Comparison of Near-Isogenic Maize Lines With and Without the Ht_1 Gene for Resistance to Four Foliar Pathogens

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ABSTRACT

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Greenhouse studies were conducted to evaluate the effect of the Ht_1 gene in conferring resistance to four foliar pathogens of maize (*Zea mays*). First, inbreds Va26, B84, and A634 were used to determine differences among near-isogenic lines. Both resistant and susceptible plants selfed once from backcross (BC) programs were compared with each other and their recurrent parents for resistance to *E. turcicum* race 2. The Ht_1 version of Va26 (BCHt_1) had fewer and smaller lesions than its recurrent parent. Other plants selfed out of the backcross program carried the recessive allele at the Ht_1 locus (BCht_1); these plants had the same size lesions as their recurrent parent and lesions were larger than the lesions on BCHt_1 plants. The BCHt_1 plants from B84 had shorter incubation periods than those of the recurrent parent or BCht_1 plants. No differences were detected with inbred A634. Second, a series of six maize hybrids either homozygous dominant or recessive for the Ht_1 gene were inoculated separately with three foliar pathogens: *Bipolaris maydis, Helminthosporum carbonum*, and *Colletotrichum graminicola*. Hybrids B73Ht_1 × MS71 and A632Ht_1 × A619Ht_1 infected with *B. maydis* had larger lesions than their near-isogenic ht_1 counterparts. When infected with *H. carbonum* race 3, H100 × Mo17Ht_1 had larger lesions than H100 × Mo17; however, A619Ht_1 × A632Ht_1 had smaller lesions than A619 × A632. No differences within hybrid pairs were detected with *Colletotrichum graminicola*.

Single-gene resistance (race-specific resistance) is a primary means of controlling plant diseases and is usually effective against some races of a pathogen, but not others. Martin and Ellingboe (14) demonstrated that the race-specific resistance gene, *Pm*4, reduced the infection efficiency of an isolate of *Erysiphe graminis* DC. f. sp. *tritici* E. Marchal with the virulence gene *p*4, as compared to the same isolate on a near-isogenic wheat (*Triticum aestivum*) line containing the recessive allele, *pm*4. Nass et al (15), found that three near-isogenic lines, *Pm*3a, *Pm*4, and *MA*, had lower disease efficiency and sporulation than the recurrent parental line, Chancellor, when inoculated with an isolate of *E. g. tritici* having the virulence genes for the three wheat lines. Royer et al (20) found that wheat line CI 14118 with the resistance gene *Pm*2 from Ulka does not express the same level of resistance to compatible races of *E. g. tritici* as does the near-isogenic line CI 14119 with *Pm*2 from CI 12632. This indicates either that the resistance genes are not identical or that other differences in resistance exist between the lines (19).

One of the criticisms of previous studies on possible residual effects of resistance genes is that the differences in reaction between near-isogenic lines were not shown to be due to a single gene rather than other resistance genes carried through a backcross program (1).

Northern leaf blight of maize (NLB) caused by *Exserohilum turcicum* (Pass.) Leonard and Suggs (perfect state = *Setosphaeria turcica* (Kuttrell) Leonard and Suggs) is a common pathogen of maize (*Zea mays* L.) in the United States. For over 15 yr, the disease was controlled by the use of the single resistance gene, Ht_1, which had been transferred from Ladyfinger popcorn or the corn inbred GE440 via a backcross procedure into many commercial maize inbreds (5,8). In 1974, a new race of *E. turcicum*, designated as race 2, was reported to produce susceptible lesions on plants with the Ht_1 gene (2). In 1979 this race was found in western Indiana (22) and in 1982 was found throughout the United States corn belt (11).

The objectives of this study were to separate the effect of the Ht_1 gene, which is conferring resistance to *E. turcicum* race 2, from other resistance genes potentially transferred during the backcross procedure and to determine if there were differences among six pairs of maize lines, with or without the Ht_1 gene, for resistance to three other foliar pathogens, *Bipolaris maydis* (Nisik.) Shoemaker, *Colletotrichum graminicola* (Ces.) G. W. Wils and *Helminthosporium carbonum* (Ullstrup).

