Effects of Qualitative and Quantitative Resistance on the Development and Spread of Northern Leaf Blight of Maize Caused by *Exserohilum turcicum* Races 1 and 2

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ABSTRACT

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The effects of qualitative and quantitative resistance on the development and spread of northern leaf blight (NLB) of maize caused by *Exserohilum turcicum* races 1 and 2 was studied in 1984 and 1985 in a 2 × 2 × 2 factorial with the following treatments: the hybrids A619 × A632 and B73 × Mo17, the genes *Ht*1 and *ht*1; and races 1 and 2 of *E. turcicum*. In 1985, A619*Ht*2 × A632 was also evaluated. NLB severity gradients were studied for each treatment. In both years, host genotype-pathogen race combinations were classified into resistant and susceptible groups based on NLB severity and spread. Hybrid-race combinations differed significantly between resistant and susceptible groups but did not differ significantly within groups. Both versions of B73 × Mo17 inoculated with either race and A619*Ht*1 × A632*Ht*1 inoculated with race 1 were resistant. The severity of NLB on resistant hybrids was less than 3% at all sampling times and distances from the foci. All versions of A619 × A632 except A619*Ht*1 × A632*Ht*1 inoculated with race 1 were susceptible. In plots of susceptible hybrids, NLB severity was below 20% at all times and distances except for 0.8 m from the foci at 8, 9, and 10 wk after inoculation. High levels of qualitative and quantitative resistance appear to be equally effective in limiting the spread and development of NLB although genes for qualitative resistance may not be effective against all pathogen races and in some hybrid combinations, such as the *Ht*2 gene in A619 × A632. Severe NLB epidemics appear to require large amounts of initial inoculum and/or environmental conditions which result in abundant secondary inoculum production.

Additional key words: corn, disease gradients, *Helminthosporium turcicum*, northern corn leaf blight, *Zea mays*.

Epidemic development of northern leaf blight (NLB) of maize (*Zea mays* L.), caused by *Exserohilum turcicum* (Pass.) Leonard and Suggs, depends on the ability of the fungus to infect, grow, and reproduce on *Z. mays* and on the ability of *E. turcicum* conidia to disperse within the crop and subsequently cause infection. Wind, rain, and diurnal conidial dispersion have been described for *E. turcicum* (13). Conidia are formed most abundantly on cool, moist nights (1,13,17,18). Production of conidia also depends greatly on host resistance (8).

Quantitative and qualitative resistance to NLB are known. Quantitative resistance was observed as variation in inbred response to inoculation and was found to be inherited polygenically (2,9). Population improvement procedures were effective in selection for quantitative resistance which reduces lesion number (10,19). Single dominant genes, which are qualitatively expressed as lesion types, also have been identified and backcrossed into maize inbred lines (3,5,6,8). The *Ht*1 gene conditions a chlorotic-lesion response and has been used extensively in commercial hybrids. The *Ht*2 gene conditions a chlorotic-lesion response with more chlorosis and necrosis than the response conditioned by *Ht*1. The *Ht*3 gene from *Tripsacum floridanum* Porter & Vassy has been incorporated into maize and conditions a reaction similar to that conditioned by *Ht*2. The *Ht*N gene prolongs incubation and latent periods. Sporulation is suppressed or delayed with each of these qualitative types of resistance (8).

Races of *E. turcicum* that are virulent on maize possessing *Ht* genes occur naturally in the continental United States (22,24). Races 1, 2, and 3 have the virulence formulas (effective/ineffective host genes): *Ht*1*Ht*2*Ht*3*Ht*N/0, *Ht*2*Ht*3*Ht*N/*Ht*1, and *Ht*1/*Ht*2*Ht*3, respectively (14,15,22). The reaction of the *Ht*N gene to race 3 has not been reported. Races 1 and 2 are prevalent in the United States corn belt (11). Although race 2 is virulent on maize possessing the *Ht*1 gene, lesion length and NLB severity are less on some genotypes that possess *Ht*1 compared with near-isogenic lines that are recessive at this locus (16).

