Race of *Helminthosporium turcicum* Not Controlled by *Ht* Genetic Resistance in Corn in the American Corn Belt

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ABSTRACT

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A race of *Helminthosporium turcicum* capable of overcoming the *Ht* monogenic resistance in *Zea mays* has been found in inbreds and hybrids near Brook, Indiana. The resistant chlorotic lesions conditioned by the *Ht* gene as a response to infection by the common U.S. race of *H. turcicum* now appear as completely susceptible lesions as a response to infection by this newly found race. Comparisons of several sources of the *Ht* gene and several isolates of the pathogen indicate race differences. On NN14B lesions were resistant with both old and new races.

Northern corn leaf blight, caused by *Helminthosporium turcicum* Pass. (= *Exserohilum turcicum* (Pass.) Leonard & Suggs, *Trichometasphaeria turcica* Luttrell [7,9]), has been economically significant on corn (*Zea mays* L.). Bergquist and Masias (1) reported races 1 and 2 of the pathogen in Hawaii. The *Ht* gene conditions a chlorotic lesion reaction that greatly reduces lesion size and sporulation of race 1 (4). Race 2 is virulent to corn lines carrying the *Ht* gene. A second major gene, Ht_2, conditions a similar chlorotic lesion reaction to both races (5). Other races (8,10) and other monogenic sources of resistance to *H. turcicum* have been reported (3,5,6).

This article reports the first known occurrence, in the corn belt of the continental United States, of a second race virulent on lines containing the widely used *Ht* gene. The pathogen was first observed in seed parent inbred lines in a seed corn production field near Brook, Indiana, in July 1979. The inbred was a derivative of A632 and contained the *Ht* gene. Only lesions indicating a completely susceptible response were found; no chlorotic lesions were found on any plant. The total lack of chlorotic lesions indicated the presence of one race in the field, rather than a mixture of races. The level of infection was 4 by Elliott and Jenkins' scale (2), and plant growth was just past anthesis. Other inbred and hybrids with the *Ht* gene within a few miles of Brook had similar infections.

MATERIALS AND METHODS

Laboratory experiments were done to verify the infecting pathogen from Brook as a race other than that previously known in the continental United States. Isolates 1851, 1981, and 2101 were all *H. turcicum* isolates that induce the chlorotic lesion response on corn materials protected by the *Ht* gene; they were compared with the fungus obtained from Brook.

In the first experiment (Table 1), short rows of 10 kernels each of inbred materials N28, N28Ht, H93, R168, and Mp490 were planted in the greenhouse. (N28Ht and H93 contain the *Ht* gene for resistance.) Spores washed from lesions on leaves collected in the production field at Brook were squirted into the whorl of 5-wk-old seedlings in the north half of each row. The south half of each row was treated similarly with spores from laboratory isolate 1981, which had been cultured on oats. After 14 days, reactions of inbreds to the isolates were compared.

In the second experiment (Table 2), remnant seed from the seed lot used to plant the production field at Brook and from other inbreds were planted in four 10-cm pots and grown in the growth chamber (six seeds per pot). Other corn materials included were A632Ht, A632, B73Ht, B73, NN14A(Ht), and NN14B (Ht_2). Two pots of each inbred were inoculated with isolate 1851, cultured on oats, and two pots were inoculated with the Brook material. Inoculum was prepared by washing two heaping teaspoons of cultured oat material (isolate 1851) in 40 ml of distilled water. The Brook inoculum consisted of 8–10 lesions excised from leaves, submerged in 40 ml of distilled water, and scraped to loosen the spores. Ten-day-old plants were inoculated with the spore suspension, and reactions to the inoculum were compared 11 days later.

Experiments were done to check for any differential effect due to cytoplasmic male sterility sources and to check several inbreds that had been converted to the homozygous *Ht* gene by backcrossing by different breeders (Table 3). Isolate 2101, cultured on potato-dextrose agar, was compared with isolate 2102 lesions from Brook. The inocula were calibrated to concentrations of 25,000 spores/ml with a hemocytometer. The inoculation method and timing were the same as for the second experiment.

