Resistance Conferred by the *Ht1* Gene in Sweet Corn Infected by Mixtures of Virulent and Avirulent *Exserohilum turcicum*

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ABSTRACT

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The *Ht1* gene conveys a chlorotic-lesion resistant reaction in corn infected by avirulent races of *Exserohilum turcicum*, the causal agent of northern corn leaf blight (NCLB). The widespread deployment of the *Ht1* gene in field corn grown in North America since the 1960s resulted in an increased frequency of *E. turcicum* race 1, which is virulent against the *Ht1* gene. The objective of this study was to assess the value of resistance conveyed by the *Ht1* gene when initial inoculum consisted of different ratios of virulent and avirulent *E. turcicum*. Forty-two sweet corn hybrids with the *Ht1* gene and 42 sweet corn hybrids without *Ht1* were grown in five trials each in 2003 and 2004. In each trial, plants were inoculated with culture suspensions consisting of different percentages of *E. turcicum* race 0 and race 1, including: 100:0, 90:10, 75:25, 50:50, and 0:100. Severity of NCLB was rated visually from 0 to 100% leaf area infected when plants were about 3 to 4 weeks past the mid-silk growth stage. The *Ht1* gene reduced severity of NCLB by as much as one-third when virulent isolates comprised 25% or less of the initial inoculum. Reduction in NCLB severity due to the *Ht1* gene was more substantial on hybrids with susceptible backgrounds than on those with general resistance. When virulent isolates comprised 50% of the initial inoculum, NCLB severity was similar for hybrids with and without the *Ht1* gene if hybrids had equivalent levels of general resistance (measured as NCLB severity from trials inoculated entirely with race 1). To accurately classify NCLB reactions of maize lines relative to their most probable performance in the United States, inoculum should consist of at least 50% *E. turcicum* race 1.

Northern corn leaf blight (NCLB), caused by *Exserohilum turcicum* (Luttrell) K.J. Leonard & E.G. Suggs; teleomorph *Setosphaeria turcica* (Pass.) K.J. Leonard & E. G. Suggs, occurs on maize (*Zea mays* L.) throughout the world, especially in humid areas with moderate temperatures. In the United States, NCLB is one of the predominant diseases of sweet corn. The fungus overwinters on crop debris infected the previous season. Practices that decrease the amount of crop debris (e.g., crop rotation, fall plowing) also decrease NCLB by reducing the amount of primary inocula arising from debris. Foliar fungicides can be applied to control secondary spread of *E. turcicum* from infected leaves in the lower canopy. In some cases, fungicidal control of NCLB is economical, especially if high-valued, susceptible sweet corn hybrids are being grown in humid environments. In most situations, host resistance is the most effective and efficient method to control NCLB.

Resistance to NCLB often is classified into two categories. General resistance

(i.e., quantitative, polygenic, or partial resistance) reduces NCLB severity without the occurrence of qualitatively distinct phenotypes. General resistance appears to be under polygenic control and effective against all biotypes of the fungus. Loci affecting general resistance to NCLB have been mapped with translocation stocks and by QTL mapping on all 10 maize chromosomes (26). Monogenic resistance, conveyed by *Ht* genes, results in distinct phenotypes in response to infection by avirulent races of *E. turcicum*. The *Ht1* gene, identified from the popcorn cultivar Ladyfinger and the field corn inbred GE440 (6), confers a chlorotic-lesion reaction that reduces sporulation and lesion size. The *Ht2* and *Ht3* genes also confer chlorotic-lesion resistance (7,9). A recessive gene, *ht4*, found in a line derived from the maize synthetic BS19, conditions symptoms that appear as circular, chlorotic halos (2). The *HtN* gene, identified from a Mexican landrace, Pepitilla, and the *HtM1* gene, identified in the inbred H102 selected from a cross of C123 × 'Mayorbela', increase incubation and latent periods without the occurrence of a qualitative difference in lesion type (5,22).

Specific virulence occurs in *E. turcicum* in relation to the *Ht* genes (14). Based on six different host resistance genes, there are 64 potential races of *E. turcicum*; therefore, races are identified by ineffec-

tive host genes (14). Race 0 of *E. turcicum* is avirulent against all of the *Ht* genes (14). Race 1 is virulent against the *Ht1* gene. Various other combinations of virulence also occur (4,14,23). The widespread use of the *Ht1* gene in field corn grown in North America since the 1960s resulted in an increased frequency of race 1 in the population of *E. turcicum* in the United States in the 1980s (9,14,15,24). Virulence against *Ht2*, *Ht3*, and *HtN* (i.e., race 23N) occurs at a low frequency (14,18,23) even though these resistance genes have not been deployed widely. In the past decade, race 1 consistently has been more frequent than race 0 in surveys of corn pathogens in the U.S. Corn Belt (21).

