Reactions of Eighty-Four Sources of Chlorotic Lesion Resistance in Corn to Three Biotypes of *Helminthosporium turcicum*

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ABSTRACT

Perkins, J. M., and Hooker, A. L. 1981. Reactions of eighty-four sources of chlorotic lesion resistance in corn to three biotypes of *Helminthosporium turcicum*. Plant Disease 65:502-504.

Eighty-four sources of chlorotic lesion resistance were evaluated in the seedling stage for reaction to three biotypes of *Helminthosporium turcicum*. Resistance was found to race 1 in 76 sources, to the race 2 biotype from Brook, IN, in 36 sources, and to race 3 in 82 sources. The degree of resistance expression differed.

Additional key words: gene *Ht*, gene *Ht2*, gene *Ht3*, gene *HtN*, northern corn leaf blight, physiologic races

Many sources of chlorotic lesion resistance in corn (*Zea mays* L.) to northern leaf blight caused by *Helminthosporium turcicum* Pass. have been identified (4–7). This type of resistance was first observed in the inbred GE440 (4). Resistance is inherited as a single dominant gene (3,4) designated *Ht* (4). A gene identified in the popcorn cultivar Ladyfinger is also at the *Ht* locus (4). Two other major genes conferring chlorotic lesion resistance, *Ht2* and *Ht3*, appear to be at independent loci (5,12). Another type of single-gene resistance is conditioned by the gene *HtN* (2). Unlike the chlorotic lesion type, gene *HtN* mainly prevents formation of lesions on adult plants. In the evaluation of diverse sources of germ plasm, many additional sources of resistance have been found (6,7,11). Most have dominant genes at or closely linked to the *Ht* locus (9,15).

Three physiologic races of *H. turcicum* have been described. Race 1, avirulent to plants having gene *Ht*, is the common race in the U.S. corn belt and elsewhere (8). In 1972 an isolate of *H. turcicum* virulent to plants having *Ht* was observed in Hawaii (1); this isolate, a virulent to plants with gene *Ht2*, was designated race 2 (10). During the summer of 1979 a second isolate of *H. turcicum* virulent to plants having gene *Ht* was observed in Indiana (14). In this respect the isolate is similar but not identical to race 2. *H. turcicum* race 3 was collected in South Carolina in 1976 (13); this race is virulent to plants having genes *Ht2* and *Ht3* but avirulent to plants having *Ht*.

Virulence formulas have been constructed for races of *H. turcicum* (10). Race 1, avirulent to corn having genes *Ht*, *Ht2*, and *Ht3*, would be designated by the formula *Ht Ht2 Ht3*/0 (effective/ineffective genes). Based on previous evaluations, race 2 from Hawaii and the biotype from Indiana would be designated by the formula *Ht2 Ht3*/*Ht*. Race 3 is designated as *Ht*/*Ht2 Ht3* (13).

Many sources of chlorotic lesion resistance to *H. turcicum* have been identified at the Illinois Agricultural Experiment Station by using race 1 as the test pathogen. The purpose of this study was to evaluate many of these sources for their reaction to races 2 and 3 of *H. turcicum* and to compare reactions to all three races.

MATERIALS AND METHODS

We used three races of *H. turcicum* to evaluate 84 sources of resistance, 11 backcross selections of *Ht*, *Ht2* or *Ht3*, and 3 susceptible inbreds. Because the number of sources was large, the study was divided into five factorial experiments. Each experiment had two replicates and included two inbred checks, ROh43Ht and RB37Ht. Ten plants from each source was tested with a race. The three races used in the study were a field isolate of race 1 from Illinois, an isolate referred to as race 2 in this paper obtained near Brook, IN (14) and virulent to corn having gene *Ht*, and race 3 from South Carolina (13).

All disease reactions were obtained in the greenhouse. Seedlings in the four to five leaf stage were sprayed with conidial suspensions in water (approximately 30,000 spores per milliliter) of each isolate. Inoculum concentrations were such that lesions occurred on all plants but the seedlings were not excessively damaged. The plants were incubated in a

Table 1. Seedling reaction[a] of resistance sources in *Zea mays* to three biotypes of *Helminthosporium turcicum*

