Effects of the *Ht*, *Ht*2, and/or *Ht*3 Genes in Three Maize Inbreds on Quantitative Resistance to *Exserohilum turcicum* Race 2

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ABSTRACT

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Three nonreciprocal diallel sets, each composed of the recurrent parent (A619, B37, or Pa91) and three nearly isogenic lines, each carrying the *Ht*, *Ht*2, or *Ht*3 gene, were evaluated for quantitative resistance to *Exserohilim turcicum* race 2 in 1983 and 1984. Parents and crosses from the A619 diallel were not significantly ($P = 0.05$) different from A619 for lesion number or lesion length; however, the parent and crosses carrying the *Ht*2 gene had significantly lower area under disease progress curve (AUDPC) values than A619. The parent and crosses from the B37 diallel carrying the *Ht*3 gene had significantly fewer lesions, shorter lesions, and lower AUDPC values than B37. Parents and crosses from the Pa91 diallel did not have significantly smaller lesions or lower AUDPC values than Pa91.

Northern leaf blight, caused by *Exserohilum turcicum* (Pass.) Leonard & Suggs (teleomorph: *Setosphaeria turcica* (Luttrell) Leonard & Suggs), is one of the most prevalent foliar diseases of maize (*Zea mays* L.) in the midwestern United States. Three dominant genes, which condition chlorotic-type lesion resistance to *E. turcicum* (4,5), and three races of the pathogen (1,9,13) have been identified.

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The *Ht* gene conditions resistance to races 1 and 3 but not to race 2, whereas the *Ht*2 and *Ht*3 genes condition resistance to races 1 and 2 but not to race 3. Sporulation on chlorotic-type lesions is suppressed, thereby reducing secondary spread of the pathogen. Plants carrying the *Ht*2 or *Ht*3 gene produce chlorotic-type lesions when inoculated with an avirulent race of *E. turcicum*, but the chlorosis can be extensive and on some inbreds the chlorosis may extend the entire length of a leaf (6). The *Ht*2 and *Ht*3 genes have been incorporated, via backcrossing, into five public inbreds (W. L. Pedersen, *unpublished*); however, their level of quantitative resistance to *E. turcicum* race 2 has not been determined.

Nelson (8) suggested combining "defeated" or ineffective resistance genes into a single cultivar to enhance "rate-reducing" resistance. Leath and Pedersen (7) observed shorter lesions and lower area under disease progress curve (AUDPC) values for three maize hybrids carrying the *Ht* gene than for the corresponding hybrids without the *Ht* gene when inoculated with *E. turcicum* race 2. If the *Ht* gene functions quantitatively against *E. turcicum* race 2, maize lines carrying the *Ht*2 or *Ht*3 gene and the *Ht* gene should have higher levels of quantitative resistance than lines with only the *Ht*2 or *Ht*3 gene.

The objectives of this study were to evaluate three maize inbreds carrying the *Ht*2 or *Ht*3 gene for quantitative resistance to *E. turcicum* race 2 and to determine if combining the *Ht* gene with either the *Ht*2 or *Ht*3 gene increases the level of quantitative resistance to *E. turcicum* race 2.

MATERIALS AND METHODS

Three nonreciprocal diallel sets, each composed of a recurrent parent (A619, B37, or Pa91) and three nearly isogenic lines (isolines), were made in 1982. The isolines contained either the *Ht*, *Ht*2, or *Ht*3 gene. Therefore, each set included the four parent isolines (P) and six crosses [number of crosses = $P(P - 1)/2$]. The recurrent parents (A619, B37, and Pa91) and crosses with isolines carrying only the *Ht* gene were susceptible to *E. turcicum* race 2, whereas parents and crosses carrying the *Ht*2 and/or *Ht*3 genes were resistant.

Field plots consisted of three rows 4 m long and 0.76 m apart with 13 plants per row. Plots were planted on 11 May in 1983 and 1984 at the Agronomy-Plant Pathology South Farm, Urbana, IL. The

Table 1. Mean lesion number, lesion length, and AUDPC[a] values for crosses from the A619 diallel in 1984

Evaluation component	Genotype	*ht1ht2ht3*	*Ht1ht2ht3*	*ht1Ht2ht3*	*ht1ht2Ht3*
Lesion number[b]	*ht1ht2ht3*	21.5[c]	27.0	17.0	15.5
	Ht1ht2ht3	···	24.4	14.2	15.7
	ht1Ht2ht3	···	···	11.4	12.9
	ht1ht2Ht3	···	···	···	13.6
LSD (0.05) = 11.6					
Lesion length[b] (mm)	*ht1ht2ht3*	80.9	94.2	87.4	72.6
	Ht1ht2ht3	···	87.3	91.8	68.0
	ht1Ht2ht3	···	···	97.6	104.4
	ht1ht2Ht3	···	···	···	64.0
LSD (0.05) = 23.1					
AUDPC[d]	*ht1ht2ht3*	127.4	144.2	100.8	109.2
	Ht1ht2ht3	···	124.9	86.5	103.2
	ht1Ht2ht3	···	···	77.8	78.6
	ht1ht2Ht3	···	···	···	104.2
LSD (0.05) = 24.4					

[a] Area under disease progress curve.
[b] Means of four replicates and five subsamples.
[c] The bottom numbers in columns are values from parents of a diallel cross.
[d] Means of four replicates.

