Interaction of *Ht* and Partial Resistance to *Exserohilum turcicum* in Maize

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ABSTRACT

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Components of northern leaf blight resistance in maize due to race-specific resistance controlled by the *Ht* gene, partial resistance derived from inbred H99, and a combination of the two kinds of resistance, were studied subsequent to inoculation with *Exserohilum turcicum* race O. Lesion types, number of lesions (lesion number), percent leaf area affected (severity), and area under the disease progress curve (AUDPC) based on lesion number and severity were assessed in field studies conducted at two locations in Uganda and one location in Ohio in 1993. Lesion types observed were consistent for genotypes across locations. In general, significant differences among genotypes for data based on lesion number and severity were consistent for AUDPC based on lesion number and severity, respectively, at all locations. In Ohio, both *Ht* and partial resistance were effective in limiting disease development. In Uganda, susceptible inbreds (A619, A635, and B73) generally had higher severity than genotypes with partial resistance (H99, Mo17, and Babungo 3). However, there was a difference in response among genotypes depending on disease intensity at each location. *Ht* resistance and moderate partial resistance did not greatly affect lesion number at the higher disease intensity location, compared with the susceptible inbreds, but at the lower disease intensity location genotypes with partial resistance had fewer lesions than susceptible inbreds or the *Ht* conversions of the susceptible inbreds. At both plot locations, genotypes with partial resistance had lower severity than the susceptible inbreds or *Ht* conversions of the susceptible inbreds. Hybrids derived from crossing H99 with genotypes with moderate levels of partial resistance (Mo17 and Babungo 3) did not have significantly lower lesion numbers than hybrids of susceptible inbreds crossed with H99, but severity was significantly lower on these hybrids at the high disease intensity location. Results indicate that the level of partial resistance in H99 would be as effective in controlling northern leaf blight as using *Ht* resistance, or a combination of *Ht* resistance and moderate levels of partial resistance as found in Mo17.

Northern leaf blight, incited by the fungus *Exserohilum turcicum* (Pass.) K. J. Leonard & E. G. Suggs (teleomorph = *Setosphaeria turcica* (Luttrell) K. J. Leonard & E. G. Suggs), is a major disease of maize (*Zea mays* L.) in the U.S. (9,13–15, 17,25,39) and Uganda, Africa (1). Five races of *E. turcicum* have been reported to overcome specific *Ht* resistance genes in the U.S. (3,26,42) and others have been reported from matings of races in the laboratory (10). Races 0 and 1 are most prevalent, whereas races 23, 2N, and 23N are rare (10). Welz et al. (41) reported that races 0, 2, 2N, 23, and 23N exist in Uganda, but only race 0 is prevalent (1,4).

Five dominant genes controlling monogenic resistance (*Ht*, *Ht2*, *Ht3*, *HtM*, and *HtN)* have been identified in maize (17,25, 34,35). Monogenic resistance is characterized by the formation of chlorotic lesions, a delay in appearance of necrosis, and a marked reduction in sporulation (32). The *Ht* gene, and to a lesser extent the *HtN* gene, have been incorporated into commercial hybrids (25,32). Subsequent to the occurrence of races capable of overcoming *Ht* resistance in the U.S. during the early 1980s (3,36,38), resistance in most Corn Belt hybrids has been based on polygenic, partial resistance (7,25). Partial resistance is characterized by an increase in latent period, a reduction in lesion size and number, and infection efficiency (1,5,16). Quantitatively, partial resistance ranges from a high level with few, small lesions to a low level with many, large sporulating lesions (9,18,32). Partial resistance is conditioned by relatively few genes in some cases (18), but in others it is controlled by many genes, some with major effects (19). The degree of resistance expressed by lines with the *Ht* gene is influenced by the level of partial resistance in the line (16,20,29, 32). Incorporation of the *Ht* gene into a background with partial resistance, like that of Mo17, confers the most effective resistance to *E. turcicum*, as displayed by reduced sporulation and number and size of lesions (20).

Polygenic, partial resistance is considered to be more durable since single-gene resistance is vulnerable to the development of new races (27,38,40). Partial resistance is not easily overcome by new races (21, 22,28) and a combination of monogenic *Ht* resistance with partial resistance permits additive or complementary interallelic interactions that may enhance the overall level of resistance (18,40).