Source	*H. turcicum* biotype Race 1	Race 2	Race 3
Group 1[b]			
Ky217	8.0	7.2	7.2
B14	7.6	8.0	6.5
B37	6.7	7.2	7.5
Pa73-3	5.1	5.2	5.5
Group 2[c]			
B1138 T	1.7	1.7	1.9
BS8-265	1.9	1.7	1.7
BS8-260	2.0	1.6	1.9
Pa75-24	2.0	1.7	2.1
Pa73-1	2.0	1.9	1.7
BS8-261	2.0	1.9	1.9
AWF4	2.0	1.9	2.0
BS8-264	2.0	2.0	1.7
Oh43Ht3	2.0	2.0	1.7
CBSA	2.0	2.0	1.9
Oh43Ht2	2.0	2.0	1.9
Va26Ht3	2.0	2.0	1.9
BS8-263	2.0	2.0	2.0
MP311	2.0	2.0	2.0
TZU39	2.0	2.0	2.0
407	2.0	2.0	2.0
NN14B	2.0	2.0	2.2
BS8-268	2.0	2.1	2.0
081	2.0	2.1	2.0
ROh43HtB	2.0	2.5	2.0
B37HtN	2.0	2.6	1.7
BS8-262	2.0	2.9	1.9
ROh43HtA	2.0	3.1	2.0
Oh43CTf	2.1	2.0	2.5
BC10	2.1	3.0	2.0
K4 · Ky36-11	2.2	2.0	2.0
Alexho	2.2	2.0	2.6
R109BR+	2.2	3.2	2.6
BS8-258	2.4	1.7	2.0
BC13	2.4	3.1	2.4
492	2.5	2.4	1.7
R134	2.6	3.0	2.7
BZU158	2.9	2.5	3.5
BS8-267	2.9	3.5	3.0
K720	2.9	3.6	2.9
GA199	3.2	3.9	4.0
Ky55	3.5	3.5	2.7
Pr	4.2	4.5	4.2
H52	4.7	3.6	4.6
AA8Ht	4.7	4.2	4.4
AWF5W	5.0	4.4	4.5
Group 3[d]			
231A	2.0	8.0	2.0
700	2.0	8.0	2.0
221	2.1	8.0	2.0
167A	2.0	7.6	2.0
231	2.1	7.6	2.0
866A	2.1	7.6	2.0
221A	2.2	7.6	2.0
336	2.7	8.0	2.1

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The research was supported by funds from the Illinois Agricultural Experiment Station.

0191-2917/81/06050203/$03.00/0

Table 1. *(continued from preceding page)*

Source	*H. turcicum* biotype		
	Race 1	Race 2	Race 3
167C	2.7	8.0	2.0
700A	2.7	8.0	2.0
EES650	2.4	7.6	3.2
RB37HtA	2.0	7.2	2.0
RB37HtB	2.0	7.2	2.0
713A	2.0	7.2	2.0
899	2.9	8.0	3.0
BC9A	2.9	8.0	2.0
224	2.9	8.0	2.0
BTU32	3.1	8.0	3.1
492A	2.7	7.6	2.0
W37A	2.0	6.9	2.0
231B	3.2	8.0	2.1
535A	2.1	6.9	2.1
NK51036	2.5	7.2	2.1
NN14A	2.6	7.2	2.2
MexMix	2.6	7.2	2.1
GE440	2.0	6.5	2.0
GRO48	2.1	6.5	2.0
415A	2.1	6.5	2.0
Ladyfinger	2.0	5.9	2.0
NK75	4.2	8.0	4.0
BC15	2.0	5.7	2.0
440	2.0	5.7	2.0
415B	2.4	5.5	2.1
Pop35	2.0	5.1	2.0
EES647	3.4	6.2	2.0
R109BHtA	2.0	4.2	2.0
MoL	2.0	4.1	2.0
R109B	2.2	4.2	2.9
AWF5B	2.0	3.9	2.0
461	4.7	6.5	3.2
R109BHtB	2.0	3.6	2.0
R109BR−	2.7	4.2	3.4
BW	2.0	3.5	2.0
BC9	2.2	3.5	2.1
Group 4[e]			
B37Ht2	2.2	2.0	3.2
Group 5[f]			
B37Ht3	4.6	2.1	1.9
Group 6[g]			
011	7.2	7.2	5.0
P1031	6.1	8.0	4.4
Oh43	5.9	5.5	3.4
BS8-266	5.7	5.9	3.2
P1026	5.7	7.6	4.5
866C	5.5	7.6	3.9
R109BS	4.6	3.9	2.4

LSD (0.05) = 1.1

[a] Averages of four observation times when lesions were evaluated as: chlorotic without necrosis = 1; chlorotic with some necrosis = 2, chlorotic with considerable necrosis = 3, necrotic with chlorotic border = 5, and wilted and necrotic = 8.
[b] Lines susceptible to races 1, 2, and 3.
[c] Lines resistant to races 1, 2, and 3.
[d] Lines resistant to races 1 and 3 and susceptible to race 2.
[e] Lines resistant to races 1 and 2 and susceptible to race 3.
[f] Lines resistant to races 2 and 3 and susceptible to race 1.
[g] Lines resistant to race 3 and susceptible to races 1 and 2.

mist chamber for approximately 12 hr and then moved to greenhouse benches. Data concerning lesions were recorded at 2- to 3-day intervals between 10 and 19 days after inoculation. Lesion types were: O;—chlorotic fleck, R+—chlorotic lesion without necrosis, R—chlorotic lesion with some necrosis, R⁻—chlorotic lesion with considerable necrosis, I—necrotic lesions surrounded by a chlorotic border, and S—wilted and necrotic lesion without chlorosis.

