

Single and Combined Effects of Monogenic and Polygenic Resistance on Certain Components of Northern Corn Leaf Blight Development

A. D. Raymundo and A. L. Hooker

Former graduate research assistant and professor, respectively, Department of Plant Pathology, University of Illinois, Urbana 61801.
Present address of senior author: Plant Pathology Laboratory, Institute of Plant Breeding, University of the Philippines at Los Baños,

College, Laguna, Philippines 3720.
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ABSTRACT

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The dominant genes *HtA*, *HtC*, and *HtD* singly or in combination with high, intermediate, and low polygenic resistance were studied to determine their effects on incubation period, latent period, lesion size, infection efficiency, sporulation capacity, and infection rate of *Helminthosporium turcicum* causing northern corn leaf blight. Gene *HtC* restricted the enlargement of lesions significantly, regardless of genetic background. Gene *HtD* had the same effect, although not as marked as that of *HtC* except when the inbred background had a high polygenic resistance. Gene *HtC* was as effective as *HtA* in limiting lesion size in inbred backgrounds of high polygenic resistance. Lesion size restriction by gene *HtA* homozygous in Oh43 × B37 was not significantly different than when it was heterozygous in

A632 × A619. In terms of infection rate, *HtA* homozygous in Oh43 × B37 was considerably more effective in inhibiting disease development than when it was heterozygous in A632 × A619. In terms of infection rate, *HtC* differed significantly from *HtD* in inbred backgrounds with moderate polygenic resistance, but not in backgrounds with high polygenic resistance. Genes *HtC* and *HtD* in combination with high polygenic resistance were as highly effective in limiting disease development as when homozygous *HtA* in Oh43 × B37. Gene *HtC* was better than gene *HtD* in suppressing sporulation when present in inbreds having intermediate polygenic resistance. Gene *HtC* was as effective as gene *HtA* in reducing sporulation.

Additional key words: epidemiology, *Helminthosporium turcicum*.

Several types of resistance to *Helminthosporium turcicum* Pass. are known in corn (*Zea mays* L.) (6). Resistance conferred by many genes occurs in various types of germ plasm (13) and commercial hybrids. Single dominant genes are present in many genotypes (7,8) and gene *Ht* is now extensively used in U.S. hybrids.

Polygenic resistance affects the development of epidemics by reducing the number and size of lesions. Quantitatively, it ranges from high (in which few lesions form) to low (in which many large sporulating lesions are present) (1,12).

Monogenic resistance, determined by the dominant gene *Ht*, is a unique type (3-5). It is characterized by the formation of small chlorotic lesions, a delay in appearance of necrosis, and a marked reduction in sporulation. Hilu and Hooker (2) reported that sporulation was delayed 50-80 hr on resistant-type lesions and spore density per unit area was reduced 60-fold compared with susceptible-type lesions. Chlorotic lesions averaged one-fourth the size of susceptible-type lesions. These characteristics decrease the probability of a destructive epidemic.

Gene *Ht* has been incorporated into inbred lines possessing polygenic resistance (6). In these lines, the degree of resistance expressed either as lesion number or lesion type is enhanced. Additional sources of the chlorotic-lesion resistance have been identified and the genes for resistance have been introduced by backcross breeding into a large number of inbred lines (11). Plants with genes from certain sources have small chlorotic lesions with little necrosis while plants with genes from other sources have lesions that are larger and with a greater area necrotic. A number of these genotypes were utilized in this study to determine the effect of various *Ht* genes in different inbred backgrounds on certain epidemic components of northern corn leaf blight.

