Reduction in Yield Loss Using Incomplete Resistance to *Pyrenophora teres* f. *teres* in Barley

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ABSTRACT

Steffenson, B. J., Webster, R. K., and Jackson, L. F. 1991. Reduction in yield loss using incomplete resistance to *Pyrenophora teres* f. *teres* in barley. Plant Dis. 75:96-100.

Disease development and yield loss due to the net blotch pathogen (*Pyrenophora teres* f. *teres*) were investigated in two barley genotypes, UC 603 and Kombar. These genotypes show moderately high to high (moderately susceptible to susceptible) infection responses (based on lesion size and degree of associated chlorosis) to *P. t.* f. *teres* in both the seedling (growth chamber study) and adult plant (field study) stages, but disease development is much reduced on UC 603 in the field. During the severe epidemic season of 1985–1986, terminal severity and area under the disease progress curve values were high (100% and 4,153, respectively) for plots of Kombar not protected with a fungicide and moderate (29.6% and 934, respectively) for unprotected plots of UC 603. Yield losses in plots where net blotch was not controlled with the fungicide propiconazole were 35.3% for Kombar and 3.2% for UC 603. Net blotch severity was lower during the second season (1986–1987); nevertheless, yield loss was 31% in unprotected plots of Kombar compared with 5.3% for UC 603. Kernel weight and size were the yield components most affected by *P. t.* f. *teres*. Propiconazole effectively controlled net blotch in the sprayed treatments; disease severities were all less than 2%. The data indicate that the incomplete resistance of UC 603 can be highly effective in reducing yield loss due to *P. t.* f. *teres*.

Net blotch, caused by *Pyrenophora teres* Drechs. f. *teres* Smedeg. (anamorph *Drechslera teres* (Sacc.) Shoemaker f. *teres* Smedeg.), is a common disease of barley (*Hordeum vulgare* L. emend Bowden) throughout most of the cereal-growing regions of the world. The disease has usually been considered of minor importance but in some regions has become a significant factor in reducing production (23). Yield losses ranging from 9 to 77% have been reported in barley (1,5,6,9,12–19,21–24,27). In California, an epidemic of net blotch developed on the cultivar Kombar in the winter of 1979, and yield losses were so severe that some growers did not harvest their grain. The area planted to Kombar dropped precipitously after the epidemic, from 123,849 ha in 1978 to 3,645 ha in 1981. Unfortunately, no reliable estimates of yield loss were documented for this epidemic.

Disease resistance has been a major strategy in controlling net blotch of barley in California. In a previous study, several barley genotypes were evaluated for incomplete resistance to *P. t.* f. *teres*, and one promising entry identified from this work was the cultivar UC 603 (26). UC 603 shows moderately high to high (moderately susceptible to susceptible) infection responses (based on the size of the lesion and degree of associated chlorosis) to the net blotch pathogen in the seedling (growth chamber study) and adult plant (field study) stages but sustains less disease than fully susceptible genotypes in the field (26). Genotypes with incomplete resistance (also partial or slow-rusting resistance in the cereal rust systems) do endure a moderate level of disease during epidemics and may not be acceptable to growers who believe that yield will be significantly reduced by even moderate levels of disease. However, moderate levels of disease do not necessarily lead to significant reductions in yield, as demonstrated in the barley:leaf rust pathogen system (11). No known studies have been advanced on the ability of genotypes possessing incomplete resistance to attain satisfactory yields in the barley:net blotch pathogen system. The objectives of this study were to determine: 1) the degree of protection against yield loss conferred by the incomplete resistance of UC 603 and 2) the potential yield loss due to net blotch in the susceptible cultivar Kombar.

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Accepted for publication 5 July 1990 (submitted for electronic processing).

