Characterization of Race-Nonspecific Resistance to *Exserohilum turcicum* Races 0 and 1 in Maize OhS10 S_1 Progenies

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ABSTRACT

Pratt, R. C., Adipala, E., and Lipps, P. E. 1993. Characterization of race-nonspecific resistance to *Exserohilum turcicum* races 0 and 1 in maize OhS10 S_1 progenies. Plant Dis. 77:1227-1232.

Three field studies were conducted in 1990 and 1991 to characterize the type and level of resistance of 149 OhS10 selfed progenies to races 1 (tested in Ohio) and 0 (tested in Uganda) of *Exserohilum turcicum*, causal agent of northern leaf blight (NLB). Maize plants were inoculated in a controlled fashion, and disease severity was assessed once late in the season using a 0–5 visual rating scale. Visual estimates of percent leaf area affected (PLAA) and counts of lesions were made four to six times following inoculation. Host responses to infection were also characterized by determining lesion size, apparent infection rate, and area under the disease progress curve (AUDPC) calculated from PLAA and lesion number. Progenies ranged in degree of resistance from susceptible to highly resistant. The top tenth percentile of progenies were selected for study based on the 0–5 rating scale. The relative ranking of resistance to both races, and characterization of their host responses, indicated that selected progenies displayed a high level of race-nonspecific resistance. The 14 most resistant progenies displayed fewer lesions, lower PLAA ratings, lower rating scores (0–5), lower AUDPC values, and lower apparent infection rates than did the resistant inbred check (Mo17). Lesion lengths on the resistant progenies did not differ from those on Mo17. Disease assessment methods were generally highly correlated with one another.

Northern leaf blight (NLB), caused by the fungus *Exserohilum turcicum* (Pass.) K.J. Leonard & E.G. Suggs, is a potentially destructive disease in many maize (*Zea mays* L.) growing regions of the world (6,34). When epidemics begin before silking and environmental conditions favor disease development, yield loss in excess of 40% may result (26, 27,35). Resistance is the primary means of controlling NLB. Race-specific and race-nonspecific sources of resistance to *E. turcicum* are available in dent and sweet maize germ plasm (9,16,23). Monogenic, race-specific resistance, under the control of *Ht*, *Ht*2, and *Ht*3 genes, is expressed as chlorotic-lesion type with marked reduction in sporulation (12,13); and *Ht*N is expressed as delayed lesion development in adult plants (10). Rate-reducing, or race-nonspecific, resistance is typically characterized by fewer lesions and a lower rate of disease development than is observed in susceptible genotypes (1,15,16,30). Race-nonspecific resistance of maize to *E. turcicum* is considered to be inherited polygenically (15–18).

Widespread use of the *Ht*1 gene in commercial maize cultivars produced in the United States has apparently contributed to a change in virulence of the *E. turcicum* population (25). Since 1980, five physiological races of *E. turcicum* have been identified (21,36). Race 0 was formerly predominant in Ohio, but race 1 is now widespread in Ohio and other midwestern states of the United States (19,21,22,33). The importance of single gene resistance to the control of *E. turcicum* has diminished because of extensive chlorosis associated with the *Ht* genes and lack of durability of race-specific resistance. The development of multiple races in the United States emphasizes the need for a more durable type of resistance (21) for control of NLB.

Many improved open-pollinated cultivars of maize with potential for cultivation in Uganda are susceptible to NLB (2). Only race 0 has been identified in Uganda (2), so emphasis on the identification of improved cultivars with polygenic, race-nonspecific resistance instead of monogenic, race-specific resistance will assist in the suppression of new races of NLB. Several maize open-pollinated cultivars from Uganda that display race-nonspecific resistance to races 0 and 1 recently have been described (1), and additional germ plasm is needed to support breeding efforts.

Polygenic resistance is desirable not only because it is effective against all physiological races of *E. turcicum* (15),

Manuscript No. 126-93, Ohio State University, Ohio Agricultural and Development Center, Wooster 44691. The mention of firm names of trade products does not imply that they are endorsed or recommended by Ohio State University over other firms or similar products not mentioned.

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Accepted for publication 11 September 1993.

but also because it can be used in combination with monogenic resistance. When combined, monogenic and polygenic resistance would be expected to increase the durability of monogenic resistance and also enhance expression of resistance (15,20).

