# Reaction of Maize Cultivars from Uganda to *Exserohilum turcicum*

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Portion of a thesis submitted by the first author in partial fulfillment of the requirements for the Ph.D. degree of The Ohio State University.

Salaries and research support provided by state and federal funds appropriated to the Ohio Agricultural Research and Developmental Center, The Ohio State University, and grant monies from the Rockefeller Foundation and USAID/Uganda Manpower for Agricultural Development (MFAD). Manuscript no. 82-92.

We wish to acknowledge the technical assistance of A. Johnston, J. P. Takan, and M. W. Ogenga-Latigo. We also thank I. W. Deep, R. C. Pratt, W. Shane, and T. Wheeler for reviewing the manuscript.

Accepted for publication 19 November 1992.

## ABSTRACT

Adipala, E., Lipps, P. E., and Madden. L. V. 1993. Reaction of maize cultivars from Uganda to *Exserohilum turcicum*. Phytopathology 83:217-223.

Several disease variables were used to characterize the type and level of disease reactions of Ugandan maize cultivars to northern leaf blight, caused by *Exserohilum turcicum*, in 1989 seedling studies and in 1990 and 1991 field trials. Maize cultivars differed significantly ($P \leq 0.001$) in susceptibility as determined by the rate of disease increase ($r$) for the logistic model, area under disease progress curves, final lesion numbers, and severity ratings. Estimates of $r$ were similar in moderately resistant and susceptible inbreds, Mo17, B73*Ht*1, and B73 but were significantly ($P \leq 0.001$) lower in the Ugandan cultivars Babungo 3, Kawanda composite A (KWCA), and Population 42, indicating that rate-reducing resistance characterized resistance. Similarly, epidemic onset ($y_0^*$), indicated by the intercept of the regression equation for logit in relation to time, differed among cultivars, but unlike $r$, differences were not always significant among susceptible and resistant cultivars. The high level of rate-reducing resistance in Babungo 3, Population 42, and KWCA was effective against both races 0 and 1, and was more effective than the *Ht*1 gene against race 0. In seedling studies, Babungo 3 delayed formation of necrotic lesions, had fewer and smaller lesions, supported low sporulation, and delayed sporulation by at least 3 days compared with susceptible cultivars. For most genotypes, results for seedling and adult plants were positively correlated. Regardless of assessment method, Babungo 3, Population 42, and KWCA were significantly ($P \leq 0.001$) more resistant to *E. turcicum* than the susceptible check B73, resistant polygenic check Mo17, or monogenic resistant check B73*Ht*1.

Northern leaf blight, caused by the fungus *Exserohilum turcicum* (Pass.) K. J. Leonard & E. G. Suggs (teleomorph *Setosphaeria turcica* (Luttrell) K. J. Leonard & E. G. Suggs), is an important disease of maize (*Zea mays* L.) worldwide (5). In Uganda, northern leaf blight is widespread but is particularly destructive in the wet, warm, and humid areas around Lake Victoria. The most efficient and effective control of northern leaf blight is by resistant hybrids or cultivars (7,17,23,27). Various levels of polygenic resistance are available in maize inbreds, as is monogenic resistance (3). Polygenic (7,12), rate-reducing (20,23), partial (17,26), field, or lesion number (12,13) resistance effectively reduces rate of disease increase (20,32,33). Genotypes with rate-reducing resistance range from highly resistant, in which few lesions form, to susceptible, in which many large sporulating lesions are present (7,17). Monogenic resistance (8–11) or race-specific resistance (32,33), under the control of *Ht*1, *Ht*2, and *Ht*3 genes, is characterized by formation of chlorotic lesions and reduced pathogen sporulation that delays epidemic onset, or as in the case of gene *HtN*, delays lesion formation (8).

Researchers have developed techniques for early generation selection of field-resistant plants, preferably at a young stage in the greenhouse (24). However, few studies have quantified relationships between disease resistance under greenhouse and under field conditions, probably because it is sometimes unreliable to extrapolate seedling results to field conditions (20).

Of the several disease progress models proposed for characterizing increase in disease over time of polycyclic diseases, the logistic and Gompertz models have been used most frequently (2,4). These and other models (4,16) define disease progress in terms of rate of disease increase and estimated disease level at the observed start of the epidemic (32). A related approach is to calculate the area under disease progress curve (AUDPC), which describes disease progress in terms of disease level integrated over the assessment times (4,34).

Because of the lack of efficient and economical hybrid seed production, maize researchers in Uganda have traditionally emphasized production of open-pollinated cultivars. Open-pollinated cultivars are an important component of crop production systems in many regions of the world. The type and level of resistance of Ugandan maize cultivars to *E. turcicum* have not been characterized. Yet, information on reaction to *E. turcicum* is necessary for use of resistant cultivars for disease management (6). The objectives of this study were to examine the effects of maize cultivars on northern leaf blight progress, to determine the level of resistance to *E. turcicum* in several maize cultivars under greenhouse and field conditions, and to compare expression of seedling and adult plant resistance to northern leaf blight in these cultivars. An abstract of this work has been published (1).

## MATERIALS AND METHODS

**Germ plasm.** Seeds of 10 open-pollinated maize cultivars or improved populations were obtained from Namulonge Research Station, Uganda (Table 1). The cultivars and populations were derived from low- to mid-altitude tropical germ plasm and had diverse genetic backgrounds developed for high yield and for resistance to maize streak virus. Kawanda composite A (KWCA), the main commercial cultivar in Uganda, and its maize-streak-virus-resistant selection, KWCA-SR, contained maize germ plasm from eastern and southern Africa. These cultivars and the eight improved populations had a high degree of homogeneity, because each represented reconstituted full-sib populations selected for diverse agronomic traits over a number of recurrent-selection cycles. Thus, the improved populations will be called cultivars throughout the rest of this paper. The 10 cultivars were compared to the three U.S. inbred lines B73 (susceptible to *E. turcicum*),

B73*Ht*1 (monogenic resistance to race 0) and Mo17 (polygenic resistance) (22,23,26,27). All cultivars were not used in every experiment.