MATERIALS AND METHODS

Experimental design. Experiments were conducted at the University of California Armstrong Plant Pathology Farm near Davis during 1985–1986 and 1986–1987 using genotypes UC 603 (PI 537576) and Kombar (CI 15694); both are six-rowed spring feed barleys. UC 603 is a recently released cultivar (1988) that possesses incomplete resistance to *P. t.* f. *teres* (26), and Kombar is a cultivar that is susceptible to many pathotypes of *P. t.* f. *teres* in California (26). UC 603 heads about 5 days earlier than Kombar. Entries were sown in 1.2 × 6.7 m plots at a rate of 108 kg/ha of seed on 22 October 1985 and 24 October 1986. Plots of each genotype to be inoculated with *P. t.* f. *teres* or sprayed with the fungicide propiconazole and left uninoculated were assigned at random. A completely randomized design was used with five replicates per treatment during 1985–1986 and six during 1986–1987. To limit the spread of spores from plot to plot, buffer rows (6.1 m wide) of triticale (× *Triticosecale* Wittm.) cultivar Juan were planted between the barley plots. Unsprayed plots were inoculated by spreading 1.45 kg of barley straw naturally infected with *P. t.* f. *teres* over the plants on 13 January 1986 and 8 January 1987; these inoculations were made when the plants were in the seedling to early tillering stage of growth—growth stage (GS) 15 during 1985–1986 and GS 23 during 1986–1987 (29).

Propiconazole (3.6EC) was applied at a rate of 0.22 kg a.i./ha to control net blotch in the protected plots. During 1985–1986, two applications were made: on 1 February 1986 when UC 603 and Kombar were at GS 20 (early tillering) and 3 wk later when UC 603 and Kombar were at GS 31 (first node detectable) and GS 30 (pseudostem erection), respectively. The following season, three applications were made: the first on 14 February (UC 603 at GS 30 and Kombar at GS 28 [late tillering]), the second on 6 March (UC 603 at GS 33 [third node detectable] and Kombar at GS 31), and the third on 10 April (UC 603 at GS 68 [anthesis complete] and Kombar at GS 63 [midanthesis]).

Another experiment was conducted to test the effect of propiconazole on the yield of barley in the absence of net blotch. The experimental design and methods were identical to those previously described except there were three replicates in the 1985–1986 trial and four in the 1986–1987 trial, plots were not inoculated with *P. t.* f. *teres*, and buffer rows of triticale were 3 m wide. In the first season, this experiment was separated from the yield-loss experiment by 6.1 m of triticale and in the second year, by over 120 m.

Assessment of disease. Traces of net blotch were seen about 2 wk after inoculation in the plots treated with infected straw. The buffer rows of triticale were not completely effective in limiting the spread of conidia, since infections were observed in the protected plots several weeks after they were first observed in the inoculated plots. From the time infections were first observed, disease was assessed every 7–8 days throughout the growing season until the crop was mature. The percentage of leaf area affected by net blotch (both necrotic and chlorotic areas) was estimated using a rating key developed by Burleigh and Loubane (3). In the sampling procedure, 15 plants within each plot were evaluated. Three of these 15 plants were tagged and rated during every assessment period in order to more easily follow the development and subsequent senescence of individual leaves. The severity of net blotch on the top three leaves from each selected tiller was averaged to derive a mean disease severity per tiller. Three aspects of net blotch development were considered in this study: 1) terminal severity (TS), 2) area under the disease progress curve (AUDPC), and 3) the apparent infection rate (r) sensu Vanderplank (28). Terminal net blotch severity was assessed near the end of the season at GS 87 (hard dough). AUDPC and r were calculated using methods previously described (26). Differences between treatment means were tested for statistical significance ($P \leq 0.05$) using the t test.

Assessment of yield and yield components. When the plants were completely senescent (GS 90 [hardening of caryopsis]), the numbers of kernels per ear, ears per meter row, and tillers per meter row were determined. The number of kernels per ear was evaluated by taking the mean number of kernels from 10 randomly selected ears in each plot, and the other two components were enumerated from a random 1-m section of row in each plot. After the experiment was harvested, the grain weight per plot (converted to calculated yield in kilograms per hectare), thousand kernel weight (TKW), and kernel size (plumpness) were assessed. Kernel plumpness was measured by means of a malting barley kernel sizing machine that vigorously shakes a seed sample through four individual sieves with slotted perforations 19.10 mm long × 3.18, 2.78, 2.38, or 1.99 mm wide. A random 100-g sample of seed was shaken for 30 sec on the sizing device, after which the weight of seed caught in each sieve was measured. Treatment means within a cultivar and year were tested for significant differences ($P \leq 0.05$) using the t test.

