Disease Development and Yield Losses Associated with Northern Leaf Blight on Corn

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ABSTRACT

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Three single-cross corn (Zea mays) hybrids, with and without the Ht resistance gene, were inoculated with conidial suspensions of Exserohilum turcicum race 1 at one of five growth stages in 1979–1982. Greater initial lesion numbers were observed on the Ht versions of the three hybrids than on the ht versions. Area under the disease progress curve (AUDPC) was greater for the ht versions of the hybrids than for the Ht versions. A619 × A632 had the highest AUDPC values, a result of low polygenic resistance. Significant reductions in grain yields and 500-kernel weights were observed only for A619 × A632, and yield reductions ranged from 0 to 18%. Critical-point (CP) and AUDPC models were evaluated using untransformed and transformed data. The best model was obtained using AUDPC ($Y = 92.97 - 0.12$ (AUDPC), $r^2 = 0.68$). CP models using percent leaf area infected at 3, 5, and 6 wk after midsilk also gave relatively good fit ($r^2 = 0.66$).

Northern leaf blight (NLB), caused by Exserohilum turcicum (Pass.) Leonard &

Suggs (teleomorph: Setosphaeria turcica (Luttrell) Leonard & Suggs), is an important foliar disease of corn (Zea mays L.). The disease is sporadic in occurrence, depending on the environmental conditions and the level of disease resistance in the hybrid. When the disease becomes severe, yield losses of 30% or more can occur when susceptible hybrids are grown (11). The magnitude of the yield losses caused by NLB depends on two factors: the stage of plant growth when infection occurs and the severity of disease. Ullstrup and Miles (17) found the most resistant hybrid yielded 4,390 kg/ha more than the most susceptible

hybrid after inoculation with E. turcicum. Highest yield losses were observed when infection occurred before silking; however, when infection occurred 6–8 wk after silking, no yield reductions occurred. Other workers have reported significant yield reductions attributed to NLB (5,12). In addition, plants infected with leaf blight are often predisposed to root and stalk rot infections (3,12).

Yield loss models have been used to estimate losses in field crops and to relate disease development and the stage of plant growth. Raymundo and Hooker (12) found the area under the disease progress curve (AUDPC) models explained the relationship between NLB disease severity and yield loss in only 1 of 2 yr. They suggested that AUDPC models were effective for NLB only when disease levels were high.

The objectives of this study were: 1) to evaluate the effects of inoculation time and type of resistance used on the development of NLB epidemics, 2) to determine the relationship between NLB severity and grain yield loss caused by NLB, and 3) to develop yield loss models for estimating potential losses caused by E. turcicum.

MATERIALS AND METHODS

Three single-cross hybrids, A619 × A632, B73 × Oh545, and Mo17 × B73, were used in the study. They were selected based on previous studies conducted in 1976 and 1977 that indicated low, intermediate, and high levels of polygenic resistance to NLB for the three hybrids, respectively (J. M. Perkins, *unpublished*). Two nearly isogenic versions of each hybrid were used in this study; therefore, six hybrids were evaluated. One version of each hybrid was homozygous for the *Ht* gene, and the second version was homyzygous for the corresponding *ht* gene. Because Oh545*Ht* was not available, the hybrid B73*Ht* × Oh545 was heterozygous.

The study was conducted in 1979, 1980, and 1981 on the Plant Pathology Farm, Urbana, and in 1982 on the Agronomy South Farm Cruse Tract, Champaign. The experimental design was a split-split plot arranged in a randomized complete block with four replicates. Main plots were inoculation times, subplots were hybrids, and sub-subplots were the two versions (*Ht* vs. *ht*) of each hybrid. Each experimental unit consisted of three rows 9.14 m long and 0.76 m apart. Six rows of Mo17*Ht* × B73*Ht* were planted between main plots to reduce interplot interference. Plots were planted and thinned to a density of about 59,300 plants per hectare. All data were collected on the middle row of the three-row plot.