Plant breeders and pathologists are particularly challenged by the need to identify both highly resistant and race-nonspecific host responses when assessing uncharacterized germ plasm populations with a broad genetic base, e.g., open-pollinated cultivars and composite or synthetic cultivars or breeding populations (1). Resistance genes are likely to be segregating, and allelic frequencies are likely to be unknown. Breeders typically evaluate population progenies (full-sib, half-sib, or self-pollinated) (11) using a visual rating scale, e.g., 0–5 (6) or 1–9 (24). Because of the labor-intensive nature of evaluating large numbers of segregating progenies in a breeding program, breeders often conduct selection using minimal or no replication in the disease nursery and only one late-season disease assessment. Less frequently, multiple ratings of percent leaf area affected (PLAA) (24) or area under the disease progress curve (AUDPC) (6) may be undertaken. Local isolates are typically used for controlled inoculation, or nurseries are planted in areas where naturally occurring disease pressure is anticipated. If multiple races are not known in a region, breeders are precluded from testing for race-nonspecific resistance because of restrictions on using nonendemic racial isolates of the pathogen.

The objectives of our study were to determine if high levels of resistance to *E. turcicum* were present in the OhS10 population, and to ascertain whether race-nonspecific resistance could be identified by evaluation with race 1 in Ohio and race 0 in Uganda. This report further describes the host responses of resistant OhS10 S_1 progenies selected for study based on late-season disease reaction (9), with more descriptive disease assessment methods. Part of the findings reported in this paper were previously published in an abstract (8).

MATERIALS AND METHODS

Host plants. The OhS10 synthetic population is comprised of one-half tropical maize germ plasm and one-half U.S. Cornbelt germ plasm, and is a potential resource to maize breeders in both temperate and tropical regions. Development of OhS10 was initiated by William R. Findley (USDA/ARS, retired) by crossing OhS7 by OhS8. It is a broad-based population developed from 14 U.S. Cornbelt inbreds and two tropical races of maize. OhS7 is composed of (M14, H73, MS1334, A96, A239, Oh26*o*2, Pa32, W22R) × (PD(MS)6 × Tuxpeno), and OhS8 is composed of (H55, B14A, B37, Oh7B, Pa884P, CI64) × (PD(MS)6 × Tuxpeno). PD(MS)6 and Tuxpeno are Cuban Flint and Tuxpeno races of maize, respectively. Some of the inbreds used for constituting OhS10 (H55, CI64, MS1334) are considered resistant to *E. turcicum* (14,17,30). The inbred Pa32 may also have contributed some resistance to the population (14). The Tuxpeno (Mexican Dent) germ plasm is considered a good source of resistance to *Helminthosporium* (*Exserohilum*) spp. (4). We are not aware of reports describing the resistance of PD(MS)6, A96, or H73. All other genotypes are known to be susceptible (14; W. R. Findley, *personal communication*). The OhS10 (Cycle 0) population was grown at the USDA/ARS experimental farm in Isabella, Puerto Rico, in 1989; and S_1 progenies were produced by controlled self-pollination of S_0 plants. Mild visual selection pressure for desirable agronomic characteristics was imposed at flowering, during harvest of S_0 plants, and during shelling of ears. The S_1 progenies were designated S-1 to S-150.

For comparison, maize inbreds B73 (susceptible check) and Mo17 (resistant check) were included in all experiments. Inbred NN14B*Ht*2 was used in Ohio (1990), and B73*Ht*1 and inbreds Mo17-*Ht*1 were used in the race 0 experiment in Uganda (1991) to confirm the racial status of isolates used for inoculation. Additionally, susceptible (EV8428-SR), intermediate or moderately resistant (KWCA-SR), and resistant (Babungo-3) open-pollinated cultivars (1) from Uganda were included in the trial but were not included in the data analysis.

