

# Measuring the Relationship Between Northern Corn Leaf Blight and Yield Losses

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## ABSTRACT

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Selected corn hybrids with different types of resistance to *Helminthosporium turcicum* were used to study the relationship between northern corn leaf blight and grain yield losses in a 2-yr study. Differences in infection rates and yields among inoculated and uninoculated hybrids were significant in 1977. In 1979, when much less disease occurred, significant differences were observed only in the areas under disease progress curves and in apparent infection rates, and neither were correlated with yield. The area curve multiple-point model appears appropriate for explaining the relationship between disease development and losses in yield in 1977 but not in 1979.

Additional key words: *Zea mays*

Several mathematical models for describing the relationship between disease development and yield loss have been proposed (8). The critical-point model relates loss to a particular time in the epidemic period. The multiple-point model involves several times of disease assessment during epidemic development. James et al (9) developed a multiple regression equation for predicting loss in tuber yield caused by late blight of potato, and Burleigh et al (2) used the equation to estimate yield losses from wheat leaf rust caused by *Puccinia recondita* Rob. ex Desm. f. sp. *tritici*. The disease progress curve model assumes a proportionality between the area under the disease progress curve and loss in yield. This model has been found reliable in explaining the relationship between yield loss and wheat stem rust caused by *P. graminis* Pers. f. sp. *tritici* Eriks. and Henn. and leaf rust progress (1).

Northern corn leaf blight, caused by *Helminthosporium turcicum* Pass., is a major disease of corn (*Zea mays* L.) in the U.S. corn belt and many corn-growing areas of the world (7). Yield losses in susceptible hosts are considerable when infection occurs before silking and the environment favors disease (12). Quantitative information is needed, especially on the relationship between disease development and yield loss. This study analyzes the progress of northern corn leaf blight in hybrids with varying degrees of host resistance to *H. turcicum* and seeks to correlate the progress of the epidemic with grain yield in a multiple-point model.

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## MATERIALS AND METHODS

Experiments were conducted at the Agronomy South Farm of the Illinois Agricultural Experiment Station in Urbana in 1977 and 1979. In 1977, the corn hybrids A632 × A619, N28 × Mo17, and H93 × Va26 were used. A632 × A619 is susceptible to *H. turcicum* and matures early. N28 × Mo17 possesses a moderate degree of polygenic resistance to the fungus and is intermediate in maturity. H93 × Va26 has an *Ht*-type monogenic resistance (6) as well as a moderate degree of polygenic resistance and is late-maturing.

The experiment was arranged in a randomized complete block design with four replications. Each replicate consisted of two rows of 24 plants each. Rows were 76 cm apart, and two-plant hills were spaced every 38 cm within rows. To obtain a basis for calculating loss in yield, the same hybrids were planted in a check block isolated from the disease block but in the same general area. In the absence of disease, equal yields of a hybrid were expected in the two blocks, because all plots were similar in soil type and fertility.

In 1979, the hybrids used involved crosses between inbred B37 and selections of inbred B37 with alleles *Ht*, *Ht2*, or *HtN* (4,6,7) and inbreds Oh43 and Oh45. The experiment was arranged as in 1977 in a randomized complete block design with four replications but with single-row plots.

One month after emergence, each plant in the disease block was inoculated with conidia of *H. turcicum*. Inoculum was obtained from diseased leaf tissue incubated for 5 days in petri plates lined with moist tissue paper. The conidia that formed on the leaf pieces were washed from the surface with distilled water and diluted to a concentration of 35,000 conidia per milliliter of water, as determined from hemocytometer readings.

Approximately 10 ml of the inoculum suspension was directed into the whorl of each plant with a compressed-air sprayer. Inoculum was applied in the early evening, because cool temperatures and free moisture favor infection by *H. turcicum*.

Percentages of leaf area infected in each row were recorded weekly, starting at silking time. Ears from each row (except from the end plants) were bulk-harvested at maturity, hot-air dried, shelled, and weighed. In addition, 500 kernels were sampled from each plot and weighed in grams. After adjusting to 15.5% moisture content, grain yields were expressed in quintals per hectare and the 500-kernel sample weights in adjusted grams. In 1977, immediately after the ears were harvested, the stalks were split and the incidence of stalk rot was recorded, because any yield loss would be the result of the combined effects of leaf blight and premature plant death from stalk rot.

Apparent infection rates for *H. turcicum*, a measure of the progress of the epidemic, were calculated from the weekly observations of percentage of leaf area infected. We used van der Plank's (13) model,  $r = 1/(t_2 - t_1) \times ((\log_e x_2 / (1 - x_2)) - (\log_e x_1 / (1 - x_1)))$ , where  $r$  is the apparent infection rate and  $x_1$  and  $x_2$  are proportions of disease at times  $t_1$  and  $t_2$ , respectively. The area under the disease progress curve, which represents the magnitude of disease for the entire growing season, was computed from the equation,  $\text{Area} = \sum_{i=1}^n (1/2)(x_i + x_{i-1})$ , where  $x_i$  is percentage of leaf area infected at time  $i$  and  $n$  is the number of times ratings were taken.