The objective of this research was to compare the effects of qualitative and quantitative resistance on the development and spread of NLB caused by *E. turcicum* races 1 and 2.

MATERIALS AND METHODS

This study was done in 1984 and 1985 on the Pomology Research Farm, University of Illinois, Urbana, in a 1.1-ha field that was isolated from maize by at least 0.5 km in all directions. The experimental design was a randomized complete block with three replications. The treatment design was a 2 × 2 × 2 factorial with high and low levels of quantitative resistance (B73 × Mo17 and A619 × A632, respectively), qualitative resistance (*Ht*1 and *ht*1), and *E. turcicum* races 1 and 2 as factors. In 1985, A619 *Ht*2 × A632 also was evaluated. Seed was produced in 1983 and 1984 by crossing near-isolines of B73 and B73*Ht*1; Mo17 and Mo17*Ht*1; A632 and A632*Ht*1; and, A619, A619*Ht*1 and A619*Ht*2 in the following combinations: B73 × Mo17, B73*Ht*1 × Mo17*Ht*1, A619 × A632, A619*Ht*1 × A632*Ht*1, and A619*Ht*2 × A632.

Planting dates were 2 June 1984 and 10 May 1985. Planting rates were approximately 108,000 kernels per hectare. Plots were thinned to populations of approximately 54,000 plants per hectare. Plots included 16 rows that were 12.2 m in length, spaced 76 cm apart, and oriented east to west. To reduce interplot interference, plots were separated from each other on all sides by 6.1 m of a hybrid with the *Ht*1 gene and relatively high levels of quantitative resistance to NLB, FRMo17cms × FR3019rfc (Illinois Foundation Seeds, Inc., Champaign).

A pattern to study disease gradients was established in each plot

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(Fig. 1). A focus of infection was established on two plants approximately 76 cm from the southwest (windward) corner in each plot. Plants were tagged for evaluation at 0.8, 2.3, 4.6, 6.9, and 9.2 m from the focus in the compass directions: 0°, 23°, 45°, 68°, and 90°. Thus, there were five subsamples per plot for each distance except for 0.8 m for which there were two subsamples (0° and 90°).

The foci were inoculated with either race 1 or 2 of *E. turcicum* when plants were at the six- to nine-leaf stage, 5 July 1984 and 20 June 1985. In 1984, 8 ml of a spore suspension of approximately 2 $\times 10^3$ conidia per milliliter were injected into plant whorls. In 1985, approximately 2 g of ground leaf tissue that had been infected by either race 1 or 2 were placed in whorls before injecting the spore suspension. Inoculum was prepared from 2-wk-old cultures of *E. turcicum* grown on lactose-casein hydrosylate agar. Conidia were loosened with a rubber policeman. Tap water was used to dilute the inoculum.

Plants were rated weekly for percentage of the total leaf area diseased (NLB severity) and lesion type. Ratings were based on a standard diagram modified from Elliott and Jenkins (2). Data were analyzed by distance and time using analysis of variance ($P = 0.05$). When variances were not homogeneous, data were arc sine-transformed to stabilize variances. The spread of NLB was evaluated in each year for resistant and susceptible reactions. Data were fitted by least-square linear regressions to Gregory's (4) and Kiyosawa and Shiyomi's (12) linear models of disease gradients.