Field plots. Plots were established at the Ohio Agricultural Research and Development Center near Wooster, and at the Makerere University Agricultural Research Institute at Kabanyolo, Uganda. Throughout the rest of this paper, all experiments are identified as Ohio or Uganda and by the year in which they were planted. In Ohio, kernels of 150 S_1 progenies were planted on 2 May 1990 and 16 May 1991 in single-row plots in a randomized complete-block design (31) with three replicates. Each 3-m-long single-row plot consisted of approximately 15 plants. To increase the spread of *E. turcicum,* kernels of B73 were planted in every other row. Kernels of Funk's 4671 (Ohio, 1990–1991) and KWCA (Uganda, 1991) were planted as four-row borders around the entire respective experiments. Plots in Ohio had been maintained in a corn-soybean-oat-wheat rotation. After plowing in spring, the plots were fertilized with 190 kg of urea per hectare and then "cultimulched" prior to planting. After hand-planting, plots were sprayed with 4 L/ha of 43% a.i. cyanazine. In Uganda, field plots were established in fields previously planted to soybean (*Glycine max* (L.) Merr.). No fertilizers or herbicides were applied, and plots were subsequently hand-weeded when the average maturity of progenies was at growth stages (GS) V1 (first true leaf collar visible), V6 (sixth true leaf collar visible), V12 (twelfth true leaf collar visible), V18 (eighteenth true leaf collar visible), and R2 (blister stage, 10–14 days after silking) (28).

Races and inoculum preparation. Monoconidial cultures of field isolates of race 1 from Madison Seed Company, London, Ohio, and race 0 from Nakulabywe, Kampala, Uganda, were grown on Difco potato-dextrose agar (PDA) in plastic petri dishes. After 12–14 days at 21–23 C under fluorescent lights for 1 hr/day, colonized agar cultures were removed from petri dishes and placed on autoclaved oat (*Avena sativa* L.) kernels (Ohio) or sorghum (*Sorghum bicolor* (L.) Moench) seeds (Uganda) in 2-L flasks. Flasks were shaken once daily. After 10–14 days, infested seeds or kernels were air-dried for 2–4 days and stored at 6 C.

Inoculations. Treatment and spreader plants were inoculated at GS V6, V9 (ninth true leaf collar visible), and V12 by placing approximately 5 g of infested oat kernels or sorghum seeds into the whorl of each plant. In 1991 in Ohio, plants were inoculated only at GS V6 and V9. After each inoculation, 2.5 cm of water was applied for 12 hr overnight using an overhead sprinkler irrigation system. Because of the dry conditions in 1991, additional irrigations were applied on 2, 9, and 22 August.

Data collection. Disease severity was assessed using a rating scale of 0, 0.5, 1, 5, 10, 25, 50, and >75 PLAA or a 0–5 scale on a whole plant basis (five randomly selected plants per plot). End-row plants were not included in observations. A single 0–5 rating was made at GS R4 (dough stage, 24–28 days after silking); and PLAA determinations were made weekly at approximately GS V18, R1 (silking stage), R2, R3 (milk stage, 18–22 days after silking), R4, and R5 (dent stage, 35–42 days after silking). In Ohio in 1990, disease was not rated at GS R2 or R5. The mean number of lesions per leaf was determined from counts of lesions on the ear leaf, and first and second leaves above the ear leaf, at GS R3. Lesion length (cm) was measured once at GS R3. A total of 10 (Ohio, 1990) and 20 (Uganda, 1991) lesions for each progeny was measured. In 1990 in Ohio, grain moisture content at harvest was measured with a Tri-Grain Moisture Tester.

Data analysis. One progeny (S-145) did not provide a suitable stand in Ohio (1990) and therefore was excluded from the analysis. Data were tested to determine if they were normally distributed using the SAS PROC NORMAL procedure (29). Because some data were determined not to be normally distrib-

uted, square-root transformations were used for analysis of variance (ANOVA) procedures.

Based on multiple assessments of PLAA and lesion number counts, AUDPC values were calculated using the midpoint rule standardized by dividing AUDPC values by the number of days from first to last assessment for each observation plot (5). Plots representing the increase in disease severity over time were charted for selfed progenies and inbred checks. Linearized logistic ($\ln[Y/(100-Y)]$), exponential ($\ln(Y/100)$), and Gompertz ($-\ln[-\ln(Y/100)]$) disease progress curve models were fitted to disease progress data (5) in which Y represents percent disease severity. Based on goodness of fit of charted plots, coefficient of determination (R^2), F statistic, and plots of standardized residuals vs. predicted values, the exponential model was chosen to characterize disease progression.

SAS PROC GLM was used to determine differences among OhS10 progenies for various disease assessment methods at each location and for data combined from both locations. All mean comparisons were conducted at $P < 0.05$ probability level using Fisher's protected least significant difference (LSD) (31). AUDPC and slopes of regression equations describing the increase in log-transformed disease severity over time (apparent infection rates [r]) were computed as described by Campbell and Madden (5).

