Differences in Resistance Between Maize Hybrids With or Without the Ht_1 Gene When Infected With *Exserohilum turcicum* Race 2

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ABSTRACT

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There are three known races of *Exserohilum turcicum*, causal organism of northern leaf blight of maize (NLB); races 1 and 3 are avirulent and race 2 is virulent on maize with the Ht_1 gene. Field studies were conducted in 1982 and 1983 to determine the effectiveness of the Ht_1 gene in conditioning resistance to races 1 and 2 of *E. turcicum*. Lesion expansion rates were significantly smaller for hybrids with the Ht_1 gene than for hybrids made from their recurrent parents without the Ht_1 gene when inoculated with race 1. With *E. turcicum* race 2, lesion length, area, and expansion rate and area under the disease progress curve were significantly lower for the hybrid A632Ht_1 × A619Ht_1 than for its near-isogenic counterpart A632 × A619, which is susceptible to *E. turcicum*. Significant differences in quantitative resistance between hybrid sets also were detected. In greenhouse studies, similar results were obtained for lesion length and disease efficiency. Significant yield differences were detected between A632Ht_1 × A619 and A632 × A619 when inoculated with *E. turcicum* race 2.

When a host gene expresses resistance to a race of a pathogen considered virulent to the gene, based on infection type, it has been referred to as "residual resistance" (10,11). Results of previous work with powdery mildew of wheat indicated that near-isogenic lines showed resistance to a virulent race of *Erysiphe graminis* DC. f. sp. *tritici* E. Marchal when resistance genes *Pm*3c, *Pm*4, or *MA* (Michigan Amber) were present (9,10). The level of resistance was determined by comparing near-isogenic lines to their recurrent parent, Chancellor, and was based on lesion number (10) or infection efficiency (9).

Anderson reviewed the residual resistance concept (1) and noted no evidence was provided that indicated residual effects were due to single genes and not due to other differences between near-isogenic lines. Furthermore, previous work did not show these residual effects in adult plants in the greenhouse or field.

Exserohilum turcicum (Pass.) Leonard & Suggs, the imperfect stage of *Setosphaeria turcica* (Luttrell) Leonard & Suggs, causes northern leaf blight (NLB) of maize and is found throughout the northern corn belt (7). The Ht_1 gene (5) was very effective in controlling NLB for over 15 yr; however, the Ht_1 gene has not been effective against race 2 of *E. turcicum*. Currently, three physiologic races of *E. turcicum* are known. Race 1 is avirulent to plants with the Ht_1 gene (5,6). Race 2, now found in many corn-producing states (7), was isolated from plants in the U.S. corn belt for the first time in 1979. This race is virulent to plants with the Ht_1 gene (14). Smith and Kinsey (12) reported a third race of the fungus in 1980 that is avirulent to Ht_1 plants but virulent to plants with the Ht_2 and Ht_3 genes; race 1 and race 2 are both avirulent to plants carrying Ht_2 and Ht_3 genes.

The objective of this research was to determine if maize hybrids or inbreds with the Ht_1 gene were more resistant to *E. turcicum* race 2 than hybrids or inbreds without the Ht_1 gene.

MATERIALS AND METHODS

Field plots were established 12 May 1982 and 11 May 1983 in Champaign County, IL. Four maize hybrid pairs, either homozygous for the dominant allele for resistance (Ht_1) or homozygous recessive for the recessive allele (*ht*) were used. The hybrids included in this study (1982 and 1983) were: A632 × A619 (early maturity and very susceptible to *E. turcicum*), Mo17 × A634 and Mo17 × N28 (intermediate maturity and moderately susceptible), and B73 × Mo17 (intermediate-late maturity and moderately resistant to the fungus). In 1983, the following hybrids,

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heterozygous for the Ht_1 gene, also were included in the study: A632Ht_1 × A619, Mo17 × A634Ht_1, Mo17 × N28Ht_1, and B73Ht_1 × Mo17.