Because I and S lesions could not be differentiated into three distinct types, they were recorded as single classes. These were later transformed to a modified 1–9 scale and the four rating dates averaged for statistical analysis. Lesion types and their values were R+—1, R—2, R⁻—3; I—5, and S—8. The values corresponding to the missing lesion types were eliminated from analysis. Because the O; rating could indicate either a delay in lesion formation or fungal growth limited to a few cells, it was arbitrarily given a numerical rating of one greater than that of the lesion type on the next rating time. For example, an O; followed by R was given a rating of 3 but if followed by S a rating of 9.

RESULTS

Two types of lesions were initially observed on inoculated seedlings. The most common was a chlorotic lesion with varying degrees of necrosis. A chlorotic fleck was initially expressed by other sources. As the study progressed, these seedlings expressed chlorotic lesions or a susceptible reaction. Gene *HtN* in B37 conditioned chlorotic lesions in seedlings. The degree of resistance expressed varied among the sources.

Of the 84 sources, 76 were resistant to race 1; 36 and 82 sources were resistant to races 2 and 3, respectively. Sources resistant to all three races carried the major gene *HtN*.

Based on the reaction of the two inbred checks, infection of *H. turcicum* was similar in the five experiments; hence, the data were combined for statistical analyses and comparisons (Table 1). The sources studied can be divided into six groups using a rating of 5.0 or less as resistant and the LSD = 1.1. Group 1 contains lines susceptible to the three races. Lines in group 2 are resistant to all three races. Races 1 and 3 are avirulent and race 2 is virulent on sources in group 3. Races 1 and 2 are virulent and race 3 is avirulent on sources in group 6. Group 4 contains B37Ht2, which is resistant to races 1 and 2 and susceptible to race 3. Group 5 contains B37Ht3, which is resistant to races 2 and 3 and susceptible to race 1.

DISCUSSION

Although the sources of chlorotic lesion resistance to *H. turcicum* used in this study were originally identified by using only race 1, many conferred a high degree of resistance to the race 2 biotype and race 3 in the seedling stage. Previous studies (3–6,9) indicate that plants expressing chlorotic lesion resistance in the seedling stage will express resistance in the adult plant stage. A possible exception is the inbred line Ky217; it expresses chlorotic lesion resistance in the adult plant stage, which appears to be recessive in inheritance (A. L. Hooker, *unpublished*). This resistance, however, was not expressed in the seedling stage in this study (Table 1).

The large number of sources that express resistance to races 2 and 3 as well as to race 1 shows the value of plant studies for resistance. The different biotypes of *H. turcicum* now available also make it possible to more clearly differentiate alleles at the *Ht* locus.

Additional sources of chlorotic lesion resistance probably exist. Alleles conferring resistance to races 2 and 3 but not to race 1 would not have been identified since race 1 has been used in all previous genetic and breeding work. Such sources, if they exist, would be useful in breeding for resistance to *H. turcicum*.

Since a number of sources resistant to the three isolates were identified, it would seem desirable to incorporate this resistance into corn belt germ plasm and commercial hybrids. Sources with resistance conditioned by single dominant genes (5,9,11) are candidates for backcross breeding programs. Inheritance studies have not been completed on several of the sources. Some sources may carry more than one gene for resistance. Recessive genes are believed to confer resistance in other instances.

Experience with the genes *Ht, Ht2*, and *Ht3* indicate that a high level of resistance can be maintained through backcross breeding, but the degree of resistance will vary among inbred lines (A. L. Hooker and J. M. Perkins, *unpublished*). Therefore, the resistance in the sources studied may not all be equally functional when the alleles are incorporated into different genotypes.

Numerous alternatives are available for developing hybrids resistant to *H. turcicum*. Resistance expressed as varying amounts of healthy tissue and polygenic in inheritance occurs. When used with chlorotic lesion resistance, the effectiveness of each is enhanced. Various combinations of genes and alleles for chlorotic lesion resistance are also possible. All of the above should be superior to susceptible genotypes or genotypes with a low degree of polygenic resistance but a single gene for chlorotic lesion resistance.

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