**Races and inoculum.** Four races of *E. turcicum* were studied: a field isolate of race 0 from Nakulabye, Uganda; a field isolate of race 1 from Madison Seed Company, London, OH; and races 23 (ATCC 64836) and 23N (ATCC 64834) from the American Type Culture Collection (ATCC) Rockville, MD. Races 0, 1, 23, and 23N have the virulence/avirulence formula: /*Ht*1, *Ht*2, *Ht*3, *HtN*; *Ht*1/*Ht*2, *Ht*3, *Ht*N; *Ht*2, *Ht*3/*Ht*1, *Ht*N; and *Ht*2, *Ht*3, *Ht*N/*Ht*1, respectively. To ensure virulence of the isolates, fungi from stock cultures were inoculated onto maize seedlings and reisolated from lesions. Mycelial plugs of each isolate were grown on potato-dextrose agar (PDA) for 8–10 days, and then 20 ml of sterilized double-distilled water was added to each culture. Conidia were dislodged with a rubber policeman and filtered through four layers of cheese cloth. Five maize seedlings (B73) were inoculated with each race by pipetting 1 ml of a spore suspension (20,000 conidia/ml) into the whorl. Ten days after inoculation, lesions were excised, surface-disinfected in 0.5% sodium hypochlorite solution, rinsed in double-distilled water for 30 s, and kept in a humid chamber (100% relative humidity [RH]) for 24–36 h. Single conidia were removed and cultured on PDA. Conidial suspensions for inoculation of experimental plants were prepared by washing conidia from 10- to 14-day-old PDA cultures and filtering through four layers of cheese cloth. Conidia were counted with a hemacytometer, and the suspension was adjusted to the desired concentration by dilution with double-distilled water.

**Growth chamber studies.** Maize seedlings (three seedlings per pot) in the four- to six-leaf stage, were misted with approximately 1 ml/pot of a spore suspension containing 30,000 conidia/ml with a mist sprayer to give a fine deposit without runoff. Inoculated plants were kept in a humidity chamber (100% RH) for 12 h at 20 ± 2 C. Plants were then transferred to four growth chambers, maintained at 18 C night and 22 C day with a light period of 12 h/day having an average intensity of 280 ± 19 $\mu E \cdot m^{-2} \cdot s^{-1}$ (14,15). Plants were watered daily.

Leaves were removed from two seedlings per pot to determine the amount of sporulation within lesions. One seedling per pot was tagged for lesion assessments, and no leaves were removed from it. For each cultivar, incubation period was measured as the time (days) from inoculation to formation of necrotic lesions (when lesions were >1.5 cm long). Latent period, the time from inoculation to sporulation, was determined by removing diseased leaves at the same time each day and examining them microscopically for conidia of *E. turcicum*. Number of lesions per plant, lesion length (in centimeters), and sporulation were recorded 10, 13, and 16 days after inoculation.

Two sections of blighted leaf tissue (2 $cm^2$) were excised from each cultivar, rinsed in 0.5% sodium hypochlorite solution for 30 s and then in double-distilled water, and kept for 24–36 h in moisture chambers (100% RH) at 22 ± 2 C. Each leaf section was then placed into a 28-ml test tube containing 5 ml of double-distilled water. The test tube contents were rotated at 4.5 rps for approximately 30 s with a vortex shaker to dislodge conidia. The estimated average number of conidia in each suspension was based on 10 subsamples counted with a hemacytometer. Conidial counts were expressed as number of conidia per square centimeter of lesion and then transformed (ln[spores + 1]) to equalize variance for statistical analysis.

A randomized complete block design with four blocks (four growth chambers) was used. Maize cultivars and pathogen race were experimental factors. Nine of the 10 maize cultivars from Uganda and two U.S. inbreds, Mo17 and B73, were studied. Each experimental unit within a chamber treatment consisted of single pots containing three seedlings of one cultivar inoculated with one race of the pathogen. Separate growth chamber studies were conducted with races 0 and 1 and later with races 23 and 23N. Cultivars and races were randomized within each chamber. Each experiment was repeated five times with races 0 and 1 and three times with races 23 and 23N. Pooled analyses were conducted for data from races 0 and 1 and from races 23 and 23N. Analysis of variance (ANOVA) was used to evaluate the effects of cultivar, race, experiment, and their interactions on the disease variables (e.g., sporulation). Data were analyzed using Minitab procedures (Minitab, State College, PA). Fisher's least significant difference (LSD, $P = 0.05$) was used to separate treatment means, and single-degree-of-freedom contrasts were calculated for the analyzed variables to evaluate cultivars for seedling reactions.

Spearman's rank correlation coefficients were calculated to evaluate relationships among disease variables. Correlations were determined with the PROC CORR procedure of SAS (Statistical Analysis System, SAS Institute, Cary, NC) and were based on the means across replications.

**Field experiments.** Adult plant reactions to northern leaf blight were studied at Wooster, OH, in 1991, and at two locations in Uganda in 1990 and 1991. Seeds of cultivars obtained from Uganda to be used for field studies in Wooster were increased under quarantine conditions in the greenhouse at the Ohio Agricultural Research and Development Center and again in Puerto Rico. In order to maintain heterogeneity of the original germ plasm, plants were sibpollinated by controlled pollination. Seeds were then bulked and used for field studies.

Single-spore cultures of races 0 and 1 were selected for rapid growth and high spore production. After 14 days on PDA, the fungal cultures were transferred to autoclaved oat (Wooster) or sorghum (Uganda) grains in 2-L glass flasks for 10–12 days and shaken daily. Infested grains were then air-dried for 2–4 days. Plants in the center row of three-row plots were inoculated with *E. turcicum* race 0 (Uganda) or race 1 (Wooster) at growth stages (GS) 4, 5, and 6 (28) by placing approximately 50 infested grains into the whorl of each plant. At Wooster, sprinkler irrigation was applied after each inoculation for 12 h overnight to provide a total of 2.5 cm of irrigation. Because of the dry conditions in 1991, additional 12-h overnight irrigation was applied on 2, 9, and 22 August.

The 10 cultivars and the three inbred lines (B73, B73*Ht*1, and Mo17) were planted on 28 September 1990 and 28 February 1991 at Kabanyolo and Namulonge, Uganda. At Wooster, the planting date was 16 May 1991. Maize cultivars were arranged in a randomized complete block design with four blocks in Uganda and eight in Wooster. Each experimental unit consisted of three rows of a cultivar or inbred. Rows were 4-m long, 1.5-m apart in Uganda and 0.75-m apart in Wooster, and contained approximately 15 plants.