In 1982, hybrids were evaluated with race 1 and race 2 at two locations in Champaign County, IL. Both studies consisted of four hybrids (each homozygous dominant or recessive for the Ht_1 gene) arranged in randomized complete block designs with two replications at each location. The race 1 isolate was obtained from a naturally infected plant, and has been used for years as the *E. turcicum* race 1 check in the Illinois corn pathology program. The race 2 isolate was collected in the fall of 1981 from a plant in Champaign County, IL, naturally infected with *E. turcicum*. This isolate was race-typed throughout this study to confirm its description (5). In 1983, the same four hybrids (each homozygous dominant or recessive or heterozygous for the Ht_1 gene) were inoculated with race 2 and evaluated at one location in Champaign County. The design was a randomized complete block with six replications. In both years, plots consisted of three rows, 4.0 m long and spaced 0.76 m apart. Rows were thinned to approximately 60,000 plants per hectare.

On 22 June 1982, plants were inoculated with a conidial suspension derived from 2-wk-old cultures of *E. turcicum* on lactose-casein hydrolysate agar (13). Conidia were loosened with a rubber policeman and tap water was used to dilute the inoculum. The inoculum was applied at 1,200 L/ha with approximately 3,300 viable conidia per milliliter for race 2 and approximately 1,600 viable conidia per milliliter for race 1. Since comparisons between races were not intended, conidial concentrations were not standardized. Conidial viability was determined by dilution plating (10^{-2}) onto the lactose-casein hydrolysate agar and counting the number of germinated conidia after 18 hr. Inoculations were done using a boom sprayer mounted on a tractor. In the fall of 1982, leaves from plants carrying the Ht_1 gene and infected with race 2 were collected, dried, and ground. On 22 June 1983, approximately 25 cm^3 of ground leaf tissue was placed in the whorl of each plant. In both years, the plants were at the 8- to 10-leaf stage at inoculation time, and between stages 5 and 6 on Hanway's growth scale (3).

Disease evaluations were made using several methods. In 1982, the number of lesions on each of 10 consecutive plants in the center row of each plot was recorded 17 days after inoculation as a measure of disease efficiency. Additionally, 25 individual plants in each plot were tagged and an isolated lesion marked. The length and width of the lesions was measured 17 days after inoculation and every three to four days until five measurements were obtained. Lesion length, lesion area, and rates of increase were determined from these data. Lesion area was calculated according to the formula: $A = (L \times W)(0.7854)$. The rates of increase in lesion area were determined by simple linear regressions of lesion length on time (days after inoculation). In 1983, lesion length measurements were determined 27 days after inoculation by measuring the length of an isolated primary lesion on each of five consecutive plants in the center row of each plot.

In 1982 and 1983, percentages of leaf area infected were visually estimated in the center row of each plot. Estimates were made weekly for 8 wk starting 2 wk before the midsilking stage. Area under the disease progress curve (*AUDPC*) was calculated according to the equation

$$\mathrm{AUDPC} = \sum_{i=1}^{n-1} [(X_{i+1} + X_i)/2]\,[t_{i+1} - t_i]$$

in which X_i = the percentage of leaf tissue blighted at the ith observation, n = the number of observations, and t = time (weeks). In both years, $n = 8$. Yields were obtained by hand harvesting the center rows (4.0 m) of each plot. Ears were shelled and grain weights (adjusted to 15.5% moisture) were expressed as kilograms per hectare.

The same hybrids and inbreds were studied in the greenhouse. Seeds were planted in 15-cm-diameter clay pots in a soil, peat, and sand (1:1:1, v/v) potting mixture and later thinned to four plants per pot. Pots were arranged in completely randomized designs, with three and five replications (number of pots) for disease efficiency and lesion-length experiments, respectively; all experiments were repeated three times. Inoculations were done by pipetting 1 ml of inoculum, containing approximately 1,200 viable conidia per milliliter, directly into the whorl of each plant. Following inoculation, the plants were placed in a mist chamber at 100% RH for 12 hr. The number of lesions on each plant was used as an estimate of disease efficiency and the data were rank transformed before analyses of variance were performed (2). A