Correlation analysis was performed using SAS PROC CORR (29) to determine relationships among disease assessment parameters such as lesion number, lesion length, PLAA, 0–5 rating, AUDPC, and apparent infection rate. The correlation between PLAA (GS R4) and harvest moisture was also examined for the Ohio (1990) experiment only.

For each experiment, progenies were classified based on PLAA (GS R4) in the following categories relative to the susceptible and resistant checks: 1 = highly resistant (statistically significant resistance better than Mo17); 2 = resistant (not significantly different than Mo17 and significantly more resistant than B73); 3 = intermediate (significantly more susceptible than Mo17 but significantly more resistant than B73); 4 = susceptible (not significantly different than B73); and 5 = highly susceptible (significantly more susceptible than B73). A complete listing of progenies evaluated in this study and their reactions can be obtained from the first author upon request.

The progenies were also ranked based on all disease assessment indices for each experiment using the SAS PROC RANK procedure (29). Spearman's rank correlation coefficients were computed to determine the relationship between the relative ranks of the progenies' resistance to race 1 (Ohio, 1990) vs. race 0 (Uganda, 1991).

RESULTS

Natural rainfall (mean 0.4 cm/day), warm temperatures (average daily temperature = 20 C), and high relative humidity (mean daily RH 80%) prevailed during the 1990 experiment in Ohio (June through September). These environmental conditions favored development of NLB (Table 1). In Uganda (1991), climatic conditions were also favorable for the development of high levels of disease (Table 1). Because of the dry conditions in Ohio in 1991, disease did not develop on the majority of OhS10 progenies.

In both Ohio (1990) and Uganda (1991), differences in the resistance of progenies were detected using the 0–5 rating scale (Table 2), PLAA (Table 3), lesion numbers (Table 3), AUDPC values (Table 4), and apparent infection rates (Table 5). Little variation in lesion size was observed (Table 3).

Late-season (GS R4) PLAA ratings in Ohio (1990) ranged from 0.3% on S-20 to 56.7% on S-134. Of the 149 progenies, 1 was rated highly susceptible; 36 were rated susceptible; none was rated intermediate; 55 were rated resistant; and 77 were rated highly resistant. These classes were based on mean comparison tests with the check inbreds, and for that reason there was some overlap between

Table 1. Population means and standard deviations of northern leaf blight disease assessment indices recorded for 149 OhS10 S_1 progenies in Ohio, 1990 and Uganda, 1991

	Ohio (race 1)		Uganda (race 0)	
Indices	**Mean**	**SD**	**Mean**	**SD**
AUDPC (PLAA)[a]	9.38 ±	8.75	13.96 ±	7.50
AUDPC (lesion number)[b]	4.22 ±	3.28	8.09 ±	3.88
Apparent infection rate[c]	0.03 ±	0.01	0.04 ±	0.01
Severity[d]	16.67 ±	13.37	24.76 ±	11.85
0–5 Score[e]	2.57 ±	1.08	3.36 ±	0.70
Lesion length[f]	7.29 ±	1.80	11.86 ±	1.42
Lesion number[g]	6.43 ±	4.03	12.67 ±	3.93

[a] Area under the disease progress curve based on percent leaf area affected from assessments at GS V18, R1, R3, and R4 in Ohio (1990) and GS V18, R1, R2, R3, R4, and R5 in Uganda (1991).
[b] AUDPC based on mean number of lesions counted on three leaves per plant at GS R3.
[c] Apparent infection rate or slopes of regression lines representing increase in disease severity over time using the exponential model.
[d] Percent leaf area affected assessed on whole-plant basis at GS R4.
[e] Whole-plant rating based on a 0–5 scale (9).
[f] Measures of 10 or 20 lesions for each progeny from race 1 (1990) and race 0 (1991) experiments in Ohio and Uganda, respectively, measured at GS R3.
[g] Means of three leaves per plant determined at GS R3.