MATERIALS AND METHODS

Experiments were conducted with 10 genotypes in field plots at the Agronomy South Farm of the Illinois Agricultural Experiment Station, Urbana, during the summers of 1975 and 1976 and in the greenhouse and laboratory during the winter of 1976. Four single-cross hybrids, A632 × A619, A632 × A619HtA, Oh43 × B37, and Oh43HtA × B37HtA formed one group. Polygenic resistance is expressed on a low level by inbreds A632 × A619 and on an intermediate level by Oh43. The other group consisted of selections of B14A, B37, and Mo17 each having gene *HtC* from Btu-32, which conditions small chlorotic lesions, and gene *HtD* from P1031, which conditions large chlorotic lesions with a considerable degree of necrosis. Genes *HtC* and *HtD* are either identical, allelic, or closely linked to *HtA* derived from inbred GE440 (9,10). Inbreds B14A, B37, and Mo17 have low, intermediate, and high degrees of polygenic resistance, respectively.

Field experiments. These were replicated four times in a split-plot arrangement of a randomized complete block design in which the genotypes were whole plots and inoculum dosages were subplots. Each block consisted of 10 rows representing the genotypes. Each row contained 12 two-plant hills spaced every 38 cm in the row. The 24 plants in each row were divided into three groups of eight. Plants were inoculated with spore suspensions of *H. turcicum* race 1 at the rate of 20,000, 10,000, and 5,000 conidia per milliliter for groups 1, 2, and 3, respectively. Seedlings were inoculated 1 mo after emergence by inoculum applied with a compressed-air sprayer until runoff commenced. Inoculum was obtained from sporulating corn leaf tissue incubated above moist paper in covered petri dishes for 5 days. Inoculation was made at about 1900 hours when temperature had decreased and moisture to insure infection by *H. turcicum* was more likely to form on leaf surfaces.

Several epidemic parameters were measured. The incubation period (the time between inoculation and first appearance of

symptoms) was recorded for each genotype. Size of individual lesions on each genotype was measured at eight intervals starting when lesions had formed. Lesions on the ear-leaf or on the leaf immediately below were marked with a waterproof pen and the lengths and widths measured every 2 days, except when weather did not permit, for a 15-day period. Growth of the sporulating zone of lesions was similarly measured in A632 × A619 and Oh43 × B37.

When symptoms were well-defined, the number of primary lesions on each plant was counted to estimate infection efficiency. The percentage leaf area infected, on a row basis, was estimated from visual observation at four weekly intervals when secondary infection had occurred.

Greenhouse and laboratory experiments. Latent period and sporulation capacity of *H. turcicum* were determined on greenhouse-grown plants. The 10 genotypes under study were grown in 15-cm-diameter pots and sprayed until runoff with a spore suspension containing 5,000 conidia per milliliter 1 mo after emergence. Only a few separated lesions were produced with this inoculum concentration. Two weeks after inoculation, a single diseased plant of each genotype was placed in a mist chamber overnight. Six 1-mo-old seedlings of A632 × A619 were carefully placed under the infected plants at 1600 hours to catch any spores being dispersed. These trap plants were taken out of the mist chamber at 0800 hours the following day and placed on a greenhouse bench. This exposure of a new set of uninfected seedlings under the sporulating plants was repeated every other day for the next 12 days. The number of lesions that formed on the trap plants was counted 12 days after exposure and the occurrence and number taken as indications of latent period and sporulation efficiency.

Sporulation intensity was also assessed from lesion segments incubated at high humidity. Disks cut with a 5-mm-diameter cork borer at the margin of lesions were incubated in petri dishes lined with moist cellulose pads. Four disks from each genotype were transferred singly at 24-hr intervals from 24 to 168 hr to depression slides containing 0.5 ml Tween-20 and agitated to wash off spores that had formed. Conidia were counted directly from five ×100 microscopic fields in each of four depression slides.

