 77 to 91% of the control for the December and March inoculations, respectively. The 1,000-kernel weight and number of spikelets per head were not affected by inoculation at Lind in 1982.

Table 3. Number of tillers per square meter for six wheat cultivars inoculated and not inoculated with *Pseudocercosporella herpotrichoides* and the percentage of the uninoculated treatment in four trials

		Tillers/m² and %NI[c]			
Cultivar[a]	Treatment[b]	Pullman (1981)	Pullman (1982)	Lind (1982)	Pullman (1984)
VPM	I	654 (82)	368 (89)	372 (89)	618 (83)
	NI	800	419	419	738
Cappelle	I	665 (88)	393 (93)	489 (109)	586 (76)
	NI	757	421	454	800
Viking	I	521 (72)	375 (94)	416 (116)	578 (65)
	NI	733*	404	361	897
Sprague	I	653 (65)	535 (90)	648 (104)	710 (81)
	NI	1,031*	607	630	902
Daws	I	577 (70)	404 (99)	398 (92)	588 (77)
	NI	813*	415	437	807
Sel 101	I	388 (46)	412 (92)	527 (91)	719 (67)
	NI	878*	459	597	1,069
Mean[d]	I	576	415	475	631
	NI	835	454**	483 ns	870**
LSD[e]		173	...	...	...
LSD[f]		24	ns	19	ns

[a] Cultivars listed in decreasing order of resistance to *P. herpotrichoides*.
[b] Treatment: I = inoculated and NI = not inoculated.
[c] Percentage of control given in parentheses: %NI = (I/NI) × 100. The figures presented were not calculated from the I and NI means and may differ because of rounding-off.
[d] Significant difference ($P = 0.05$) between inoculated and uninoculated treatment means based on F tests. Nonsignificant cultivar × inoculation interaction allows comparison of treatment means; ns = not significant according to F tests.
[e] Protected least significant difference. Values given are for comparing inoculated and uninoculated treatments within cultivars. * = Significant different ($P = 0.05$).
[f] Protected least significant difference for comparing the percentage of NI among cultivars ($P = 0.05$).

Table 4. One-thousand-kernel weights of six wheat cultivars inoculated and not inoculated with *Pseudocercosporella herpotrichoides* and the percentage of the uninoculated treatment in four trials

		1,000-Kernel weight (g) and %NI[c]			
Cultivar[a]	Treatment[b]	Pullman (1981)	Pullman (1982)	Lind (1982)	Pullman (1984)
VPM	I	35 (96)	40 (101)	36 (99)	37 (97)
	NI	37	40	37	39
Cappelle	I	31 (79)	43 (104)	38 (100)	35 (85)
	NI	40*	42	38	41*
Viking	I	23 (84)	31 (98)	29 (100)	29 (89)
	NI	27*	31	29	33*
Sprague	I	24 (80)	31 (99)	27 (96)	31 (98)
	NI	30*	31	28	31
Daws	I	29 (74)	37 (102)	36 (101)	35 (85)
	NI	39*	37	36	41*
Sel 101	I	24 (73)	31 (92)	29 (98)	30 (91)
	NI	33*	34	30	33*
Mean	I	28	35	33	33
	NI	34	36 ns	33 ns	36
LSD[d]		2	...	...	2
LSD[e]		8	7	ns	8

[a] Cultivars listed in decreasing order of resistance to *P. herpotrichoides*.
[b] Treatment: I = inoculated and NI = not inoculated.
[c] Percentage of control given in parentheses: %NI = (I/NI) × 100. The figures presented were not calculated from the I and NI means and may differ because of rounding-off.
[d] Protected least significant difference. Values given are for comparing inoculated and uninoculated treatments within cultivars. * = Significant difference ($P = 0.05$); ns = not significant according to F tests.
[e] Protected least significant difference for comparing the percentage of NI among cultivars ($P = 0.05$).

DISCUSSION

Not surprisingly, the effect of disease on yield, averaged across cultivars, was greatest when disease severity was greatest. Lower yields at Pullman result primarily from fewer tillers per square meter and lower kernel weight. In addition, quality of the grain is reduced as reflected by reductions in test weight. When disease is severe, cultivar and disease interactions occur, and resistant cultivars have smaller reductions in tillers per square meter and kernel weight than susceptible cultivars under similar disease levels. However, when disease is less severe, interactions between cultivars and disease do not occur and lower yields are due primarily to fewer tillers per square meter. The effect of disease on yield components is related to both host resistance and the genetic yield potential of the cultivar. Cultivars with high yield potential (e.g., Daws) can overcome to a large extent the effects of severe disease and outperform resistant cultivars with less severe disease (Tables 2–4). Resistance to *P. herpotrichoides* has the same effect on yield and yield components as does delaying inoculation; i.e., as inoculation is delayed, disease becomes less severe and the relative reductions in tillers per square meter and kernels per head become smaller.

