Quantitative Resistance to *Pyrenophora teres* f. *teres* in Barley

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ABSTRACT

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Resistance to the net blotch pathogen *Pyrenophora teres* f. *teres* was studied in seven barley genotypes using four criteria: 1) infection response (size and type of lesion), 2) area under the disease progress curve (AUDPC), 3) terminal severity (TS), and 4) apparent infection rate (r). The susceptible check, Kombar, and resistant check, Tifang, differed greatly in reaction to *P. t.* f. *teres* in the field during the 2-yr study; Kombar exhibited high or compatible (susceptible to moderately susceptible) infection responses and high TS and AUDPC values, whereas Tifang exhibited low or incompatible (resistant) infection responses and low TS and AUDPC values. Atlas, Beecher, Hazera, Cape, and UC 603 displayed infection responses that indicated a degree of compatibility, but disease progress on each was significantly lower than that on Kombar as measured by AUDPC. Thus, these five genotypes possess different levels of quantitative resistance to *P. t.* f. *teres*. The environment and amount of inoculum may alter the expression of quantitative resistance since high net blotch severities were observed on the five genotypes during a severe epidemic in 1985–86 and low severities were observed during a moderate epidemic in 1986–87. Cape and UC 603 consistently exhibited the highest level of quantitative resistance as given by AUDPC and could serve as parents in programs breeding for this type of resistance in barley.

One of the most widely distributed foliar diseases of barley (*Hordeum vulgare* L. emend Bowden) throughout the world is net blotch caused by *Pyrenophora teres* Drechs. f. *teres* Smedeg. (anamorph: *Drechslera teres* (Sacc.) Shoem. f. *teres* Smedeg.). This disease is most severe in temperate regions of high rainfall and humidity, although epidemics have occurred in low rainfall areas of Western Australia (14,15). The net blotch pathogen can cause significant reductions in the yield of barley (15). In California, where many spring habit cultivars are grown during a Mediterranean-type winter season, yield losses exceeding 30% have been recorded (31). Quality factors of barley, important for the malting and animal feed industries, also can be detrimentally affected by *P. t.* f. *teres* (26,31).

The deployment of cultivars with disease resistance is one of the most effective and environmentally sound means of controlling plant diseases. This approach has been used in a number of barley production areas for the control of net blotch. In 1928, Geschele (10) made one of the first studies on the resistance of barley to the net blotch pathogen and reported that a highly resistant reaction to *P. t.* f. *teres* was inherited in a simple Mendelian manner. Since this study, a number of resistant barley genotypes have been identified from diverse regions of the world (4,8,12,13,22). In most cases, subsequent genetic studies revealed that resistance was controlled by one or a few genes that confer a distinctly low (resistant) infection response (2,9,13,17,23). This type of resistance is sometimes referred to as qualitative because of the method used to assess the effects of genes controlling the response (5,6,14). A second type of resistance that has been described in this pathosystem is quantitative (1,6) or incomplete resistance (31)—a type that can be recognized by a reduction in the percentage of leaf tissue affected by *P. t.* f. *teres* (1,5,6,14). The genetic basis of quantitative resistance to the net blotch pathogen in barley has been attributed to additive gene effects (1), additive plus additive × epistatic gene effects (5), and a single partially dominant gene plus additive gene effects (6). Genotypes with quantitative resistance can exhibit lower levels of disease by reducing the infection efficiency (5,7,11,18), lengthening the latent period (7,18), or reducing the sporulation capacity (7,11) of the pathogen. One or more of these components of resistance also can reduce the rate of disease development in the field (18). Thus, this type of resistance may be identified by comparing parameters of disease progress such as the area under the disease progress curve (AUDPC) or the apparent infection rate (34). In the *H. vulgare*/*P. t.* f. *teres* pathosystem, few studies have been advanced on the selection and performance of genotypes with quantitative resistance in the field. If genotypes with high levels of quantitative resistance were identified in barley, they could prove useful as an alternative form of resistance in breeding programs. This study was undertaken to evaluate the performance of several potential sources of quantitative resistance to *P. t.* f. *teres* in barley.