**Field disease assessments.** At each location, 10 randomly selected plants in the center row were tagged and used for successive disease assessments. Reactions to northern leaf blight were assessed at GS 8, 9.0, 9.1, 9.2, 9.3, and 9.4. Severity of northern leaf blight was visually estimated for each tagged plant as the percentage of total leaf area affected using a modified scale of 0.0, 0.5, 1.0, 5.0, 10.0, 25.0, 50.0, and ≥75% of leaf area affected (7). The number of lesions on the ear leaf and on the first and second leaf above the ear leaf were also counted on

TABLE 1. Origin and sources of maize germ plasm obtained from Uganda[a]

| Germ plasm[b] | Origin | Source |
|---|---|---|
| KWCA | Uganda | Namulonge Research Station |
| KWCA-SR | Uganda | Namulonge Research Station |
| EV8428-SR | CIMMYT[c] | IITA[d] |
| EV8429-SR | CIMMYT | IITA |
| EV8349-SR | CIMMYT | IITA |
| Population 42 | CIMMYT | IITA |
| Gusau TZB-SR | IITA | IITA |
| Jos | IITA | IITA |
| Across 83 TZM-SR | IITA | IITA |
| Babungo 3 | IITA | IITA |

[a] All seeds were obtained from Namulonge Research Station, Uganda.
[b] KWCA = Kawanda composite A, KWCA-SR = its maize-streak-resistant selection.
[c] Centro Internacional de Mejoramiento de Maiz y Trigo, Mexico.
[d] International Institute of Tropical Agriculture, Nigeria.

each tagged plant. At GS 9.2, lengths (in centimeters) of two lesions on 10 plants (total of 20 lesions/experimental unit) in the center rows were measured. Data obtained from individual plants of each experimental unit were averaged and used for statistical analyses.

In order to develop linearized models for increase in lesion number with time (see below), 1,620 lesions on 143 leaves representing all cultivars were measured at Kabanyolo in 1991. Results indicated that the maximum number of lesions per plant, based on lesion and leaf sizes, was 67. However, because leaves would most likely senesce before the maximum number of lesions developed, 50 lesions was considered as $y_{max}$ (4). Because the average maximum number of lesions per plant in a plot was generally much less than 50, use of the estimated $y_{max}$ had little effect on the results (17).

Disease progress curves for severity and lesion number were plotted for each cultivar. AUDPC was calculated for each plot and standardized by dividing by the number of days in the epidemic (4).

Linearized forms of the exponential, Gompertz, and logistic models (4,16) were fitted to disease progress data (percent disease severity and lesion number) for each replication of the field experiments. Criteria for selecting the best fitting model included examination of observed and predicted disease severity (or lesion number values) versus time, coefficient of determination ($R^2$) values for each model, standard errors of the parameter estimates, $F$ statistic testing the significance of the regression model ($P \leq 0.05$), and plots of the standardized residuals versus predicted values for regression equations of each model (4). The $R^2$ value of the regression equation for each genotype in a location indicated the proportion of total variation explained by the regression model. Determinations were made for each genotype in each location, for each year, and also across locations and years. The REG procedure of SAS was used for regression analysis.

The linearized forms of the logistic and Gompertz models were both judged appropriate based on the criteria described above. The logistic model was selected to quantify disease progression because it provided a somewhat better description of disease severity near the onset of the epidemic than the Gompertz model (2). Linearized logistic models were used with the assumption that maximum severity was 100% or 50 lesions/leaf (for lesion number calculations). Slopes of linearized regression equations ($r$) estimated the rate of disease increase, and intercept values ($y_o^*$) estimated the transformed level of disease at the onset of the epidemic (time = 0) (4,32). The logistic model provided equal fit to disease data on leaf area affected and lesion numbers. Therefore, only disease progress data for percentage leaf area affected are presented.

Data for percent leaf area affected at GS 9.3, number and lengths of northern blight lesions, $y_o^*$, $r$, and AUDPC values were analyzed by ANOVA using the general linear model procedure of SAS. For both growth chamber and field experiments, cultivar effects were further evaluated by single-degree-of-freedom contrasts and LSDs ($P = 0.05$). Separate analyses were done for each location and year. Spearman's rank correlation coefficients were calculated to determine the association among disease variables in the seedling experiment and those in the field. Correlations were performed with PROC CORR of SAS and were based on means across experiments. Additionally, cultivars were ranked separately in the growth chamber and field experiments (each year and location separately) based on composite scores for disease variables. To obtain a composite score, each variable was standardized to have a standard deviation of 5, and then the mean across variables for each cultivar was determined. PROC STANDARD of SAS was used for standardization.

## RESULTS

**Seedling resistance.** Inoculation of seedlings with four races of *E. turcicum* in the growth chamber tests revealed differences among nine Ugandan cultivars and two U.S. maize inbreds. Results of the five and three experimental repetitions with races 0 and 1, and 23 and 23N, respectively, were in general agreement based on similar ANOVA tables. Also, race and the race by host genotype interactions were not significant. Therefore, data for races 0 and 1 and races 23 and 23N were pooled for subsequent analysis.

By the third day after inoculation with the four *E. turcicum* races, chlorotic flecks had formed on most of the 10 Ugandan cultivars; however, lesions were only visible on very susceptible EV8428-S8R, EV8429-SR, B73, and EV8349-SR. By 7 days after inoculation, lesions had formed on all genotypes except Babungo 3. Chlorotic-type (resistant) lesions were produced initially on Babungo 3, but by 14 days after inoculation most lesions had turned necrotic. Resistant reactions were expressed, in part, as a delay in formation of necrotic lesions, i.e., a longer incubation period. Cultivar significantly affected incubation period ($P \leq 0.05$), but only Babungo 3 significantly delayed formation of necrotic lesions compared with the susceptible check B73 (Table 2). The majority of Ugandan cultivars showed susceptible reactions as indicated by relatively rapid formation of necrotic lesions. Cultivar significantly ($P \leq 0.001$) affected the number and length of lesions formed. Number of lesions per plant ranged from 2.2 (Babungo 3) to 7.1 (EV8428-SR) when inoculated with race 0 or 1 (Table 2). Similarly, lesion lengths ranged from 3.2 (Babungo 3) to 8.0 cm (EV8429-SR).