Table 2. Reaction of resistant OhS10 S_1 progenies to race 1 and race 0 of *Exserohilum turcicum* using a 0–5 rating scale at GS R4 in Ohio and Uganda, respectively[a]

			Ohio		Uganda	
Entries	**Mean**	**Rank**	**Race 1**	**Rank**	**Race 0**	**Rank**
S-20	1.0	1[b]	0.5	1	1.5	1
S-30	1.3	2	0.5	1	2.1	6
S-91	1.4	3	1.0	3	1.9	2
S-115	1.5	4	1.0	3	1.9	4
S-90	1.5	5	1.0	3	2.1	6
S-122	1.6	6	1.0	3	2.2	10
S-143	1.6	6	1.3	11	1.9	2
S-111	1.7	8	1.0	3	2.3	16
S-15	1.7	9	1.0	3	2.1	6
S-33	1.7	9	1.0	3	2.4	17
S-146	1.7	11	1.3	11	2.1	9
S-142	1.8	12	1.3	11	2.2	10
S-100	1.8	13	1.3	11	2.3	12
S-124	1.8	13	1.3	11	2.3	12
S-18	1.9	15	1.0	3	2.7	29
B73	4.2	139	4.0	138	4.3	145
B73*Ht*	...	...	...	...	4.0	130
Mo17	2.8	69	3.0	101	2.7	29
Mo17*Ht*	...	...	...	...	2.5	21
LSD (0.05)	1.5		0.7		0.7	

[a] Severity assessments were made on five plants per experimental unit using the 0–5 rating scale of Elliott and Jenkins (9).
[b] Values are rounded to one decimal place; however, rankings are based on original data of two decimal places.

Table 3. Severity, lesion number, and lesion length of resistant OhS10 S_1 progenies caused by race 1 and race 0 of *Exserohilum turcicum* in Ohio and Uganda, respectively

	Severity[a] (% leaf area infected)						Lesion number[b] (count)						Lesion length[c] (cm)					
Entries	**Mean**	**Rank**	**Race 1**	**Rank**	**Race 0**	**Rank**	**Mean**	**Rank**	**Race 1**	**Rank**	**Race 0**	**Rank**	**Mean**	**Rank**	**Race 1**	**Rank**	**Race 0**	**Rank**
S-20	3.3	1	0.3	1	6.3	3	2.1	1	0.1	1	4.0	2	6.1	2	2.8	2	9.5	7
S-30	3.7	3	0.3	2	7.1	4	2.4	2	0.3	2	4.4	5	6.7	4	6.0	33	7.4	1
S-91	6.2	9	2.0	7	10.5	16	2.4	3	0.6	5	4.1	3	7.5	5	5.0	12	10.0	13
S-115	4.5	4	2.9	10	6.1	2	4.1	9	2.1	15	6.1	13	8.6	36	6.1	39	11.2	55
S-90	3.6	2	1.7	5	5.6	1	3.3	4	1.0	8	5.6	7	7.9	13	4.8	9	10.9	49
S-122	5.5	6	1.6	4	9.4	13	4.2	10	1.4	11	6.9	16	7.6	10	4.6	6	10.6	33
S-143	5.8	8	3.6	16	8.0	6	3.4	5	2.6	24	4.2	4	8.7	43	6.0	36	11.4	65
S-111	5.2	5	2.0	7	8.4	9	3.4	6	1.2	9	5.6	8	6.8	5	4.9	10	8.8	3
S-15	6.6	11	3.9	17	9.3	12	4.4	12	2.8	28	6.0	10	8.8	45	6.1	43	11.4	62
S-33	6.6	12	2.3	9	11.0	17	4.8	15	2.1	16	7.4	20	8.4	27	5.4	18	11.4	65
S-146	6.3	10	4.1	19	8.6	10	3.6	8	2.3	19	4.8	6	8.5	29	5.6	23	11.4	63
S-142	7.7	17	5.3	31	10.1	15	5.7	25	5.3	73	6.1	11	7.8	12	4.7	8	10.9	40
S-100	5.8	7	2.9	11	8.7	11	4.3	11	0.8	6	7.8	22	6.6	3	3.8	4	9.5	6
S-124	8.0	19	4.3	24	11.7	20	4.4	13	2.3	18	6.5	14	8.6	33	5.7	25	11.4	67
S-18	10.3	28	1.6	3	19.0	46	5.0	17	0.5	3	9.5	33	7.6	9	4.7	7	10.5	30
B73	53.8	149	39.3	138	68.3	153	14.3	135	9.7	123	18.9	148	9.3	64	6.5	60	12.0	85
B73*Ht*	...	...	...	...	31.7	119	...	...	...	...	16.5	133	...	...	...	...	10.6	32
Mo17	19.7	88	23.3	110	16.0	36	8.4	58	7.0	95	9.8	36	7.9	13	5.3	16	10.4	25
Mo17*Ht*	...	...	...	...	7.3	5	...	...	...	...	9.6	34	...	...	...	...	7.7	2
LSD (0.05)	16.4		10.9		12.4		9.7		3.2		4.7		6.9		2.5		3.0	

[a] Percent leaf area affected assessed on whole plant basis at GS R4.
[b] Lesion numbers are means of three leaves per plant determined at GS R3.
[c] Lesion lengths are measures of 10 or 20 lesions for each progeny from race 1 and race 0 experiment in Ohio and Uganda, respectively, measured at GS R3.