MATERIALS AND METHODS

Field study. In 1984–1985, a preliminary screening for quantitative resistance was made on 159 different barley genotypes collected from the International Maize and Wheat Improvement Center (CIMMYT) in Mexico and the barley breeding programs of the University of Western Australia and the University of California at Davis (29). Our objective was to study quantitative resistance in the absence of genes with strong qualitative effects that confer low or incompatible infection responses, i.e., small restricted lesions with little or no chlorosis. Thus, we selected genotypes that exhibited high or compatible infection responses and low AUDPC values. From this initial experiment, five genotypes were selected for further study: Atlas (CI 4118), a cultivar selected from barley seed first introduced into California by Spanish settlers; Beecher (CI 6566), a cultivar derived from the cross Atlas × Vaughn; Cape (CI 1026), a cultivar originally introduced from South Africa; Hazera (CI 12673), a cultivar of Israeli origin; and UC 603 (PI 537576), a recently released cultivar (1988) from the UC Davis barley breeding program. Additionally, Kombar (CI 15694) and Tifang (CI 4407-1) were included in the study as susceptible and resistant checks, respectively. Kombar is susceptible to many isolates of *P. t.* f. *teres* from California, whereas Tifang is resistant (30). Heading dates for all of the genotypes are within about 1 wk of each other: Beecher is the

earliest and Kombar and Tifang are the latest to head.

The experiments were conducted for two consecutive seasons (1985–86 and 1986–87) at the University of California Armstrong Plant Pathology Farm near Davis. Plantings were made on 11 November 1985 and on 29 October 1986 using a completely randomized design with five replicates. For each replicate, 4.5 g of seed was sown in 2.4-m rows spaced 0.6 m from the ends and 0.8 m from the sides of the other replicates or spreader rows. Spreader rows of Kombar were planted around the outside edges of the entire plot and were inoculated at the midtillering stage of growth (growth stage [GS] 24 according to Zadoks et al [36]). These inoculations were made on 13 January 1986 and 7 January 1987 (Julian day [JD] 13 and JD 7) by scattering 4 kg of barley straw over the plants. The barley straw was collected from the previous season's crop at the farm and was infected primarily with pathotypes 3-10-15-19-21 and 3-10-15-19-20-21 of *P. t.* f. *teres* (30). Infections were detected on the spreader rows and test entries about 3 wk after inoculation. The infection response (size and type of lesion) and percent net blotch severity were assessed 11 times during the season in 1985–86 (at intervals of 5–14 days) and six times in 1986–87 (at intervals of 10–24 days) on 10 randomly selected tillers per replicate. The first assessment was made at the jointing stage (GS 31–32) and the last at the soft-to-hard dough stages of kernel development (GS 85–87). Infection responses were based on the following criteria: R = resistant (dark pinpoint lesions with no chlorosis); MR = moderately resistant (small lesions consisting of a few transverse and longitudinal streaks with no or slight chlorosis); MS = moderately susceptible (netted or solid lesions restricted in width or length, often with marginal chlorosis at the ends of the lesion); and S = susceptible (long netted or solid lesions, often with marginal chlorosis at the ends of the lesion). Descriptions for the high (susceptible and moderately susceptible) infection responses included both the typical net-type lesion and a solid (uniform) necrotic lesion because Cape and Beecher frequently exhibited the latter in the field. The final infection response readings were based on lesions from the flag and penultimate leaves. Estimation of percent net blotch severity (including both chlorotic and necrotic areas) was made according to the scale devised by Burleigh and Loubane (3). Disease severity on the top three leaves was averaged to derive a mean disease severity per tiller.