Differences in latent period and amount of sporulation were both significantly affected by cultivar ($P \leq 0.05$). Latent period was 3 days longer for Babungo 3 than for the more susceptible genotypes, EV8428-SR, EV8429-SR, and EV8349-SR. The number of spores produced per square centimeter of infected leaf area ranged from $1.8 \times 10^3$ to $29.0 \times 10^3$. Spore production for race 0 and 1 was significantly lower on Babungo 3, Population 42, and Mo17 than on EV8428-SR, EV8429-SR, EV8349-SR, and KWCA-SR (Table 2). However, marked variation in sporulation was observed on leaf sections of the same cultivar. In a separate experiment with EV8428-SR, KWCA-SR, and Babungo 3, there were no significant ($P \geq 0.05$) effects of race on incubation period, length and number of lesions, or sporulation when the four races were used in the same experiment (*data not presented*). Single-degree-of-freedom contrasts indicated that at the seedling stage, Babungo 3 and Mo17 were most resistant, Population 42, Across 83, Jos, Gusau, and KWCA-SR were intermediate, and EV8428-SR, EV8429-SR, EV8349-SR, and B73 were susceptible.

Correlation analysis indicated a significant and highly positive relationship between latent period and incubation period, lesion length and lesion number, and latent period and sporulation for races 0 and 1 and races 23 and 23N across the nine cultivars and two inbreds tested (Table 3). Significant and strongly negative correlations existed between incubation period and lesion number, lesion number and latent period, lesion length and latent period, and latent period and sporulation. Correlation coefficients for each disease variable, except sporulation, between races 0 and 1 and races 23 and 23N were also highly significant. This degree of correlation indicated a high level of consistency among races and the reactions expressed across cultivars and inbreds.

**Adult plant reactions.** Inoculation with race 0 at Kabanyolo and Namulonge in 1990 and 1991 and race 1 at Wooster produced a range of disease reactions on test cultivars and inbreds. In 1990, final disease severity ranged from 4% on Babungo 3 to 48% on B73 at Namulonge; in 1991 final disease severity ranged from 1% on Babungo 3 to 75% on B73 at Kabanyolo (Table 4). Final disease severity on B73, B73*Ht*1, and Mo17 was greatest at Kabanyolo in 1991, where disease severity on these inbreds reached 75, 59, and 40%, respectively, compared with 21% on the most susceptible Uganda line, EV8428-SR.

Final percentage of leaf area affected ($y_f$) and AUDPC values provided adequate evaluation of the reaction of the Ugandan cultivars to *E. turcicum* at Kabanyolo and Namulonge (Table 4). In 1990 and 1991, the 10 Ugandan cultivars had significantly lower $y_f$ values than B73 and B73*Ht*1 in three out of four tests at the two locations. In general, the level of disease on the more susceptible Ugandan cultivars was similar to that of Mo17. How-

TABLE 2. Characteristics of northern leaf blight and sporulation of *Exserohilum turcicum* on seedlings of nine open-pollinated maize cultivars from Uganda and inbreds B73 and Mo17 inoculated with four races of E. turcicum[a]

| Genotype | Means for races 0 and 1[b] | | | | | Means for races 23 and 23N[b] | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Incubation period[c] (days) | Lesion number[d] | Lesion length[d] (cm) | Latent period[e] (days) | Sporulation[f] | Incubation period[c] (days) | Lesion number[d] | Lesion length[d] (cm) | Latent period[e] (days) | Sporulation[f] |
| B73 | 5.6[g] | 4.7 | 6.7 | 6.9 | 8.3 | 5.9 | 3.9 | 5.9 | 6.9 | 8.6 |
| Mo17 | 8.1 | 2.2 | 4.1 | 7.8 | 7.3 | 7.0 | 1.9 | 4.3 | 8.0 | 8.3 |
| EV8428-SR | 5.3 | 7.1 | 7.8 | 5.4 | 8.4 | 5.5 | 6.0 | 8.3 | 5.4 | 9.0 |
| EV8429-SR | 5.5 | 6.4 | 8.0 | 5.6 | 8.4 | 5.8 | 5.6 | 8.9 | 5.3 | 8.7 |
| Gusau TZB-SR | 6.4 | 4.5 | 6.5 | 6.8 | 7.8 | 7.1 | 3.2 | 7.2 | 6.7 | 8.7 |
| Jos | 6.8 | 4.4 | 6.1 | 7.7 | 7.7 | 6.9 | 3.6 | 8.1 | 6.7 | 9.0 |
| Across 83 TZM-SR | 8.2 | 3.5 | 4.8 | 7.1 | 7.8 | 7.4 | 2.5 | 7.0 | 7.0 | 8.8 |
| KWCA-SR | 6.8 | 3.9 | 6.8 | 6.8 | 8.2 | 7.0 | 2.8 | 9.0 | 6.8 | 9.2 |
| EV8349-SR | 6.4 | 5.0 | 7.0 | 6.4 | 8.2 | 6.8 | 3.7 | 7.0 | 5.9 | 8.9 |
| Population 42 | 8.3 | 3.0 | 5.0 | 8.9 | 7.1 | 8.9 | 2.1 | 5.4 | 8.1 | 8.4 |
| Babungo 3 | 14.8 | 2.2 | 3.2 | 9.8 | 7.3 | 13.8 | 1.5 | 4.4 | 9.1 | 7.1 |
| LSD ($P = 0.05$) | 4.7 | 0.2 | 0.2 | 3.0 | 0.6 | 5.3 | 0.3 | 0.5 | 2.6 | 0.4 |

[a] Races 0 and 1 were field isolates and were studied separately from races 23 (ATCC 64836) and 23N (ATCC 64834). Seedlings in the four- to six-leaf stage were inoculated with a suspension (30,000 conidia/ml) and then grown in the growth chamber at 22 C day/18 C night and light intensity of approximately 280 $\mu E \cdot m^{-2} \cdot s^{-1}$.
[b] There were no significant differences in disease variables between races 0 and 1 or between 23 and 23N.
[c] Days from inoculation to formation of necrotic lesions >1.5 cm.
[d] Sixteen days after inoculation.
[e] Days from inoculation to conidium formation.
[f] Assessed 13 days after inoculation; data are transformed (ln[spores + 1]$cm^{-2}$) values.
[g] Data are means of 20 (races 0 and 1) and 12 (races 23 and 23N) values (four replicate pots per experiment, five and three repeats of the experiment, respectively).