RESULTS

Net blotch development was greater during 1985–1986 than during 1986–1987 on the unsprayed plots of Kombar and UC 603 (Fig. 1). During 1985–1986, net blotch developed rapidly on Kombar after Julian day (JD) 80 (21 March), and by JD 126 (6 May), 100% of the leaf tissue was diseased (Fig. 1A). Rapid development of net blotch occurred about 2 wk later on UC 603 than on Kombar and plateaued just below 30% severity. During 1986–1987, the rapid increase of disease on Kombar occurred at approximately the same time as the previous year, but the relative progression of disease was slower (Fig. 1B). Net blotch severity on UC 603 was nearly nil for the entire season. The TS of net blotch was high (100 and 85.4%) in the unsprayed plots of Kombar during 1985–1986 and 1986–1987, respectively, whereas the TS for UC 603 was 29.6 and 0.7%, respectively (Table 1). This same trend was also seen in the AUDPC values for these two treatments. The apparent infection rate for unsprayed plots of Kombar was significantly higher than that for unsprayed plots of UC 603 in both years. Two or three applications of propiconazole effectively controlled *P. t.* f. *teres* in this study, as the sprayed plots had no more than 1.8% TS.

Sprayed plots of Kombar significantly outyielded unsprayed plots by 35.3 and 31.0% during 1985–1986 and 1986–1987, respectively (Table 2). Significant increases of 31.6 and 18.5% in TKW were also found with sprayed plots of Kombar