Three aspects of net blotch development were investigated on the seven barley genotypes: terminal severity (TS), AUDPC, and apparent infection rate (r) sensu Vanderplank (33). Terminal net blotch severity was assessed near the end of the growing season at GS 85–87, and AUDPC was calculated using the equation:

$$\text{AUDPC} = \sum_{i=1}^{n} [(Y_{i+1} + Y_i) \times 0.5] [T_{i+1} - T_i],$$

where Y_i = percentage of leaf area affected by net blotch at the ith observation, T_i = time (in days) at the ith observation, and n = total number of observations. The apparent infection rate was estimated by regressing the logit of disease proportion (decimal form of percent disease severity) on time in days. The slope or linear regression coefficient (b) of this line was used as an estimate of the apparent infection rate as given by Vanderplank (33). Estimates of disease progress parameters were subjected to analysis of variance, and differences among treatment means were tested for statistical significance using the Tukey-Kramer method at a significance level of 0.05.

Growth chamber study. To determine the reaction of the barley genotypes in the seedling stage, an experiment was conducted using pathotypes 3-10-15-19-21 and 3-10-15-19-20-21. These pathotypes were chosen for study because they comprised 72% of the isolates collected in and around the experimental site (30). Leaf material, previously infected with pure cultures of these pathotypes, was surface-sterilized and transferred to plates containing 17.7% V8 juice agar (177 ml of V8 juice, 16 g of agar, 3 g of $CaCO_3$ per liter of H_2O). These plates were incubated on a laboratory bench at 19–22 C and received 12 h of natural sunlight (9,688–10,106 ergs cm^{-2} s^{-1}). After 14 days, conidia were harvested by adding 2 ml of water to the plates and scraping the culture with a rubber spatula. This suspension was filtered through a double layer of cheesecloth to separate large mycelial fragments from conidia, and then the spore concentration was adjusted to 2×10^4 conidia per milliliter for inoculation.

Barley genotypes were sown (five to seven seeds per clump) in metal flats (50 × 35 × 9 cm) containing UC mix (16) and arranged in a completely randomized design with four replicates per genotype. These plants were grown in a greenhouse at 15–22 C and inoculated 2 wk later when the second leaf was fully expanded. The conidial suspension was sprayed onto the plants (12 ml per flat) using a DeVilbiss atomizer (Somerset, PA). Next, the plants were placed in a mist chamber at 18–22 C (12-h photoperiod, 2,474 ergs cm^{-2} s^{-1}) where the humidity was maintained near saturation. After a 48-h moist period, the plants were transferred to a growth chamber at 21–24 C with a 12-h photoperiod (105,874 ergs cm^{-2} s^{-1}). Infection responses on the second leaf were assessed 2 wk after inoculation using the system of Tekauz (32).

RESULTS

The development of net blotch was rapid and severe on cultivars Kombar, Atlas, Hazera, and Beecher in 1985–86 (Fig. 1A). This epidemic was due in part to abundant and timely rainfall. Net blotch severity was lower on UC 603 and Cape throughout the entire season and was nearly nil on Tifang. The most rapid development of net blotch occurred after JD 84 (25 March). Disease severities were lower for all genotypes in 1986–87 (Fig. 1B). Profiles of disease progress curves for Atlas, Beecher, Hazera, Cape, and UC 603 were similar even though their severities differed. In contrast, the disease progress curve for Kombar was

