Effect of Gene *HtN* on the Development of Northern Corn Leaf Blight Epidemics

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ABSTRACT

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Corn homozygous for gene *HtN*, heterozygous for *HtN*, lacking the allele, or having *HtN* in combination with gene *Ht* or with polygenic resistance was inoculated with *Helminthosporium turcicum*. Incubation time, latent period, percentage infection, and lesion size were measured for all plant genotypes. The effect of the gene *HtN* was primarily to prolong incubation time and the latent period, thereby delaying the start of epidemics and markedly affecting inoculum production.

Disease development is determined essentially by four factors: infection, latent period, pathogen sporulation, and loss of infectious tissue (7). Resistance affects one or more of these processes and thus influences the outcome of a potential epidemic. The types of resistance currently employed against *Helminthosporium turcium* Pass., the cause of northern leaf blight of corn (*Zea mays* L.), act in diverse ways (4,5). Polygenic resistance reduces the number of lesions produced, while chlorotic-lesion resistance, conditioned by genes *Ht, Ht2,* and *Ht3,* primarily suppresses fungal growth and sporulation. A new single dominant gene, designated *HtN*, was recently reported (1). This gene, unlike the *Ht* genes, usually delays lesion formation.

The objective of this study was to determine the effect of the gene *HtN*, when homozygous or heterozygous and in different genetic backgrounds, on the development of epidemics of northern leaf blight of corn.

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MATERIALS AND METHODS

Experiments were done at the Agronomy South Farm of the Illinois Agricultural Experiment Station in Urbana during the summers of 1977 and 1979. Eight inbreds (B37, B37HtN, RB37Ht, Hy, HyHtN, RHyHt, Hy × HyHtN [Hy heterozygous for *HtN*], and B1138T) and four hybrids (B73 × B37HtN, HyHtN × RB37Ht, Hy × B73, and HyHtN × B73) were studied in 1977. Inbreds B37, B73, and Hy have intermediate to low degrees of polygenic

Table 1. Effect of genes *Ht* and *HtN* on incubation time and latent period of northern leaf blight of corn under field conditions in 1977

Genotype	Average incubation time (days)		Average latent period (days)
	Direct inoculation	Inoculated via spreader rows	
B37	15	13	14
B37HtN	74	76	No sporulation
RB37Ht	8	8	No sporulation
B73 × B37HtN	17	30	43
HyHtN × RB37Ht	10	13	No sporulation
Hy	14	13	14
HyHtN	20	26	No sporulation
RHyHt	8	8	No sporulation
Hy × HyHtN	15	13	18
Hy × B73	14	13	14
HyHtN × B73	17	18	28
B1138T	10	12	No sporulation

resistance to *H. turcicum*. Gene *Ht* imparts a chlorotic-lesion type of resistance, and B1138T has a chlorotic-fleck reaction to *H. turcicum* (2).

Two experiments were conducted in 1977. In experiment 1, plants were directly inoculated with a conidial suspension of *H. turcicum* (3.5×10^4 conidia per milliliter of water) about a month after seedling emergence. In experiment 2, the susceptible hybrid A632 × A619 was planted in rows adjacent to each test plot to provide a source of inoculum. Plants in these rows were inoculated by the ground-leaf method (3) at the same time inoculations were made in experiment 1.

Both experiments were arranged in a randomized complete block design with four replications. Each genotype was planted in two adjacent hill plots in each replicate. Hill plots were spaced every 78 cm. Each hill had five plants, providing 10 experimental plants per genotype per replicate.

In 1979, 28 hybrids among inbreds B14, B14A, B37, C103, and W22, having among them genes *Ht*, *HtN*, or neither allele, were studied. Each hybrid was planted in a single hill plot, and all plants were inoculated by the ground-leaf method (3).

In 1977, incubation time (period between inoculation and first appearance of symptoms), latent period (time from inoculation to start of sporulation), percentage of total leaf tissue infected (visual estimates) at six weekly intervals (11, 18, and 25 July; 1, 8, and 15 August), and lesion size (calculated from length and width measurements in centimeters of marked lesions) were recorded. Only percentage of infected leaf tissue was recorded in 1979.

The apparent infection rate, a measure of the progress of disease development, was calculated with van der Plank's (7) model, $r = 1/(t_2-t_1) \times (\log_e x_2/(1-x_2) - \log_e x_1/(1-x_1))$, where x_2 and x_1 are percentage of blighted leaf tissue at times t_2 and t_1, respectively. Data on increases in lesion size were analyzed by simple linear regression.