Table 4. Apparent infection rates[a] of resistant OhS10 S_1 progenies to race 1 and race 0 of *Exserohilum turcicum* in Ohio and Uganda, respectively

			Ohio		Uganda	
Entries	**Mean**	**Rank**	**Race 1**	**Rank**	**Race 0**	**Rank**
S-20	0.010[b]	1	0.003	1	0.018	2
S-30	0.014	2	0.004	2	0.024	6
S-91	0.022	15	0.015	13	0.030	30
S-115	0.015	3	0.012	5	0.018	2
S-90	0.019	6	0.012	5	0.025	11
S-122	0.020	9	0.013	7	0.028	18
S-143	0.027	41	0.024	48	0.030	30
S-111	0.018	4	0.019	26	0.017	1
S-15	0.022	15	0.016	16	0.027	16
S-33	0.020	9	0.013	7	0.026	14
S-146	0.018	4	0.016	16	0.020	4
S-142	0.019	6	0.011	4	0.027	16
S-100	0.021	12	0.019	26	0.024	6
S-124	0.024	23	0.020	31	0.029	23
S-18	0.025	27	0.014	12	0.036	84
B73	0.070	151	0.066	149	0.075	153
B73*Ht*	...	...	...	...	0.043	133
Mo17	0.045	133	0.054	139	0.035	80
Mo17*Ht*	...	...	...	...	0.024	6
LSD (0.05)	0.015		0.014		0.012	

[a] Apparent infection rate or slopes of regression lines representing increase in disease severity over time using the exponential model.
[b] Values are rounded to three decimal places; however, rankings are based on original data of six decimal places.

classes. Thus, statistically, some progenies could be members of more than one class and the combined class total exceeded 149.

In Uganda (1991), late-season (GS R4) PLAA values ranged from 5.6% on S-90 to 68.3% on B73 (Table 3). Average disease levels of the OhS10 progenies were higher in Uganda in 1991 (24.8%) than in Wooster in 1990 (16.7%). The higher disease severity in Uganda permitted better separation of progenies based on their host responses to NLB. In Uganda, no progenies were rated more susceptible than B73; 8 were rated as susceptible as B73; 46 were rated intermediate; 93 were resistant; and only 6 were rated more resistant than Mo17.

In Ohio (1991), disease severity ranged from 0.0% on 19 progenies to 33.3% on B73*Ht*1. All progenies had less than 10% leaf area affected. Average disease severity was 1.1% compared to 16.7% in 1990, and only 14 progenies had more than 4% leaf area affected. The 14 progenies with more than 4% leaf area affected in 1991 were rated susceptible in 1990 (*data not presented*).

The top tenth percentile of resistant progenies were selected for study using the average 0–5 rating from combined Wooster (1990) and Uganda (1991) data. Average ratings of the resistant progenies were less than 2 on the 0–5 rating scale (Table 2), and they displayed low PLAA values (less than 11%) (Table 3). Resistance of the selected progenies was characterized by few lesions (Table 3) and a low rate of disease increase (Table 5). Only one progeny, S-30, had lesions significantly smaller than those on Mo17 (Table 3).

NN14B*Ht*2 displayed chlorotic lesions in response to infection with *E. turcicum* in the race 1 experiment in Ohio (1990), consistent with the racial classification of the inoculum source. The presence of the *Ht*1 gene in B73*Ht*1 and Mo17*Ht*1 significantly reduced *E. turcicum* race 0 infection in Uganda compared to inbred lines without the *Ht*1 gene, thus verifying inoculation with race 0.