Main plots were inoculated on five dates, which corresponded to growth stages (GS) 2–6 (6). An uninoculated check plot was included in the design. Inoculum consisted of a condial suspension obtained from sporulating cultures of *E. turcicum* race 1 grown on lactose casein hydrolysate agar (15). Cultures were macerated with deionized water in a Waring Blendor and filtered through a double thickness of cheesecloth. The resulting suspension was adjusted to about 12,000 conidia per milliliter and sprayed over plants in each plot. Inoculations were made after 1600 hours, when air temperatures were cooling and moisture needed to ensure infection would occur on the leaves.

Ten random plants per plot were selected and total lesions per plant counted 14 days after inoculation. After the initial appearance of typical NLB symptoms, ratings of percent leaf area infected were made at weekly intervals until leaf tissue senescence began. The number of ratings varied with the inoculation time. AUDPC (14) was calculated for each plot, with time being expressed as weeks. Before harvest, the total number of plants per plot was determined. In 1980, 1981, and 1982, data on number of lodged plants were collected. Any plant that had the stalk broken at any point between the soil surface and ear-bearing node was considered lodged.

After physiological maturity, the center row of each plot was hand-harvested and mechanically shelled. Weight of shelled grain and percent grain moisture were determined. Yields were converted to kilograms per hectare of shelled grain at 15.5% grain moisture. Because of extreme drought stress in 1980, plot yields varied greatly and yield data from this year were not included in the analysis. A sample of each plot was air-dried to about 15% moisture and 500-kernel weights were determined.

Data were analyzed as a split-split plot. Fisher's least significant difference (LSD) (4) was used to separate differences among treatment means. Critical-point (CP) models using untransformed leaf blight ratings and AUDPC models were evaluated using ordinary least-squares regressions (2). Yield, expressed as a percentage of the average yield of the uninoculated check plot, was used as the dependent variable. Criteria used to select the best fitting models for estimating yield losses included: 1) coefficients of determinations (r^2), which indicate the proportion of the total variation explained by the model ($r^2 > 0.50$); 2) *F*-statistic, which tests the significance of the regression model ($P < 0.05$); and 3) *T*-statistic of each partial regression coefficient ($P < 0.05$), which tests the contribution of each partial regression coefficient to the model.

RESULTS

The average number of lesions developing after inoculation with *E. turcicum* was significantly different ($P = 0.05$) among the three corn hybrids. The hybrid with a low level of polygenic resistance, A619 × A632, generally showed the greatest number of initial lesions, ranging from an average of less than one to more than 19 lesions per plant (Table 1). The hybrids with greater polygenic resistance, Mo17 × B73 and B73 × Oh545, had fewer lesions after inoculation. Greater initial lesion numbers were observed on the three hybrids carrying the *Ht* gene than on the corresponding hybrids with the *ht* gene for inoculations at GS 2; however, the difference only was significant for A632 × A619. Differences were observed between time of inoculation for initial lesion numbers (Table 1), with the earlier inoculations having the greatest numbers of lesions.

AUDPC values ranged from 1 in check plots to 248 in plots of A619 × A632 inoculated at GS 2 (Table 1). The three hybrids carrying the *Ht* gene had lower AUDPC values than the corresponding hybrids carrying the *ht* gene. This trend

Table 1. Lesion number, percent leaf area infected (LAI) 3 wk after silk, AUDPC, and percent stalk lodging for six corn hybrids after inoculation with *Exserohilum turcicum* race 1 at one of five growth stages