## RESULTS AND DISCUSSION

Differences in disease development among the three hybrids were large in 1977. Percentages of blighted leaf tissue differed significantly at all rating dates. Six weeks after silking, the susceptible, early-maturing A632 × A619 had 97% of leaf tissue blighted because of *H. turcicum* infections; the moderately polygenic-resistant, intermediate-maturing N28 × Mo17 had 89%, and the chlorotic-lesion-resistant, late-maturing H93 × Va26 had 27%.

Overall infection rates ( $r$ ) on A632 × A619 were about 1.5 times that of N28 × Mo17 (Table 1). Infection rates generally increased weekly for A632 × A619 and N28 × Mo17. The areas under the disease progress curves were not

**Table 1.** Effect of northern leaf blight on yield and stalk rot incidence in three inoculated corn hybrids in 1977

Hybrid <sup>a</sup>	Treatment	Area <sup>b</sup>	Apparent infection rate (r) <sup>c</sup>	Quintals/hectare	Loss (%)	500-kernel weight (gm)	Loss (%)	Stalk rot (%)
A632 × A619	Inoculated	294.8	0.13	28.1	62.3	98.8	39.2	95.0
	Control	0.0	...	74.5	...	162.4	...	33.1
N28 × Mo17	Inoculated	289.1	0.08	45.1	43.3	115.9	40.0	89.5
	Control	0.0	...	79.6	...	193.2	...	18.9
H93 × Va26	Inoculated	123.4	0.04	68.0	16.7	148.2	9.2	35.2
	Control	0.0	...	81.6	...	163.2	...	3.5
LSD 0.05	(Hybrid):	16.3	0.08	4.4	...	9.6	...	15.0
	(Treatment):	...	...	3.6	...	7.8	...	12.3
Correlation coefficient <sup>d</sup>				0.89		0.97		
$b^e \pm SE^f$				0.22 ± 0.02		0.18 ± 0.01		

<sup>a</sup> A632 × A619, early-maturing and susceptible to *Helminthosporium turcicum*; N28 × Mo17, intermediate maturity and polygenic resistance to *H. turcicum*; and H93 × Va26, late-maturing and monogenic resistance to *H. turcicum*.

<sup>b</sup> Area under the disease progress curve calculated from the equation,  $Area = \sum_{i=1}^n (1/2(x_i + x_{i-1}))$ , where  $x_i$  is percentage infection at time  $i$  and  $n$  is the number of times ratings were taken.

<sup>c</sup> From van der Plank's (13) model,  $r = 1/(t_2 - t_1) \times ((\log_e x_2 / (1 - x_2)) - (\log_e x_1 / (1 - x_1)))$ .

<sup>d</sup> Calculation of percentage loss with area; both values are significant ( $P = 0.01$ ).

<sup>e</sup> Regression coefficient of percentage loss on area.

<sup>f</sup> Standard error of regression coefficient.

**Table 2.** Effect of northern leaf blight on yield of selected corn hybrids in 1979

Hybrid	Area <sup>a</sup>	Apparent infection rate (r) <sup>b</sup>	Quintals/hectare	Yield change <sup>c</sup> (%)	500-kernel weight (gm)	Yield change <sup>c</sup> (%)
Oh43 × B37	97.6	0.069	76.8	...	152.2	...
Oh43Ht × B37	48.3	0.042	89.9	+17.1	158.4	+ 4.1
Oh43Ht × B37Ht	53.4	0.038	87.2	+13.5	161.9	+ 6.4
Oh43Ht2 × B37Ht	45.6	0.028	76.6	- 0.3	151.9	- 0.2
Oh43Ht2 × B37Ht2	46.4	0.044	76.7	- 0.1	167.4	+ 9.9
LSD 0.05	11.3	0.017	ns <sup>d</sup>	...	ns	...
Oh45 × B37	49.0	0.059	88.1	...	166.7	...
Oh45 × B37Ht	46.4	0.045	88.9	+ 0.9	174.9	+ 4.9
Oh45 × B37Ht2	28.4	0.033	89.6	+ 1.7	175.7	+ 5.4
Oh45 × B37HtN	12.6	0.024	97.1	+10.2	190.3	+14.2
LSD 0.05	16.5	0.016	ns	...	13.4	...

<sup>a</sup> Area under the disease progress curve calculated from the equation,  $Area = \sum_{i=1}^n (1/2(x_i + x_{i-1}))$ , where  $x_i$  is percentage infection at time  $i$  and  $n$  is the number of times ratings were taken.

<sup>b</sup> From van der Plank's (13) model,  $r = 1/(t_2 - t_1) \times ((\log_e x_2 / (1 - x_2)) - (\log_e x_1 / (1 - x_1)))$ .

<sup>c</sup> Calculation based on Oh43 × B37 in Oh43 group and Oh45 × B37 in Oh45 group.