TABLE 1. Five assessments of northern leaf blight on four maize hybrid sets with or without the Ht_1 gene grown at two locations in Champaign County, IL, in 1982 following inoculation with *Exserohilum turcicum* race 1

Hybrid	Disease efficiency[w] (lesions/plant)	Lesion expansion[w] (mm^2/day)	Lesion length[w] (mm)	Leaf tissue blighted[x] (%)	*AUDPC*[y]
A632 × A619	3.5 a[z]	4.3 a	55.8 a	20.0 a	49.0 a
A632Ht_1 × A619Ht_1	3.0 a	1.2 d	28,0 c	10.8 b	24.4 b
Mo17 × A634	1.4 bc	3.2 b	4.8 ab	4.5 c	15.4 bc
Mo17 Ht_1 × A634Ht_1	2.9 a	0.7 d	31.8 c	3.0 c	9.5 c
Mo17 × N28	1.1 bc	3.0 b	45.7 b	5.0 c	13.7 bc
Mo17 Ht_1 × N28Ht_1	1.8 b	0.8 d	31.8 c	5.0 c	14.5 bc
B73 × Mo17	0.9 c	1.9 c	33.7 c	3.5 c	12.4 c
B73Ht_1 × Mo17Ht_1	1.0 bc	0.5 d	26.5 c	5.0 c	14.6 bc

[w] Disease efficiency, lesion expansion, and lesion length are based on data from 10, 25, and 25 plants, respectively, from the center row of three-row plots averaged over two locations and two replications.
[x] Represents the percentage of leaf tissue destroyed on the upper two-thirds of plants in the center row of three-row plots 5 wk after the midsilk stage.
[y] *AUPDC* (area under the disease progress curve) is based on visual estimates of leaf tissue blighted in the center row of three-row plots. See Materials and Methods section for the equation.
[z] Means within a column followed by the same letter are not significantly different according to Bayes' least significant difference procedure, $k = 100$.

TABLE 2. Five assessments of northern leaf blight on four maize hybrid sets of paired maize hybrids (one of each pair with, and the other without gene Ht_1) following inoculation with *Exserohilum turcicum* race 2. The plants were grown at two locations in Champaign County, IL, in 1982

Hybrid	Disease efficiency[w] (lesions/plant)	Lesion expansion[w] (mm^2/day)	Lesion length[w] (mm)	Leaf tissue blighted[x] (%)	*AUDPC*[y]
A632 × A619	4.6 a[z]	5.6 a	71.1 a	30.0 a	88.0 a
A632Ht_1 × A619Ht_1	4.1 a	4.9 a	74.6 a	30.0 a	80.9 a
Mo17 × A634	1.6 c	3.2 b	49.8 b	10.0 b	22.8 b
Mo17Ht_1 × A634Ht_1	2.8 b	3.3 b	56.0 b	6.2 b	20.5 bc
Mo17 × N28	1.7 c	3.1 b	56.1 b	6.8 b	18.6 bc
Mo17Ht_1 × N28Ht_1	1.7 c	3.0 b	54.3 b	6.9 b	21.2 b
B73 × Mo17	1.0 c	1.8 c	31.0 c	4.0 b	11.8 c
B73Ht_1 × Mo17Ht_1	1.0 c	1.6 c	26.8 c	4.6 b	15.9 bc

[w] Disease efficiency, lesion expansion, and lesion length are based on data from 10, 25, and 25 plants, respectively, from the center row of three-row plots averaged over two locations and two replications.
[x] Represents the percentage of leaf tissue destroyed on the upper two-thirds of plants in the center row of three-row plots 5 wk after the midsilk stage.
[y] *AUPDC* (area under the disease progress curve) is based on visual estimates of leaf tissue blighted in the center row of three-row plots. See Materials and Methods section for the equation.
[z] Means within a column followed by the same letter are not significantly different according to Bayes' least significant difference procedure, $k = 100$.