DISCUSSION

All experiments verified that the Brook isolate of *H. turcicum* differed from isolates frequently encountered previously in the continental United States. Microscopic observations indicated morphology fitting descriptions of *H. turcicum*. All symptoms on hosts were considered classic for those of northern corn leaf blight. Koch's postulates successfully demonstrated the pathogenicity of the fungus.

The first experiment gave indication of a variation in this fungus that allowed it to overcome *Ht* gene resistance, as already noted in field observations at Brook. The finding of only completely susceptible lesions in the field indicated a nearly pure infection with an *H. turcicum* variant that could overcome the chlorotic lesion resistance. A mixture of lesion types would have indicated a mixture of races in the field.

The second experiment involved remnant seed used in the production field at Brook and eliminated the possibility of seed labeling confusion that might have accounted for the susceptible lesions

Table 1. Comparison of infections caused by spores washed from lesions on leaves collected near Brook, Indiana, and infections made by *Helminthosporium turcicum* isolate 1981

Inbred	*Ht* Gene	Inoculum source: Brook lesions	Inoculum source: Isolate 1981
N28	Absent	S[a]	S
N28Ht	Present	S	R[b]
H93	Present	S	R
Mp490	Absent	S	S
R168	Absent	S	S

[a] S = Susceptible reaction type lacking chlorotic tissue and lesion unrestricted in size.
[b] R = Resistance exhibited by restriction of lesion size and chlorotic tissues.

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Table 2. Comparison of disease reactions on inbreds with and without resistance to *Helminthosporium turcicum*

Inbred	Known single dominant gene	Fungus source: Brook lesions	Fungus source: Isolate 1851
A632Ht	*Ht*	S[a]	R[b]
A632	None	S	S
A632Ht derivative[c]	*Ht*	S	R
B73Ht	*Ht*	S	R
B73	None	S	S
NN14A	*Ht*	S	R
NN14B[d]	Ht_2	R	R

[a]S = Susceptible reaction.
[b]R = Resistant reaction.
[c]Remnant seed from the production field planted near Brook, Indiana.
[d]Presence of the Ht_2 gene may or may not be responsible for the resistant lesions resulting from infections by inoculum from Brook materials.

Table 3. Inbred conversions to the homozygous *Ht* by different breeders

Pedigree	Isolate[a] 2101	Isolate[a] 2102[b]
A619	S[c]	S
A619Ht(15)[d]	R	S
A619Ht(16)	R	S
A619PS cytoplasm *Ht*[e]	R	S
A632	S	S
A632Ht(11)	R	S
A632Ht(14)	R	S
A632C cytoplasm *Ht*	R	S
N28	S	S
N28Ht(16)	R	S
N28Ht(11)	R	S
N28T cytoplasm *Ht*	R	S

[a]Inoculations were at concentrations of 25,000 spores/ml.
[b]This isolate was from materials from Brook, Indiana.
[c]S = Susceptible lesion response, R = resistant lesion response.
[d]Numbers in parentheses designate conversions by different breeders.
[e]Only three male sterile cytoplasms were tested.

rather than chlorotic lesions at Brook. It also demonstrated that NN14B possessed resistance for the race of *H. turcicum* at Brook. Although the NN14B is a source of the Ht_2 gene (5), it is not clear whether the Ht_2 gene is responsible for the resistant reaction observed with the Brook isolate.

The third experiment involved inbreds converted to the *Ht* gene through backcrossing by different breeders. The results supported the previous experiments showing that the Brook race negated the effectiveness of the *Ht* gene regardless of its origin. The three cytoplasms tested had no effect on the disease reaction, compared with their normal counterpart.

We have not yet been able to differentiate the Brook race of *H. turcicum* from race 2 found in Hawaii, although further testing may do so. No corn planted immediately near the production field is known to have come from Hawaii and the field had no history of any material from Hawaii. Lesions on corn containing the *Ht* gene were observed at four different locations in Indiana within a month of the initial discovery. Similar findings on material from Martinsburg and Landisville, Pennsylvania, indicate that the pathogen may be widespread. Such a spread indicates that the pathogen has either mutated simultaneously over a wide geographic area or that it has been present for some time but not previously observed.

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