<sup>d</sup> ns = nonsignificant.

significantly different between A632 × A619 and N28 × Mo17, but both were more than twice that of H93 × Va26 (Table 1).

Percentage of rotted stalks was significantly higher in the *H. turcicum* inoculated plots compared to the control (Table 1). This indication that northern corn leaf blight predisposes corn plants to attack by stalk rot organisms has been reported before (3). Incidence of stalk rot was not significantly different between A632 × A619 and N28 × Mo17, but both hybrids sustained more stalk rot than H93 × Va26 (Table 1). Premature plant death due to stalk rot needs to be considered in any assessment of yield losses from leaf diseases.

Higher losses in yield were obtained from A632 × A619 and N28 × Mo17 than from H93 × Va26. On a quintal per hectare basis, losses were 62.3% for A632

× A619, 43.3% for N28 × Mo17, and 16.7% for H93 × Va26 compared with the controls (Table 1). These figures were significantly different ( $P = 0.05$ ). Unusually small and mostly unfilled ears were harvested from severely infected plants. Although lower percentage losses were calculated from all hybrids on the 500-kernel weight basis than on the quintal per hectare basis, differences were significant ( $P = 0.05$ ).

In 1979, conditions for northern corn leaf blight development were unfavorable. The highest percentage of leaf tissue blighted was only 37% on susceptible Oh43 × B37 plants. Within the Oh43 × B37 group of hybrids, the susceptible hybrid differed significantly in area under the disease curve and infection rate from the resistant hybrids with genes *Ht1* or *Ht2* or both (Table 2). No significant differences in yield or 500-kernel weight

were observed among these latter hybrids all having monogenic resistance to *H. turcicum*.

In hybrids involving Oh45, the highest percentage of blighted leaf tissue was 15%. Hybrid Oh45 × B37HtN was the most resistant, with only 3% of leaf tissue infected 6 wk after silking. This hybrid did not differ significantly from Oh45 × B37Ht2 in area and infection rate but was significantly better than Oh45 × B37 and Oh45 × B37Ht.

Yields (quintals per hectare) and 500-kernel weights, except for the 500-kernel weight differences in the Oh45 group, did not differ significantly among hybrids. Yield changes were erratic in the Oh43 group when quintals per hectare and 500-kernel weights were compared. In the Oh45 group, a similar trend was seen from both categories of percentage change in yield.

The area under the disease progress curve was highly correlated with percentage loss in yield in 1977 (Table 1); correlation coefficients of 0.89 and 0.97 were obtained on quintals per hectare and 500-kernel weight bases, respectively. These values were highly significant. Regression coefficients, estimates of the unit change in loss brought about by a unit change in area, were 0.22 and 0.18, respectively, for quintals per hectare and 500-kernel weight bases.

In 1979, correlation coefficients between area and yield were not significant. The values were 0.04 and -0.63 for quintals per hectare and 500-kernel weight bases, respectively.

Differences in disease development and subsequent loss in yield among the hybrids were determined primarily by the resistance mechanism in each hybrid. For instance, the low level of disease and the correspondingly low yield loss in H93 × Va26 were effects of the combined moderate polygenic and monogenic *Ht* types of *H. turcicum* resistance and perhaps a greater resistance to stalk rot (Table 1).

Maturity, however, also played a role. For instance, N28 × Mo17 had a lower percentage loss than A632 × A619, even though it had a higher percentage of blighted leaf tissue 2 wk after 75% silking and had nearly as high a level of disease throughout the season. Rapid dry-matter accumulation starts at silking time (5), and clearly this process was affected more adversely in A632 × A619, because the grain-fill period was shorter than in N28 × Mo17.

Number of kernels per ear is an important component of yield loss. Because considerably shorter ears were harvested from inoculated plants, greater losses were recorded in quintals per hectare, which reflects total yield, than in 500-kernel weights, which reflect mainly seed size (Tables 1 and 2). Similarly, the difference in percentage loss between A632 × A619 and N28 × Mo17 was

considerably greater on a quintal per hectare basis than on a 500-kernel weight basis.

Position of leaves infected relative to crop growth is essential in assessing the disease-loss relationship (10). Upper leaves contribute more photosynthate to the plant than lower leaves. Leaf tissue blighted early in the grain-filling period has a greater effect on yield than an equivalent amount of leaf tissue blighted later in the grain-filling period (12).

The disease progress curve model satisfactorily explained the relationship between northern corn leaf blight and corn yield losses in 1977 but not in 1979. The area under the curve relationship may only be useful when disease levels are quite high (unlike 1979), so that the effects of specific genotypes can be distinctly expressed. The area curve model, which accounts for the magnitude of disease during the season, appears appropriate when dealing with relatively

long epidemics and plants with a long period of dry-matter accumulation, such as corn (5). The multiple-point model could conceivably also be used if the problem of autocorrelation among the independent variables, usually ratings of fungal infection at different dates, eg, on the same plants, is solved. Failure to follow this fundamental statistical assumption of independent factors could lead to misinterpretations of results (11).

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