Table 2. Effect of different genotypes on the development of northern leaf blight of corn in 1977

Genotype	Inoculation method	Percentage leaf tissue blighted[a]						Apparent infection rate (r)[b]
		7/11	7/18	7/25	8/1	8/8	8/15	
B37	Direct[c]	8.1	23.8	41.3	54.4	74.4	86.9	0.12
	Spreader[d]	· · ·	10.0	20.6	38.1	59.4	83.1	0.13
B37HtN	Direct	0.0	0.0	0.0	0.0	0.0	0.0	0.00
	Spreader	· · ·	0.0	0.0	0.0	0.0	0.0	0.00
RB37Ht	Direct	5.8	8.1	10.5	12.8	16.3	19.0	0.04
	Spreader	· · ·	4.8	6.1	8.9	12.1	13.0	0.04
B73 × B37HtN	Direct	1.8	1.8	1.8	3.0	4.0	5.8	0.03
	Spreader	· · ·	0.0	0.0	0.3	1.3	1.5	0.10
HyHtN × RB37Ht	Direct	1.8	1.8	1.8	2.8	3.9	5.0	0.03
	Spreader	· · ·	1.0	1.0	1.3	2.3	2.8	0.04
Hy	Direct	9.9	24.4	44.4	58.1	75.6	92.5	0.14
	Spreader	· · ·	10.0	22.5	42.5	71.3	92.5	0.16
HyHtN	Direct	1.6	1.6	1.6	2.6	5.0	6.5	0.04
	Spreader	· · ·	0.0	0.0	1.0	4.0	4.5	0.14
RHyHt	Direct	5.63	8.9	12.5	15.6	18.3	20.0	0.04
	Spreader	· · ·	5.6	9.3	13.4	18.1	21.8	0.05
Hy × HyHtN	Direct	5.5	8.4	12.5	20.6	29.4	43.1	0.07
	Spreader	· · ·	1.8	4.0	10.0	20.6	30.6	0.11
Hy × B73	Direct	7.0	13.1	20.6	30.6	43.8	61.9	0.09
	Spreader	· · ·	3.1	9.3	21.9	36.8	49.4	0.12
HyHtN × B73	Direct	2.8	3.0	3.5	8.3	16.9	26.3	0.07
	Spreader	· · ·	0.5	0.5	1.9	5.6	13.8	0.12
B1138T	Direct	1.0	1.0	1.5	2.0	3.0	3.0	0.03
LSD (0.05)	Direct	2.0	3.0	6.6	7.7	9.2	11.0	0.02
	Spreader	· · ·	1.3	2.7	5.5	6.3	8.7	0.02

[a] Means of four replicates.
[b] Calculated from the equation (7), $r = 1/(t_2-t_1) \times (\log_e x_2/(1-x_2) - \log_e x_1/(1-x_1))$.
[c] All plants directly inoculated with conidial suspension on 7 June 1977.
[d] Inoculum from inoculated spreader rows of susceptible hybrid A632 × A619. Lesions on spreader rows began sporulating on 30 June 1977.

Table 3. Effect of different genotypes on the increase in size of lesions of northern leaf blight of corn in 1977

Genotype	Lesion size (cm^2)[a]						Corr. coeff.[b]	b[c] ± SE[d]
	7/6	7/13	7/18	7/23	7/28	8/2		
B37	0.00	0.71	3.00	4.22	6.88	10.42	0.97*	0.383 ± 0.051
B37HtN	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.000 ± 0.000
RB37Ht	0.01	0.44	0.51	0.58	0.60	0.72	0.91*	0.023 ± 0.005
B73 × B37HtN	0.00	0.00	0.00	0.00	0.00	0.64	0.64	0.002 ± 0.001
HyHtN × RB37Ht	0.00	0.01	0.04	0.15	0.17	0.18	0.94*	0.008 ± 0.001
Hy	0.00	0.64	3.62	6.10	10.11	10.11	0.97*	0.436 ± 0.056
HyHtN	0.00	0.00	0.00	0.00	0.41	1.22	0.77	0.038 ± 0.016
RHyHt	0.01	0.44	0.58	0.67	0.68	0.69	0.88*	0.023 ± 0.006
Hy × HyHtN	0.00	0.31	1.72	2.69	3.71	6.20	0.96*	0.225 ± 0.032
Hy × B73	0.00	1.20	3.92	6.47	11.26	15.47	0.97*	0.587 ± 0.075
HyHtN × B73	0.00	0.00	0.00	0.15	1.34	5.76	0.75	0.173 ± 0.077
B1138T	0.00	0.00	0.02	0.05	0.05	0.05	0.92*	0.002 ± 0.001
LSD (0.05)	0.00	0.33	0.77	1.41	3.17	4.35		

[a] Means of four replicates; five lesions per replicate.
[b] Correlation coefficient of lesion size with days; * indicates significance at 0.05 level.
[c] Regression coefficient of lesion size on days.
[d] Standard error of regression coefficient.