Despite the occurrence of virulence, many sweet corn hybrids presently grown in North America carry the *Ht1* gene (17). The *Ht1* gene also is prevalent in field corn grown in the United States. The value of resistance conferred by the *Ht1* gene may vary from field to field depending on the proportion of avirulent and virulent inoculum.

The value of the *Ht1* gene against *E. turcicum* race 0 was estimated in previous research (8,16,25). Ullstrup (25) observed that the extensive chlorosis associated with the *Ht1*-resistant reaction had a debilitating effect on yield as compared to protection conveyed by polygenic resistance. However, NCLB severity was decreased by the *Ht1* gene, and yield losses for *Ht1* hybrids ranged from 6 to 28% compared with losses ranging from 21 to 56% for susceptible hybrids that did not carry the *Ht1* gene. Hooker and Kim (8) assessed NCLB severity for 28 pairs of hybrids nearly isogenic for *Ht1* and *ht* following inoculation with *E. turcicum* race 0. They concluded that *Ht* resistance and polygenic resistance complemented each other. When their disease ratings for *Ht1* versions of hybrids are plotted against the disease ratings for the *ht* isolines, the slope coefficient is 0.67, indicating that Hooker and Kim observed about a one-third decrease in NCLB severity (rated on a 0 to 5 scale) when hybrids with the *Ht1* gene were infected by *E. turcicum* race 0. In a similar study in which three inbreds and three hybrids nearly isogenic for the *Ht1* gene were evaluated in Ohio and Uganda, Lipps et al. (16) observed less severe NCLB on lines with *Ht1*. When disease severity (rated from 0 to 100% leaf area infected) from the *Ht1* isolines is plotted against disease severity from the *ht* isolines, the

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slope coefficient is 0.7, indicating that Lipps et al. also observed about a one-third decrease in NCLB severity when lines with the *Ht1* gene were infected by race 0. Hooker and Kim (8) and Lipps et al. (16) noted that the absolute value of the *Ht1* gene was greater in hybrids with susceptible background than in hybrids with general resistance.

The *Ht1* gene appears to have little or no value against *E. turcicum* race 1. Leath and Pedersen (11–13) observed small differences in lesion size, lesion number, and AUDPC between *Ht1* and *ht1* versions of some maize inbreds and hybrids inoculated with race 1, but differences did not occur for all lines (e.g., B73). They concluded that the *Ht1* gene may contribute a small degree of “residual” resistance against *E. turcicum* race 1 (20). Campaña and Pataky (1) were unable to demonstrate a change in the frequency of the *Ht1* gene in populations of sweet corn selected for general resistance to *E. turcicum* race 1 even though general resistance was improved about 14% per cycle for four or five cycles of recurrent selection. They concluded that the *Ht1* gene did not convey sufficient levels of general resistance to *E. turcicum* race 1 to be selected for in population improvement breeding programs that have been shown to improve levels of general resistance to NCLB (10).

Since primary inoculum of *E. turcicum* comes predominantly from crop debris, the racial composition of *E. turcicum* populations may differ among regions or even among individual corn fields; however, in most situations, initial inoculum in North America is likely to be a mixture of races 0 and 1. The value of the *Ht1* gene against mixed populations of avirulent and virulent *E. turcicum* (races 0 and 1, respectively) has not been documented clearly. The objective of this study was to assess the value of resistance conveyed by the *Ht1* gene when initial inoculum consisted of different ratios of *E. turcicum* races 0 and 1.

MATERIALS AND METHODS

The value of resistance conveyed by the *Ht1* gene when initial inoculum was comprised of different ratios of *E. turcicum* races 0 and 1 was assessed in 2003 and 2004 in field trials at the University of Illinois South Farms in Champaign and Urbana, IL. Forty-two sweet corn hybrids with the *Ht1* gene and 42 sweet corn hybrids without *Ht1* were selected to represent a range of general resistance to NCLB (17). Hybrids were grown in five separate trials each year. Each trial was planted in a separate field within a 2- or 4-day period (20 to 21 May 2003 and 18 to 21 May 2004). Fields were at least 1.2 km apart. Each trial included three replicates of hybrids arranged in a randomized complete block design. An experimental unit consisted of a single row of a hybrid with approximately 12 plants per row.