Significant differences in the number of kernels per head and 1,000-kernel weight were found only when disease was severe. Under such conditions, 1,000-kernel weight was lower and the number

of kernels per head increased slightly in inoculated plots. The increased number of kernels per head is probably due to compensation, either among plants or among tillers of a single plant as has been previously suggested (7,13). As disease severity increases, the proportion of tillers with severe lesions increases, but even with severe disease, some healthy or nearly healthy tillers are still present (Figs. 2 and 3). Presumably, these healthy tillers are subject to less competition than similar tillers on completely healthy plants and produce more kernels per head. The presence of healthy tillers among infected tillers may explain the lack of differences in number of kernels per head and 1,000-kernel weight at lower disease levels. Compensation by healthy tillers on plants with mild lesions may continue until disease becomes so severe that no healthy tillers remain.

The 1982 Lind plots provided an environment very different from the Pullman plots. Average annual precipitation is 25 cm at Lind compared with 53 cm at Pullman. The relatively dry environment must subject plants to additional stress. Once infection occurs, colonization and death of infected tillers appears to proceed rapidly so that tillers are either relatively healthy or nearly dead (Fig. 3). Under these circumstances, the expected primary effect of strawbreaker foot rot might be a reduction of tillers per square meter; however, significant differences in the number of tillers per square meter were not detected until disease became severe (Tables 3 and 7). The number of kernels per head was the only other yield component affected at Lind and, like tillers per square meter, was only reduced when disease was severe (Table 7). The data from the cultivar trials at Lind are consistent with those from the date of inoculation study; yield reductions could be accounted for by fewer tillers per square meter and kernels per head (Tables 3 and 6), although significant differences were not detected between treatments or among cultivars. Environmental stresses, especially moisture stress, can lead to abortion of tillers on healthy plants (9,10). The severity of stress determines the number of tillers that can be supported, and the timing of stress determines which tillers are aborted (9,10). At Lind, all cultivars probably produce excess tillers that are later aborted. Infected tillers may be aborted first in inoculated plots, whereas healthy tillers are aborted in uninoculated plots, thereby resulting in similar numbers of tillers per square meter. Most of these are relatively healthy and probably have similar numbers of kernels per head. Only at the most severe level of disease do reductions in tillers per square meter and kernels per head become significant (Table 7).

The distributions of tillers in lesion classes emphasize the importance of determining severity of strawbreaker foot rot lesions rather than percentage of infected tillers (Figs. 2 and 3). In each case, there was 100% infected tillers, yet the effect on yield and yield components were quite different depending on lesion severity.

Our results support the conclusions of Scott and Hollins in England (13); differences in yield between resistant and susceptible cultivars could not be explained by differences in lodging alone. For example, both VPM and Sel 101 lodged severely in 1981 and 1984, yet inoculation had a much greater effect on yield of Sel 101 than on yield of VPM. Lodging was not the primary determinant of yield, although it was related, and the direct effects of the pathogen on yield must have been much greater on the very susceptible Sel 101 than on VPM. In contrast to other conclusions (14), however, we found that the degree of yield reduction was not always closely associated with lodging. At Lind, yield was severely reduced but lodging was

Table 5. Test weight (volumetric weight) of six winter wheat cultivars inoculated and not inoculated with *Pseudocercosporella herpotrichoides* and the percentage of the uninoculated treatment in four trials

		Test weight (kg/m^3 and %NI)[c]			
Cultivar[a]	Treatment[b]	Pullman (1981)	Pullman (1982)	Lind (1982)	Pullman (1984)
VPM	I	721 (99)	735 (101)	718 (99)	730 (97)
	NI	721	730	721	750
Cappelle	I	708 (95)	744 (101)	705 (100)	713 (94)
	NI	747	736	704	757
Viking	I	704 (95)	759 (99)	736 (105)	714 (96)
	NI	745	763	704	745
Sprague	I	720 (95)	775 (99)	739 (99)	718 (98)
	NI	762	785	745	732
Daws	I	716 (93)	778 (99)	744 (99)	735 (94)
	NI	772	779	748	779
Sel 101	I	721 (92)	775 (98)	757 (99)	720 (95)
	NI	780	794	759	759
Mean[d]	I	715	762	734	722
	NI	754**	766 ns	730 ns	754**
LSD[e]		3	2	2	ns

[a] Cultivars listed in decreasing order of resistance to *P. herpotrichoides*.
[b] Treatment: I = inoculated and NI = not inoculated.
[c] Percentage of control given in parentheses: %NI = (I/NI) × 100. The figures presented were not calculated from the I and NI means and may differ because of rounding-off.
[d] ** = Significant difference between inoculated and uninoculated treatment means based on *F* tests ($P = 0.05$); ns = not significant according to *F* tests.
[e] Protected least significant difference for comparing the percentage of NI among cultivars ($P = 0.05$).

