Relationship of Dominant Genes in Corn for Chlorotic Lesion Resistance to *Helminthosporium turcicum*

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ABSTRACT

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Dominant genes for chlorotic lesion resistance to *Helminthosporium turcicum* in each of 27 inbred lines of corn are at or closely linked to the *Ht* locus.

Additional key words: gene *Ht*, northern leaf blight, *Zea mays* L.

Chlorotic lesion resistance to northern leaf blight of corn (*Zea mays* L.), caused by *Helminthosporium turcicum* Pass., is often monogenic dominant in inheritance. Genes *Ht* (2) and *Ht2* (3) at unlinked loci have been identified. Inbred GE440 and the cultivar Ladyfinger popcorn each have gene *Ht* (2). An additional 29 inbred lines carry single dominant genes for resistance to *H. turcicum* (4). In Yugoslavia, lines BC9, BC10, and others have single dominant genes for resistance to this pathogen (5).

This paper reports data on the relationship of the genes in 27 lines to the *Ht* and *Ht2* loci.

MATERIALS AND METHODS

Twenty-two of the 27 monogenic resistant lines were crossed with inbreds that had gene *Ht* as a result of backcrossing. Lines BW and 713 were also crossed with inbred NN14B, which has gene *Ht2*. Sister inbred lines from the same cultivar and several resistant lines were intercrossed. The resulting resistant × resistant single crosses were advanced to the F_2 generation and crossed with a susceptible inbred or hybrid to produce test cross populations.

Seedling reactions and adult plant reactions of the F_2 and test cross populations were determined by inoculating plants with *H. turcicum*. The methods were described previously (1–3).

The chi-square test was used to test observed ratios in segregating populations for goodness of fit to expected ratios.

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RESULTS AND DISCUSSION

When intercrossed, sister lines to AWF, 167, 221, 231, 415, 700, 713, and 866, derived from different individual plants of each cultivar, did not segregate

Table 1. Plants resistant to *Helminthosporium turcicum* in F_2 populations[a]

Cross		Source of data[b]	Resistant plants (no.)
Resistant inbreds × inbreds with gene *Ht*			
MOL		G	460
011		G	116
BC9		F	112
BC10		F	95
BTU32		G	74
P35		G	143
W37A		F	90
W37A		G	94
Gro48		G	419
BZU158		F	83
221		F	203
461		F	87
EES647		F	72
EES650		F	91
EES650		G	221
713		F	80
713		G	113
866		G	119
899		G	104
Resistant lines			
P1	**P2**		
W37A	BTU32	F	69
W37A	NK75	F	130
W37A	415	F	93
W37A	415	G	124
W37A	866	F	87
EES647	BW	F	89
EES650	BW	F	71
EES650	BTU32	F	87
EES650	NK75	F	65
EES650	EES647	F	29
EES650	EES647	G	80
713	BZU158	F	62

[a] None of the plants were susceptible.
[b] F = field, G = greenhouse.

Table 2. Resistance to *Helminthosporium turcicum* in test cross populations

Cross		Source of data[a]	Resistant plants (no.)
Resistant inbreds × inbreds with gene *Ht*			
AWF		F	89
AWF		G	215[b]
BW		G	238
MOL		G	215
011		F	87
BC9		F	81
BC10		F	77
BTU32		G	236
P35		G	159
W37A		F	97
W37A		G	522
Gro48		G	334
081		G	196
BZU158		F	168
BZU158		G	209
167		F	173
167		G	186
221		F	169
231		G	217[c]
EES650		F	250
EES650		G	218[d]
700		G	196[c]
713		F	95
713		G	546
866		F	87
866		G	233
899		G	115
Resistant lines			
P1	**P2**		
W37A	BW	F	83
W37A	BW	G	273
W37A	BTU32	F	98
W37A	NK75	F	164[c]
W37A	415	F	98
W37A	415	G	130
W37A	866	G	201
W37A	NK51036	G	210[c]
EES647	BW	F	99
EES647	EES650	G	107
EES650	AWF	F	84
EES650	AWF	G	117
EES650	BW	F	90
EES650	BW	G	114
EES650	MOL	G	107
EES650	011	F	92
EES650	BTU32	F	103
EES650	TZU39	G	240
EES650	Gro48	F	78
EES650	Gro48	G	227
EES650	NK75	F	82
EES650	NK75	G	231
EES650	231	F	86
EES650	415	F	92
EES650	415	G	118
EES650	535a	G	233
EES650	700	G	115
EES650	713	F	86
EES650	713	G	214
EES650	866	G	227
EES650	899	G	120
EES650	NK51036	G	231
713	MOL	F	82
713	BZU158	F	260

[a] F = field, G = greenhouse.
[b] Two additional plants were susceptible.
[c] One additional plant was susceptible.
[d] Six additional plants were susceptible.

for chlorotic lesion resistance to *H. turcicum*. Therefore, we concluded that only one gene for chlorotic lesion resistance occurred in each cultivar.

When crossed with lines having gene *Ht* or when crossed with each other, the 27 resistant lines did not segregate for chlorotic lesion resistance in F_2 (Table 1) or test cross (Table 2) populations. Because all 27 resistant lines were included in these and other crosses (data not reported), the dominant genes for chlorotic lesion resistance to *H. turcicum* in each source included in this study must be identical, allelic, or closely linked to the known *Ht* locus. The results with sweet corn lines 535a, EES647, and EES650 confirm the conclusions of Wilson and Rhodes (6).

Ten F_2 populations or test crosses derived from crossing BW and 713 with NN14B, having gene *Ht2*, segregated in a ratio of 15:1 resistant/susceptible in the F_2 generation and 3:1 resistant/susceptible in test cross populations ($P = 0.30$–0.98). These data further confirm that NN14B carries a gene for chlorotic lesion resistance to *H. turcicum* that segregates independently from the *Ht* locus (3).

A few plants in the F_2 or test cross populations were classified as susceptible. These were usually in seedling tests and are believed to be the result of misclassification of small plants rather than the result of segregation. Seed mixtures were also possible. Where apparent segregation was observed for any line, segregation was not confirmed in the field, in crosses with other lines, or in test crosses involving another susceptible line or hybrid.

Based on two kinds of evidence, we believe that the alleles at the *Ht* locus in some lines differ from those in other lines. In several F_2 and test cross populations, two classes of resistant plants were observed. The observed ratios agreed with ratios expected for two alleles at the same locus. Secondly, when the genes from the different lines were incorporated by backcrossing into susceptible inbreds, the degrees of resistance also were different. Lesions on plants with genes from 011, NK75, BZU158, and NK51036 enlarged rapidly and had large necrotic centers with narrow chlorotic margins. In contrast, lesions on plants with genes from BW, MOL, BTU32, W37A, Gro48, 535a, 700, and 713 enlarged slowly and had smaller necrotic centers with considerable chlorosis at the margins. The latter group of lines are believed to be more suitable sources of resistance for breeding programs and commercial hybrids. Because the dominant genes for chlorotic lesion resistance to *H. turcicum* are present in different endosperm types, sweet corn and popcorn breeders do not need to use dent corn as a source of resistance.

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