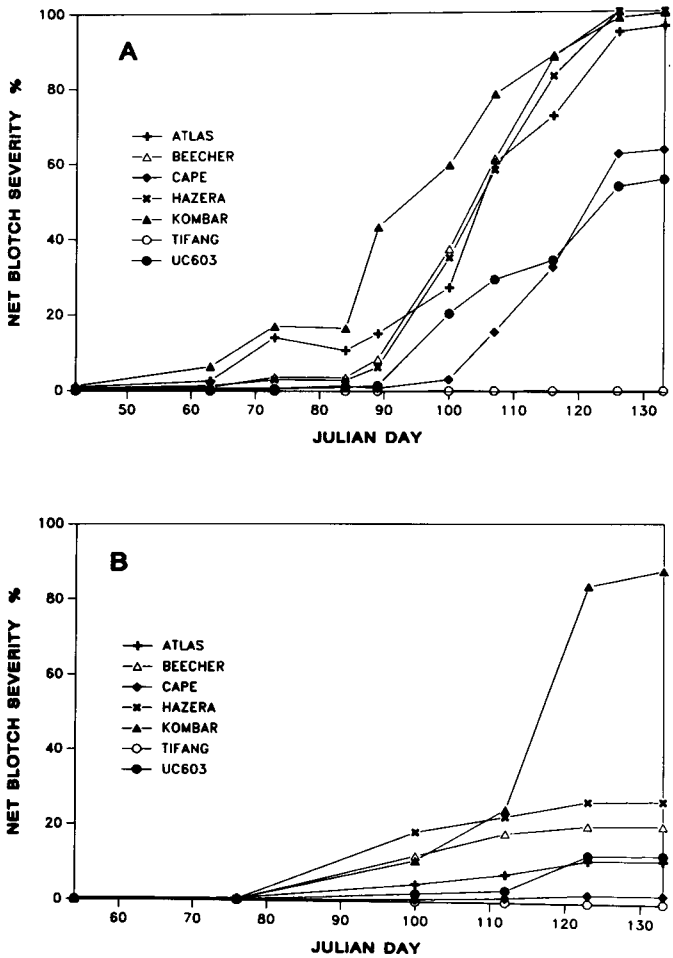

Fig. 1. Disease progress curves for seven barley genotypes infected with *Pyrenophora teres* f. *teres* in the field from **A**, 1985–86, and **B**, 1986–87. Net blotch severity was based on the average infection percentage of the top three leaves. The plotted points are the means of five replicates.

TABLE 1. Terminal net blotch severity, area under the disease progress curve (AUDPC), estimated apparent infection rate (r), and infection response for seven barley genotypes infected with *Pyrenophora teres* f. *teres* in the field during 1985–86 and 1986–87

Genotype	Terminal severity[u] (%)		AUDPC[v]		Apparent infection rate[w]		Infection response[x]	
	1985–86	1986–87	1985–86	1986–87	1985–86	1986–87	1985–86	1986–87
Hazera	100.0 a[y]	26.9 b	2,904 b	1,019 b	0.17 a	0.10 b	S	MS-MR-S
Beecher	100.0 a	20.5 bc	3,017 b	753 b	0.18 a	0.10 b	S	MS-S
Kombar	99.5 a	87.5 a	3,958 a	1,808 a	0.11 b	0.14 a	S-MS	S-MS
Atlas	96.0 a	11.3 cd	2,960 b	358 c	0.11 b	0.07 bc	S	S-MS
Cape	63.5 b	2.3 de	1,255 c	69 c	0.14 ab	0.05 c	S-MS	MS-MR
UC 603	55.7 c	12.7 c	1,433 c	278 c	0.14 ab	0.09 b	MS-S	S-MS
Tifang	0.2 d	0.1 e	7 d	5 c	...[z]	...	R	R

[u] Terminal severity was assessed at growth stages 85–87 (according to the scale of Zadoks et al [36]) using the net blotch rating scale of Burleigh and Loubane (3).

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

[w] Apparent infection rate (r) was estimated by the linear regression coefficient (b) of the logit transformation of disease proportion plotted against time in days.

[x] R = Resistant (dark pinpoint lesions with no chlorosis); MR = moderately resistant (small lesions consisting of a few transverse and longitudinal streaks with no or slight chlorosis); MS = moderately susceptible (netted or solid lesions restricted in width or length, often with marginal chlorosis at the ends of the lesion); and S = susceptible (long netted or solid lesions, often with marginal chlorosis at the ends of the lesion).

[y] Means followed by different letters within a column are significantly different at $P = 0.05$ according to the Tukey-Kramer method. The means are based on five replicates.