The effect of incubation time at high humidity and stage of plant

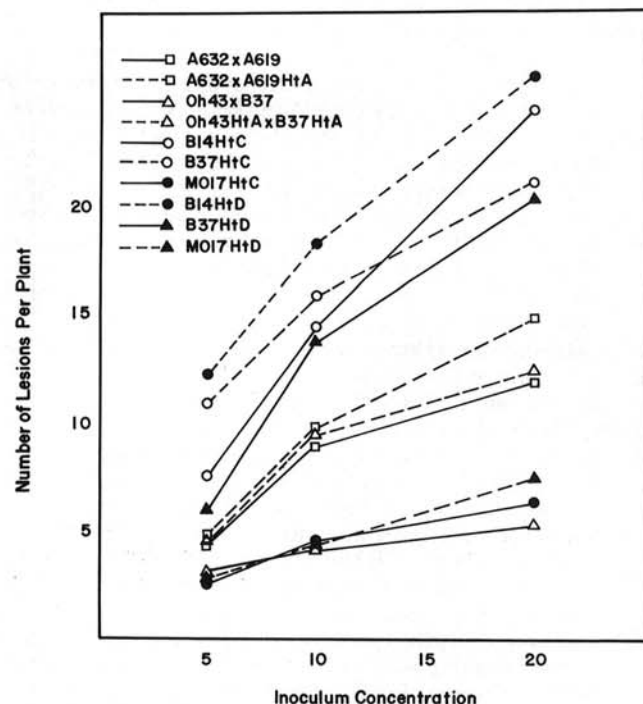


Fig. 1. Number of northern corn leaf blight lesions produced on individual plants of 10 corn genotypes inoculated with *Helminthosporium turcicum* at three inoculum concentrations (5, 10, and 20 × 10³ spores per milliliter) in 1976.

growth on blight development was also studied in the greenhouse. One-month-old seedlings of the 10 genotypes planted in flats were sprayed until runoff with a spore suspension of 5,000 conidia per milliliter and placed in a mist chamber. After 2, 4, 8, 12, and 16 hr under high humidity, the flats containing six seedlings of each genotype were removed and put on a greenhouse bench. Lesions that formed were counted 12 days after inoculation. Three-, 4-, and 5-wk-old seedlings of each genotype, grown in 15-cm-diameter pots, were likewise inoculated with 5,000 spores per milliliter, incubated in a mist chamber for 16 hr, and then placed on a greenhouse bench. Similarly, the resulting lesions were counted 12 days after inoculation.

Data analysis. All data were subjected to analysis of variance. Greenhouse observations on sporulation intensity of *H. turcicum* by using trap plants, effect of incubation time at high humidity, and effect of plant age on number of blight lesions were analyzed as completely randomized design experiments. Field data on lesion number were analyzed as factorial experiments, but other field studies were treated as randomized complete block designs. In addition, a simple linear regression analysis was performed on all data except on lesion number and percentage leaf area infected. The equation, $r = 1 / (t_2 - t_1) (\log_e x_2 / (1 - x_2) - \log_e x_1 / (1 - x_1))$ (14) was used to calculate the infection rate in the case of percentage leaf area infected.

RESULTS

Field experiments. The average incubation period for *H. turcicum* infection was 3 days with the inbreds having gene *HtD*, 4 days with the inbreds having gene *HtC* and the hybrids having gene *HtA*, 7 days with A632 × A619, and 9 days with Oh43 × B37. Generally, more lesions resulted from higher than from lower inoculum concentrations (Fig. 1). In 1975, differences among inoculum dosages were significant except in the case of Oh43 × B37 and of M017HtC when dosages of 5,000 and 10,000 spores per milliliter were compared. At concentrations between 10,000 and 20,000 spores per milliliter, nonsignificant differences were obtained with Oh43HtA × B37HtA, M017HtC, and all genotypes with the gene *HtD*. In 1976, comparisons among all three dosages showed significant differences.