Correlation analysis indicated highly significant relationships among disease severity assessments, lesion numbers, AUDPC, and the 0–5 rating scale (Table 6). Lower, but still significant, correlations between lesion size and other assessment methods were observed in Ohio (1990) and Uganda (1991). The average lesion size on progenies was markedly higher in Uganda (1991) than in Ohio (1990) (Table 3), which likely accounts for the reduced degree of association between lesion length and other host responses. We observed only nonsignificant correlations among the disease assessment indices and percent grain

Table 5. Area under the disease progress curve (AUDPC) for percent leaf area affected (PLAA) and lesion number of resistant OhS10 S_1 progenies to race 1 and race 0 of *Exserohilum turcicum* in Ohio and Uganda, respectively

	AUDPC (PLAA)[a]						AUDPC (lesion number)[b]					
			Race 1		Race 0				Race 1		Race 0	
Entries	Mean	Rank	Mean	Rank	Mean	Rank	Mean	Rank	Mean	Rank	Mean	Rank
S-20	1.5[c]	1	0.1	1	2.9	1	0.9	2	0.0	1	1.8	3
S-30	1.7	2	0.2	2	3.3	2	0.8	1	0.2	4	1.5	1
S-91	2.6	4	0.7	5	4.6	10	1.1	3	0.2	4	1.9	4
S-115	2.7	5	1.7	19	3.6	3	1.9	10	1.4	28	2.5	7
S-90	2.3	3	1.0	9	3.7	4	1.5	5	0.4	9	2.7	9
S-122	3.0	7	0.9	6	5.2	13	1.8	6	0.6	11	3.0	11
S-143	2.7	5	1.5	13	3.8	5	1.3	4	0.9	17	1.7	2
S-111	3.4	12	0.9	6	6.0	19	2.8	23	0.3	7	5.3	45
S-15	3.1	9	1.9	21	4.3	7	1.9	10	1.6	34	2.1	5
S-33	3.8	15	1.6	16	6.0	19	2.7	22	1.3	24	4.0	23
S-146	3.4	12	2.3	25	4.5	9	2.2	14	1.3	24	3.1	13
S-142	4.2	17	3.0	36	5.4	15	3.3	32	3.4	76	3.2	15
S-100	3.1	9	0.9	6	5.4	15	2.4	17	0.3	6	4.6	32
S-124	3.5	14	1.8	20	5.3	14	2.0	13	1.1	20	2.9	10
S-18	4.5	22	0.6	4	8.4	35	2.2	14	0.1	3	4.4	28
B73	23.6	138	15.6	120	31.7	149	9.2	122	5.9	115	12.4	135
B73*Ht*	...	...	...	...	18.1	120	...	...	...	...	10.3	110
Mo17	8.6	62	8.7	93	8.6	39	3.3	32	3.2	73	3.5	18
Mo17*Ht*	...	...	...	...	4.3	7	...	...	...	...	3.7	20
LSD (0.05)	7.3		7.4		6.6		3.6		2.7		4.4	

[a] AUDPC (PLAA) based on percent leaf area affected from assessments at GS V18, R1, R3, and R4 in Ohio (1990) and GS V18, R1, R2, R3, R4, and R5 in Uganda (1991).
[b] AUDPC (lesion number) based on mean number of lesions counted on three leaves per plant at GS R3.
[c] Values are rounded to one decimal place; however, rankings are based on original data of four decimal places.

moisture content at harvest ($r = -0.16$ to 0.18, data not presented; all nonsignificant). When the relative ranks of resistance of progenies to race 0 and 1 were compared, the Spearman's rank correlation coefficients for all indices except AUDPC (PLAA) and lesion number were in the range ($r = 0.65$ to 0.75) and highly significant ($P<0.0001$). Lesion number and AUDPC (PLAA) ranks also were positively correlated but not as highly as the other indices ($r = 0.35$, $P < 0.0001$ and $r = 0.18$, $P < 0.05$, respectively).

Table 6. Correlation coefficients for relationships among northern leaf blight assessment indices recorded for 149 S_1 progenies in Ohio, 1990 and Uganda, 1991

Assessment index	PLAA[a]	(0–5)	Lesion (no.)	Lesion (length)	AUDPC[b] (PLAA)	AUDPC[c] (no.)
Ohio (1990)						
0–5	0.95**[d]					
Lesion no.	0.95**	0.92**				
Lesion length	0.78**	0.78**	0.78**			
AUDPC[b]	—[e]	0.91**	0.94**	0.79**		
AUDPC[c]	0.92**	0.89**	—	0.77**	0.94**	
r	—	0.89**	0.83**	0.65**	—	0.76**
Uganda (1991)						
0–5	0.89**					
Lesion no.	0.86**	0.95**				
Lesion length	0.25**	0.28**	0.30**			
AUDPC[b]	—	0.89**	0.88**	0.24**		
AUDPC[c]	0.91**	0.89**	—	0.26**	0.94**	
r	—	0.76**	0.70**	0.30**	—	0.60**

[a] Percent leaf area affected assessed on five randomly selected plants per plot at GS R4.
[b] Area under the disease progress curve calculated from severity ratings at V18, R1, R2, R3, R4, and R5 in Uganda and V18, R1, R3, and R4 in Ohio.
[c] Calculated from average number of lesions on 15 leaves per replicate.
[d] ** = Significant at the $P < 0.01$ level or less.
[e] — = Data confounded so correlation analysis could not be performed.