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MATERIALS AND METHODS

To evaluate the effect of the Ht_1 gene on resistance to *E. turcicum* race 2, seeds of near-isogenic inbred sets of maize with or without Ht_1 were obtained for inbreds Va26, B84, and A634. Seed was from backcross programs with each line having been crossed to an Ht_1 donor plant and then crossed six times to its recurrent parent (ht_1). This sixth backcross was designated BC_6. The Ht_1 donor parents for Va26, B84, and A634 were ROh43Ht_1, B73Ht_1, and A632Ht_1, respectively. The Ht_1 gene from either inbred GE440 or cultivar Ladyfinger popcorn may be considered identical (5). Seed from BC_6 was selfed once (S_1). The S_1 seeds, which segregated 3 resistant: 1 susceptible (to race 1), and seed of the recurrent parent were used in this study. Seeds were planted in 15-cm-diameter clay pots with six seeds per pot; 48 seeds per inbred. Two weeks after planting, the fourth leaf of each seedling was inoculated with three 10-μl drops of a conidial suspension of *E. turcicum* race 1 as previously described (13). The drops contained approximately 500 conidia (50×10^3 conidia per milliliter). Two weeks after the race 1 inoculation, plants were evaluated for the presence of the Ht_1 gene based on the presence of chlorotic-type lesions (4). The plants were thinned to two to four plants per pot, containing plants either of genotype ht_1ht_1 or Ht_1. A minimum of eight pots of each type were retained. No separation was made between plants homozygous dominant (Ht_1 Ht_1) or heterozygous Ht_1 ht_1. Immediately after plants were evaluated with race 1, all leaves with symptoms of NLB were removed and plants were reinoculated with race 2 by pipetting 1 ml of a conidial suspension into the whorl of emerging leaves. The inoculum was prepared by placing leaf tissue from greenhouse-grown plants infected with *E. turcicum* race 2 in a moist chamber for 4 days. Conidia were washed from the tissue with distilled water, filtered through cheesecloth, and the suspensions were diluted to a final concentration of 5,150 viable conidia per milliliter. Viability was greater than 90% as determined from dilution plating (10^{-2}) on lactose-casein hydrolysate agar at 18 hr after inoculation (21). Inbreds B84 and Va26 with the Ht_1 gene were inoculated with *E. turcicum* races 1 and 2 or race 2 alone as described. Comparisons were made for incubation period, disease efficiency, and lesion length. All experiments with *E. turcicum* were repeated three times.

Incubation period was determined by counting the numbers of lesions present at 10 and 14 days after inoculation. The number of lesions present at day 10 was divided by the number present at day 14. Germ plasm with a short incubation period would have relatively more lesions formed early which would result in higher day 10:day 14 ratios. Data were arc sine transformed when appropriate. The decision to transform was based on inspection of residuals plotted against predicted values and normal probability plots of residuals for both the original and transformed data.

Disease efficiency (15) was based on the total number of lesions present 14 days after inoculation. A rank transformation of these counts was used prior to analyses (3). The length of one lesion on each single plant subsample per pot was measured 14 days after inoculation. For analysis of variance, the experiment was considered to be completely random with eight replications and unequal numbers of subsamples.

To determine if differences in resistance to pathogens other than *E. turcicum* existed between hybrids with and without the Ht_1 gene, seeds of six hybrid sets were planted, six seeds per pot, in 15-cm-

TABLE 1. Comparison of three near-isogenic lines in two inbred sets of maize for resistance to *Exserohilum turcicum* race 2 based upon three assessments of disease under greenhouse conditions

Inbred	Incubation period[x] (% lesions developed 10 days postinoculation)	Disease efficiency[y] (lesion/plant)	Lesion length[z] (mm)
Va26			
Rec. parent	0.50	6.5	49
BCht_1	0.60	4.2	50
BCHt_1	0.44	2.5	37
FLSD ($P = 0.10$)	0.29	3.2	10
B84			
Rec. parent	0.52	9.2	62
BCht_1	0.36	10.2	62
BCHt_1	0.68	9.7	62
FLSD ($P = 0.10$)	0.16	3.4	9

[x] Incubation period was calculated as the percentage of lesions present 14 days postinoculation that were present 10 days after inoculation.
[y] Disease efficiency represents the total lesions per plant averaged over single plant subsamples for eight replications.
[z] Means are from one lesion averaged over single-plant subsamples in each of eight replications.