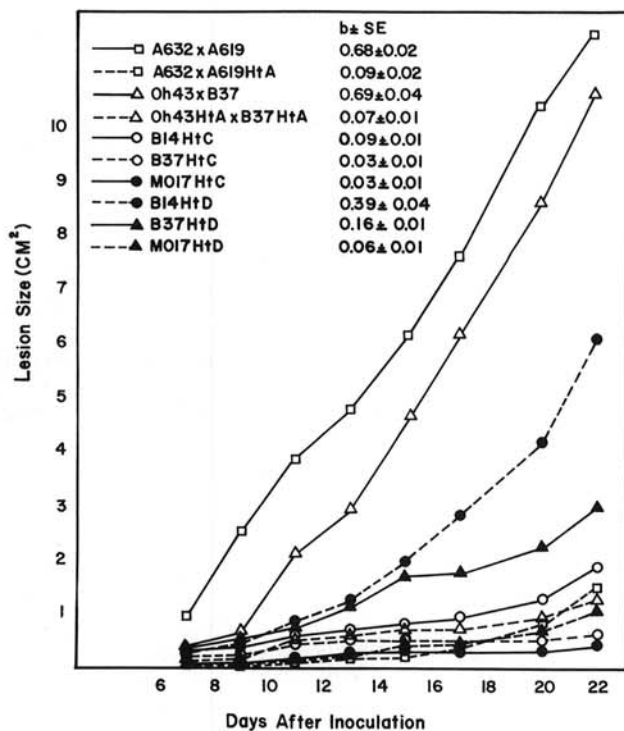


Fig. 2. Size of northern corn leaf blight lesions on 10 corn genotypes at eight intervals after inoculation with *Helminthosporium turcicum* spores in 1975.

Number of lesions differed significantly on plants of various genotypes (Fig. 1). Genotypes B14A and B37 with either the *HtC* or *HtD* gene had the greatest number of lesions. In both years, Mo17 with gene *HtC* or *HtD* had significantly fewer lesions compared to the other genotypes. Within the same inbred background, lesion number on plants having the gene *HtC* and *HtD* was not significantly different. The number of lesions on A632 × A619 was not significantly different from its isogenic counterpart with *HtA*, but Oh43 × B37 had significantly more lesions than Oh43HtA × B37HtA.

Among groups of genotypes, significant differences were observed in lesion size at different days after inoculation (Fig. 2). The two hybrids without *HtA* had distinctly larger lesions compared to all other genotypes. Lesions on the hybrids containing the gene *HtA* were not significantly different from each other, even though one hybrid was heterozygous and the other homozygous for *HtA*. Gene *HtC* from Btu-32 restricted the development of lesions significantly, regardless of genetic background. The same can be said for gene *HtD* from P1031, although its effect was not as marked as *HtC* except with inbred Mo17HtD. Lesions on Mo17HtD, with a high degree of polygenic resistance, were similar in size to lesions on B14AHtC and B37HtC, both of which have lower degrees of polygenic resistance. However, lesions on plants having the gene *HtD* were sporulating. Comparing B14A, B37, and Mo17 having genes *HtC* and *HtD*, only B14A had significantly larger lesions, presumably due to its low level of polygenic resistance. Gene *HtC* was as effective as gene *HtA* in limiting lesion size when the inbred background had in addition a high degree of polygenic resistance as does Mo17HtD. Lesion size was not significantly correlated with lesion number. Combined analysis of data showed a nonsignificant genotype × year interaction indicating that the effect of genotypes in both years was similar.

Lesions followed a definite expansion pattern (Fig. 2). Over the eight 2-day intervals of measurement, lesion size increase was rapid in the case of the hybrids A632 × A619, Oh43 × B37, and in B14AHtD and B37HtD. This can be seen from the slope coefficient *b*, which is a measure of the relative rate of lesion expansion among genotypes. In both years, correlations between size of lesion and rate of expansion were highly significant.