In each trial, plants were inoculated with culture suspensions consisting of different percentages of *E. turcicum* race 0 and race 1, including: 100:0, 90:10, 75:25, 50:50, and 0:100. Inoculum was produced by culturing at least six different isolates each of race 0 and race 1 on lactose casein hydrolysate agar (LCA). A mixture of isolates, including two standard isolates 74-105 and 82-82 (obtained from J. M. Perkins, Monsanto Co., Waterloo, IL), was used in order to account for slight differences that might occur in aggressiveness (i.e., general virulence) between isolates. Isolates 74-105 and 82-82 (race 0 and race 1, respectively) were selected previously for profuse sporulation and aggressiveness (i.e., high degree of general virulence). The other 10 isolates used in these studies originated from particularly large lesions collected at various times from hybrids with moderate to resistant reactions in sweet corn disease nurseries in Illinois. Cultures isolated from those lesions were compared visually to 74-105 and 82-82 for sporulation on LCA. Greenhouse-grown plants with and without the *Ht1* gene were inoculated with isolates that sporulated abundantly in order to confirm the racial designation of the isolates. After racial identification, the fungi were maintained on dried leaf tissue collected from plants inoculated in the greenhouse. Prior to producing inoculum, *E. turcicum* was isolated from leaf tissue, and the racial designations of the isolates were confirmed again by inoculating greenhouse-grown plants with and without the *Ht1* gene. Mother cultures grown on LCA were ground in sterile water, and 100-mm-diameter petri plates of LCA were flooded with 2 ml of the culture suspensions. Flooded cultures were grown at room temperature (approximately 24 to 26°C) under diurnal fluorescent light for 12 to 15 days. Inocula were produced only from LCA plates that were covered entirely by sporulating cultures of uniform age. Plates used for inocula were selected for uniform growth and abundant sporulation. For each inoculation, 79 profusely sporulating cultures of *E. turcicum* race 0 were ground in 2 liters of water, and 47 profusely sporulating cultures of race 1 were ground in 1 liter of water. The 79 cultures of race 0 consisted of 13 cultures of each of five isolates and 14 cultures of the sixth isolate. The 47 cultures of race 1 consisted of 8 cultures of each of five isolates and 7 cultures of the sixth isolate. The resulting culture suspensions were strained through four layers of cheesecloth. Five sets of inoculum with the desired proportions of mycelial fragments and conidia of race 0 and race 1 were then produced by blending culture suspensions in the following quantities (ml of race 0 : ml of race 1) 635:0, 571:54, 476:135, 317:270, and 0:541. Thus, three of the five sets of inoculum were produced from an equivalent of about 25 culture plates con-

sisting of a mixture of all 12 isolates. Inocula for the trials with ratios of 100:0 and 0:100 of race 0 and race 1 were produced from about 25 culture plates of a mixture of 6 isolates of each race. Each set of concentrated inoculum was diluted to 12 liters. Conidial concentration of inoculum was not measured; however, the number of conidia per plate was quantified from 10 plates each of isolates 74-105 and 82-82 grown under the same conditions used to produce inocula. After grinding plates individually in 100 ml of water, straining ground cultures through four layers of cheesecloth, and diluting to either 300 or 500 ml, conidia were counted using a hemacytometer. The number of conidia per plate for isolates 74-125 and 82-82 averaged $3.68 \times 10^6 \pm 0.42 \times 10^6$ and $3.38 \times 10^6 \pm 0.39 \times 10^6$, respectively, and ranged from 3.0 to 4.1×10^6 for isolate 74-125 and from 2.8 to 4.2×10^6 for isolate 82-82. Thus, these procedures produced diluted inoculum with about 7.4×10^4 conidia per ml plus mycelial fragments. About 4 ml of diluted inoculum was sprayed directly into the whorl of every plant in a trial. Plants between the three- and six-leaf stages were inoculated twice (19 and 25 June 2003) or three times (15, 18, and 23 June 2004). All five trials were inoculated at the same times.

Severity of NCLB was rated visually from 0 to 100% leaf area infected on a row basis when plants were about 3 to 4 weeks past the mid-silk growth stage (i.e., within 1 week of sweet corn harvest maturity). The 0 to 5 scale of Elliott-Jenkins (3), modified to illustrate percent leaf area infected (19), was used as a standard diagram for disease assessments. Each row was given a separate rating by two people. If the two ratings differed by more than 10%, each person re-evaluated the row. The two ratings were averaged for each experimental unit. Trials were rated between 2 and 8 August 2003 and between 27 July and 3 August 2004. Each row also was rated qualitatively (i.e., yes or no) for the occurrence of chlorotic lesions that result when hybrids with the *Ht1* gene are infected by *E. turcicum* race 0.