[z] The apparent infection rate was not calculated because of the large error associated in estimating low disease severities.

markedly steeper as net blotch severity increased from about 24% on JD 112 (22 April) to over 80% on JD 123 (3 May). Tifang was again highly resistant as disease severity was almost nil.

The TS of net blotch on Hazera, Beecher, Atlas, and Kombar was over 95% in 1985–86 (Table 1). Terminal severities on Cape and UC 603 were significantly lower than those of the high disease group. AUDPC values for Kombar were significantly higher than those for Hazera, Beecher, and Atlas, and this group in turn had significantly higher values than those for Cape and UC 603. The resistant check, Tifang, exhibited low TS and AUDPC values of 0.2% and 7, respectively. Beecher and Hazera had the highest apparent infection rates, and these values were significantly higher than those for Kombar and Atlas. All genotypes exhibited some compatible to moderately compatible (susceptible or moderately susceptible) infection responses with the exception of Tifang, which gave an incompatible (resistant) response.

In the second season of the experiment, the highest TS was on Kombar (87.5%) and the lowest on Tifang (0.1%); the other genotypes ranged from 2.3 to 26.9% (Table 1). Three groups of statistical significance were discerned with respect to AUDPC: Kombar at the high level; Hazera and Beecher at the middle level; and UC 603, Atlas, Cape, and Tifang at the low level. The highest rate of disease development was exhibited by Kombar ($r = 0.14$) and the lowest by Cape ($r = 0.05$). Compatible to moderately compatible infection responses were observed on all entries except Tifang.

Moderately high to high (7,8 to 9,10) infection responses were commonly observed on Kombar, Atlas, Hazera, and Beecher to pathotypes 3-10-15-19-21 and 3-10-15-19-20-21 in the seedling stage (Table 2). Tifang exhibited low (2,1 or 1,2) and Cape moderately low (5,4) infection responses to both of the pathotypes. The infection response of UC 603 was moderately high to pathotype 3-10-15-19-21 and intermediate (mixture of infection responses 5 and 6) to pathotype 3-10-15-19-20-21. Higher infection responses were observed more often on Kombar, Hazera, UC 603, and Tifang to pathotype 3-10-15-19-21 than to 3-10-15-19-20-21.

TABLE 2. Mode and range of infection responses on seedlings of seven barley genotypes to pathotypes 3-10-15-19-21 and 3-10-15-19-20-21 of *Pyrenophora teres* f. *teres* in a growth chamber

Genotype	Pathotype 3-10-15-19-21[y]		Pathotype 3-10-15-19-20-21	
	Mode	Range	Mode	Range
Kombar	9,10[z]	8–10/(7)	8,7	7–9/(10)
Atlas	9,8	8–10	9,8	8–10
Hazera	8,9	7–9	8,7	7–9/(5)
Beecher	8,9	6–9/(10)	8,9	7–9/(5,10)
UC 603	8,7	6–9/(5)	5,6	4–6/(7)
Cape	5,4	4–5/(6)	5,4	4–5/(6)
Tifang	2,1	1–3	1,2	1–2/(3)

[y] Pathotypes were previously described by Steffenson and Webster (30). The infection responses observed on Cape to pathotype 3-10-15-19-20-21 in this study were lower than those previously described (30).

[z] Infection responses were assessed using the scale of Tekauz (32). The mode represents the two most common (most prevalent type listed first) infection responses observed on the barley genotypes to the individual pathotypes, whereas the range includes all infection responses identified, including those rarely observed (in parentheses).

