

Exserohilum turcicum Virulent on Corn with the *Ht* Resistance Gene in Ohio

P. E. LIPPS, Assistant Professor, Ohio Agricultural Research and Development Center, Wooster 44691, and R. E. HITE, Plant Pathologist, Animal and Plant Health Inspection Service, USDA, Department of Plant Pathology, The Ohio State University, Columbus 43210

ABSTRACT

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Isolates of *Exserohilum turcicum* from commercial and seed production cornfields in Hardin, Mercer, and Preble counties, Ohio, caused necrotic lesions on inbreds and hybrids of *Zea mays* with the *Ht* gene. When these isolates were inoculated onto inbred NN14B containing the *Ht2* gene, resistant-type, chlorotic lesions developed indicating the isolates were race 2. None of the isolates were virulent on NN14B carrying the *Ht2* gene and none were avirulent on lines carrying the *Ht* gene; therefore race 3 has not as yet been found in Ohio.

Additional key words: *Helminthosporium turcicum*, maize, monogenic resistance, northern corn leaf blight

Northern corn leaf blight caused by the *Exserohilum turcicum* (Pass.) Leonard and Suggs state of *Setosphaeria turcica* (Luttrell) Leonard and Suggs has been controlled principally through the use of corn (*Zea mays* L.) hybrids resistant to this pathogen. The development of races virulent on hybrids containing the widely used resistance gene *Ht* is a potentially dangerous situation that warrants

monitoring in major corn producing regions (7).

The dominant *Ht* gene has provided effective control of northern corn leaf blight in the continental United States for nearly 20 yr (3,7,8). In 1974 a new race of *E. turcicum* was reported from Hawaii (1). This race was virulent on plants containing the *Ht* gene but avirulent on plants with *Ht2* gene (1). This race was designated race 2 and was represented by the virulence formula *Ht2/Ht* (effective/ineffective genes) (6). Race 2 or a race that responded similarly was reported in Indiana in 1979 (8). This was the first report of a naturally occurring race virulent on plants with the *Ht* resistance gene in the continental United States (7,8). Subsequent investigators have reported another biotype, race 3, with a virulence formula *Ht/Ht2, Ht3* (7). In this study, however, the *Ht2* gene could

not be differentiated from the *Ht3* resistance gene (7).

This paper reports the occurrence of a race of *E. turcicum* virulent on corn lines carrying the *Ht* resistance gene in Ohio. In early September 1980, leaves with large necrotic lesions typical of the susceptible-type lesions caused by *E. turcicum* were collected in commercial and seed production fields in Hardin, Mercer, and Preble counties of western Ohio. Inbreds and hybrids from these fields were known to have the *Ht* gene. The level of infection was light, with only a few lesions on leaves late in the season, and no yield loss was expected.

MATERIALS AND METHODS

Single lesion isolates were obtained by surface sterilizing small pieces of necrotic leaf tissue in 0.5% sodium hypochlorite for 1 min, plating on Difco potato-dextrose agar (PDA), and transferring hyphal tips onto PDA. Inoculum was prepared by growing the isolates on PDA in the dark at 23 C for 14 days. Cultures were then flooded with sterile, distilled water, and conidia were scraped from the agar surface with a rubber policeman. The resulting suspension was filtered through two layers of cheesecloth, and the concentration was adjusted to 10,000 conidia per milliliter.

Six isolates of *E. turcicum* were used in inoculation studies: 46-80 from Mercer County; 48-80 from Hardin County; 50-80, 51-80, and 52-80 from Preble County;

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Table 1. Response of corn lines carrying the *Ht* or *Ht2* resistance gene to six isolates of *Exserohilum turcicum* from Ohio

Corn line	Isolate					
	46-80	48-80	50-80	51-80	52-80	204-79
G4444 ^a	N ^b	N	N	N	N	N
G4444A <i>Ht</i>	N	N	N	N	N	C
B73 × Mo17	N	N	N	N	N	N
B73 <i>Ht</i> × Mo17 <i>Ht</i>	N	N	N	N	N	C
A632	N	N	N	N	N	N
A632 <i>Ht</i>	N	N	N	N	N	C
B37	N	N	N	N	N	N
B37 <i>Ht</i>	N	N	N	N	N	C
NN14B <i>Ht2</i>	C	C	C	C	C	C

^a Funk brand G4444 and G4444A*Ht*.