Size of sporulating zone differed between hybrids (Fig. 3). In 1975, significant differences in size of sporulating zones were observed between A632 × A619 and Oh43 × B37 at the early, but

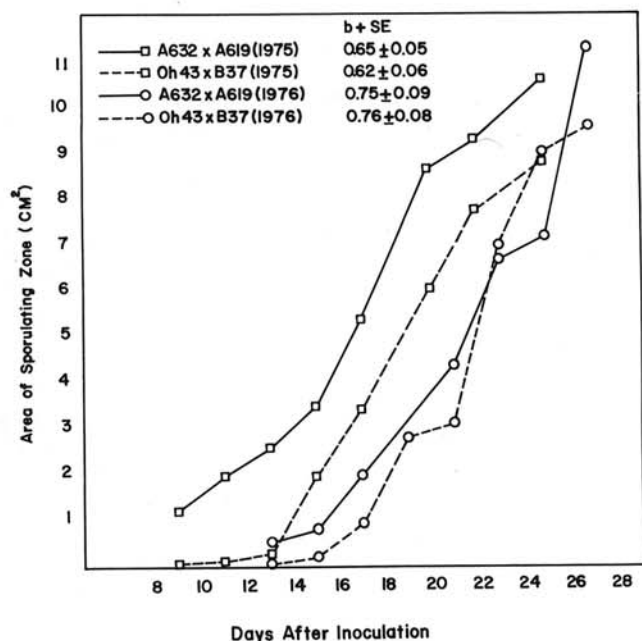


Fig. 3. Area of sporulating zones of northern corn leaf blight lesions on two corn hybrids at eight intervals after inoculation with *Helminthosporium turcicum* spores in 1975 and 1976.

not at the later, dates of measurement. In 1976, differences in size were noted at both the first and last dates of observation.

Infection rates (*r*), calculated from four weekly disease ratings, were low (Fig. 4). Genotype differences in *r* values, however, occurred especially in 1976. Significant differences in percentage leaf area infected were obtained among genotypes at several intervals after inoculation in 1975 and 1976. The hybrid Oh43 × B37 was more effective than A632 × A619 in suppressing disease development. Fewer lesions were observed in the former hybrid (Fig. 1). Homozygous *HtA* in the hybrid Oh43 × B37 and heterozygous *HtA* in the hybrid A632 × A619 were considerably better than their isogenic counterparts in inhibiting disease progress. Gene *HtC* differed significantly from gene *HtD* with inbred backgrounds B14A and B37, but not with Mo17. Genes *HtC* and *HtD*, in combination with Mo17 possessing a high degree of polygenic resistance, were as effective as homozygous *HtA* in limiting disease development. At the later stage of the epidemic, the effects of the three inoculum dosages were impossible to distinguish since secondary infection had already occurred.

Greenhouse and laboratory experiments. Differences in latent period and sporulation intensity were observed among greenhouse-grown plants having different genotypes (Table 1). Latent period, the time from lesion formation to the onset of sporulation, was delayed by 2 days and 1 day in genotypes with the genes *HtA*, *HtC*, and *HtD* when hybrids A632 × A619 and Oh43 × B37, respectively, were taken as points of reference. Sporulation intensity, measured by the number of lesions that formed on trap plants, varied significantly among plant genotypes at each of the seven intervals when spores were trapped (Table 1). Sporulation was substantially reduced by the *Ht* genes and *HtC* was more effective than *HtD* in this respect. Sporulation reduction was greater in Mo17 than in B14A when both contained the same *Ht* gene. Considerable differences in sporulation were observed among disks cut from lesions on plants of the ten genotypes incubated at high humidity for 24–168 hr (Table 2). Sporulation was abundant on lesions of