DISCUSSION

Atlas, Hazera, Beecher, Cape, and UC 603 exhibited infection responses that indicated some degree of compatibility (i.e., exhibited moderately high to high infection responses) to *P. t.* f. *teres* in the field, yet disease progress, as measured by AUDPC, was significantly lower on each than that on the susceptible check, Kombar (Table 1). Thus, these five genotypes possess different levels of quantitative resistance in the field. In this study, the use of the term quantitative resistance is not intended to imply anything about the genetics, components, pathotype-specificity, or durability of the resistance. Quantitative resistance is a practical term for describing a type of resistance that can be assessed by estimating the percentage of tissue affected by the net blotch pathogen. This kind of resistance also exhibits a continuous range of variability. We consider quantitative resistance synonymous with incomplete resistance (a term used to describe the resistance of UC 603 in another study [31]).

The rating scales for classifying infection responses in the seedling (growth chamber study) and adult plant stages (field study) were based on lesion size and type (degree of chlorosis). Genotypes with quantitative resistance could be easily differentiated from Tifang with qualitative resistance on the basis of the infection response at both growth stages (Tables 1 and 2). Tifang possesses two genes for resistance to *P. t.* f. *teres* (13) and, in this study, exhibited low or incompatible (resistant) infection responses. In contrast, the other genotypes exhibited distinctly higher levels of compatibility (higher infection responses) to the pathogen. The seedling reaction of Kombar, Beecher, Atlas, Hazera, UC 603, and Cape can vary because low infection responses were reported for all of these genotypes to at least a few pathotypes of *P. t.* f. *teres* in California (30). Thus, genes conferring low infection

responses to other pathotypes are present in these genotypes and may be different from those conferring the quantitative resistance already described in this study. The effect of these seedling resistance genes on disease development in adult plants is not known; however, most of the infection responses observed on genotypes in the field were of the susceptible to moderately susceptible type, indicating a high degree of compatibility between the pathotypes of *P. t.* f. *teres* present in the nursery and the barley genotypes (except Tifang).

The apparent infection rate was closely associated with values for TS and AUDPC in 1986–87. This relationship was not readily apparent in the first season of this study since UC 603 and Cape had higher *r* values than Kombar despite having significantly lower TS and AUDPC values. Generally, fully susceptible genotypes (as measured by TS and AUDPC) exhibit the highest apparent infection rates; however, high *r* values can occur on genotypes with reduced disease levels when there is a rapid increase of disease from a low to a moderate level within a short period of time. In some cases, the calculation of *r* can result in a loss of information on the disease progress curve, and this can increase the experimental error (24). This result also was observed in our study, particularly for the cultivar Cape. The coefficient of determination (R^2) for the estimate of *r* on Cape was only 0.64 and 0.77 for the first and second season, respectively. Thus, the apparent infection rate may not be the most reliable parameter for assessing the slow development of net blotch on barley. This conclusion also was reported by other investigators in the wheat/stem rust pathogen (21,35) and wheat/powdery mildew pathogen systems (24). An important shortcoming of the apparent infection rate sensu Vanderplank (33) is that the development of disease does not always conform to a logistic model. This may have been a factor in this study. Park and Lim (19) correctly stated that disease progress curves cannot be accurately fitted using the logistic function when the asymptotic maximum disease proportion is less than one, and they outlined an improved procedure for estimating disease progress curves using Richards generalized rate parameters. An attempt was made to describe the disease progress curves observed in this study by the methods of Park and Lim (19), but convergence for the model using empirically estimated asymptotes was not achieved for all genotypes.

TS assessments also may have limitations for the identification of quantitative resistance. Using TS as the sole criterion, differences between Hazera and the fully susceptible genotype Kombar would not have been detected during the first season. Hazera exhibited a very high TS value, but sustained less disease than Kombar during the early part of the season (Fig. 1A). The slower development of disease on Hazera was revealed by the AUDPC value, which measures the cumulative effects of resistance over the course of the entire season. TS and AUDPC can be highly correlated as demonstrated in 1986–87. If such a relationship exists, quantitative resistance could be easily identified by a single disease assessment made near the end of the growing season instead of the multiple assessments necessary for the calculation of AUDPC. In considering both years of this experiment, AUDPC was the most reliable statistic for assessing quantitative resistance to *P. t.* f. *teres* in barley. This result is in agreement with research done in other pathosystems (21,24,35).