Data were analyzed for each trial individually by analysis of variance using SAS software. Waller-Duncan Bayesian mean separation values (BLSD $k = 100$) were calculated to compare NCLB severity among hybrids. General resistance of hybrids was estimated from the severity of NCLB in the trials inoculated solely with race 1 (i.e., the 0:100 trial).

Groups of hybrids with and without the *Ht1* gene were compared by analysis of covariance. Severity of NCLB from the trial in which initial inoculum was comprised of 100% race 1 (i.e., the 0:100 trial) was the covariate (i.e., the baseline measure of the general resistance of each hybrid). The presence or absence of the *Ht1* gene (i.e., groups of hybrids with or with-

out the *Ht1* gene) was a qualitative variable in the covariance analysis. The covariance analysis was done for each of the four trials in which race 0 comprised a portion of the initial inoculum (i.e., 100:0, 90:10, 75:25, and 50:50).

RESULTS

Severity of NCLB ranged from 1 to 59% among hybrids in the five trials in 2003 and from 3 to 75% among hybrids in the five trials in 2004 (Table 1). Even though five trials were planted and inoculated at

the same times within years, severity differed among the five trials each year apparently due to differences among fields in soil type, plant growth, environmental conditions within the crop canopy, etc. In 2003, mean NCLB severity was 18.4, 28.2, 21.2, 27.5, and 21.7% in trials in which initial inoculum consisted of 100, 90, 75, 50, and 0% race 0, respectively. In 2004, mean NCLB severity was 42.4, 30.9, 32.1, 32.5, and 22.7% in trials in which initial inoculum consisted of 100, 90, 75, 50, and 0% race 0, respectively. Mean separation

values for comparison of hybrids within trials ranged from 6.5 to 8.3% in 2003 and from 5.3 to 6.5% in 2004 (Table 1).

General resistance, measured as NCLB severity from the trial inoculated with 100% race 1, varied substantially among hybrids. The range of severity based on hybrid means overlapped considerably between groups of hybrids with and without the *Ht1* gene, although the number of hybrids with very low and very high severity differed among the two groups (Figs. 1 and 2). Based on mean NCLB severity

Table 1. Severity of northern corn leaf blight^a (NCLB) within 1 week of fresh market harvest maturity for groups of 42 sweet corn hybrids with the *Ht1* gene and 42 sweet corn hybrids without the *Ht1* gene evaluated in five separate trials in 2003 and 2004 in which plants were inoculated with culture suspensions comprised of different percentages of *Exserohilum turcicum* races 0 and 1

Percentage of races in initial inoculum		2003				2004			
		Hybrids without <i>Ht1</i> (%)	Hybrids with <i>Ht1</i> (%)	Range ^b (%)	BLSD ^c (%)	Hybrids without <i>Ht1</i> (%)	Hybrids with <i>Ht1</i> (%)	Range (%)	BLSD (%)
Race 0	Race 1								
100	0	24.3	12.0	1–45	6.5	50.8	33.1	12–75	6.5
90	10	34.3	21.6	3–59	8.1	38.5	22.5	6–67	5.5
75	25	25.0	17.2	2–45	8.3	40.0	23.4	6–68	5.8
50	50	31.7	23.1	4–56	6.9	37.5	27.0	10–58	6.3
0	100	24.9	18.2	1–45	6.9	26.6	18.2	3–42	5.3

^a Severity of NCLB assessed as a percentage of total leaf area infected on an experimental unit basis.

^b Range = minimum and maximum NCLB severity for all 84 hybrids in a trial inoculated with a particular mixture of races 0 and 1.

^c BLSD = Waller-Duncan Bayesian least significant difference ($k = 100$) for comparison of hybrid means.