TABLE 2. Resistance of six hybrid maize sets with or without the Ht_1 gene to infection by *Bipolaris maydis* race O as assessed by mean disease efficiency and lesion length

Hybrid	Disease efficiency[x] (lesions/plant)	Lesion length[y] (mm)
B73Ht_1 × MA71	3.8 ab[z]	12.8 a
B73 × MS71	2.9 b	10.3 b
A619Ht_1 × A632Ht_1	3.8 ab	12.0 a
A619 × A632	2.3 b	9.3 c
Mo17Ht_1 × N28Ht_1	3.4 ab	9.9 bc
Mo17 × N28	3.6 ab	9.2 c
B73Ht_1 × Mo17Ht_1	3.3 ab	9.9 bc
B73 × Mo17	4.0 a	9.9 bc
A634Ht_1 × Mo17Ht_1	3.4 ab	9.4 c
A634 × Mo17	3.2 ab	8.8 c
H100 × Mo17Ht_1	2.7 ab	9.4 c
H100 × Mo17	3.2 ab	8.7 c

[x] Disease efficiency is based on the number of lesions per single plant subsample in each of four replications.
[y] Means are from one lesion per single plant subsample in each of four replications.
[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. Resistance of six sets of paired maize hybrids, one with and the other without, the Ht_1 gene for resistance to infection by *Helminthosporium carbonum* race 3 as determined by incubation period and lesion length

Hybrid	Incubation period[x]	Lesion length (mm)[y]
B73Ht_1 × MS71	0.58 abc[z]	13.4 a
B73 × MS71	0.67 a	12.8 ab
H100 × Mo17Ht_1	0.54 abc	12.7 ab
H100 × Mo17	0.53 abc	10.3 c
Mo17Ht_1 × N28Ht_1	0.65 ab	11.5 abc
Mo17 × N28	0.46 abc	10.7 bc
Mo17Ht_1 × A634Ht_1	0.63 abc	11.1 bc
Mo17 × A634	0.41 c	0.6 cd
B73Ht_1 × Mo17Ht_1	0.62 abc	10.1 cd
B73 × Mo17	0.58 abc	10.8 bc
A619Ht_1 × A632Ht_1	0.44 bc	8.1 d
A619 × A632	0.41 c	10.2 c

[x] Incubation period is on a per plant basis from four subsamples and six replications and is expressed as the percentage of lesions present 14 days postinoculation as compared to the number present 21 days postinoculation.
[y] Means are from one lesion per single plant subsample in each of six replications.
[z] Means within a column followed by the same letter are not significantly different according to Bayes' least significant difference procedure ($k = 50$).

diameter clay pots. The hybrids were homozygous at the Ht_1 allele, except B73 Ht_1 × MS71 and H100 × Mo17 Ht_1 were heterozygous. Seedlings were thinned to four plants per pot prior to inoculation. The potting medium was a mixture of soil:peat:perlite (2:2:1, v/v). Inoculum was prepared by placing leaf tissue from plants infected with *B. maydis* race O in a moist chamber for 4 days. Conidia were washed from the tissue with distilled water and filtered through cheesecloth. The isolate of *B. maydis* used in this study was originally recovered in 1982 from east central Illinois. Plants were inoculated 20 days after planting by placing three 10-μl drops of the conidial suspension adjusted to 11,600 viable conidia per milliliter on the leaf surface (13). Viability was greater than 95% as determined 18 hr after inoculation by dilution plating (10^{-2}) on lactose-casein hydrolysate agar.

Two other leaf pathogens were used in similar studies: *H. carbonum* race 3 (17), causal organism of northern leaf spot of maize and *C. graminicola*, the causal organism of maize anthracnose. Isolates of both fungi were recovered from naturally infected plants in central Illinois. Plants were inoculated with 10-μl drops as described earlier. Inoculum was prepared by washing conidia of *C. graminicola* and *H. carbonum* from 14-day-old cultures growing on oatmeal and potato-dextrose agar, respectively (21). Inoculum concentration was 14.45×10^3 viable conidia per milliliter with 97% viability on lactose-casein hydrolysate agar for *H. carbonum*. Approximately 2.0×10^5 conidia per milliliter were used for inoculations with *C. graminicola* (23). Inoculations with *B. maydis*, *H. carbonum*, and *C. graminicola* were all repeated three times.