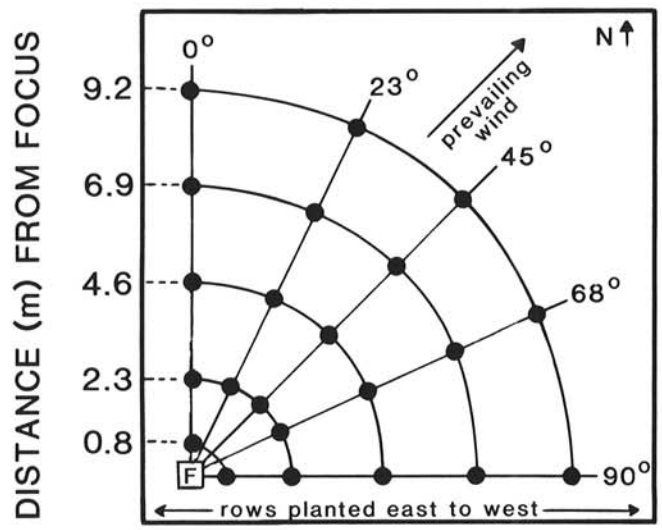

Fig. 1. Sampling pattern used to study northern leaf blight severity gradients from a focus (F) of infection. Plants sampled at all points (•) where rays and arcs interesect.

RESULTS

Susceptible- or resistant-type NLB lesions were observed first at the foci 2–3 wk after inoculation with race 2 in 1984 and with both races in 1985. In 1984, NLB did not develop from initial inoculations with race 1. Foci were reinoculated with race 1 on 21 July 1984, but disease development was delayed and insufficient to include in the analyses. Maximum NLB severity at the foci ranged from 20 to 50% for race 2 in 1984 and from 6 to 70% for both races in 1985 (Fig. 2).

In 1984, the main effect of quantitative resistance was signifcant at all times and distances for which NLB severity was greater than 4% on at least one genotype. The main effect of qualitative resistance and the quantitative × qualitative interaction term were

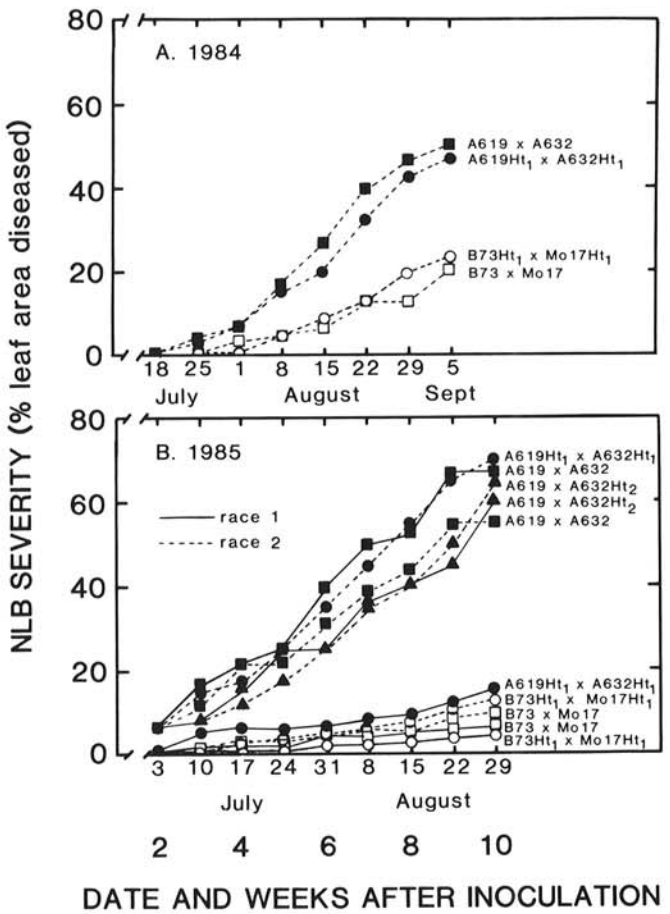

Fig. 2. Development of northern leaf blight at a focus of infection in which maize plants were inoculated with **A,** *Exserohilum turcicum* race 2 in 1984 and **B,** Either *E. turcicum* race 1 or 2 in 1985.