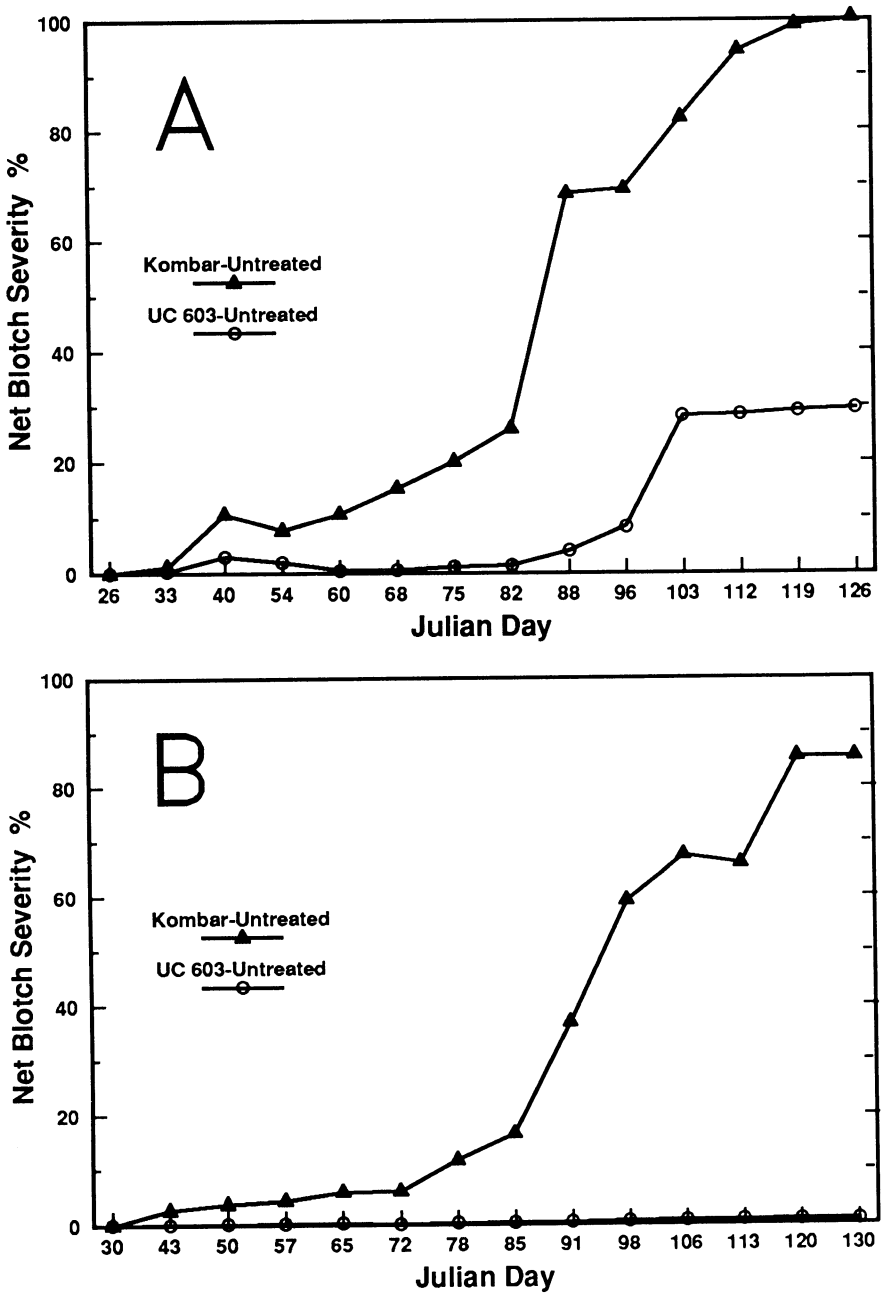

Fig. 1. Disease progress curves for unsprayed plots of barley cultivars Kombar and UC 603 inoculated with *Pyrenophora teres* f. *teres* during **(A)** 1985–1986 and **(B)** 1986–1987. Net blotch severities were assessed using the scale of Burleigh and Loubane (3) and are the means of infection percentages for the top three leaves. The data points are the means of five replicates during 1985–1986 and six replicates during 1986–1987.

during the two seasons. Sprayed plots of UC 603 showed increases of 3.2 and 5.3% for yield and 3.9 and 3.5% for TKW during 1985–1986 and 1986–1987, respectively, but these values were not significantly different from those of unsprayed plots. The percentage of thin kernels was significantly greater in the unsprayed plots of Kombar; 91.1% more kernels passed through the 19.10 × 2.38 mm sieve during 1985–1986 and 46.2% during 1986–1987. Sprayed plots of UC 603 had a higher percentage of plump kernels than unsprayed plots, but the differences were not significant in either year. Other yield components assessed in this study included the number of kernels per ear, ears per meter row, and tillers per meter row (data not shown); however, no significant differences were detected in any of these components for either genotype.

Net blotch was not excluded from the experiment to test the effect of propiconazole on the yield of barley in the absence of disease. Unsprayed plots of Kombar were heavily infected by *P. t.* f. *teres*; TS values reached 100% during 1985–1986 and 83.1% during 1986–1987 (Table 3). The corresponding AUDPC values were also high for this treatment. The TS and AUDPC values were low for UC 603 and sprayed plots of Kombar. In the first season of this experiment, the unsprayed plots of Kombar had an apparent infection rate that was more than double that found the next year.

Yield of Kombar from plots sprayed with propiconazole was about 26% greater than that from unsprayed plots, but this difference was only significant in the second season (Table 4). Additionally, TKW and the percentage of plump kernels were significantly higher for the sprayed plots of Kombar in both years. No significant differences were found for yield or any yield component in the UC 603 treatments even though some differences between means were quite large during 1985–1986.