TABLE 3. Spearman's rank correlation coefficients among assessments used to quantify northern leaf blight reaction in seedlings of nine open-pollinated maize cultivars from Uganda and inbreds B73 and Mo17 inoculated with *Exserohilum turcicum*[a]

| Assessment | Incubation period[b] | Lesion number | Lesion length | Latent period[c] | Sporulation |
|---|---|---|---|---|---|
| Races 0 and 1 | | | | | |
| Lesion number | −0.94***[d] | ... | | | |
| Lesion length | −0.89*** | 0.92*** | ... | | |
| Latent period | 0.90*** | −0.91*** | −0.93*** | ... | |
| Sporulation | −0.90*** | 0.89*** | 0.88*** | −0.91*** | ... |
| Races 23 and 23N | | | | | |
| Lesion number | −0.92*** | ... | | | |
| Lesion length | −0.53 | 0.65* | ... | | |
| Latent period | 0.82*** | −0.89*** | −0.78* | ... | |
| Sporulation | −0.47 | 0.55 | 0.84*** | −0.91*** | ... |
| Races 0 and 1 versus 23 and 23N | 0.92*** | 0.98*** | 0.77** | 0.92*** | 0.53 |

[a] Pooled data for races 0 and 1 and races 23 and 23N, across cultivars and inbreds.
[b] Days from inoculation to formation of necrotic lesions >1.5 cm.
[c] Days from inoculation to conidium formation.
[d] Asterisks indicate significance levels: * $P \leq 0.05$, ** $P \leq 0.01$, *** $P \leq 0.001$.

ever, $y_f$ and AUDPC values for Babungo 3, Population 42, KWCA, and KWCA-SR were significantly lower than those of Mo17 in each test.

Estimates of $r$ were significantly ($P \leq 0.001$) affected by cultivar in both 1990 and 1991 at each location (Table 4). In 1990 and 1991, $r$ was similar for B73 and B73*Ht*1, and was significantly higher for these two inbreds than for Babungo 3, Population 42, and KWCA.

Disease onset, as indicated by $y_o^*$ (= logit of disease severity [$y$] at time 0), was not significantly affected by cultivar at Kabanyolo in 1990 (Table 4). At Namulonge in 1990, however, all Ugandan cultivars responded with a significantly larger $y_o^*$ ($P \leq 0.001$) than B73*Ht*1, Mo17, and B73. However, differences among Ugandan cultivars were not always significant. For example, Babungo 3 had $y_o^* = -20.3$ compared with $y_o^* = -19.1$ for EV8428-SR, the more susceptible cultivar.

Fewer lesions formed on Babungo 3, Population 42, and KWCA than on Mo17, B73*Ht*1, and B73 (Table 5). This relationship was consistent across locations in 1990 and 1991. In both 1990 and 1991, B73*Ht*1 had fewer lesions than B73 in Uganda. Lesion length was significantly ($P \leq 0.001$) affected by maize genotype but, in contrast to seedling results, differences were not associated with overall genotypic resistance to *E. turcicum*. For example, Babungo 3 had longer lesions than B73.

Reactions of the maize cultivars to race 1 were evaluated at Wooster in 1991. Dry conditions prevailed in Wooster, and generally only low disease levels developed. Disease was restricted to inoculated leaves. Even with low levels of disease, cultivar effects were significant for final severity, AUDPC, $r$, $y_o^*$, lesion number, and lesion length ($P < 0.001$) (Tables 5–7). There were no significant differences among the majority of Ugandan cultivars, partly because of the low disease levels, but both B73 and B73*Ht*1 had significantly higher $r$ and AUDPC values and more lesions than the Ugandan cultivars.

At Wooster, only lesions indicating a completely susceptible response developed on B73*Ht*1. In contrast in Uganda, only chlorotic lesions formed on B73*Ht*1 and necrotic lesions formed on B73, as expected on the basis of races used (race 0 in Uganda, 1 in Wooster).

Correlations among disease variables from seedling tests and inoculated adult plant trials were inconsistent (Table 6). Significant relationships were obtained for incubation period on seedlings and disease severity, AUDPC, and lesion length on adult plants; lesion number on seedlings and disease severity, AUDPC, and lesion length on adult plants; and latent period of seedlings and lesion length on adult plants. Latent period and lesion length on seedlings showed little relationship with most adult plant disease variables.

For adult plants, ranks of cultivars for resistance were similar for different locations and years (Table 7). There also was similarity in the ranks for seedling and adult plants. However, expression of resistance to *E. turcicum* differed between seedlings and adult plants of a few cultivars. Mo17, the polygenic resistant check, was resistant as seedlings and as adult plants in Wooster. In Uganda, however, Mo17 was more susceptible than the majority of Ugandan cultivars.

## DISCUSSION

In this study, resistance to northern leaf blight in the Ugandan cultivars, especially Babungo 3, KWCA, and Population 42, was

expressed as lower $r$, AUDPC, and final severity. Such resistance is termed rate-reducing resistance (18,19,20,29), and although the genetics of resistance was not studied here, this resistance is considered to be under the control of several genes (9,12,13,18), stable, and effective against all physiological races of *E. turcicum* (12).

Differences in response to northern leaf blight among cultivars were apparent between adult plant tests and seedling tests. Low

TABLE 4. Final severity ($y_f$) of northern leaf blight, caused by *Exserohilum turcicum*, standardized area under disease progress curve (AUDPC), slope ($r$), and intercept ($y_o^*$) for regression of logit transformation of percentage leaf area affected over time for 10 open-pollinated cultivars from Uganda and three inbreds at two locations in Uganda, 1990 and 1991, and Wooster, 1991[a]