TABLE 3. Four assessments of northern leaf blight and grain yields on four sets of three maize hybrids homozygous recessive, heterozygous, or homozygous dominant for the Ht_1 gene following inoculation with *Exserohilum turcicum* race 2 in 1983 at one location in Champaign County, IL.

Hybrid	Disease efficiency[v] (lesions/plant)	Lesion length[v] (mm)	Leaf tissue blighted[w]	*AUDPC*[x]	Yield[y] (kg/ha)
A632 × A619	14.7 a[z]	87.4 a	58.5 a	248.2 a	2,104.9 d
A632Ht_1 × A619	14.7 a	88.3 a	58.7 a	258.7 a	3,024.8 c
A632Ht_1 × A619Ht_1	13.7 a	72.6 bc	44.5 ab	206.9 b	2,593.6 cd
Mo17 × A634	5.4 b	75.2 ab	46.8 bc	120.7 c	4,699.1 a
Mo17 × A634Ht_1	6.0 b	75.5 ab	39.6 cd	111.8 cd	5,934.8 a
Mo17Ht_1 × A634Ht_1	7.2 b	79.5 ab	37.6 de	103.7 cd	5,739.7 a
Mo17 × N28	4.5 b	61.4 cd	30.5 e	83.3 de	4,285.0 b
Mo17 × N28Ht_1	5.8 b	62.7 cd	30.6 e	81.3 de	4,536.1 b
Mo17Ht_1 × N28Ht_1	5.0 b	51.3 de	30.7 e	81.5 de	4,360.6 b
B73 × Mo17	1.4 c	50.2 de	20.1 f	47.8 f	4,754.9 b
B73Ht_1 × Mo17	1.9 c	46.7 e	20.2 f	49.9 f	4,255.9 b
B73Ht_1 × Mo17Ht_1	2.0 c	45.7 e	20.1 f	58.8 ef	4,682.5 b

[v] Disease efficiency and lesion lengths are based on data from 10 and five plants, respectively, and were obtained from the center row of three-row plots averaged over six replications in 1983. Disease efficiency data were analyzed following a rank transformation.
[w] Represents the percentage of leaf tissue destroyed on the upper two-thrids of plants in the center row of three-row plots 5 wk after the midsilk stage.
[x] *AUDPC* (area under the disease progress curve) is based on visual estimates of leaf tissue blighted in the center row of three-row plots. See Materials and Methods section for the equation.
[y] Grain yields were adjusted to 15.5% moisture.
[z] Means within a column followed by the same letter are not significantly different according to Bayes' least significant difference procedure, $k = 100$.

TABLE 4. Analysis of variance for two assessments of northern leaf blight and yield from four sets of three maize hybrids, A632× A619, Mo17×A634, Mo17 × N28, and B73 × Mo17, which are homozygous dominant, heterozygous, or homozygous recessive for the Ht_1 allele from one location in Champaign County, IL, in 1983

Source of variation	df	Mean squares: Leaf tissue blighted[w]	*AUDPC*[x]	Yield (kg/ha)
Block	5	0.026	3,539.0**	2,800,273**
Hybrid	11	0.126**[y]	33,681.0**	7,986,999**
Ht_1Ht_1 vs ht_1ht_1 in all hybrids	1	0.013	1,814.2	1,761,265
Ht_1Ht_1 and Ht_1ht_1 vs ht_1ht_1	1	0.010	562.5	2,958,362*
Ht_1Ht_1 vs ht_1ht_1 in three hybrids[z]	1	0.017	3,620.0*	2,574,499*
Error	55	0.007	817.2	707,885

[w] Percentage of leaf tissue blighted 5 wk after the midsilk stage and transformed with the arcsine transformation.
[x] *AUDPC* (area under the disease progress curve) is based on visual estimates of leaf tissue blighted in the center row of three-row plots in 1983. See Materials and Methods section for the equation.
[y] Mean squares followed by asterisks (* and **) have significant F tests at $P = 0.05$ and 0.01, respectively.
[z] Contrasts Ht_1Ht_1 hybrids of A632 × A619, Mo17 × A634, and Mo17 × N28 against their ht_1ht_1 counterparts.

