Inheritance of Virulence in Helminthosporium turcicum to Monogenic Resistant Corn

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ABSTRACT

The progeny from a cross between isolates of *Helminthosporium turcicum* avirulent or virulent to monogenic (Ht_1) resistant corn segregated in a 1:1 ratio indicating that virulence in the pathogen is monogenic in inheritance. Corn gene Ht_2 conditioned resistance to both parental isolates. We propose that *H. turcicum* biotypes avirulent to corn carrying genes Ht_1 and Ht_2 be designated by the virulence formula $Ht_1Ht_2/0$ (formula number US1) and biotypes avirulent to corn carrying gene Ht_2 but virulent to corn carrying gene Ht_1 be designated by the virulence formula Ht_2/Ht_1 (formula number US2). The *Zea mays-H. turcicum* host-pathogen system fits the gene-for-gene model.

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Additional key words: Trichometasphaeria turcica, Zea mays L., chlorotic-lesion resistance, gene Ht_1, gene Ht_2, virulence formula, gene-for-gene model.

Northern leaf blight, caused by *Helminthosporium turcicum* Pass. (perfect stage: *Trichometasphaeria turcica* Luttrell), occurs sporadically in most temperate, humid areas where corn (*Zea mays* L.) is grown. It is known as one of the more serious leaf diseases in the USA Corn Belt and can cause yield losses of more than 50% (10).

H. turcicum has been isolated from various wild and cultivated species of the Gramineae, as well as from corn. The fungus is heterothallic and the perfect stage can be produced in culture by mating compatible isolates (7). Robles (9) reported races 1 and 2 of *H. turcicum* based on differential pathogenicity of the isolates to ten South American corn selections. However, the pathogenicity was determined quantitatively and no information on the genetic bases of resistance was given. A study (8) on pathogenic potential in *H. turcicum* showed that some ascospore progenies from crosses of highly pathogenic isolates were capable of producing large lesions on corn with polygenic resistance.

The chlorotic-lesion type of resistance in some sources is conditioned by a single dominant gene Ht_1 (3). Currently, another gene, Ht_2, is under investigation in our laboratory and is being incorporated into elite corn inbreds. Gene Ht_1 has conditioned resistance to a wide spectrum of *H. turcicum* isolates collected from 13 host species of the Gramineae in various areas throughout the world (4) and has conditioned resistance in disease nurseries in many areas of the world. Repeated host passage of a mixture of isolates did not change virulence (5). Gene Ht_1 is now used widely in USA and European corn hybrids. It conditions an effective degree of resistance to *H. turcicum* in the field. Recently, however, some isolates of *H. turcicum* from Hawaii have produced susceptible lesions on corn inbreds carrying gene Ht_1. This report describes some of our preliminary studies on monoascosporic progenies from crosses of *H. turcicum* isolates avirulent and virulent to corn plants carrying gene Ht_1.

All work was done in the laboratory and special precautions were taken to dispose of all cultures, equipment, and plant materials, since no *H. turcicum* isolates virulent to monogenic resistant plants have been found in Illinois. Fifteen monoconidial avirulent isolates of *H. turcicum* were obtained from naturally infected corn plants in central Illinois. A monoconidial virulent isolate was obtained from a lesion on a corn leaf collected in the Hawaiian Islands. Each of 15 avirulent isolates was mated with the virulent isolate. The techniques used to produce perithecia and isolate ascospore progeny have been described (6). The 15 crosses were incubated at 24 C for 21-25 days in the dark. Two crosses produced mature perithecia. Ninety-two monoascosporic cultures were obtained from one mating. The virulence or avirulence of the parental and monoascosporic isolates was determined by the lesion type produced on the nearly isogenic inbreds ROh43Ht_1 and Oh43. All plants were grown in the greenhouse to the 4- to 5-leaf stage before transfer to the laboratory. For inoculation, three or four small drops of conidial suspension, adjusted to 3,000-5,000/ml, were placed in each leaf whorl with small disposable pipettes. Plants were covered for 16 h with plastic bags which served as small moist chambers.

Seedling reactions based on lesion types were classified 12 days after inoculation. Forty-three ascospore isolates produced susceptible lesions on both inbreds like those caused by the virulent parental isolate. Forty-nine ascospore isolates produced resistant chlorotic-type lesions on inbred ROh43Ht_1 and susceptible wilt-type lesions on inbred Oh43 similar to those caused by the avirulent parental isolate. These data afford a good fit to a calculated 1:1 ratio (χ^2=0.39; P=0.70-0.50), indicating that virulence of *H. turcicum* to the monogenic (Ht_1) resistant corn is conditioned by a single gene in the fungus. All 16 parental isolates used in this study were avirulent to corn seedlings having gene Ht_2.

A determination of physiologic specialization is most precise when pathogen isolates are tested on host plants with known genes for disease reaction. This was done in our study. Furthermore, we have demonstrated the genetic difference between two kinds of biotypes on a qualitative basis. We propose that biotypes of *H. turcicum* avirulent to corn carrying genes Ht_1 and Ht_2 be designated by the virulence formula $Ht_1Ht_2/0$ (effective/ineffective genes; formula number US1) and biotypes avirulent to gene Ht_2, but virulent to gene Ht_1 be designated by the virulence formula Ht_2/Ht_1 (formula number US2) (2). Both biotypes are virulent to corn plants having neither gene Ht_1 nor Ht_2.

This host-pathogen system, *Z. mays-H. turcicum,* fits the gene-for-gene model (1). Resistance or susceptibility in corn is monogenic in inheritance, and virulence or avirulence in the pathogen is also monogenic in inheritance. These corresponding genes in the host and pathogen interact to condition disease reaction.

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