| | Kabanyolo | | | | Namulonge | | | | Wooster[d] | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Genotype | $y_f$[b] | AUDPC[c] | $r$ | $y_o^*$ | $y_f$ | AUDPC[c] | $r$ | $y_o^*$ | $y_f$ | AUDPC[c] | $r$ | $y_o^*$ |
| 1990 | | | | | | | | | | | | |
| B73 | 53.6 | 123.7 | 0.041 | −6.1 | 47.9 | 75.4 | 0.082 | −29.3 | ... | ... | ... | ... |
| B73*Ht1* | 38.1 | 78.4 | 0.041 | −5.7 | 19.7 | 25.4 | 0.071 | −26.9 | ... | ... | ... | ... |
| Mo17 | 30.2 | 47.9 | 0.011 | −6.1 | 23.3 | 23.5 | 0.086 | −31.5 | ... | ... | ... | ... |
| EV8428-SR | 23.1 | 37.4 | 0.010 | −5.0 | 24.8 | 52.1 | 0.050 | −19.1 | ... | ... | ... | ... |
| EV8429-SR | 20.1 | 32.1 | 0.009 | −5.8 | 23.3 | 61.5 | 0.039 | −14.9 | ... | ... | ... | ... |
| Gusau TZB-SR | 17.1 | 29.9 | 0.010 | −6.3 | 21.9 | 42.0 | 0.052 | −19.6 | ... | ... | ... | ... |
| Jos | 13.0 | 22.7 | 0.008 | −5.7 | 16.3 | 30.8 | 0.048 | −18.8 | ... | ... | ... | ... |
| Across 83 TZM-SR | 11.7 | 25.3 | 0.008 | −5.5 | 12.6 | 20.9 | 0.049 | −19.4 | ... | ... | ... | ... |
| KWCA-SR | 11.9 | 15.5 | 0.009 | −6.5 | 7.2 | 13.5 | 0.040 | −16.6 | ... | ... | ... | ... |
| EV8349-SR | 10.7 | 12.9 | 0.010 | −6.7 | 9.0 | 18.6 | 0.043 | −17.5 | ... | ... | ... | ... |
| KWCA | 8.1 | 12.7 | 0.008 | −6.3 | 7.0 | 16.9 | 0.033 | −14.3 | ... | ... | ... | ... |
| Population 42 | 11.0 | 9.8 | 0.011 | −7.5 | 8.2 | 16.5 | 0.041 | −16.9 | ... | ... | ... | ... |
| Babungo 3 | 6.2 | 7.9 | 0.009 | −6.8 | 3.5 | 2.7 | 0.048 | −20.3 | ... | ... | ... | ... |
| LSD ($P = 0.05$) | 7.1 | 21.8 | 0.010 | NS | 4.5 | 7.5 | 0.011 | 3.8 | ... | ... | ... | ... |
| 1991 | | | | | | | | | | | | |
| B73 | 75.0 | 84.4 | 0.080 | −11.5 | 58.3 | 61.2 | 0.095 | −14.8 | 27.9 | 22.6 | 0.099 | −26.3 |
| B73*Ht1* | 59.4 | 23.0 | 0.109 | −17.5 | 37.1 | 25.4 | 0.099 | −16.6 | 27.5 | 23.2 | 0.100 | −26.4 |
| Mo17 | 40.0 | 14.2 | 0.087 | −14.9 | 19.5 | 15.2 | 0.076 | −13.5 | 1.3 | 1.3 | 0.024 | −10.0 |
| EV8428-SR | 20.8 | 19.3 | 0.057 | −9.4 | 19.8 | 18.7 | 0.065 | −12.2 | 3.2 | 3.8 | 0.044 | −14.5 |
| EV8429-SR | 15.7 | 13.6 | 0.050 | −9.7 | 17.1 | 13.7 | 0.076 | −14.0 | 2.3 | 4.0 | 0.030 | −10.8 |
| Gusau TZB-SR | 14.4 | 12.9 | 0.051 | −9.9 | 15.5 | 11.7 | 0.058 | −11.3 | 1.0 | 1.4 | 0.022 | −9.6 |
| Jos | 7.5 | 6.7 | 0.042 | −9.4 | 10.9 | 10.4 | 0.063 | −12.4 | 1.0 | 1.3 | 0.023 | −9.3 |
| Across 83 TZM-SR | 8.2 | 7.2 | 0.042 | −9.0 | 8.1 | 7.9 | 0.044 | −9.4 | 1.3 | 1.9 | 0.024 | −10.0 |
| KWCA-SR | 5.6 | 6.0 | 0.034 | −8.1 | 6.9 | 6.1 | 0.051 | −10.9 | 1.2 | 1.6 | 0.020 | −9.1 |
| EV8349-SR | 5.1 | 5.7 | 0.039 | −8.9 | 10.0 | 9.0 | 0.060 | −12.0 | 0.5 | 0.5 | 0.013 | −7.8 |
| KWCA[e] | 3.1 | 2.5 | 0.030 | −8.0 | 4.0 | 3.8 | 0.048 | −10.7 | ... | ... | ... | ... |
| Population 42 | 1.8 | 1.5 | 0.029 | −8.1 | 4.1 | 3.4 | 0.042 | −10.0 | 0.1 | 0.4 | 0.008 | −6.4 |
| Babungo 3 | 1.0 | 1.0 | 0.014 | −6.2 | 1.5 | 1.7 | 0.025 | −7.7 | 0.0 | 0.1 | 0.003 | −5.2 |
| LSD ($P = 0.05$) | 6.8 | 7.66 | 0.016 | 2.5 | 6.1 | 7.6 | 0.022 | 3.4 | 2.1 | 2.3 | 0.011 | 2.3 |

[a] Plants were inoculated with race 0 (Uganda) or race 1 (Wooster) at GS 4, 5, and 6. Data are means of four replicates.
[b] Final severity rating at growth stage (GS) 9.3, where severity was estimated as percentage leaf area affected.
[c] AUDPC based on severity ratings at GS 8, 9.0, 9.1, 9.2, 9.3, and 9.4. Area divided by time span of disease assessments to obtain standardized values.
[d] No trials were conducted in Wooster in 1990.
[e] KWCA (Kawanda composite A) was not studied at Wooster.

TABLE 5. Mean final number and length of northern leaf blight lesions on 10 open-pollinated maize cultivars and three inbreds at two locations in Uganda, 1990 and 1991, and Wooster, 1991[a]