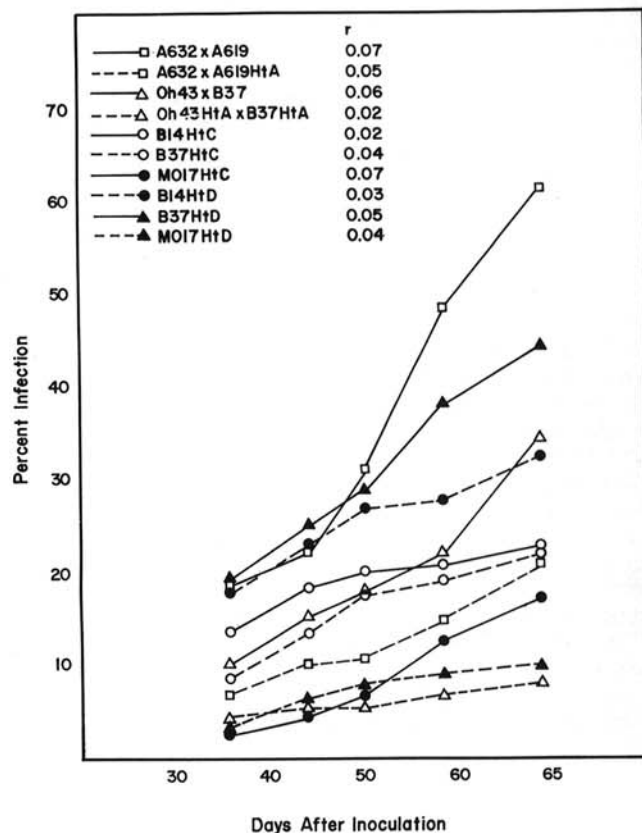


Fig. 4. Percentage of leaf area infected and apparent infection rates (*r*) on 10 corn genotypes at five intervals after inoculation with *Helminthosporium turcicum* spores in 1976.

TABLE 1. Sporulation of *Helminthosporium turcicum* on 10 corn genotypes as indicated by the number of lesions produced on trap plants (A632 × A619) exposed at seven intervals after inoculation of inoculum source plants

Genotype (inoculum sources)	Number of lesions produced on trap plants ^a by inoculum collected at intervals (days) after inoculation of inoculum source plants							<i>r</i> ^b	<i>b</i> ^c	SE ^d
	10	12	14	16	18	20	22			
A632 × A619	0.67	8.67	38.67	45.00	48.83	55.67	64.83	0.95	5.29	±0.359
A632 × A619HtA	0.00	0.00	4.50	13.00	14.17	21.83	28.50	0.97	2.47	±0.192
Oh43 × B37	0.00	0.67	13.67	26.00	33.50	35.67	35.67	0.94	3.40	±0.259
Oh43HtA × B37HtA	0.00	0.00	0.33	2.67	4.83	1.17	1.33	0.47	0.19	±0.040
B14AHtC	0.00	0.00	0.83	1.00	6.17	18.00	25.17	0.88	2.08	±0.208
B37HtC	0.00	0.00	0.67	2.00	4.17	5.67	6.17	0.97	0.59	±0.048
Mo17HtC	0.00	0.00	1.00	2.67	3.17	1.50	2.67	0.77	0.23	±0.025
B14AHtD	0.00	0.00	0.83	20.67	32.00	35.61	36.67	0.94	3.79	±0.315
B37HtD	0.00	0.00	3.67	6.50	23.67	37.33	35.83	0.93	3.60	±0.314
Mo17HtD	0.00	0.00	0.67	5.00	3.83	5.00	23.50	0.77	1.49	±0.178
FLSD ^e (<i>P</i> = 0.05)	0.44	1.99	3.47	4.44	5.80	5.65	7.60			

^a Means of observations on six replicate plants.

^b Correlation coefficient of lesion number with days; all values are significant, *P* = 0.01.

^c Regression coefficient of lesion number on days.

^d Standard error of regression coefficient.

^e Fisher's Least Significant Difference.