Fig. 1. Severity of northern corn leaf blight (NCLB) in 2003 on sweet corn hybrids with and without the *Ht1* gene following inoculation with culture suspensions consisting of various ratios of *Exserohilum turcicum* races 0 and 1: **A**, 100% race 0. **B**, 90% race 0, 10% race 1. **C**, 75% race 0, 25% race 1. **D**, 50% race 0, 50% race 1. Severity of NCLB following inoculation with 100% race 1 (x-axis) was used as a covariate to account for different levels of general resistance among hybrids.

from trials in both years, 21 hybrids with the *Ht1* gene and 11 hybrids without the *Ht1* gene had relatively high levels of general resistance (i.e., <15% NCLB severity from inoculation with race 1). Two hybrids with the *Ht1* gene and 18 hybrids without the *Ht1* gene were relatively susceptible (i.e., >30% NCLB severity from inoculation with race 1). Severity of NCLB on hybrids inoculated solely with race 1 was linearly correlated ($r = 0.84$) among years. Similarly, mean severity from both trials was linearly correlated ($r = 0.89$) with the 1 to 9 (resistant to susceptible) reactions assigned previously to hybrids based on multiple years of evaluation (17).

In each trial, mean severity was significantly greater for the group of hybrids without *Ht1* than for the group with *Ht1* (Table 1). In 2003, the ratios of mean NCLB severity for groups of hybrids without and with *Ht1* were 2.03, 1.59, 1.45, 1.37, and 1.37 for trials in which initial inoculum consisted of 100, 90, 75, 50, and 0% race 0, respectively. Thus, in the trial inoculated with race 0, mean NCLB severity was twice as much on the group of hybrids without *Ht1* as compared to the hybrids with *Ht1*, but in the trial inoculated with race 1, mean severity was only 37% greater on hybrids without *Ht1* as com-

pared to hybrids with *Ht1*. In 2004, the ratios of mean NCLB severity for groups of hybrids without and with *Ht1* were 1.53, 1.71, 1.71, 1.39, and 1.46 for trials in which initial inoculum consisted of 100, 90, 75, 50, and 0% race 0, respectively.

Hybrid mean severity was highly correlated among trials (Table 2). Correlation coefficients ranged from 0.90 to 0.94 in 2003 and from 0.94 to 0.98 in 2004 for comparisons among the four trials in which race 0 comprised a portion of the initial inoculum (i.e., 100, 90, 75, and 50% race 0). When hybrid means from the trial inoculated entirely with race 1 were compared with hybrid means from the other

four trials, correlation coefficients increased from 0.87 to 0.96 in 2003 and from 0.86 to 0.93 in 2004 as the percentage of the initial inoculum comprised of race 1 increased among trials from 0 to 50%.

Covariance analyses differed among the eight trials in which race 0 comprised a portion of the inoculum (Figs. 1 and 2). The covariate, general resistance as measured by NCLB severity from the trial inoculated with 100% race 1, was highly significant in all analyses. The qualitative variable, the presence or absence of the *Ht1* gene, was significant in six analyses, but it was not significant in the analyses of

Table 2. Correlations of hybrid means for northern corn leaf blight severity from five trials in 2003 and 2004 in which 42 sweet corn hybrids with the *Ht1* gene and 42 sweet corn hybrids without the *Ht1* gene were inoculated with culture suspensions consisting of different percentages of *Exserohilum turcicum* races 0 and 1

Percentages of races 0 and 1 in initial inoculum	Percentages of races 0 and 1 in initial inoculum				
	100:0	90:10	75:25	50:50	0:100
100:0	...	0.90 ^a	0.92	0.90	0.87
90:10	0.95 ^b	...	0.93	0.94	0.92
75:25	0.95	0.98	...	0.94	0.94
50:50	0.94	0.94	0.95	...	0.96
0:100	0.86	0.89	0.91	0.93	...

^aCorrelation coefficients above the diagonal are from trials in 2003.

^bCorrelation coefficients below the diagonal are from trials in 2004.

Fig. 2. Severity of northern corn leaf blight (NCLB) in 2004 on sweet corn hybrids with and without the *Ht1* gene following inoculation with culture suspensions consisting of various ratios of *Exserohilum turcicum* races 0 and 1: **A**, 100% race 0. **B**, 90% race 0, 10% race 1. **C**, 75% race 0, 25% race 1. **D**, 50% race 0, 50% race 1. Severity of NCLB following inoculation with 100% race 1 (x-axis) was used as a covariate to account for different levels of general resistance among hybrids.

the two trials in which inoculum was comprised of equal proportions of race 0 and race 1. There was a significant interaction between general resistance and the presence or absence of the *Ht1* gene in four analyses, those for trials inoculated with 100% race 0 and 75% race 0 in 2003 (Fig. 1A and C) and 90% race 0 and 75% race 0 in 2004 (Fig. 2B and C).