The 2-yr field study allowed for the evaluation of quantitative resistance during severe (1985–86) and moderate (1986–87) epidemics of net blotch. Disease levels varied greatly on Atlas, Hazera, Beecher, Cape, and UC 603 between the two seasons of the experiment due, perhaps, to the environment and amount of inoculum. The variability observed in the expression of resistance is not surprising because this host/parasite interaction can be markedly altered by various environmental factors and the concentration of inoculum (27). Large differences also were observed among genotypes with quantitative resistance, especially during the first season, as AUDPC values for Cape and UC 603 were significantly lower than those for Atlas, Hazera, and Beecher. These results are similar to those of Skou and Haahr (28), who differentiated levels of resistance among cultivars based on the pattern of epidemic development and a point score representing a level of attack by the pathogen. The seedling infection responses on UC 603 and Cape to pathotypes 3-10-15-19-21 and 3-10-15-19-20-21 indicated a lower degree of compatibility compared with those observed on Atlas, Hazera, and Beecher (Table 2). The infection response is usually considered a qualitative assessment of disease, but it can be correlated with components of quantitative resistance (32). UC 603 and Cape may provide the highest level of quantitative resistance identified in this study. Before UC 603 was released to growers as a commercial cultivar in 1988, it consistently exhibited lower severities of net blotch than susceptible check cultivars in breeding nurseries (C. W. Schaller, *personal communication*). Moreover, the quantitative (or incomplete) resistance of UC 603 can be highly effective in reducing yield loss due to *P. t.* f. *teres* during severe epidemics (31).

Since the available seed for some genotypes was limited, the field experiments were arranged with the test entries adjacent to each other and to the spreader rows in a small disease nursery. An experiment arranged in this fashion can result in the underestimation of quantitative resistance as discussed by Vanderplank (33) and Parlevliet and Van Ommeren (20). Burleigh and Loubane (3) studied the development of net blotch as affected by different plot sizes. They contend that if 40- × 40-m plots represent an area where maximum disease development is attained, then 20- × 20-m and 10- × 10-m plots sustain only 56 and 54% of maximum disease severity, respectively. Even the smallest of these plot sizes are prohibitively large and expensive for most screening programs, and thus plot size compromises must be made. The quantitative resistance described in this study was probably underestimated, but was still readily detectable in the field during both seasons.

This study is a preliminary investigation on quantitative resistance to *P. t.* f. *teres* in barley. Further research should be initiated on the genetics, components, and pathotype-specificity of this resistance. It is important to determine the genetics of quantitative resistance so that this character can be efficiently manipulated in breeding programs. In two recent studies (1,5), genes with additive effects were found to confer quantitative resistance to the net blotch pathogen in barley. These genes can be exploited in breeding programs (1,5). Indeed, Sharp (25) and his associates have successfully used recurrent selection populations to pyramid major and minor gene resistance into both six- and two-rowed barley types.

Investigations on the components contributing to quantitative resistance also can be valuable. For example, if a strong correlation is found between a particular resistance component (such as receptivity [18]) and quantitative resistance in the field, this component could serve as a trait for selecting this type of resistance in the greenhouse. Correlations between in vitro disease assessments on detached leaves and quantitative resistance have been documented in this pathosystem (1,6). This screening technique should prove valuable to the breeder because it is rapid and inexpensive.

Resistance that is effective against a wide spectrum of pathotypes may last longer than types exhibiting strong differential reactions to different genotypes of the pathogen. This attribute could not be determined in the field for this study because the inoculum consisted of a composite of pathotypes. However, in the seedling stage, UC 603 differed markedly in infection response to pathotypes 3-10-15-19-21 and 3-10-15-19-20-21, suggesting that this resistance may be pathotype-specific. Nevertheless, genotypes such as UC 603 or Cape may prove useful as parents in programs breeding for this type of resistance in barley.

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