DISCUSSION

The incomplete resistance of UC 603 effectively reduced yield loss due to *P. t.* f. *teres* in both years of this study. In contrast, Kombar showed yield losses of 31–35% (Table 2). Frequent and heavy rains fell during the first season of the study, contributing to a severe epidemic of net blotch and high TS and AUDPC values for Kombar. The incomplete resistance of UC 603 reduced the TS and AUDPC values to less than one-third of those found with Kombar (Table 1). Infection responses of Kombar and UC 603 ranged from moderately high to high in both the growth chamber and the field (26). These high infection responses (large lesion types) were also observed for these two cultivars in this study. Thus, the lower disease severity and infection rate in UC 603 than in Kombar were probably due to components of resistance that reduce the rate of epidemic development. During 1986–1987, environmental conditions were not as conducive to net blotch development as in the previous season, yet Kombar was severely diseased (TS = 85.4%) and UC 603 was not (TS = 0.7%). The low disease severity on UC 603 may have been due to a combination of two factors: 1) the cumulative reduction of infection among plants during a season when conditions for net blotch were not always optimal and 2) the failure of conidia to infect new susceptible tissue on the upper portions of plants.

Most of the barley in California is sown from October to December and harvested from late May through June. Cool, wet conditions commonly prevail during the early months of barley growth. These conditions favor the development of net blotch, but the impact of this disease on barley yield has not been previously documented for the Mediterranean-type climate of California. In this study, net blotch significantly decreased yield by over 30% in the experimental plots of Kombar during a severe and moderate epidemic. It is possible that losses of greater magnitude may have occurred in commercial fields of Kombar when a severe net blotch epidemic spread over California in 1979. Yield losses of 30% or more have been documented by others in the *H. vulgare*/*P. t.* f. *teres* system (9,14,15,17,21). No statistically

Table 1. Terminal disease severity, area under the disease progress curve (AUDPC), and apparent infection rate for propiconazole-treated and untreated plots of barley cultivars Kombar and UC 603 inoculated with *Pyrenophora teres* f. *teres*[u]

Year / Cultivar	Percent terminal severity[v]		AUDPC[w]		Apparent infection rate[x]	
	Treated	Untreated	Treated	Untreated	Treated	Untreated
1985–1986						
Kombar	0.5***[y]	100.0 a	19.9***	4,153.7 a	...[z]	0.12 a
UC 603	0.1***	29.6 b	3.6***	934.1 b	...	0.09 b
1986–1987						
Kombar	1.8***	85.4 a	72.5***	3,147.4 a	...	0.10 a
UC 603	0.1***	0.7 b	2.2***	32.7 b	...	0.05 b

[u] Treated plots were sprayed with propiconazole twice during 1985–1986 and three times during 1986–1987 and were not inoculated with *P. t.* f. *teres*. Untreated plots were not sprayed with propiconazole and were inoculated with *P. t.* f. *teres*. There were five replicates per treatment during 1985–1986 and six during 1986–1987.

[v] Assessed at Zadok's growth stage 87 using the net blotch rating scale of Burleigh and Loubane (3).

[w] AUDPC $= \Sigma_{i=1}^{n} [Y_{i+1} + Y_i) \times 0.5] [T_{i+1} - T_i]$, where Y_i = net blotch severity (in percent) at the ith observation, T_i = time in days of the ith observation, and n = total number of observations.

[x] Estimated by the linear regression coefficient (b) of the logit transformation of disease proportion plotted against time in days.

[y] *** = Significantly less than untreated at $P = 0.0001$ using the t test. Values followed by different letters within a year are significantly different at $P = 0.05$.

[z] Not calculated because of large error associated in estimating low disease severities.

Table 2. Yield and yield components for propiconazole-treated (T) and untreated (U) plots of barley cultivars Kombar and UC 603 inoculated with *Pyrenophora teres* f. *teres*[t]

Year / Cultivar	Yield (kg/ha)[u]			Thousand kernel weight (g)			Kernels passing sieve (%)[v]			Kernels per ear[w]			Ears per meter row[x]		
	T	U	Difference (%)	T	U	Difference (%)	T	U	Difference (%)	T	U	Difference (%)	T	U	Difference (%)
1985–1986															
Kombar	4,005	2,592	35.3*[y]	54.5	37.3	31.6*	4.2	47.3	91.1**	57.8	53.6	7.3 NS	...[z]	...	...
UC 603	3,541	3,426	3.2 NS	43.9	42.2	3.9 NS	5.4	11.4	52.6 NS	50.5	51.4	1.8 NS	...	...	...
1986–1987															
Kombar	6,306	4,353	31.0*	41.1	33.5	18.5*	36.1	67.1	46.2*	55.6	55.0	1.1 NS	69.7	62.8	9.9 NS
UC 603	6,599	6,246	5.3 NS	34.0	32.8	3.5 NS	41.8	49.4	15.4 NS	45.1	47.2	4.4 NS	124.8	137.0	8.9 NS