RESULTS AND DISCUSSION

Dry weather at the beginning of the 1977 cropping season delayed the onset of northern leaf blight of corn, as is apparent in the long incubation period of the disease on directly inoculated plants (Table 1). In experiment 2, lesions on plants in the spreader rows began sporulating 4 wk after inoculation. We assume that the test plants were inoculated with spores from the spreader rows at this time. Average incubation times for the different genotypes between the two experiments did not differ, except for B73 × B37HtN and HyHtN, where the differences were 13 and 6 days, respectively.

Genetic background influenced the expression of gene *HtN* considerably. Compared with the susceptible counterpart, *HtN* in the inbred Hy background prolonged incubation time by 1 wk in experiment 1 and 2 wk in experiment 2. The effect of *HtN* in prolonging incubation time appeared to be most strongly expressed in the B37 background. Distinct leaf blight lesions appeared on B37HtN about 60 days after they appeared on the susceptible inbred B37.

The effect of gene *HtN* when heterozygous in delaying symptom production decreased drastically. The early symptom effect usually seen with gene *Ht* as chlorotic lesions seemed to dominate the effect of *HtN* in hybrids heterozygous for both genes.

No sporulation was observed on the genotypes B37HtN, RB37Ht, HyHtN × RB37Ht, HyHtN, RHyHt, and B1138T. Sporulation occurred when inbreds B37HtN and HyHtN were crossed with B73; however, the latent period was extended by about 2 wk compared with plants of nearly isogenic susceptible crosses.

After the early dry period, environmental conditions generally favored leaf blight development during the remaining 1977 season. The pattern of northern leaf blight progress was basically the same on plants directly inoculated and plants infected with inoculum from adjacent diseased plants in the spreader rows (Table 2). The degree of infection in each genotype was of the same level in both experiments. Apparent infection rates in each genotype in the two experiments were basically proportionate in relation to other genotypes.

Within each experiment, disease development differed markedly among genotypes. Inbred B37HtN had no infection at the last rating date, 69 days after inoculation. A few lesions, however, were observed on some plants about a week after this date. At this time, the leaves were still green, while 90% of the foliage of the counterpart B37 was blighted. Inbred RB37Ht, the resistant counterpart to B37 and nearly isogenic for gene *Ht*, had the shortest incubation period but a slow infection rate, 0.04 per unit per day, such that only 19% of the leaf tissue was eventually blighted.

The single-cross B73 × B37HtN had a very low level of infection. Lesion type also differed from the susceptible wilt-type with this genotype and with HyHtN × B73—lesions were smaller and lacked the pronounced wilting at the margins. Sporulation in these lesions was also less profuse and distinct from lesions on susceptible plants.

The combination of the genes *HtN* and *Ht* (HyHtN × RB37Ht) did not differ significantly from a combination of *HtN* with a polygene system (B73 × B37HtN). The inbred background of *HtN* exerted a major effect, as can be discerned by comparing the levels of infection of HyHtN × B73 and B37HtN × B73. Among the hybrids, Hy × B73 had the highest degree of disease development, as measured by the final amount of blighted tissue; gene *HtN* reduced this by 50%.

The regression coefficient (b) of lesion size on days, indicating the rate of lesion growth, was highest with Hy × B73 (Table 3). Except with genotypes B73 × B37HtN, HyHtN, and HyHtN × B73, lesion size increase was significantly correlated with the slope. No slope was computed with B37HtN, because no lesions were available for measurement during the time of data collection.

Among genotypes involving versions of inbred B37, those with the gene *HtN* did not differ significantly in final lesion size from those with gene *Ht* or those with gene *HtN* in combination with polygenic resistance. Such was not the case with genotypes involving versions of inbred Hy. For instance, HyHtN and RHyHt differed significantly from Hy × HyHtN and HyHtN × B73 in lesion size, apparently because of the greater susceptibility of Hy.

Lesions on B1138T, with a slope of 0.002, remained virtually unchanged, which is consistent with the finding of Hilu and Hooker (2). Disease level increased through increase in the number of lesions.

Conditions for the development of northern leaf blight were not as favorable in 1979 as in 1977. The highest percentages of infected leaf tissue were 30.0, 38.3, and 41.7 in the susceptible hybrids B37 × B14A, W22 × B37, and W22 × B14A, respectively (Table 4). The lowest infection, 1.0%, was on C103HtN × B37. The magnitude of infection was generally very low in hybrids involving inbred C103, which has a high degree of polygenic resistance. Infection was also particularly low when the gene *HtN* was present.