TABLE 5. Assessment of resistance to *Exserohilum turcicum* race 2 in two maize hybrid backgrounds under greenhouse conditions

Hybrid	Incubation period[w] (Percentage of lesions present 15 days after inoculation)	Disease efficiency[x] (lesions/plant)	Lesion length[y] (mm)
A632 × A619	44.3 a[z]	2.4 a	64.2 a
A632Ht_1 × A619	11.4 b	1.0 bc	49.1 bc
A632Ht_1 × A619Ht_1	16.7 b	2.0 ab	52.2 b
B73 × Mo17	16.4 b	1.6 ab	51.4 bc
B73Ht_1 × Mo17Ht_1	6.5 b	0.4 c	40.1 c

[w] Incubation period expressed as a percentage and determined by dividing number of lesions present 15 days after inoculation by the total number present 21 days after inoculation and multiplying by 100. Data were transformed with arcsine transformation prior to analysis.
[x] Number of lesions per plant 15 days after inoculation; analyses were conducted on rank-transformed data.
[y] Four lesions per plant (subsample) were measured and averaged for each subsample.
[z] Means within a column followed by the same letter are not significantly different according to Bayes' least significant difference procedure, $k \times 100$.

second inoculation method involved placing three 10-μl drops per plant (approximately 500 viable conidia per drop) on the third leaf as previously described (8); it was used to estimate incubation periods and lesion length. Conidial viability was determined as described for field studies. Plants were held at 100% RH for 12 hr immediately after inoculation. Incubation period was determined by comparing the ratio of lesion numbers present 13, 15, and 17 days after inoculation to the total number of primary lesions present 21 days after inoculation. Incubation period data were arcsine transformed before analyses of variance were performed. The decision to transform was based on inspection of residuals plotted against predicted value and normal probability plots of residuals for both the original and transformed data. Lesion length measurements were made 21 days after inoculation.

RESULTS

Hybrids with the Ht_1 gene, except B73Ht_1 × Mo17Ht_1, had significantly shorter lesions and significantly slower rates of lesion expansion than the same hybrids without the Ht_1 gene when inoculated with *E. turcicum* race 1 in 1982 (Table 1). Significant differences existed among, but not within, sets of hybrid pairs for the five disease assessments for *E. turcicum* race 2 in 1982, except Mo17 × A634 had significantly fewer lesions per plant than Mo17Ht_1 × A634Ht_1 (Table 2).

In 1983, lesions were significantly shorter and *AUDPC* values were significantly lower for A632Ht_1 × A619Ht_1 than the same hybrid with heterozygous or homozygous recessive alleles carried at the Ht_1 locus (Table 3). Yields were significantly lower for A632 × A619 than the A632Ht_1 × A619 or A632Ht_1 × A619Ht_1. No differences were observed within hybrid sets for Mo17 × A634, Mo17×N28, or B73×Mo17 for the four disease assessments of *E. turcicum* race 2 or grain yield in 1983.

Analyses of variance showed that mean squares for hybrids were highly significant for leaf tissue blighted, *AUDPC*, and grain yields of four hybrid sets inoculated with *E. turcicum* (Table 4). Single-degree-of-freedom comparisons between means for hybrids with homozygous dominant alleles versus hybrids with homozygous recessive alleles at the Ht_1 locus were not significant for leaf tissue

TABLE 6. Greenhouse evaluation of disease-producing efficiency of *Exserohilum turcicum* race 2 on four inbred lines of maize

Inbred	Disease efficiency[y]	
	13 days	15 days
Mo17ht_1ht_1	6.1 a[z]	6.4 a
Mo17Ht_1ht_1	3.8 abc	4.5 abc
Mo17Ht_1Ht_1	3.2 bcde	4.3 abc
B73ht_1ht_1	1.7 c	5.1 abc
B73Ht_1ht_1	3.4 bcd	6.2 ab
B73Ht_1Ht_1	4.8 ab	5.7 abc
N28ht_1ht_1	2.9 cde	4.6 abc
N28Ht_1ht_1	1.9 de	3.8 c
N28Ht_1Ht_1	2.6 cde	4.0 bc

[y] Disease efficiency expressed as mean number of lesions per plant averaged over four single-plant subsamples per pot, four replications, and repeated three times.