| | Final lesion number[b] | | | | | Mean lesion length[c] (cm) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Kabanyolo | | Namulonge | | Wooster | Kabanyolo | | Namulonge | | Wooster |
| Genotype | 1990 | 1991 | 1990 | 1991 | 1991 | 1990 | 1991 | 1990 | 1991 | 1991 |
| B73 | 37.9 | 18.8 | 23.0 | 14.2 | 18.4 | 11.7 | 12.9 | 13.0 | 12.8 | 13.3 |
| B73*Ht1* | 29.9 | 11.1 | 18.4 | 9.2 | 17.2 | 10.9 | 8.7 | 8.4 | 8.4 | 14.2 |
| Mo17 | 11.4 | 8.3 | 12.2 | 8.9 | 0.9 | 12.0 | 12.0 | 12.2 | 11.8 | 13.2 |
| EV8428-SR | 12.3 | 7.9 | 14.1 | 9.8 | 1.1 | 18.0 | 23.4 | 21.8 | 22.6 | 26.7 |
| EV8429-SR | 11.7 | 6.7 | 12.6 | 8.0 | 0.9 | 19.0 | 21.2 | 18.6 | 19.8 | 18.8 |
| Gusau TZB-SR | 11.6 | 6.4 | 12.2 | 9.3 | 0.3 | 19.6 | 19.8 | 18.9 | 19.8 | 17.4 |
| Jos | 8.5 | 3.7 | 8.8 | 7.1 | 0.2 | 16.6 | 17.1 | 16.7 | 19.1 | 16.1 |
| Across 83 TZB-SR | 9.4 | 3.3 | 9.6 | 6.9 | 0.2 | 15.9 | 17.1 | 18.1 | 17.6 | 14.4 |
| KWCA-SR | 4.8 | 2.5 | 4.5 | 3.2 | 0.4 | 15.9 | 19.9 | 18.8 | 21.2 | 17.1 |
| EV8349-SR | 5.3 | 3.2 | 5.0 | 5.1 | 0.1 | 12.6 | 15.6 | 16.2 | 15.0 | 16.6 |
| KWCA[d] | 4.0 | 1.6 | 3.3 | 3.9 | ... | 19.3 | 19.9 | 19.8 | 20.1 | ... |
| Population 42 | 4.0 | 1.1 | 3.0 | 3.0 | 0.0 | 15.0 | 16.2 | 17.6 | 17.3 | ...[e] |
| Babungo 3 | 3.4 | 0.6 | 1.8 | 2.6 | 0.0 | 16.4 | 18.6 | 18.8 | 19.3 | ...[e] |
| LSD ($P = 0.05$) | 4.2 | 1.0 | 2.9 | 2.0 | 1.1 | 3.5 | 4.5 | 4.5 | 4.8 | 5.7 |

[a] Plants were inoculated at growth stages (GS) 4, 5, and 6 with *Exserohilum turcicum* races 0 (Uganda) or 1 (Wooster).
[b] Data represent the mean number of lesions on the ear leaf of 10 plants per experimental unit with four replicate blocks used at Kabanyolo and Namulonge and eight at Wooster. Lesion numbers counted at GS 9.3.
[c] Lesion length measured at GS 9.2 from 20 lesions on 10 plants per experimental unit with four replicate blocks at Kabanyolo and Namulonge and eight at Wooster.
[d] KWCA was not studied at Wooster.
[e] Too few lesions formed for valid comparisons, and data from these cultivars were not used in statistical analysis.

correlations between disease assessments on seedling and adult plants for *E. turcicum* on maize (21), *Puccinia graminis* f. sp. *avenae* on *Avena sterilis* (30), and on other pathosystems reviewed by Parlevliet (20) have been reported. However, several disease variables for seedlings were correlated with disease variables measured on adult plants in the field. For several genotypes, overall rankings were also similar for the field and growth chamber studies. For instance, Babungo 3, the most resistant cultivar tested at seedling stage, was also the most resistant genotype tested under field conditions. Resistance in Babungo 3 at the seedling stage was expressed as a delay in formation of necrotic lesions, fewer and shorter lesions, a longer latent period, and significantly less sporulation of *E. turcicum* within lesions. In the field, resistance was expressed as a significant reduction in the number of lesions but without a corresponding reduction in lesion length. Therefore, all the various manifestations of rate-reducing resistance as expressed in Ugandan cultivars may not be apparent in seedling plants.

TABLE 6. Spearman's rank correlation coefficients among assessments used to quantify northern leaf blight reactions in seedling and adult maize plants inoculated with *Exserohilum turcicum* races 0, 1, 23, or 23N (seedlings) and 0 or 1 (adult plants)[a]

| Adult plant variables | Seedling variables | | | | |
|---|---|---|---|---|---|
| | Incubation period[b] | Lesion number | Lesion length | Latent period[c] | Sporulation |
| Wooster[d] | | | | | |
| Severity | −0.70**[e] | 0.73* | 0.55 | −0.53 | 0.35 |
| AUDPC[f] | −0.67* | 0.70** | 0.52 | −0.52 | 0.28 |
| $r$[f] | −0.73** | 0.65* | 0.26 | −0.45 | 0.15 |
| $y_o^*$[f] | 0.70** | −0.64* | −0.25 | 0.45 | −0.12 |
| Lesion number | 0.74** | 0.60* | 0.32 | −0.44 | 0.31 |
| Lesion length | −0.43** | 0.62* | 0.80** | −0.86** | 0.59 |
| Uganda[d] | | | | | |
| Severity | −0.64*** | 0.52*** | 0.04 | −0.34* | 0.10 |
| AUDPC | −0.60*** | 0.56*** | 0.06 | −0.32* | 0.07 |
| $r$ | −0.21 | −0.03 | 0.14 | −0.19 | 0.45** |
| $y_o^*$ | 0.02 | 0.16 | −0.14 | 0.10 | −0.40** |
| Lesion number | −0.12 | 0.23 | 0.66*** | −0.42** | 0.40** |
| Lesion length | −0.62** | 0.55*** | 0.09 | −0.36** | 0.08 |

[a] Data averaged for races 0, 1, 23, and 23N (seedlings), and for locations and years in Uganda (adult plant resistance).
[b] Days from inoculation to formation of necrotic lesions >1.5 cm.
[c] Days from inoculation to conidium formation.
[d] Races 0 and 1 were studied in Uganda and Wooster, respectively (adult plant resistance); seedling studies were conducted at The Ohio Agricultural Research and Developmental Center, Wooster.
[e] Asterisks indicate significance level: * $P \leq 0.05$, ** $P \leq 0.01$, *** $P \leq 0.001$.
[f] Standardized area under disease progress were (AUDPC), slope ($r$), and intercept ($y_o^*$) for regression of logit transformation of percentage leaf area affected on time.