Except for W22 × C103, no significant differences in infection were found among hybrids with the inbred C103 as one parent, regardless of the other genes for resistance that were present. The crosses between B37 or W22 and B14 or B14A involving genes *Ht* and *HtN* were not significantly different, but all differed significantly from B37 × B14A and W22 × B37 with neither gene.

In the crosses studied, gene *HtN* when homozygous did not result in significantly less disease than when heterozygous or when gene *Ht* or both *Ht* and *HtN* were present, except in the crosses involving W22 and B14. Conceivably, the effects of the different genes on disease development might be more distinct when conditions are more favorable for leaf blight.

The effect of the gene *HtN* offers a unique departure from other known types of resistance to *H. turcicum*. The characteristic prolongation of incubation time not only delays the start of an epidemic but also drastically affects inoculum availability. Our results show that infection would be expected to start at a time well past the period of critical grain yield accumulation. Genotypes with the gene *HtN* also had fewer lesions, smaller lesions, and smaller sporulation zones. All these attributes contribute to the effectiveness of resistance.

Genes for resistance to *H. turcicum* can be combined and used in various ways. Gene *HtN* was not as effective when heterozygous as when homozygous. It was more effective in a background with a moderate degree of polygenic resistance than in a background with few or no genes for polygenic resistance. With a high degree of polygenic resistance, the combination could conceivably confer a much higher degree of resistance. Such strategies have been proposed by Hooker (6). More stable, longer-lasting protection can be achieved when "multicomponent" systems such as the combination of genes

Table 4. Effect of different hybrids on the development of northern leaf blight of corn in 1979

Hybrid	Percentage leaf tissue blighted[a] Weeks after silking 1	2	3	4	5	6	Apparent infection rate (r)[b]
B37 × B14A	3.0	6.0	8.3	11.7	15.7	30.0	0.08
RB37Ht × B14A	2.0	3.3	4.3	6.3	9.0	11.3	0.05
B37 × B14HtN	0.7	1.3	1.3	2.0	2.7	5.7	0.05
RB37Ht × B14HtN	1.0	2.0	2.3	2.7	4.3	8.7	0.06
B37HtN × B14HtN	0.0	0.3	1.0	1.0	1.0	2.3	0.04
C103 × B37	1.0	1.3	2.0	3.3	4.7	6.3	0.05
RC103Ht × B37	1.0	1.7	2.0	3.3	3.3	3.7	0.04
C103HtN × B37	0.0	0.0	1.0	1.0	1.0	1.0	0.00
C103 × B14A	1.0	1.0	1.7	2.7	4.3	6.3	0.05
RC103Ht × B14A	1.0	1.7	2.0	3.3	4.3	6.7	0.05
C103HtN × B14A	1.0	1.3	1.7	2.0	3.3	5.3	0.05
C103 × B14HtN	0.3	1.0	1.0	1.0	1.3	2.7	0.03
RC103Ht × B14HtN	0.0	0.7	1.0	1.0	1.0	1.3	0.01
C103HtN × B14HtN	0.0	0.0	1.0	1.0	1.0	1.3	0.01
W22 × B37	2.4	4.0	8.3	9.7	20.0	38.3	0.09
RW22Ht × B37	2.0	3.7	4.3	5.7	8.0	10.7	0.06
W22HtN × B37	0.3	1.0	2.0	2.3	4.7	7.0	0.06
W22 × B14A	4.0	8.3	12.3	16.7	30.0	41.7	0.09
RW22Ht × B14A	1.3	2.7	2.7	4.3	6.3	8.3	0.06
W22HtN × B14A	1.3	2.7	4.3	5.7	10.0	17.3	0.08
W22 × B14HtN	1.3	3.0	5.0	6.0	11.7	24.0	0.09
RW22Ht × B14HtN	0.7	1.0	1.3	1.7	2.0	4.7	0.04
W22HtN × B14HtN	0.0	0.4	1.0	1.0	1.3	3.3	0.05
W22 × C103	1.0	2.7	3.7	5.7	9.0	14.0	0.08
RW22Ht × C103	1.0	1.7	2.0	2.3	3.0	4.3	0.04
W22HtN × C103	0.0	0.3	1.0	1.0	1.0	1.7	0.02
RW22Ht × C103HtN	0.7	0.7	1.0	1.3	1.3	1.3	0.01
W22HtN × C103HtN	0.0	0.3	0.7	1.0	1.0	1.3	0.01
LSD (0.05)	1.0	2.0	2.1	2.4	4.5	9.1	0.03

[a] Means of four replicates.

[b] Calculated from the equation (7), $r = 1/(t_2-t_1) \times (\log_e x_2/(1-x_2) - \log_e x_1/(1-x_1))$.

Ht N and *Ht* and polygenes are used in corn inbreds and hybrids exposed to *H. turcicum* infection.

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