Past studies on combinations of *Ht* and partial resistance to *E. turcicum* have compared reactions of different inbred lines and hybrids (24,29,30). These studies did not permit direct comparisons of different types of resistance because genetic background effects were not held constant. The objective of this study was to document the level of partial resistance expressed by inbred H99 and to evaluate the effect of combining partial resistance of inbred H99 with *Ht* resistance and other sources of partial resistance on northern leaf blight development.

MATERIALS AND METHODS

Host genotypes. Inbred lines A619, A619*Ht*, A635, A635*Ht*, B73, B73*Ht*, Mo17, and H99 were used in field studies after verifying the presence of the *Ht* genes in greenhouse studies (12). A synthetic line (OhS10 S_1-90) (31), and an open-pollinated experimental cultivar (Babungo 3) were also included in the study. Babungo 3 was developed by the International Institute of Tropical Agriculture, Nigeria.

Inbred H99 was developed from Illinois Synthetic 60C (Syn 60C) by the Purdue Agricultural Experiment Station (11). It was selected based on its general agronomic performance, and little published information is available on its reaction to diseases. It is generally accepted that H99 has a relatively high level of partial resistance to *E. turcicum* as confirmed in previous inbred screening tests (P. E. Lipps, *unpublished*). H99 has been used to a limited extent in production of U.S. maize hybrids

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(43), but has been used widely to develop new inbred lines (11). Inbred line H99 was crossed with A619, A619*Ht*, A635, A635*Ht*, B73, B73*Ht*, Mo17, OhS10 S_1-90, and open-pollinated cultivar Babungo 3 to constitute hybrid genotypes with differing combinations of factors for resistance to *E. turcicum*. All lines were evaluated in Uganda. Lines A635, A635*Ht*, A635 × H99, A635*Ht* × H99, Babungo 3, and Babungo 3 × H99 were not tested in Ohio.

The genotypes under study were divided into six groups based on resistance components in order to assess the effect of *Ht* and partial resistance on development of northern leaf blight (Table 1). Group I consisted of inbred lines with the lowest levels of resistance (A619, A635, and B73) and were designated susceptible, group II consisted of genotypes with partial resistance (Mo17, OhS10 S_1-90, and Babungo 3), and group III consisted of the near-isogenic *Ht* lines of inbreds in group I. Genotypes in groups I, II, and III were crossed with H99 to form groups IV, V, and VI, respectively.

Field experiments. Field experiments were conducted at the Namulonge Agricultural and Animal Production Research Institute (Namulonge) and the Kamenyamiggo District Farm Institute (Kamenyamiggo) in Uganda and at the Ohio Agricultural Research and Development Center (OARDC) near Wooster (Wooster). Throughout the rest of this paper the experiments will be identified by their locations. The two field locations in Uganda represented two agroecological zones (1). The Namulonge location has a relatively dry climate (800 to 1,100 mm of annual precipitation) with temperatures of 25 to 30°C and <70% relative humidity most days. The Kamenyamiggo location is relatively wet (1,400 mm of annual precipitation) with temperatures of 20 to 25°C and >80% relative humidity most days. In Uganda, fields were disk plowed twice and then harrowed prior to planting. Superphosphate and urea were broadcast just before planting at 45 kg of P_2O_5 and 30 kg of N per ha, respectively. An additional 30 kg of N per ha was applied 4 weeks after planting. Plants were hand weeded three times during the growing season. Seeds were hand planted at Namulonge on 30 March 1993 and at Kamenyammigo on 6 April 1993, in a randomized complete block design with two replications at each location. Each entry was represented by one row per replicate. The rows were 5 m long spaced 75 cm apart, with 22 plants per row. The periphery of the experiment was planted to Longe 1, a commercial open-pollinated maize variety. At Wooster, the field was plowed in the spring, fertilized with 448 kg of ammonium nitrate and 112 kg of 0-18-36 (N-P_2O_5-K_2O) granular fertilizer per ha and incorporated with a disk prior to planting. Weeds were controlled by a preplant application of 4.2 liters/ha of 43% a.i. cyanazine plus 4.6 liters/ha of 85.1% a.i. butylate. Seeds were hand planted on 26 May 1993 in single-row experimental units in a randomized complete block design with three replications. Each 3-m-long single row consisted of approximately 15 plants. To increase the spread of *E. turcicum*, seeds of B37 × B73 were planted in the three center rows of the plot and seeds of B73 were planted as two-row borders around the entire periphery of the plot.