RESULTS

No differences were detected for incubation period, disease efficiency or lesion length when B84 and Va26 with the Ht_1 gene were inoculated with *E. turcicum* races 1 and 2 or race 2 alone. No differences between inbred lines of Va26 with or without Ht_1 were detected for incubation period, but the backcross line with Ht_1 (BCHt_1) had both fewer and smaller lesions than its recurrent parent (Table 1). The other Va26 line selfed from the backcross program carried the recessive allele at the Ht_1 locus (BCht_1) and had longer lesions than the BCHt_1 plants. However, the BCht_1 plants were intermediate between Va26 and the BCHt_1 plants for disease efficiency (Table 1). In lines of B84 no differences in lesion lengths or lesion numbers were detectable. However the BCHt_1 plants had a shorter incubation period than B84 plants or plants of the BCht_1 group (Table 1). The A634 lines selfed from the backcross program produced few Ht_1 plants (BCHt_1), probably as a result of small sample size. When plants of A634 and the BCht_1 were compared for lesion lengths and disease efficiency, no significant differences were detected.

Differences existed both among and within hybrid sets for resistance to *B. maydis*. Hybrids B73Ht_1 × MS71 and A619Ht_1 × A632Ht_1 had larger lesions than did their ht_1 counterparts (Table 2). Differences existed among hybrids with regard to disease efficiency for *B. maydis*; however, there were no differences within hybrids. No differences were detected with incubation period.

The same six hybrid sets also were evaluated for resistance to *H. carbonum* (Table 3), and differences among hybrid sets were detected when incubation period was used to assess disease; no differences were detected within hybrid sets (Table 3). Hybrid H100 × Mo17Ht_1 had longer lesions than H100 × Mo17, while A619Ht_1 × A632Ht_1 had smaller lesions than its near-isogenic counterpart, A619 × A632. No differences within sets were detected with *C. graminicola*.

DISCUSSION

Inoculation with *E. turcicum* race 1 was used to identify plants with the Ht_1 gene from the selfing of plants from the BC_6 generation. Inbreds B84 and Va26 with the Ht_1 gene were inoculated with race 1 and when the chlorotic-type lesion was visible, the inoculated leaves were removed. These plants and uninoculated control plants were then inoculated with race 2. No differences were detected for incubation period, disease efficiency and lesion length between plants inoculated with races 1 and 2 or race 2 alone. Therefore, no acquired or induced resistance was observed with this technique of identifying plants with the Ht_1 gene.

In inbred Va26, the BCHt_1 version had shorter lesions than Va26 and BCht_1. With both race 1 and 2, Ht_1 lines have been reported to have smaller lesions than their ht_1 counterparts (5,7,12). The number of lesions per plant is usually controlled by multiple genes (5,7–10); although BCHt_1 had fewer lesions than Va26, it did not have fewer than BCht_1. This would indicate that Ht_1 may be conditioning resistance to race 2 in a manner similar to race 1, but that quantitative resistance traits also were transferred during the backcrossing.

The selfing procedure employed in these studies produced resistant plants. Although the resistant plants were either heterozygous or homozygous dominant for the gene Ht_1, this did not appear to influence results dramatically. However, residual resistance may be less apparent in the heterozygous version of Ht_1. Hooker (5) observed that plants homozygous for the Ht_1 allele have fewer and smaller lesions than plants heterozygous for Ht_1 when inoculated with *E. turcicum* race 1.