^b N = necrotic, susceptible-type lesion. C = chlorotic, resistant-type lesion.

and 204-79 from Wayne County. Isolate 204-79 was known to cause the chlorotic-lesion response on corn lines carrying the *Ht* gene.

Two kernels each of corn hybrids B73 × Mo17, B73*Ht* × Mo17*Ht*, Funk brand G4444, G4444A*Ht*, and inbreds A632, A632*Ht*, B37, B37*Ht*, and NN14B*Ht2* were planted in 10-cm-diameter pots containing autoclaved Wooster silt loam and peat moss (5:1, v/v). Plants were grown in the greenhouse at 16–28 C under supplemental light (1,050 lux). Four plants in the four to five leaf stage were inoculated by placing 1 ml of conidial suspension in the leaf whorl. Reactions of lines with and without the *Ht* gene were compared after 10–14 days. The experiment was then repeated.

RESULTS AND DISCUSSION

Inoculation tests demonstrated that isolates 46-80, 48-80, 50-80, 51-80, and 52-80 were virulent on corn lines with the *Ht* gene (Table 1). These isolates caused necrotic, susceptible-type lesions (2) on corn lines with and without the *Ht* resistance gene, and isolates could not be differentiated on the corn lines tested. Isolate 204-79 produced chlorotic lesions on plants with the *Ht* gene and necrotic lesions on plants without this gene. Inbred NN14B*Ht2*, homozygous for *Ht2*, responded with chlorotic lesions to all

isolates tested. Based on these tests, isolates 46-80, 48-80, 50-80, 51-80, and 52-80 were designated race 2 with a virulence formula *Ht2/Ht* (6). Isolate 204-79 was avirulent on lines carrying *Ht*; therefore, this isolate corresponded to race 1, with a virulence formula *Ht, Ht2/0* (6). None of the isolates were virulent on NN14B*Ht2*, and none were avirulent on lines carrying the *Ht* gene; therefore race 3 (7) has not as yet been found in Ohio (Table 1).

We believe that the occurrence of race 2 will probably increase due to the widespread use of the monogenic resistance gene *Ht* in commercial corn hybrids. The direct substitution of *Ht2* for *Ht* gene would give effective control of race 2, but it would also select for the buildup of race 3 (7). Smith and Kinsey (7) suggested that, since races exist that are virulent on genes *Ht*, *Ht2*, and *Ht3* singly, the most effective use of monogenic resistance would be to combine genes *Ht* and *Ht2* or *Ht* and *Ht3* in commercial hybrids. The most probable outcome of continued use of monogenic resistance will be selection for new races of *E. turcicum* and demand for a number of new genes for their control.

A significant attribute of monogenic resistance is the suppression of sporulation that reduces the production of secondary inoculum (2–5,9). Ullstrup (9) questioned

the usefulness of monogenic resistance when large amounts of inoculum are blown into an area. In a 3-yr test, the average yield of single crosses with polygenic resistance was 961 kg/ha more than single crosses carrying monogenic resistance (9). The lower yield of lines carrying monogenic resistance was attributed to extreme chlorosis expressed in the resistant-type lesions (9). The utilization of polygenic resistance has been limited by the difficulty of transferring multigenic resistance to inbreds without changing maturity, standability, harvest grain moisture, and other factors (5). Evidence reviewed here suggests that future research should be directed toward combining monogenic and polygenic resistance to control the occurrence and spread of *E. turcicum* (5,7).

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