Inoculum preparation. *E. turcicum* isolates obtained from infected maize tissue at Namulonge, Kamenyamiggo, and Wooster were used to prepare inoculum for these respective plot locations. Portions of leaf tissues were surface sterilized in 1% sodium hypochlorite for 30 s, rinsed in distilled water, and then placed under high humidity under fluorescent light for 3 days to initiate sporulation. Single conidia were then picked from conidiophores with a glass needle and placed onto Difco potato dextrose agar in petri plates. Cultures were maintained at 20°C with 12 h of fluorescent light (320 μE $s^{-1}m^{-2}$) for 14 days. Colonized agar sections from potato dextrose agar cultures were placed in plastic bags containing sterilized sorghum (*Sorghum vulgare* L.) (Uganda) or oat (*Avena sativa* L.) (Ohio) kernels. The bags were shaken every other day to ensure complete colonization of the kernels. Infested kernels were air dried on laboratory benches for 3 to 4 days and thereafter kept dry until used. Virulence of the isolates used to inoculate plots was confirmed in greenhouse inoculation tests on B37, B37*Ht*, B37*Ht*2, and B37*Ht*3, and A619, A619*Ht*, A619*Ht*2, and A619*Ht*3. Virulence spectrum of all isolates corresponded to race 0 (26).

Inoculations and disease evaluation. Experimental plants were inoculated on 17 May at Namulonge, on 15 May at Kanmenyamiggo, and 8 July 1993 at Wooster, by placing about 50 infested kernels into the whorl of each plant at the V10 to V12 growth stage (33). Disease severity was assessed as the percent leaf area affected (severity) on each plant, using a scale of 0, 1, 3, 5, 10, 20, 30, 40, 50, 60, 70, and ≥75% leaf area affected. The scale used was modified from those of Elliott and Jenkins (9), Klenke et al. (23), and Adipala et al. (2). Severity was assessed 28, 41, 53, and 60 days after inoculation at Kamenyammigo, 28, 43 and 55 days after inoculation at Numalonge, and 46, 55, 63, and 71 days after inoculation at Wooster. The number of lesions per plant (lesion number) was counted 19, 28, 40, 52, and 61 days after inoculation at Kamenyammigo and 28, 43, and 55 days after inoculation at Namulonge. The number of lesions per plant was not recorded at OARDC.

Experimental design and data analysis. Data obtained from individual plants of each replication were averaged and used for statistical analysis. Disease progress curves for severity and lesion number were plotted for each maize line. Area under the disease progress curve (AUDPC) was computed for both lesion number and severity by the method of Campbell and Madden (6) and Ceballos et al. (8). AUDPC was computed for each plot and standardized by dividing by the number of days in the epidemic (6).

Data from Wooster were not analyzed because data were highly skewed due to preponderance of zero values for each genotype except the susceptible inbreds. Data from Namulonge and Kamenyamiggo were analyzed separately and together for lesion number, severity, and AUDPC with PROC ANOVA of SAS (SAS Institute, Inc., Cary, NC). Non-orthogonal contrasts (37) among the resistance type groups were made with PROC GLM of SAS. The additivity of monogenic *Ht* and RNS resistance was evaluated by examining significant statistical differences among groups of entries according to the resistance type combinations and the variance ratios from the single degree of freedom comparisons. Type III mean square for the entry by location interaction was used as the error term.

Table 1. Reactions of maize genotypes with *Ht* and partial resistance to *Exserohilum turcicum*

Resistance type group	Resistance type[a]		Representative genotypes
	Ht	Partial	
I	S	S	A619, A635 and B73
II	S	R[a]	Mo17, OhS10 S_1-90, and Babungo 3
III	R	S	A619*Ht*, A635*Ht*, and B73*Ht*.
IV	S	R	A619 × H99, A635 × H99, and B73 × H99
V	S	R	Mo17 × H99, Babungo 3 × H99, and OhS10 S_1-90 × H99
VI	R	R	A619*Ht* × H99, A635*Ht* × H99, and B73*Ht* × H99
Check	S	R	H99

[a] R = resistant reaction, S = susceptible reaction, where genotypes with *Ht* resistance exhibited chlorotic lesions (R) and those without *Ht* resistance expressed susceptible-type, necrotic lesions (S). Genotypes with partial resistance had fewer and smaller lesions (R) than those with low levels of partial resistance with many large lesions (S).