Table 6. Number of kernels per head of six wheat cultivars inoculated and not inoculated with *Pseudocercosporella herpotrichoides* and the percentage of the uninoculated treatment in four trials

		Number of kernels/head and %NI[c]			
Cultivar[a]	Treatment[b]	Pullman (1981)	Pullman (1982)	Lind (1982)	Pullman (1984)
VPM	I	20 (125)	28 (108)	16 (101)	21 (105)
	NI	16	26	16	20
Cappelle	I	25 (108)	25 (93)	12 (85)	25 (130)
	NI	24	27	14	21
Viking	I	43 (126)	48 (106)	22 (85)	34 (131)
	NI	35	47	26	26
Sprague	I	19 (85)	29 (91)	15 (93)	28 (117)
	NI	23	32	17	25
Daws	I	35 (115)	37 (86)	18 (103)	35 (118)
	NI	31	45	17	31
Sel 101	I	43 (149)	31 (79)	13 (84)	27 (107)
	NI	32	40	17	26
Mean[d]	I	31	33	16	28
	NI	27 ns	36 ns	18 ns	25**
LSD[e]		ns	ns	ns	ns

[a] Cultivars listed in decreasing order of resistance to *P. herpotrichoides*.
[b] Treatment: I = inoculated and NI = not inoculated.
[c] Percentage of control given in parentheses: %NI = (I/NI) × 100. The figures presented were not calculated from the I and NI means and may differ because of rounding-off.
[d] ** = Significant difference between inoculated and uninoculated treatment means according to *F* tests ($P = 0.05$); ns = not significant according to *F* tests.
[e] Protected least significant difference for comparing the percentage of NI among cultivars ($P = 0.05$); ns = not significant according to *F* tests.

Table 7. Effect of date of inoculation with *Pseudocercosporella herpotrichoides* on yield and yield components of Nugaines winter wheat in 1982 at Lind, WA

Date	Percentage of uninoculated control[a]					
	Disease index[b]	Yield (g/m)	Tillers/ m^2	Kernels/ head	1,000-Kernel weight	Spikelets/ head
3 November	3.7	48	65	77	97	109
3 December	2.5	70	90	82	97	100
19 February	1.2	91	97	91	97	100
16 March	1.2	94	86	91	100	100
Control	0.3	100	100	100	100	100
LSD[c]	0.3	25	22	14	ns	ns

[a] Data expressed as a percentage of the uninoculated control.
[b] Disease index based on a scale of 0–4, where 0 = healthy tiller with no lesions and 4 = severe lesion girdling the tiller or dead tiller.
[c] Protected least significant difference for comparing means within columns ($P = 0.05$); ns = not significant according to F tests.

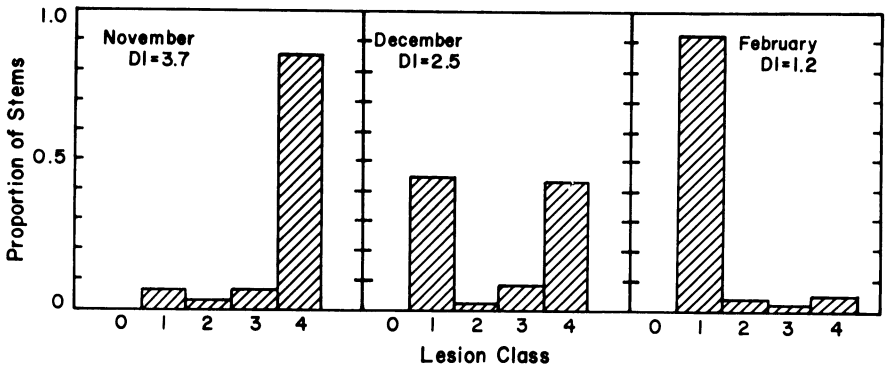

Fig. 3. Distribution of tillers in lesion severity classes of Nugaines wheat inoculated at different dates with *Pseudocercosporella herpotrichoides* at Lind, WA, 1982. Disease index based on a scale of 0–4, where 0 = healthy tiller and 4 = dead tiller.

negligible (Tables 2 and 7).

Screening for resistance to *P. herpotrichoides* in the field has relied on measurement of yield differences between inoculated and uninoculated plots. However, yield is controlled by many factors other than disease and is subject to large effects of environment necessitating testing at several locations over many years. Disease index measurements are reliable from year to year, have the advantage of identifying, with certainty, resistant genotypes, and are therefore better indicators of resistance than measuring yield differences in inoculated plots. In this study, differentiation of cultivars for resistance to *P. herpotrichoides* was better at Lind than at Pullman (Fig. 1). Screening genotypes for resistance to *P. herpotrichoides* in the field might be hastened by using early inoculation to induce intense disease pressure under which cultivar and disease interactions appear, by measuring disease index, and by growing cultivars under arid conditions such as occur at Lind.

The development of cultivars with high levels of resistance to *P. herpotrichoides* should allow earlier seeding than is currently feasible, resulting in less disease, higher yield potential, and less soil erosion. It appears, however, that even with the level of resistance present in VPM-1, there will be occasions when fungicides will be needed to achieve the full yield potential.

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