[t] Treated plots were sprayed with propiconazole twice during 1985–1986 and three times during 1986–1987 and were not inoculated with *P. t.* f. *teres*. Untreated plots were not sprayed with propiconazole and were inoculated with *P. t.* f. *teres*.

[u] Yield from 1.2 × 6.7 m plots with five replicates during 1985–1986 and six during 1986–1987.

[v] Percentage of 100-g seed sample falling through a sieve with perforations of 19.10 × 2.38 mm after 30 sec of shaking on the seed sizing device.

[w] Based on 10 random samples from each plot.

[x] Based on a random 1-m row section from each plot.

[y] * = Significant at $P = 0.01$, ** = significant at $P = 0.001$, NS = not significant at $P = 0.05$.

[z] Not assessed.

significant differences were detected for yield or any yield component with the UC 603 treatments. In each season, however, sprayed plots were consistently higher than unsprayed plots in yield, TKW, and the percentage of plump kernels.

Yield losses in this study were chiefly due to a reduction in TKW; the other yield components of kernels per ear, ears per meter row, and tillers per meter row were not significantly affected (Tables 2 and 4). There is conflict in the literature regarding the yield components affected by *P. t.* f. *teres*. Some investigators (1,9,18,21,22,24,27) indicate that grain weight is primarily affected, whereas others (5,6,8,12–14) report that both grain weight and grain number are affected. Piening and Kaufman (19) attributed yield loss in barley to a decrease in grain number, not weight. In Morocco, Burleigh et al (4) found that yield loss from *P. t.* f. *teres* in barley was due primarily to a reduction in ear number and kernel weight. The yield components affected by *P. t.* f. *teres* depend on the time of onset, severity, and duration of disease on barley in addition to complex physiologic processes of the host as described by Gaunt (7).

Kernel plumpness was also significantly reduced by the net blotch pathogen in this study, and this agrees with the results of Amelung (1), Shipton (22), and Smedegård-Petersen (24). The kernel size or plumpness is an important quality factor at the market because the maximum limits of thin barley (i.e., that seed which passes through a sieve having 19.10 × 2.38 mm perforations) are 10% for U.S. grade No. 1, 15% for No. 2, 25% for No. 3, 35% for No. 4, and 75% for No. 5 (2). According to this criterion, the unsprayed plots of Kombar would be graded as No. 5 six-rowed barley in both years, representing a substantial loss in the value of the grain produced. UC 603 had a high percentage of thin barley during 1986–1987. This may have resulted from an excessive number of ears per meter row that did not allow for maximum kernel development or possibly from water stress that occurred late in the season during this dry year (C. W. Schaller, *personal communication*).

A number of fungicides have been shown to be phytotoxic or phytotonic on plants, and this effect can confound yield loss estimates that are attributed solely to disease (10). An experiment was designed to test the effect of propiconazole on the yield and yield components of barley in the absence of disease, but natural infections by the net blotch pathogen occurred in the plots. However, some useful data were obtained from the UC 603 plots in this experiment. During 1985–1986, there was only a trace of infection in the unsprayed UC 603 plots (TS of 0.1%) and complete disease control was achieved in the sprayed plots (Table 3). This difference was statistically significant because the standard error among the readings for the two treatments was very low, and thus small differences were judged significant. Physiologically, it is doubtful that a TS of 0.1% and AUDPC of 1.1 would have a significant effect on yield or any yield parameter. Indeed, the data in Table 4 bear this out. For yield, there was a 20.9% difference between sprayed and unsprayed plots of UC 603. The source of this variability is unknown, but we should note that only three replicates were included in the experiment. Disease severity was greater in the UC 603 plots during the second season (1.7% for the unsprayed treatment vs. 0.1% in the sprayed plots); these differences, as well as those for yield, were not significant, however. Propiconazole has been reported to delay the senescence of leaves (1,12,27), and with Kombar and UC 603, the time of delay in leaf senescence was similar. Although propiconazole may extend the duration of green leaf tissue, it did not significantly affect yield or any yield component of UC 603 under the conditions of this study. Thus, the reductions for yield and yield components reported herein are due primarily to the effect of the net blotch pathogen. These results agree with those found for barley in Morocco by Burleigh et al (4).