Table 2. Mean lesion number, lesion length, and AUDPC[a] values for crosses from the B37 diallel in 1983 and 1984

Evaluation component	Genotype	*ht1ht2ht3*	*Ht1ht2ht3*	*ht1Ht2ht3*	*ht1ht2Ht3*
Lesion number[b]	*ht1ht2ht3*	9.2[c]	7.9	10.5	2.8
	Ht1ht2ht3	···	8.4	7.2	3.2
	ht1Ht2ht3	···	···	10.6	2.2
	ht1ht2Ht3	···	···	···	0.9
LSD (0.05) = 3.9					
Lesion length[b] (mm)	*ht1ht2ht3*	78.2	67.8	59.7	44.0
	Ht2ht2ht3	···	81.7	56.1	34.3
	ht1Ht2ht3	···	···	68.5	34.8
	ht1ht2Ht3	···	···	···	17.8
LSD (0.05) = 25.7					
AUDPC[d]	*ht1ht2ht3*	88.6	70.6	59.1	46.5
	Ht1ht2ht3	···	71.6	70.1	31.0
	ht1Ht2ht3	···	···	99.4	25.4
	ht1ht2Ht3	···	···	···	17.6
LSD (0.05) = 25.7					

[a] Area under disease progress curve.
[b] Means of four replicates and five subsamples.
[c] The bottom numbers in columns are values from parents of a diallel cross.
[d] Means of four replicates.

Table 3. Mean lesion number, lesion length, and AUDPC[a] values for crosses from the PA91 diallel in 1983 and 1984

Evaluation component	Genotype	*ht1ht2ht3*	*Ht1ht2ht3*	*ht1Ht2ht3*	*ht1ht2Ht3*
Lesion length[b] (mm)	*ht1ht2ht3*	47.6[c]	54.9	47.1	48.9
	Ht1ht2ht3	···	40.9	45.4	52.6
	ht1Ht2ht3	···	···	65.8	44.7
	ht1ht2Ht3	···	···	···	39.8
LSD (0.05) = ns					
AUDPC[d]	*ht1ht2ht3*	26.8	27.1	23.9	28.7
	Ht1ht2ht3	···	19.5	23.8	36.9
	ht1Ht2ht3	···	···	21.6	19.1
	ht1ht2Ht3	···	···	···	24.0
LSD (0.05) = 10.1					

[a] Area under disease progress curve.
[b] Means of four replicates and five subsamples.
[c] The bottom numbers in columns are values from parents of a diallel cross.
[d] Means of four replicates.

crosses from each diallel were arranged in randomized complete block designs with four replicates.

Inoculum was prepared by flooding 2-wk-old cultures of *E. turcicum* race 2 growing on lactose-casein-hydrolysate agar (12) with distilled water, scraping with a rubber policeman, and diluting with distilled water. Plants were inoculated at growth stage 5 (3), and inoculum was applied with a boom sprayer at 1,200 L/ha with a conidial concentration of about 2,000/ml. Conidial viability was >90% as determined by dilution plating on lactose-casein-hydrolysate agar and counting the germinated conidia after 18 hr.

The number of lesions on all leaves of five consecutive plants in the center row of each plot (lesion number) and the length of five random primary lesions per plot were recorded 17 days after inoculation. Blight ratings were initiated 1 wk before midsilking and continued weekly for five rating dates. Blight ratings were based on visual estimates of the total percentage of leaf tissue blighted in the center row of each plot. When estimates of leaf blight severity were made, both necrotic and highly chlorotic lesions were included; however, only necrotic areas were measured for estimates of lesion length. AUDPC values were calculated as described by Tooley and Grau (11), except time (T_i) was expressed in weeks after planting.

Data for lesion number, lesion length, and AUDPC were combined over years (1983 and 1984), and a combined analysis of variance was performed. Comparisons among the crosses within each diallel were made using Fisher's least significant difference, $P = 0.05$ (10).

RESULTS

Parents and crosses carrying the *Ht2* and/or *Ht3* genes from the three diallel sets had chlorotic-type lesions, whereas other parents and crosses had necrotic (no chlorosis) lesions when inoculated with *E. turcicum* race 2. All parents and crosses from the A619 diallel lacked vigor and were extremely chlorotic in 1983. Because necrotic and chlorotic lesions were difficult to differenciate from general chlorosis, data from the A619 diallel in 1983 were not included. Also, because of the extreme chlorosis or "bleeding" (6) associated with the *Ht* genes in Pa91 when infected with *E. turcicum*, it was not possible to accurately assess lesion number and those data are not presented.

Parents and crosses carrying the *Ht2* and/or *Ht3* genes from the A619 diallel were not significantly ($P = 0.05$) different from the recurrent parent for lesion number or lesion length (Table 1). However, the parents and crosses carrying the *Ht2* gene had significantly lower AUDPC values than A619.