RESULTS

Lesion types. Lesion type expression was consistent in Uganda and Ohio. All inbreds containing the *Ht* gene expressed only chlorotic-type lesions. Non-*Ht* inbreds

and genotypes with partial resistance displayed susceptible, necrotic lesions. The *Ht* gene in combination with partial resistance produced only chlorotic-type lesions.

Comparisons of location effects. In Uganda, disease intensity was generally higher at Kamenyamiggo than at Namulonge, but the shapes of disease progress curves based on lesion number and severity at Kamenyamiggo were similar to those at Namulonge, thus only curves from Kamenyamiggo are presented (Fig. 1A,B). The number of lesions increased on all genotypes during the first 4 weeks after inoculation followed by little increase in lesion number on genotypes in resistance type groups I, II, III, IV, and VI (Fig. 1A). Genotypes in group V (inbreds with partial resistance × H99) and H99 continued to increase in lesion number through 7 weeks after inoculation. In contrast, severity increased on all genotypes throughout the assessment time (Fig. 1B). This indicated that the increase in disease level during later assessments was primarily due to increase in lesion size for susceptible genotypes (group I), those with monogenic *Ht* resistance (group III) and those crossed with partially resistant H99 (groups IV and VI). Lesions on H99 and genotypes with partial resistance crossed with H99 (group V) remained small throughout the assessment period and the amount of increase in disease severity was due to increased lesion numbers of relatively small size.

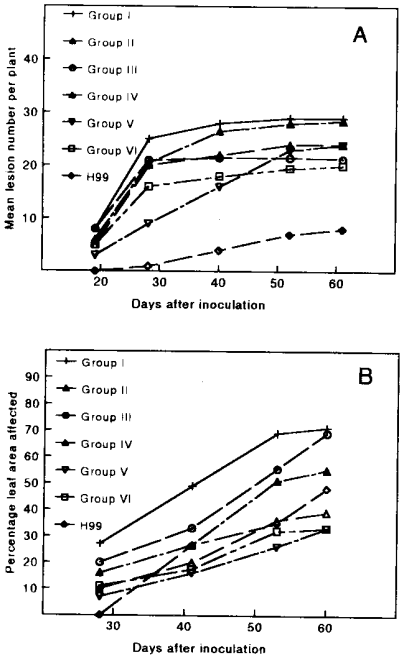

Fig. 1. Northern leaf blight progress on representative resistance type groups (see Table 1) at Kamenyamiggo District Farm Institute, Uganda, 1993. Disease progress recorded as the increase in mean number of lesions per plant **(A)** and percent of leaf area affected per plant **(B)** by *Exserohilum turcicum* over assessment dates.

At Wooster, disease progress occurred only on the susceptible inbreds A619 and B73, and on the partially resistant inbred Mo17. Only a few lesions developed on A619*Ht;* no lesions developed on any other line in the test (disease progress curves not presented).

Disease severity in Uganda was higher at Kamenyamiggo than at Namulonge (Tables 2 and 3). Lesion numbers and severity were similar to AUDPC values based on lesion number and severity, respectively, at each location (Table 3). In comparison, the level of disease observed on susceptible inbreds (group I) at Wooster was as high as that observed at Kamenyamiggo, but lines that had partial and/or *Ht* resistance (groups II to VI) had little or no disease development. Analysis of combined data for Kamenyamiggo and Namulonge showed locations, replications within locations, and entries to be statistically significant ($P \leq 0.05$) for lesion number (Table 2). Locations and entries were highly significant ($P = 0.001$) for severity, but replicates within locations were not significant. Genotype × location interaction, which was not statistically significant for lesion number, was significant for severity.

Comparisons of individual genotypes. At Kamenyamiggo, OhS10 S_1-90 had the highest lesion number (31 lesions per plant), but A619 had the highest severity (63%) (Table 3). Lesions on OhS10 S_1-90 were relatively small, amounting to a severity value (37%) significantly below that of A619. At Namulonge, A619 had three times as many lesions as OhS10 S_1-90 (18 and 6, respectively), but A619 and OhS10 S_1-90 had 27 and 12% leaf area affected, respectively. H99 had the lowest lesion number and severity of entries at Kamenyamiggo and Namulonge, but lesion number and severity did not differ significantly from Mo17 ×x H99, OhS10 S_1-90 × H99, A619*Ht* × H99, and B73*Ht* × H99 at both locations. Only three entries (OhS10 S_1-90 × H99, A619*Ht* × H99, and B73*Ht* × H99) did not differ statistically from H99 for AUDPC based on lesion number and severity at both locations in Uganda (Table 3).