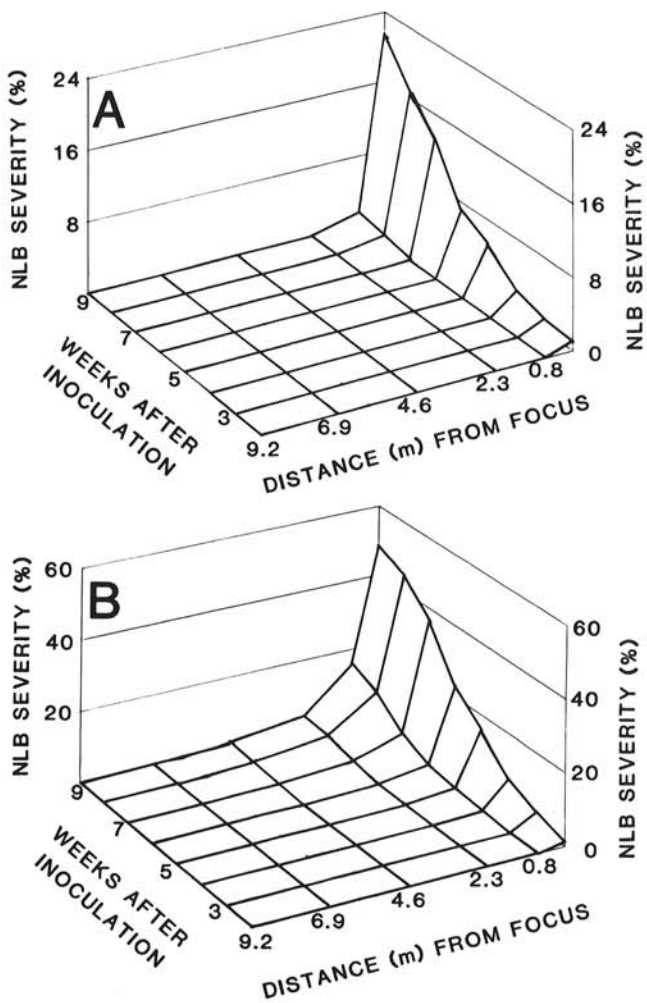

Fig. 3. Spatial and temporal development of northern leaf blight from a focus of infection in 1984 for maize hybrid-*Exserohilum turcicum* race combinations that were **A,** Resistant; and **B,** susceptible.

not significant. Consequently, genotypes were classified into two groups based on resistance to race 2 (Fig. 3). A619 × A632 and A619*Ht*1 × A632*Ht*1 were susceptible and did not differ significantly. B73 × Mo17 and B73*Ht*1 × Mo17*Ht*1 were resistant and did not differ significantly.

In 1985, for the factorial treatment analysis, the main effects of quantitative resistance, qualitative resistance, and races; the first-order interaction of qualitative resistance × races, and the second-order interaction of quantitative resistance × qualitative resistance × races were significant at all times and distances for which NLB severity was greater than 3% on at least one genotype. In addition, NLB severity on A619*Ht*2 × A632 was significantly different than severity on A619*Ht*1 × A632*Ht*1 inoculated with race 1 and both versions of B73 × Mo17 inoculated with both races. Genotype-race combinations again were classified into two groups (Fig. 4). A619 × A632 and A619*Ht*2 × A632 inoculated with either race, and A619*Ht*1 × A632*Ht*1 inoculated with race 2 did not differ significantly and were classified as susceptible (Fig. 4A). The greatest variation among susceptible hybrids was between A619 × A632 and A619*Ht*2 × A632 inoculated with race 1 (Fig. 5). Both versions of B73 × Mo17 inoculated with either race, and A619*Ht*1 × A632*Ht*1 inoculated with race 1 did not differ significantly and were classified as resistant (Fig. 4B).