DISCUSSION

The range of reactions of OhS10 selfed progenies to *E. turcicum* race 1 was probably due to the broad genetic base of OhS10, which includes both resistant and susceptible germ plasm (Table 1). Selection at the tenth percentile using the results of a single late-season disease severity assessment (0–5 rating scale) resulted in the identification of resistant progenies, essentially all of which expressed rate-reducing resistance. The low lesion numbers of resistant progenies (equal to or lower than that of Mo17) points to a race-nonspecific, likely polygenic basis of resistance (36).

This type of resistance, because of its apparent polygenic character, may be very useful for control of NLB. The resistant OhS10 progenies could be useful in recurrent selection programs to incorporate resistance to *E. turcicum* in agronomically acceptable cultivars.

In a previous study with OhS9 and OhS10 populations conducted in Puerto Rico (32), both populations were equally susceptible to NLB and were more susceptible than the resistant Pioneer brand hybrid X304-C. However, the average rating of OhS10 was only 1.35, and that of X304-C was 0.7, using the 0–5 scale. These results represented the mean reaction of OhS10 and did not reveal the frequencies of resistant alleles. In our study, mean comparisons with susceptible and resistant checks indicated a higher frequency of resistant than of susceptible progenies.

B73*Ht*1 had lower NLB severity than B73 in Uganda, but the level of resistance conferred by the *Ht* gene was low compared to the level of resistance identified in OhS10, e.g., S-20, S-30, S-90, or S-115, or in the cultivar Babungo-3, a resistant open-pollinated cultivar (1). A high level of resistance to race 0 was obtained with the combination of the *Ht* gene and polygenic resistance (Mo17*Ht*1).

Assessment of severity (PLAA or 0–5 rating) required less time than counting lesions. Both indices were, however, highly correlated with lesion counts and were effective in assessing differences among progenies (Table 6). Our data suggest that one late-season assessment using the 0–5 rating scale was adequate for identifying genotypes resistant to *E.*

turcicum. However, using only one late rating would probably underestimate the resistance expressed by progenies early in the epidemic from tasseling through GS R2. Additionally, single late-season ratings at the end of the growing season may misrepresent disease severities of genotypes that require longer growing seasons (7). The use of an earlier disease rating at GS R2 or R3, or calculating AUDPC from multiple rating times, would identify such genotypes. An inherent problem concerning the use of AUDPC values, apart from the many assessments required (3), is the inability of AUDPC to distinguish between genotypes with different disease progress curves but similar AUDPC values. The apparent infection rate and identification of the best disease progress model will allow better factors to be selected. Moreover, AUDPC models are effective for identifying genotypes resistant to NLB only when disease levels are high (26,27), as was the case in Ohio (1990) and Uganda (1991). Selection of the appropriate disease assessment index depends on the level of accuracy required, the type of resistance desired, and assessment time limitations.

Ceballos et al (6) observed that, in general, later maturing maize genotypes with the same genetic background expressed more resistance than did early ones. We observed low correlations between disease assessment indices and harvest moisture values. Possible bases for these contrasting observations may include either a larger range in maturity or resistance conferred primarily by certain late maturing progenitors in the populations studied by Ceballos et al (6). We conclude that high levels of resistance may be obtained from the OhS10 population throughout its range of maturity.

ACKNOWLEDGMENTS

We thank Audrey Johnston and David Lubaale for valuable technical support. We also thank I. W. Deep, M. Ellis, L. V. Madden, and K. Garland Campbell for reviewing the manuscript, and Jim Hacker and Burt Bishop for assistance with statistical analyses. Salaries and research support were provided by state and federal funds appropriated to the Ohio Agricultural Research and Development Center and Ohio State University, and by grant monies from the Rockefeller Foundation and USAID/Uganda Manpower for Agricultural Research and Development (MFAD).

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