At Wooster, A619, B73, and Mo17 were the only entries that had appreciable levels of disease. All other entries had negligible lesion numbers or severity.

Contribution of *Ht* and partial resistance factors. Lesion number and severity values for individual genotypes with and without *Ht* resistance (A619*Ht* and A635*Ht* versus A619 and A635) indicated that the presence of the *Ht* gene reduced the severity of disease, but differences were not always significant or consistent across environments (Table 3). The *Ht* gene apparently did not confer added resistance to B73 because lesion number and severity were not significantly reduced.

Inbreds Mo17 and Babungo 3, with partial resistance, had disease severity levels similar to their respective crosses with H99 and the susceptible *Ht* conversion crosses with H99 at both locations in Uganda (Table 3). AUDPC based on lesion number for H99 did not differ significantly from that observed for Mo17 or Babungo 3 at either location in Uganda. However, H99 had a significantly lower AUDPC value based on severity than these two lines at Namulonge, but not at Kamenyamiggo (Table 3). Genotypes containing both *Ht* and partial resistance had significantly lower AUDPC based on severity than their inbred parents with *Ht* resistance. Resistance conditioned by *Ht* or partial resistance appeared to be highly effective in limiting disease development at Wooster, compared with inbreds without resistance factors.

Non-orthogonal comparison. Differences among the six resistance type groups were examined with non-orthogonal comparisons in order to assess the effect of *Ht* and partial resistance of H99 on the level of resistance. Non-orthogonal comparisons for AUDPC based on lesion number and severity are not presented because of similar results from comparisons using lesion number and severity data. Results of non-orthogonal comparisons among resistance type groups evaluated at Namulonge were generally similar to those at Kamenyamiggo for severity, but some differences occurred between location for lesion number (Table 4).

Lesion number comparisons at Kamenyamiggo showed group I (susceptible inbreds) versus groups V (inbreds with par-

Table 2. Analysis of variance for reaction of 19 maize genotypes with *Ht*, partial, or both types of resistance to *Exserohilum turcicum* race O at Kamenyamiggo District Farm Institute and Namulonge Agricultural and Animal Research Institute in Uganda, 1993

		Lesion number		Percent leaf area affected	
Source of variation	df	Mean square	Variance ratio[a]	Mean square	Variance ratio[a]
Locations	1	2,094.65	121.69***	7,096.62	44.30***
Replicates (locations)	2	80.14	4.66*	11.17	0.70 NS
Entries	18	108.11	6.28*	337.77	21.10***
Entry × location	18	28.40	1.65 NS	57.53	3.59***
Error	36	17.21		16.01	
Total	75				

[a] Statistical difference: * =$P \leq 0.05$, ** = $P \leq 0.01$, and *** = $P \leq 0.001$; NS = no significant difference.

Table 3. Reaction of maize genotypes representing resistance-type groups to *Exserohilum turcicum* at Kamenyamiggo District Farm Institute (Kamen.), Namulonge Agricultural and Animal Research Institute (Namu.), Uganda , and the Ohio Agricultural Research and Development Center (Wooster), Ohio, 1993

	Lesion number[a]		Percent leaf area affected[a]			AUDPC[b] Lesion number		AUDPC[b] Percent leaf area affected		
Group - resistance type	**Kamen.**	**Namu.**	**Kamen.**	**Namu.**	**Wooster**	**Kamen.**	**Namu.**	**Kamen.**	**Namu.**	**Wooster**
Group I - susceptible										
A619	29	18	63	27	58	31	18	62	27	31
A635	25	22	53	28	. . .	27	21	53	27	. . .[c]
B73	19	13	47	19	43	21	13	44	19	21
Mean	25	18	54	25	51	26	18	53	24	26
Group II - partial resistance										
Mo17	14	6	28	15	23	14	6	25	15	8
OhS10 S_1-90	31	6	37	12	0	34	5	38	12	0
Babungo 3	15	5	19	12	. . .	16	4	18	12	. . .
Mean	20	6	28	13	12	21	5	26	13	4
Group III - *Ht* conversions of susceptible										
A619*Ht*	18	12	44	22	2	19	12	40	22	1
A635*Ht*	20	12	46	21	. . .	22	12	42	21	. . .
B73*Ht*	22	17	44	22	1	23	17	41	22	0
Mean	20	14	45	22	2	21	13	41	22	1
Group IV - susceptible x H99										
A619 × H99	23	7	36	17	0	25	6	35	17	0
A635 × H99	23	9	37	19	. . .	24	9	35	18	. . .
B73 × H99	23	5	39	13	0	24	5	35	13	0
Mean	23	7	38	16	0	24	7	35	16	0
Group V - partial resistance × H99										
Mo17 × H99	17	4	23	8	0	18	4	21	7	0
OhS10 S_1-90 × H99	15	5	22	10	0	16	5	21	10	0
Babungo 3 × H99	15	4	20	12	. . .	16	3	18	12	. . .
Mean	16	4	22	10	0	16	4	20	10	0
Group VI - *Ht* conversions of susceptible × H99										
A619 *Ht* × H99	15	5	29	10	0	17	5	28	10	0
A635 *Ht* × H99	19	6	27	15	. . .	20	5	25	14	. . .
B73 *Ht* × H99	14	5	19	9	0	15	5	18	8	0
Mean	16	5	25	11	0	17	5	24	11	0
Check - partial resistance										
H99	5	2	28	5	0	5	2	26	4	0
LSD (P = 0.05)	12	4	10	6	. . .	12	4	10	6	. . .