UC 603 sustained disease levels as high as 29% without significant yield loss in this study. Similar results have been demonstrated with the slow-rusting type of resistance in the barley:leaf rust pathogen (11), wheat:stem rust pathogen (20), and wheat:leaf rust pathogen (25) systems. Thus, satisfactory yields can still be obtained with genotypes possessing incomplete resistance even though they may sustain moderate amounts of disease.

Table 3. Terminal disease severity, area under the disease progress curve (AUDPC), and apparent infection rate for propiconazole-treated and untreated plots of barley cultivars Kombar and UC 603 naturally infected with *Pyrenophora teres* f. *teres*[y]

Year	Percent terminal severity		AUDPC		Apparent infection rate	
Cultivar	Treated	Untreated	Treated	Untreated	Treated	Untreated
1985–1986						
Kombar	0.3***[z]	100.0 a	4.3***	2,864.0 a	...	0.24 a
UC 603	0.0***	0.1 b	0.0***	1.1 b	...	...
1986–1987						
Kombar	1.4***	83.1 a	84.1***	2,792.9 a	...	0.11 b
UC 603	0.1 NS	1.7 b	1.3 NS	58.0 b	...	...

[y] Similar to Table 1 except that plots were not inoculated with *P. t.* f. *teres* and there were three replicates per treatment during 1985–1986 and four during 1986–1987.
[z] *** = Significantly less than untreated at $P = 0.0001$, NS = not significant at $P = 0.05$. Values followed by different letters within a year (terminal severity and AUDPC) or between years (apparent infection rate) are significantly different at $P = 0.0001$.

ACKNOWLEDGMENTS

We thank Hal Cushing, Gene Prewett, and Bill Paquin for excellent technical assistance. This research was supported in part by the California Crop Improvement Association and the University of California, Davis Computer Center.

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Table 4. Yield and yield components for propiconazole-treated (T) and untreated (U) plots of barley cultivars Kombar and UC 603 naturally infected with *Pyrenophora teres* f. *teres*[y]

Year	Yield (kg/ha)			Thousand kernel weight (g)			Kernels passing sieve (%)			Kernels per ear			Ears per meter row		
Cultivar	T	U	Difference (%)	T	U	Difference (%)	T	U	Difference (%)	T	U	Difference (%)	T	U	Difference (%)
1985–1986															
Kombar	3,725	2,754	26.1 NS	50.5	36.2	28.3**[z]	6.8	53.6	87.3**	54.2	60.2	10.0 NS	...	...	...
UC 603	3,525	2,790	20.9 NS	41.6	39.1	6.0 NS	6.8	12.6	46.0 NS	48.1	44.3	7.9 NS	...	...	...
1986–1987															
Kombar	7,040	5,150	26.8*	44.0	35.7	19.0*	24.5	59.1	58.5*	65.6	63.1	3.8 NS	59.8	61.5	2.8 NS
UC 603	7,083	6,464	8.7 NS	35.9	36.9	2.7 NS	27.4	26.5	3.3 NS	55.9	54.1	3.2 NS	108.0	95.5	11.6 NS

[y] Similar to Table 2 except that plots were not inoculated with *P. t.* f. *teres* and there were three replicates per treatment during 1985–1986 and four during 1986–1987.
[z] * = Significant at $P = 0.01$, ** = significant at $P = 0.001$, NS = not significant at $P = 0.05$.

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