It seems unlikely that a single gene for qualitative resistance to *E. turcicum* (Ht_1) would condition susceptibility to other leaf blight pathogens in some hybrids but not in others. This would suggest that the increased susceptibility of the Ht_1 versions of B73 × MS71 and A619 × A632 to *B. maydis* is not due to Ht_1. Similarly, it is unlikely that the Ht_1 gene would condition susceptibility or resistance to *H. carbonum* depending on the inbred into which the gene is incorporated. There is evidence that Ht_1 is expressed at different levels in different backgrounds (7) when inoculated with *E. turcicum* but none to indicate that Ht_1 acts in a completely different manner depending on background. When hybrids were infected with *H. carbonum*, the effect of the Ht_1 gene was not clear. In one hybrid, A619 × A632, the Ht_1 version was more resistant; however, in the hybrid H100 × Mo17 the opposite was true. Although the expression of Ht_1 is influenced by the genetic background in which it occurs, a difference this dramatic has not been shown with Ht_1. Data from a previous study showed no effect of Ht_1 when plants were inoculated with race 2 of *H. carbonum* (6). This may be due to differences in pathogenicity between isolates of races 2 and 3, differences in sample size, or to differences in inoculation method.

This study would indicate that both the Ht_1 gene and other resistance genes carried through a backcross program can be acting together. To detect residual gene resistance, we regarded race 2 virulent on Ht_1 because of the infection type (susceptible lesion) race 2 produced on Ht_1 plants compared to the infection type produced by race 1 (chlorotic-type lesions). One explanation of these results is the possible differences between isolates of *E. turcicum* race 2 for parasitic fitness (aggressiveness). Royer et al (20) have shown differences in partial resistance to different isolates of the same race of *E. g.* f. sp. *tritici* on more than one isoline of Chancellor winter wheat. In this study, all greenhouse experiments were repeated with a second isolate of *E. turcicum* race 2. The second isolate, originally recovered from Indiana (22), ensured results reported here were applicable to isolates from at least two locations. Work by Pedersen (*unpublished*) showed these two isolates are relatively aggressive when evaluated with 11 other race 2 isolates for latent period and lesion length, under greenhouse conditions. The race 2 isolates used in this study were hyphal-tipped regularly to maintain purity. Each time inoculum was produced directly from leaf tissue, the tissue was from greenhouse-grown plants inoculated with a culture that had been hyphal-tipped; this ensured a single isolate for each inoculation.

The likelihood of transferring quantitative genes for resistance to *E. turcicum* into the recurrent parent while crossing with the Ht_1 donor seems great. Hooker and Perkins commented that in their backcross programs (where seed for this study originated) alleles for resistance to other races of *E. turcicum* could be selected although the germ plasm under selection had only been inoculated with race 1 (9). Some of the quantitative genes for resistance to *E.*

turcicum in maize are on the long arm of chromosome 2 (10). The Ht_1 gene also is on the long arm of chromsome 2 (18) and this make the transfer of such genes likely while incorporating Ht_1. Further, current theory (4) would indicate that backcrossing with selection would enhance the probability of recovering favorable alleles from the donor plant. The likelihood of this increases when the donor and recurrent parent differ in the number of loci with alleles for resistance. However, the continued transfer of these genes into the new inbred depends on selection. At Illinois, in backcrossing Ht_1, selection was made only for lesion-type and no intended selection was made for size or numbers of lesions (J. M. Perkins, *personal communication*). Therefore, genes other than Ht_1 would be less likely to be recovered after numerous backcrosses as half of them would be replaced with alleles from the recurrent parent with each generation of backcrossing. The possibility of linkage confuses the situation. If a linkage did not break or if it was not broken until late backcross generations, any resistance alleles near Ht_1 would be carried with this gene.

The results (based on infection type) of this study would indicate that a single gene may condition resistance to a pathogen even after the pathogen has overcome the major effect of the resistance gene. This type of resistance, residual gene resistance, was detected between near-isogenic maize inbred and hybrid sets under field and greenhouse conditions. Quantitative genes for resistance also are contributing to the differences detected between near-isogenic lines. The possibility that genes tightly linked to Ht_1 are conditioning much of the resistance attributed to Ht_1 cannot be discounted. The idea of continuing to use genes once a pathogen has gained virulence to them (16,19) may be of value for control of NLB of maize. However, the levels of resistance discussed here are small compared to the levels of quantitative resistance found in many commercial maize hybrids and therefore may be unnoticeable in the field under less-than-epiphytotic conditions.

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