[a] Mean lesion number per plant and/or mean percent leaf area affected reported across assessment dates at each location.
[b] Area under the desease progress curve (AUDPC) (6).
[c] Entry not evaluated at Wooster.

tial resistance × H99) and VI (inbreds with *Ht* plus partial resistance × H99), and group IV (susceptible × H99) versus groups V (*Ht* inbreds × H99) and VI (RNS resistant × H99) contrasts to be significantly different. The rest of the group contrasts at Kamenyamiggo, based on lesion numbers, were not significant. There appeared to be additive benefit from the presence of the *Ht* gene and partial resistance from H99 as shown by the statistically significant contrasts for lesion number and severity between group VI and groups I, III, and IV.

The effect of partial resistance from H99 on the level of disease development was evaluated by comparing the disease assessments from the combined groups I, II, and III (with no contribution from H99) with the combined groups IV, V, and VI (Table 4). The contribution of resistance in H99 resulted in highly significant differences in each variable for all comparisons except lesion number and AUDPC based on lesion number at Kamenyamiggo.

DISCUSSION

Ht and partial resistance gene interactions were examined with H99 as a common parent. Inbred line H99 possessed a high level of partial resistance that was expressed as few susceptible-type lesions in the field. Incorporation of partial resistance from H99 into hybrids with susceptible inbreds A619 and A635 with or without the *Ht* gene (groups IV and VI) improved resistance as determined by lesion number and severity estimates at Numalonge, but only as determined by severity at Kamenyamiggo (Table 3).

The *Ht* conversions of A619, A635, and B73 did not consistently improve the response of the inbreds through reduction of the number of lesions or severity. We were unable to demonstrate the role played by the *Ht* gene in reducing the amount of initial inoculum (13,40). The daily increase in lesion number may have been affected by the amount of inoculum produced on oat or sorghum kernel inoculum within plots throughout the duration of the epidemic or by inoculum produced in lesions on plants in adjacent rows resulting in interplot interference. The level of this interference was not determined. However, natural inoculum was not a factor at Wooster because non-inoculated B73 plants surrounding the plot were free of any lesions. It was possible that natural inoculum may have influenced final disease assessments in Uganda. Regardless of the level of possible interference, the effect of resistance factors on disease development was evident at each location. All lines with *Ht* resistance produced only chlorotic lesions, so races capable of overcoming this resistance were not present at any location.

At Kamenyamiggo, *E. turcicum* spread rapidly so that by 4 weeks after inoculation the susceptible genotypes (A619, A635, and B73) had relatively high disease severities and were killed prematurely by *E. turcicum*. As the lesion area increased, the number of new lesions initiated by secondary inoculum was limited by coalescence of primary lesions resulting in less area

Table 4. Non-orthogonal comparisons among representative resistance type groups, based on the mean number of lesions per plant and mean percent leaf area affected by *Exserohilum turcicum*, at Kamenyamiggo District Farm Institute and Namulonge Agricultural and Animal Research Institute, Uganda, 1993