| | | Hybrid | | | | | |
| | | A632 × A619 | | B73 × Mo17 | | B73 × Oh545 | |
Parameter	GS[a]	*Ht/Ht*	*ht/ht*	*Ht/Ht*	*ht/ht*	*Ht/ht*	*ht/ht*
Lesion number	2	19.5[b]	10.9	9.5	5.5	9.4	6.3
	3	8.6	8.6	3.7	3.5	4.0	3.5
	4	2.4	2.7	1.6	1.2	1.1	1.1
	5	0.6	0.6	0.5	0.3	0.6	0.2
	6	0.3	0.4	0.2	0.1	0.2	0.2
LSD ($P = 0.05$)[c]				1.6			
% LAI 3 wk	2	6	38	3	7	3	7
after silk	3	5	18	3	5	2	7
	4	1	3	1	1	1	1
	5	0	1	0	0	0	0
	6	0	1	0	0	0	0
	Control	0	1	0	0	0	0
LSD ($P = 0.05$)				4			
AUDPC	2	52	248	19	54	18	59
	3	41	126	16	39	16	50
	4	10	28	4	8	4	9
	5	3	7	1	2	1	2
	6	3	9	2	3	2	3
	Control	2	7	1	1	1	1
LSD ($P = 0.05$)				6			
Stalk lodging	2	9	16	3	3	6	8
	3	7	8	2	3	6	6
	4	6	5	4	3	7	7
	5	6	5	4	3	6	4
	6	5	2	2	3	5	3
	Control	7	2	4	2	8	4
LSD ($P = 0.05$)				4			

[a] Growth stage (8).
[b] Means based on four replicates per year.
[c] Least significant difference (LSD) is used for all pairwise comparisons.

continued throughout the season, with the differences in AUDPC values between the hybrids carrying the *Ht* gene or the *ht* gene becoming greater. Differences in percent stalk lodging were observed among the hybrids carrying the *ht* gene (Table 1). The hybrid A619 × A632 showed an 800% increase in stalk lodging when inoculated at GS 2 compared with the uninoculated check.

Grain yields ranged from 8,352 to 12,115 kg/ha (Table 2) during the study. Yield reductions ranged from 0 to 18% for A619 × A632; however, no differences were observed in grain yields for any of the other hybrids, either with or without the *Ht* gene. Significant losses occurred only when the AUDPC value was 126 or greater.

Significant reductions in 500-kernel weights also were observed for the hybrid A619 × A632 (Table 2). Maximum kernel weight losses were 20 and 13% in 1979 and 1981, respectively, compared with the uninoculated check.

CP and AUDPC models were developed for A619 × A632 to estimate potential yield losses caused by *E. turcicum*. Both untransformed and transformed data were used in the analysis; however, better models were obtained using untransformed data. More than one model adequately described the relationship between percent yield and disease severity. The best model was obtained using AUDPC ($Y = 92.97 - 0.12$ (AUDPC), $r^2 = 0.68$). CP models using untransformed percent leaf area infected (LAI) at ratings 3, 5, and 6 wk after midsilk gave relatively good fit ($r^2 = 0.66$).

DISCUSSION

Use of monogenic resistance conferred by the *Ht* gene was effective in reducing NLB development in corn hybrids with a low level of polygenic resistance. Plants carrying the *Ht* gene produced small chlorotic lesions compared with large lesions found on plants carrying the *ht* gene. In this study, use of the *Ht* gene in a hybrid susceptible to NLB produced a disease reaction equivalent to that of a hybrid with a high degree of polygenic resistance. A negative effect of the *Ht* gene was an increase in initial lesion numbers in all hybrids at GS 2. In plants with chlorotic lesion resistance, such as *Ht*, the invading hyphae of *E. turcicum* have rarely been reported to penetrate the xylem, and lesion enlargement is limited (9). This may reduce competition between individual infection sites, resulting in more initial lesions on hybrids carrying the gene *Ht*.

Comparison of AUDPC values from A619 × A632, with and without the *Ht* gene, indicated that use of single-gene resistance such as *Ht* can reduce disease development. The inoculation of A619 × A632 at GS 2 resulted in an average value of 248 compared with a value of 52 for A619 × A632*Ht/Ht*. This represents a 79% reduction in disease when single-gene resistance was used in conjunction with low polygenic resistance. The AUDPC value of 52 for A619 × A632*Ht/Ht* was comparable with an AUDPC value of 54 for the GS 2 inoculation of B73 × Mo17. Therefore, the use of the *Ht* gene in susceptible hybrids can give a level of resistance equivalent to hybrids with a high level of polygenic resistance. The combination of monogenic resistance and polygenic resistance produced the lowest AUDPC values, 19 for Mo17 × B73*Ht/Ht* and 18 for B73 × Oh545*Ht/ht*.