The marked reduction in rate of disease progress in resistant cultivars Babungo 3, Population 42, and KWCA compared with Mo17, B73*Ht*1, and B73 suggested that these cultivars possessed high levels of rate-reducing resistance to *E. turcicum*. Although B73*Ht*1 delayed onset of northern leaf blight in Uganda, disease subsequently developed and was accompanied by numerous lesions with extensive chlorosis. Although smaller lesions were formed on B73*Ht*1, the extensive chlorosis accounted for the high severity and AUDPC values. In some cases, rate of disease increase and AUDPC were higher in B73*Ht* than in B73. These results confirm earlier findings that damage to genotypes with *Ht* resistance genes can be as high as to genotypes without *Ht* genes (21–23,25,26,31). Severe chlorosis associated with the *Ht* gene accounted for lower yields of hybrids with *Ht* genes than in those with polygenic resistance (31). In studies with race 0, Perkins (21) also found *Ht*1 to be effective under moderate disease intensities. To increase effectiveness of single-gene (*Ht*) resistance (8,22,23,26), a combination of polygenic resistance to reduce lesion numbers and monogenic resistance to suppress sporulation may be preferred. In our study with Ugandan cultivars, rate-reducing resistance present in cultivars like Babungo 3 appeared effective alone but could be used with other control strategies such as effective rotation and residue management.

Examination of the disease progress curves, AUDPCs, final severities, and numbers of lesions revealed that the cultivar effects on epidemics of northern leaf blight were fairly consistent in both years. Rates of disease progress were similar across locations in 1991 but different between locations in 1990. Overall, severity of northern leaf blight increased more rapidly in 1991 than in 1990, probably because of the wetter conditions in 1991 in Uganda. Furthermore, although significant differences between genotypes

TABLE 7. Composite scores and rankings of 10 open-pollinated maize cultivars and three inbreds for resistance to four races (0, 1, 23, and 23N) of *Exserohilum turcicum* at seedling stage, and two races (0 and 1) at adult plant stage in two locations in Uganda and in Wooster, 1989, 1990, and 1991[a,b]

| Genotype | Seedling plants | Adult plants | | | | | |
|---|---|---|---|---|---|---|---|
| | | Kabanyolo | | Namulonge | | Wooster | |
| | 1989 | 1990 | 1991 | 1990 | 1991 | 1991 | Pooled |
| B73 | 16.6 (7)[c] | 27.7 (13) | 29.1 (13) | 22.1 (13) | 23.6 (13) | 16.5 (8) | 7.6 (11) |
| B73*Ht*1 | ...[d] | 25.6 (12) | 25.0 (12) | 21.1 (12) | 21.1 (12) | 20.0 (12) | ...[d] |
| Mo17 | 13.9 (4) | 20.8 (11) | 22.6 (11) | 20.8 (11) | 20.6 (11) | 13.9 (4) | 5.4 (7) |
| EV8428-SR | 19.5 (11) | 20.2 (10) | 20.6 (10) | 20.5 (10) | 20.4 (10) | 19.4 (11) | 7.2 (10) |
| EV8429-SR | 18.3 (10) | 19.7 (9) | 19.7 (9) | 20.3 (9) | 20.3 (9) | 18.3 (10) | 6.8 (9) |
| Gusau TZB-SR | 16.1 (6) | 19.5 (8) | 19.5 (8) | 20.0 (8) | 20.1 (8) | 16.1 (7) | 5.8 (8) |
| Jos | 16.6 (7) | 18.5 (6) | 18.6 (7) | 19.9 (7) | 19.9 (7) | 16.6 (8) | 5.1 (6) |
| Across 83 TZM-SR | 13.8 (3) | 18.8 (7) | 18.5 (6) | 19.8 (6) | 19.8 (5) | 13.8 (3) | 4.3 (5) |
| KWCA-SR | 17.0 (9) | 17.9 (4) | 17.9 (4) | 19.8 (6) | 19.9 (7) | 17.0 (9) | 4.2 (4) |
| EV8349-SR | 16.0 (5) | 17.9 (4) | 18.1 (5) | 19.7 (4) | 19.8 (5) | 16.0 (5) | 5.4 (3) |
| KWCA | ...[d] | 17.4 (3) | 17.7 (3) | 19.3 (3) | 19.3 (3) | ...[d] | ...[d] |
| Population 42 | 12.8 (2) | 17.2 (2) | 17.6 (2) | 18.9 (2) | 19.2 (2) | 12.8 (2) | 3.9 (2) |
| Babungo 3 | 9.8 (1) | 16.5 (1) | 17.2 (1) | 16.9 (1) | 17.2 (1) | 9.8 (1) | 3.1 (1) |

[a] Seedling studies were conducted at The Ohio Agricultural Research and Development Center (OARDC), Wooster; seedlings in the four- to six-leaf stage were inoculated with a suspension (30,000 conidia) and then grown in the growth chamber at 22 C day/18 night and light intensity of approximately 280 $\mu E \cdot m^{-2} \cdot s^{-1}$. Adult plants were inoculated with races 0 (Uganda) and 1 (Wooster) at growth stages (GS) 4, 5, and 6.
[b] Scores were computed from means standardized to have a standard deviation of 5. Standardized variables of incubation period, latent period, and lesion number were averaged for seedling composite scores; standardized variables of percentage leaf area affected at GS 9.3, area under the disease progress curve, lesion number, and slope ($r$) of the logit transformation of percentage leaf area affected were averaged for adult plant composite scores.
[c] Overall composite score for seedling and adult plant resistance. Figures in parentheses represent rank order from 1 to 13, with 1 = most resistant and 13 = most susceptible.
[d] Not studied at seedling stage (B73*Ht*1 and KWCA) or in Wooster (KWCA).

were shown using $r$ and $y_0^*$, $r$ and $y_0^*$ could not separate genotypes as well as final lesion numbers, final severity ratings, and AUDPC.

Necrotic (susceptible) lesions were produced in all Ugandan maize genotypes in response to inoculations with races 0, 1, 23, and 23N. Chlorotic lesions were produced only in genotype B73 with *Ht*1 in Uganda. When inoculated with race 1 in Wooster, B73*Ht*1 developed necrotic lesions. In the seedling stage, necrotic lesions developed on all Ugandan cultivars 14 days after inoculation. Lack of significant cultivar by race interactions suggested that there was no race specificity in cultivar differences and that the resistance expressed in the Ugandan maize cultivars was not under control of any of the known *Ht* resistance alleles (9–11).

This study identified useful sources of resistance to northern leaf blight. On the basis of correlations between seedling and adult plant results for some variables, seedling tests can help identify sources of chlorotic resistance and indicate susceptible genotypes, but field tests are necessary to fully characterize rate-reducing resistance. On seedlings, incubation period and lesion number, and on adult plants, percentage of leaf area affected and lesion number seem to be useful for assessing rate-reducing resistance to northern leaf blight.

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