TABLE 2. Sporulation of *Helminthosporium turcicum* on lesion segments from 10 corn genotypes incubated at high humidity

Genotype	Number of conidia ^a produced on lesion segments after intervals (hours) at high humidity							<i>r</i> ^b	<i>b</i> ^c	SE ^d
	24	48	72	96	120	144	168			
A632 × A619	23.50	208.00	425.00	426.00	661.50	788.00	1185.50	0.97	7.26	±0.454
A632 × A619HtA	0.00	0.00	0.00	4.50	26.75	50.00	78.00	0.90	0.53	±0.074
Oh43 × B37	20.00	77.25	86.00	131.50	181.25	588.00	873.75	0.87	0.32	±0.059
Oh43HtA × B37HtA	0.00	0.00	0.00	2.00	7.25	21.25	56.75	0.80	0.32	±0.059
B14AHtC	2.00	1.00	0.25	10.75	19.75	95.00	108.25	0.86	0.78	±0.199
B37HtC	0.00	0.00	0.00	0.00	12.25	13.50	63.00	0.76	0.33	±0.068
Mo17HtC	0.00	0.00	1.25	5.50	20.75	22.25	56.75	0.87	0.34	±0.051
B14AHtD	12.75	44.00	83.75	90.50	145.25	293.00	608.75	0.86	3.49	±0.491
B37HtD	5.00	14.75	28.25	48.25	70.75	159.75	338.25	0.86	1.98	±0.292
Mo17HtD	0.00	0.00	0.25	5.75	29.75	47.50	59.75	0.92	0.45	±0.056
FLSD ^e (<i>P</i> = 0.05)	9.36	36.29	70.85	77.20	70.49	66.96	135.53			

^a Means of four replications.

^b Correlation coefficient of number of conidia with hours of incubation; all values are significant, *P* = 0.01.

^c Regression coefficient of number of conidia on hours of incubation.

^d Standard error of regression coefficient.

^e Fisher's Least Significant Difference.

A632 × A619 and Oh43 × B37. Differences among lesion segments from Oh43 × B37 and B14AHtD were not significant. Lesions from Oh43HtA × B37HtA and Mo17HtC had the fewest spores. Among the three genotypes containing *HtC* and *HtD*, lesions from inbred Mo17 had the lowest number of spores. Generally, *HtC* was better than *HtD* in suppressing sporulation in all genotypes. The gene *HtA* was as effective as *HtC* present in inbreds B37 and Mo17. Correlation coefficients between conidia produced and exposure to high humidity ranged from 0.80 to 0.97.

Incubation time at high humidity and stage of plant growth influenced infection efficiency. Number of hours at high humidity, however, had less effect than plant age on number of lesions. Number of lesions increased with time from 2 to 16 hr when inoculated plants were incubated at high humidity, but the differences among genotypes were significant only at 16 hr when B37HtC, B37HtD, and the hybrid Oh43 × B37 had more lesions than the other genotypes. Differences in lesion number among genotypes at each plant age were significant. No significant variation among plant ages was obtained.

DISCUSSION

Genes for resistance had a marked effect on certain epidemic components of northern corn leaf blight. Sporulation suppression, deemed the most important characteristic of the genetic mechanism under study, was apparent in various resistance combinations. This

characteristic, which determines the magnitude of effective spores produced per day per lesion, is extremely important in the dynamics of epidemics of northern corn leaf blight. The effects of different *Ht* genes were significant. The genetic background affected the expression of the *Ht* genes. In addition, the daily increase and ultimate size of lesions and of the sporulating zone also affected the amount of inoculum produced in the field. These are crucial points in the selection of a type of resistance to be used in agriculture. Field experience over large areas for several years indicates clearly that the use of chlorotic-lesion resistance alone prevents *H. turcicum* from causing a significant amount of infection in temperate areas of the world. However, we prefer a combination of polygenic resistance, which reduces the number of lesions produced and monogenic resistance, which suppresses sporulation. Some *Ht* alleles are more effective than others in suppressing sporulation. Combinations of the *Ht* genes with *Ht2* (7) and other forms of resistance are possible and are likely to result in greater stability and longer protection against *H. turcicum*.

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