The parents and crosses from the B37 diallel carrying the *Ht*3 gene had significant ($P = 0.05$) reductions in lesion number, lesion length, and AUDPC values compared with B37 (Table 2). Both lesion length and AUDPC were significantly reduced for the parent isoline (B37*Ht*3*Ht*3) compared with the heterozygous cross (B37*Ht*3*ht*3). There also was a significant difference between the parent B37 isoline carrying the *Ht*2 gene and the heterozygous cross for AUDPC.

No significant differences were observed between the recurrent parent Pa91 and other parents or crosses in the Pa91 diallel for lesion length or AUDPC (Table 3).

DISCUSSION

The first objective of this study was to evaluate the level of resistance to *E. turcicum* provided by the *Ht*2 or *Ht*3 gene in three maize inbreds. Although chlorotic-type (resistant) lesions were produced on all parents and crosses carrying the *Ht*2 or *Ht*3 gene from the three diallel sets, only the parent and crosses carrying the *Ht*2 gene from the A619 diallel and the parent and crosses carrying the *Ht*3 gene from the B37 diallel had significantly lower AUDPC values than their recurrent parent, A619 or B37. Parents and crosses from the three diallel sets carrying the *Ht*2 and/or *Ht* 3 genes, except the parent and crosses carrying the *Ht*3 gene from the B37 diallel, had severe chlorosis associated with the resistant lesions. Therefore, sporulation of primary lesions and secondary spread of the pathogen may have been reduced, but the extreme chlorosis associated with the lesions accounted for the high AUDPC values for the resistant inbreds. Some chlorosis also occurred in the Pa91 diallel; however, the resistant and susceptible lesions were very small and secondary spread from primary lesions was low so AUDPC values were very low. A gene dosage effect was observed for lesion length and AUDPC for B37*Ht*3*Ht*3 vs. B37*Ht*3*ht*3. Gene dosage effects have been reported for the *Ht* gene (2) and the *Ht*2 gene (6).

The second objective was to determine if combining the *Ht* gene with either the *Ht*2 or *Ht*3 gene would increase the level of quantitative resistance to *E. turcicum* race 2. Crosses carrying the *Ht* and *Ht*2 genes or *Ht* and *Ht*3 genes from all three diallel sets were not significantly different from corresponding parents (homozygous for the *Ht*2 or *Ht*3 gene) or crosses (heterozygous for the *Ht*2 or *Ht*3 gene) for the parameters evaluated. Therefore, no evidence of "residual gene resistance" (8) associated with the *Ht* gene was observed in this study. However, combining the *Ht* gene with the *Ht*2 or *Ht*3 gene would provide resistance to all three races of *E. turcicum* as suggested by Smith and Kinsey (9).

LITERATURE CITED

1. Bergquist, R. R., and Masias, O. R. 1974. Physiologic specialization in *Trichometasphaeria turcica* f. sp. *zeae* and *T. turcica* f. sp. *sorghi* in Hawaii. Phytopathology 64:645-649.
2. Dunn, G. M., and Namm, T. 1970. Gene dosage effects on monogenic resistance to northern corn leaf blight. Crop Sci. 10:352-354.
3. Hanway, J. J. 1963. Growth stages of corn (*Zea mays* L.). Agric. J. 55:487-492.
4. Hooker, A. L. 1977. A second major gene locus in corn for chlorotic lesion resistance to *Helminthosporium turcicum*. Crop Sci. 17:132-135.
5. Hooker, A. L., Nelson, R. R., and Hilu, H. M. 1965. Avirulence of *Helminthosporium turcicum* on monogenic resistant corn. Phytopathology 55:462-463.
6. Hooker, A. L., and Perkins, J. M. 1980. Helminthosporium leaf blights of corn—the state of the art. Proc. Annu. Corn Sorghum Res. Conf. 35:68-87.
7. Leath, S., and Pedersen, W. L. 1984. Evaluation of the residual effects of the Ht_1 gene with respect to *Exserohilum turcicum* race 2 in maize inbreds and hybrids. (Abstr.) Phytopathology 74:818.
8. Nelson, R. R. 1978. Genetics of horizontal resistance to plant diseases. Annu. Rev. Phytopathol. 16:359-378.
9. Smith, D. R., and Kinsey, J. G. 1980. Further physiologic specialization in *Helminthosporium turcicum*. Plant Dis. 64:779-781.
10. Steel, R. G. D., and Torrie, J. H. 1960. Principles and Procedures of Statistics. McGraw-Hill, New York. 481 pp.
11. Tooley, P. W., and Grau, C. R. 1984. Field characterization of rate-reducing resistance to *Phytophthora megasperma* f. sp. *glycines* in soybeans. Phytopathology 74:1201-1208.
12. Tuite, J. 1969. Plant Pathological Methods. Burgess Publishing, Minneapolis, MN. 239 pp.
13. Turner, M. T., and Johnson, E. R. 1980. Race of *Helminthosporium turcicum* not controlled by *Ht* genetic resistance in the American corn belt. Plant Dis. 64:216-217.