[z] Means within a column followed by the same letter are not significantly different according to Bayes' least significant difference procedure, $k = 100$. Analyses were performed on rank-transformed data.

blighted, *AUDPC*, or grain yield. Comparisons of hybrids homozygous dominant or heterozygous versus homozygous recessive for the Ht_1 gene were significant for grain yield but not leaf tissue blighted or *AUDPC*. However, comparisons of A632Ht_1 × A619Ht_1, Mo17Ht_1 × A634Ht_1, and Mo17Ht_1 × N28Ht_1 against the same hybrids without the Ht_1 gene were significant for *AUDPC* and grain yield.

Greenhouse studies supported results obtained from field studies; however, differences often were not as apparent. Lesions appeared significantly sooner on A632 × A619 than on either the homozygous or heterozygous Ht_1 versions of the same hybrid (Table 5). With regard to disease efficiency, B73Ht_1 × Mo17Ht_1 had significantly fewer lesions than the B73 × Mo17 ($P \leq 0.05$). Seventeen days after inoculation the homozygous Ht_1 versions of both hybrids (A632Ht_1 × A619Ht_1 and B73Ht_1 × Mo17Ht_1) had significantly smaller lesions than the same hybrids without the Ht_1 gene under greenhouse conditions.

When inbred lines were evaluated, no differences between original and Ht_1 versions of the inbred lines A632 or A619 were detected. However, inbred Mo17 had significantly more lesions and significantly higher disease efficiency than the same inbred with the Ht_1 allele heterozygous or homozygous dominant. Conversely, B73Ht_1 had significantly higher disease efficiency than B73 (Table 6).

DISCUSSION

In the study of race 1 of *E. turcicum*, the effect of the Ht_1 gene was very clear (Table 1). The data indicated that the techniques used were effective in evaluating resistance to NLB, and therefore should have been appropriate for the analysis of race 2 data.

Lesion number expansion and *AUDPC* from race 2 inoculations indicate that resistance of the A632Ht_1 × A619Ht_1 was superior to A632 × A619. Differences were more pronounced when lesion length and *AUDPC* were used to estimate disease. Since lesion length and width were used to calculate lesion expansion rate, and lesion length and lesion area were highly correlated ($r = 0.99$), lesion length appears to be as effective in assessing disease as lesion area.

Results of this study indicate that some Ht_1-converted hybrids are more resistant to *E. turcicum* race 2 than the original versions of these same hybrids. The difference was consistently significant with the most susceptible hybrid, A619 × A632. This suggests that the level of quantitative resistance affects the expression of "residual resistance."

The conidial suspension was used in 1982 to give a uniform inoculation for disease efficiency studies and to produce discrete lesions for determining rates of lesion expansion. In 1983, ground leaf tissue was used to ensure a higher level of initial infection (4) so disease severities on susceptible hybrids would be high enough to affect grain yield (Table 3). Although disease levels were different in 1982 and 1983, the ranking for the four hybrid sets was consistent in both years.

Under greenhouse conditions, the inbred Mo17Ht_1Ht_1 was more resistant to *E. turcicum* race 2 than Mo17, while the opposite was noted for the B73 series. This inconsistency is difficult to explain if only the Ht_1 gene is considered. This study indicates some maize hybrids and inbreds with the Ht_1 gene are more resistant to *E. turcicum* race 2 than near-isogenic hybrids without the Ht_1 gene; however, this difference was significant only in moderately or very susceptible hybrids or inbreds.

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