When initial inoculum was comprised of 100, 90, or 75% race 0, comparisons of hybrids with and without *Ht1* were relatively similar among trials (Figs. 1A to C and 2A to C). For the four trials in which the interaction was significant, NCLB severity did not differ between hybrids with and without the *Ht1* gene if the hybrid had general resistance (i.e., low severity from the trials inoculated with 100% race 1). For hybrids that were moderately susceptible to susceptible to race 1 (e.g., severity >30% from trials inoculated with 100% race 1), NCLB severity was greater on hybrids without *Ht1* than on those with *Ht1* if initial inoculum was comprised of 100, 90, or 75% race 0. For the two trials in which the interaction was not significant (Figs. 1B and 2A), NCLB severity differed between hybrids with and without *Ht1*. Severity of NCLB from 100% race 0 inoculum in the trial in 2004 was nearly 10% greater on hybrids without *Ht1* than on hybrids with *Ht1* if the hybrids had equivalent levels of general resistance (Fig. 2A). Similarly, NCLB severity from 90% race 0 and 10% race 1 inoculum in 2003 was about 6.5% greater on hybrids without *Ht1* than on hybrids with *Ht1* if the hybrids had equivalent levels of general resistance (Fig. 1B).

When initial inoculum was comprised of 50% race 0 and 50% race 1, there was no difference in NCLB severity between hybrids with and without the *Ht1* gene if hybrids had equivalent levels of general resistance (Figs. 1D and 2D). In both years, the relationship between the covariate and NCLB severity from the trial inoculated with 50% race 0 and 50% race 1 was described by a single regression equation for both groups of hybrids.

DISCUSSION

The widespread deployment of the *Ht1* gene in field corn since the 1960s has caused selection for virulence in populations of *E. turcicum* in North America (14,23,24). Despite the prevalence of race 1, many sweet corn hybrids and some field corn hybrids still carry the *Ht1* gene. The effectiveness of the *Ht1* gene was affected by the frequency of virulent isolates in these experiments. When virulent isolates comprised 50% of the initial inoculum, severity of NCLB on hybrids with and without the *Ht1* gene was similar if hybrids had equivalent levels of general resistance. When virulent isolates were 25% or less of the initial inoculum, the *Ht1* gene conveyed some resistance, particularly to

hybrids that had susceptible to moderately susceptible backgrounds.

In the past decade, race 1 has been isolated more frequently than race 0 in samples of *E. turcicum* collected from monitor plots located throughout the U.S. Corn Belt (21). In most years, race 1 has comprised greater than 50% of the samples collected. If these samples are representative of populations of *E. turcicum* found throughout North America, the *Ht1* gene contributes little or no value to reduced severity of NCLB based on the results from our trials. However, when the population of *E. turcicum* is comprised of 25% or less race 1, the *Ht1* gene contributes to lower NCLB severity, particularly among hybrids with low levels of general resistance (i.e., moderately susceptible or susceptible).

In addition to conditioning a chlorotic lesion response, the *Ht1* gene reduces sporulation. Thus, Hooker and Kim (8) suggested that resistance conveyed by the *Ht1* gene to *E. turcicum* race 0 may be greater in large fields as compared to small plots where rows of plants with the *Ht1* gene are infected by inocula from neighboring rows of susceptible plants. In our trials inoculated with 90, 75, and 50% race 0, there appeared to be a similar proportion of chlorotic lesions on leaves in the lower, middle, and upper canopies, although the number of chlorotic and necrotic lesions was not counted in all trials. Possibly, an abundance of secondary inoculum of race 0 was produced on plants without the *Ht1* gene in these trials.

Many corn pathologists and breeders classify hybrids and inbreds for reactions to NCLB on a continuous scale from resistant to susceptible (e.g., 1 to 9) based on severity of symptoms. Based on the results from this study, there appears to be little concern that lines with high or moderate levels of general resistance will be misclassified regardless of whether the plants are infected with *E. turcicum* race 0, race 1, or a mixture of races. These results are similar to those of Lipps et al. (16), who observed little difference in severity of partially resistant corn with or without the *Ht1* gene. However, when moderately susceptible to susceptible lines with the *Ht1* gene are evaluated in trials with a high proportion of race 0 inoculum, NCLB severity may be as much as one-third less than when these lines are evaluated in trials with race 1 inoculum. In such cases, lines with moderately susceptible to susceptible reactions to NCLB may appear to have moderate or moderately susceptible reactions, respectively, but it is unlikely that they would be mistaken for resistant or moderately resistant lines. By ensuring that race 1 comprises at least half of the inoculum in these evaluations, pathologists and breeders can accurately classify reactions

of maize lines to NCLB relative to their most likely performance in North America.

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