The appearance of race 2 of *E. turcicum* (1,16), which is virulent on hybrids carrying the gene *Ht*, requires the consideration of other sources of single-gene resistance. The *Ht2* and *Ht3* genes confer resistance to race 2 (9,13). Many other sources of race 2 resistance have been identified, and their reactions have been reported (10,11). Polygenic resistance was also effective in reducing NLB development. Although B73 × Oh545 and B73 × Mo17 were selected on the basis of previous studies that indicated they had intermediate and high levels of polygenic resistance to NLB, the results of this study showed little difference in resistance level. Very little NLB development was observed on these two hybrids compared with A619 × A632. This suggests the level of polygenic resistance in many of the inbreds commonly used in hybrid production (18) is adequate to reduce disease development in the field. Because polygenic resistance is conditioned by several genes and is non-race-specific, it should be effective against new races such as race 2 of NLB.

The highest level of resistance was obtained with the combination of polygenic resistance with the *Ht* gene. Although the level of resistance of the combination was greater than either polygenic or monogenic alone, there appeared to be no need for the combination because either alone provided adequate resistance.

Yield reductions resulting from *E. turcicum* inoculations were observed only for A619 × A632. No significant yield reductions were observed for Mo17 × B73 or B73 × Oh545. This suggests hybrids with intermediate to high levels of polygenic resistance should not incur yield losses in the field. The lack of a yield reduction in A619*Ht* × A632*Ht* indicated single-gene resistance is useful in conjunction with low levels of polygenic resistance. Use of the *Ht* resistance gene will reduce disease development, resulting in little or no yield loss.

Significant differences in 500-kernel weights occurred in the same 2 yr significant grain yield losses were observed on A619 × A632, suggesting reduced kernel weight contributed to yield losses. The reductions in kernel weight were probably a result of the loss of active photosynthetic leaf area caused by NLB. The loss of active leaf area results in less photosynthate available during the grain-fill period, producing smaller kernels. This reduced kernel weight also contributes to grain yield losses. Reduction in the number of kernels per ear resulting from early disease infection might also contribute to grain yield losses. Data collected in 1982 showed no differences in kernels per ear.

Yield loss models were developed using untransformed data from the hybrid A619 × A632. The model using AUDPC explained the relationship between percent yield and disease severity when yield was expressed as the percentage of the average yield of the uninoculated check plots of A619 × A632 for 1979, 1981, and 1982. Hence, AUDPC would be the best choice in attempting to predict yield losses. However, this method required multiple

Table 2. Grain yield (kg/ha) and 500-kernel weights of six hybrids after inoculation with *Exserohilum turcicum* at one of five growth stages in 1979, 1981, and 1982

Parameter	GS[a]	A632 × A619		B73 × Mo17		B73 × Oh545	
		Ht/Ht	*ht/ht*	*Ht/Ht*	*ht/ht*	*Ht/ht*	*ht/ht*
Yield (kg/ha)	2	9,378[b]	8,352	11,113	11,287	11,655	11,300
	3	9,678	8,921	11,511	11,185	12,115	11,478
	4	9,716	9,450	11,505	11,250	11,630	11,349
	5	9,552	9,648	11,691	11,204	11,571	11,597
	6	10,156	9,851	11,374	11,259	11,814	11,944
	Control	9,882	10,165	11,506	11,110	11,269	11,374
LSD ($P = 0.05$)				960			
500-Kernel wt	2	148	144	144	146	140	137
	3	153	147	145	148	145	143
	4	156	153	144	147	141	141
	5	153	152	146	148	141	146
	6	154	155	144	149	144	147
	Control	154	154	146	148	143	141
LSD ($P = 0.05$)[c]				4			

[a] Growth stage (8).
[b] Means based on four replicates per year.
[c] Least significant difference (LSD) is used for all pairwise comparisons.

ratings during the growing season. CP models using percent LAI at 3, 5, and 6 wk after midsilk also gave relatively good estimates of potential yield losses. CP models have the advantage of requiring only one rating during the growing season.