		Kamenyamiggo		Namulonge	
		Mean squares[a]			
Sources of variation	df	Lesion no.	Percent leaf area affected	Lesion no.	Percent leaf area affected
Replicates	1	147.08*	1.62 NS	13.15 NS	80.13***
Entries	18	74.08*	3,11.87***	62.43***	83.43***
Contrasts:					
Group I vs Group II	1	67.26 NS	2,092.73***	445.54***	391.47***
Group I vs Group III	1	61.38 NS	290.38***	52.67***	24.97 NS
Group I vs Group IV	1	10.03 NS	846.38***	354.14***	215.31***
Group I vs Group V	1	233.11*	3,209.51***	538.01***	649.45***
Group I vs Group VI	1	221.02*	2,591.32***	474.52***	554.20***
Group II vs Group III	1	0.13 NS	824.03***	191.84***	218.71***
Group II vs Group IV	1	25.35 NS	277.34***	5.24 NS	26.14 NS
Group II vs Group V	1	49.94 NS	118.94*	4.36 NS	32.47 NS
Group II vs Group VI	1	44.43 NS	26.61 NS	0.45 NS	14.11 NS
Group III vs Group IV	1	21.79 NS	145.26*	133.66***	93.63***
Group III vs Group V	1	55.26 NS	1,569.11***	254.01***	419.73***
Group III vs Group VI	1	49.45 NS	1,146.80***	211.01***	343.90***
Group IV vs Group V	1	146.44*	759.54***	19.15*	116.88***
Group IV vs Group VI	1	136.89*	475.78***	8.79 NS	78.64**
Group V vs Group VI	1	0.16 NS	33.03 NS	1.99 NS	3.77 NS
Group (I+VI) vs (III+IV)	1	7.47 NS	11.38 NS	9.21**	7.49 NS
Group (I+V) vs (II+IV)	1	7.61 NS	165.38*	139.97***	40.27*
Group (I+II+III) vs (IV+V+VI)	1	99.37 NS	1,818.60***	418.47***	504.83***
Error	18	30.75	24.06	66.27	7.96
Total	37	2,033.94	6,048.27	1,123.70	1,665.74

[a] Statistical significance: * = $P \leq 0.05$, ** = $P \leq 0.01$, *** = $P \leq 0.001$; NS = no significant difference.

available for infection and lesion development. Thus, later assessments may have been affected by a high competition for space on the leaves. We observed that the number of lesions developing on susceptible genotypes increased during the first three assessment times; subsequent increases in lesion numbers were negligible. However, percent leaf area affected increased throughout the assessment period on all genotypes. Apparently, the increase in disease severity near the end of the epidemic was primarily due to increase in lesion size and not lesion number. H99 and hybrids composed of genotypes with partial resistance crossed with H99 had few, small lesions, but disease increase resulted from both increased lesion numbers and, to some degree, lesion size. Regardless, there was a clear relationship between the severity of disease and the level of partial resistance as demonstrated by the slow rate of disease development in groups II and V.

Combination of partial resistance, which reduces the number of lesions produced, and *Ht* resistance, which suppresses sporulation, is projected to result in greater stability and longer protection against *E. turcicum* than result from high levels of partial resistance alone (29,32,40). We observed that a combination of *Ht* and partial resistance limited lesion numbers (group VI versus group I) only in the less severe epiphytotic at Namulonge. However, severity was reduced in genotypes with a combination of low to moderate partial resistance and *Ht* resistance (group I versus group VI and group III versus group VI). In previous studies we observed that the *Ht* gene did not reduce lesion numbers on B73 or Mo17, but did reduce percent leaf area affected on B73 under severe disease pressure (31). Apparently, the chlorotic-type lesion resistance did not limit damage to the host in these studies. This concurs with conclusions of Ullstrup (39) and Pataky (29) that the *Ht* gene confers less protection to the host than partial resistance under conditions favorable for epidemics.

It would be essential that high levels of partial resistance be identified prior to initiating a breeding program to develop durable, yet highly effective levels of resistance in commercial hybrids. In environments where disease epidemics are frequent and severe, there appears to be no apparent advantage in combining *Ht* with high levels of partial resistance to control northern leaf blight. Use of high levels of partial resistance alone will likely be sufficient, especially where recurrent selection is applied to improve a population gradually for certain traits including resistance to *E. turcicum*. A combination of the *Ht* gene with partial resistance from H99 appears better than use of either resistance alone only in genotypes with low levels of partial resistance, like A619, A635, and their isogenic lines. There was no added advantage in crossing H99 with Mo17 and Babungo 3 genotypes, which already provided sufficient levels of partial resistance.

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