The results of this research show the importance of polygenic resistance in preventing yield losses caused by NLB. The hybrids with a high level of polygenic resistance showed no significant yield or kernel weight reductions. Single-gene resistance, incorporated into a hybrid with low polygenic resistance, also prevented yield losses. Evaluation of yield loss models indicated the most favorable time to estimate potential yield losses in hybrids with low polygenic resistance was during the period 3–6 wk after silking.

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. Draper, N. R., and Smith, H. 1981. Applied Regression Analysis. John Wiley & Sons, New York. 709 pp.
3. Fajemisin, J. M., and Hooker, A. L. 1974. Top weight, root weight, and root rot of corn seedlings as influenced by three Helminthosporium leaf blights. Plant Dis. Rep. 58:313-317.
4. Federer, W. T. 1955. Experimental Design. Macmillan, New York.
5. Fisher, D. E., Hooker, A. L., Lim, S. M., and Smith, D. R. 1976. Leaf infection and yield loss caused by four Helminthosporium leaf diseases of corn. Phytopathology 66:942-944.
6. Hanway, J. J. 1963. Growth stages of corn (*Zea mays*, L.). Agron. J. 55:487-492.
7. Hilu, H. M., and Hooker, A. L. 1964. Host-pathogen relationship of *Helminthosporium turcicum* in resistant and susceptible corn seedlings. Phytopathology 54:570-575.
8. Hooker, A. L. 1975. *Helminthosporium turcicum* as a pathogen of corn. Rep. Tottori Mycol. Inst. Jpn. 12:115-125.
9. Lim, S. M., Kinsey, J. G., and Hooker, A. L. 1974. Inheritance of virulence in *Helminthosporium turcicum* to monogenic resistant corn. Phytopathology 64:1150-1151.
10. Pedersen, W. L., Perkins, J. M., Radtke, J. A., and Miller, R. J. 1986. Field evaluation of corn inbreds and selections for resistance to *Exserohilum turcicum* race 2. Plant Dis. 70:376-377.
11. Perkins, J. M., and Hooker, A. L. 1981. Reactions of eighty-four sources of chlorotic lesion resistance in corn to three biotypes of *Helminthosporium turcicum*. Plant Dis. 65:502-504.
12. Raymundo, A. D., and Hooker, A. L. 1981. Measuring the relationship between northern corn leaf blight and yield losses. Plant Dis. 65:325-327.
13. Smith, D. R., and Kinsey, J. G. 1980. Further physiologic specialization in *Helminthosporium turcicum*. Plant Dis. 64:779-781.
14. Tooley, P. W., and Grau, C. R. 1984. Field characterization of rate-reducing resistance to *Phytophthora megasperma* f. sp. *glycinea* in soybean. Phytopathology 74:1201-1208.
15. Tuite, J. 1969. Plant pathological methods: fungi and bacteria. Burgess Publishing, Minneapolis, MN. 239 pp.
16. Turner, M. T., and Johnson, E. R. 1980. Race of *Helminthosporium turcicum* not controlled by *Ht* genetic resistance in corn in the American corn belt. Plant Dis. 64:216-217.
17. Ullstrup, A. J., and Miles, S. R. 1957. The effects of some leaf blights of corn on grain yield. Phytopathology 47:331-336.
18. Zuber, M. S., and Darrah, L. L. 1980. 1979 U.S. corn germplasm base. Proc. Annu. Corn Sorghum Res. Conf. 35:234-249.