The spread of NLB was limited on both resistant and susceptible hybrids (Figs. 3 and 4). On the resistant hybrids in both years, NLB severity was below 3% at all times and distances except for the foci (Figs. 3B and 4B). On the susceptible hybrids in 1984, NLB severity at 9 wk after inoculation of the foci averaged 16%, 4%, 1.5%, and 0.5% at 0.8, 2.3, 4.6, and 6.9 m from the foci, respectively (Fig. 3A). No disease was observed at 9.2 m from the foci. On the susceptible hybrids in 1985, NLB severity was below 20% at all times and distances from the foci except for 8, 9, and 10 wk after inoculation at 0.8 m from the foci for which mean NLB severity averaged 23%, 31%, and 35%, respectively (Fig. 4A). NLB severity was slightly higher at all distances within rows (90° rays) than between rows (0° rays) which may have resulted from row orientation and/or wind direction.

Neither Gregory's or Kiyosawa's linear models accurately described NLB severity gradients although acceptable results were obtained when data for the susceptible hybrids in 1985 were fit to Gregory's model (Fig. 6). The slope of disease gradients from Gregory's model for susceptible hybrids in 1985 ranged from −0.913 to −1.179. Residuals from both models usually indicated underestimation of severity at distances nearest and furthest from the focus. At intermediate distances, severity was overestimated.

DISCUSSION

Qualitative and quantitative resistance in the maize genotypes evaluated in this study had similar effects on the spread and development of NLB. Disease severity and spread were similar within groups of resistant and susceptible genotype-race combinations in both years.

The significant first- and second-order interaction terms in 1985 can be explained by the effects of the *Ht*1 gene on races 1 and 2 in the two genetic backgrounds. The *Ht*1 gene had very little effect on the severity of NLB caused by race 1 or 2 in B73 × Mo17 due to the high level of quantitative resistance in this hybrid. Although the effect of the *Ht*1 gene against race 1 was not significant in this

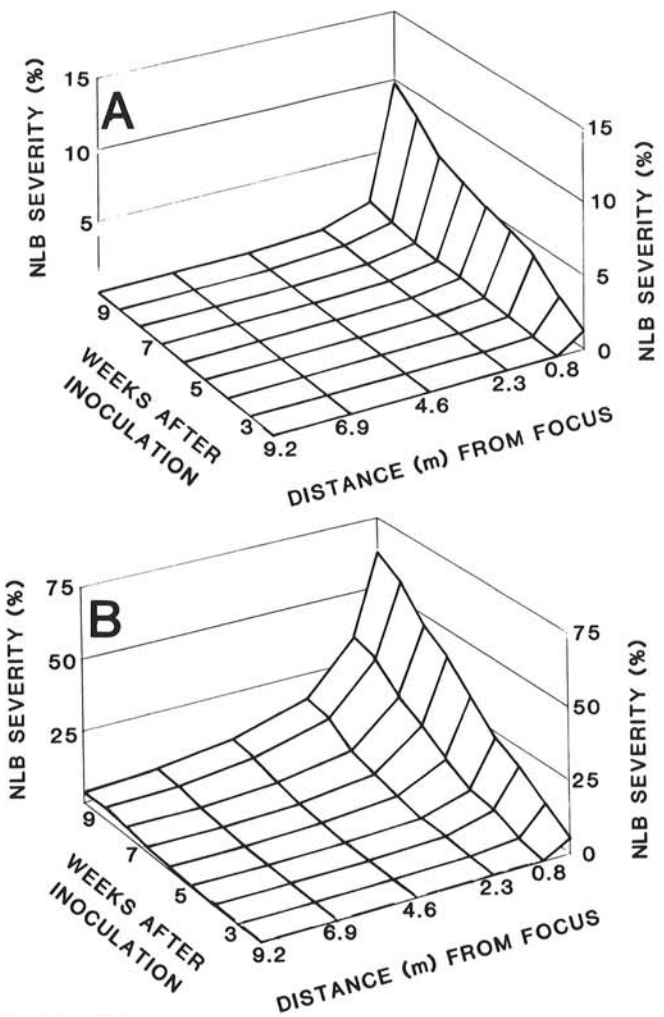

Fig. 4. Spatial and temporal development of northern leaf blight from a focus of infection in 1985 for maize hybrid-*Exserohilum turcicum* race combinations that were **A,** Resistant; and **B,** Susceptible.

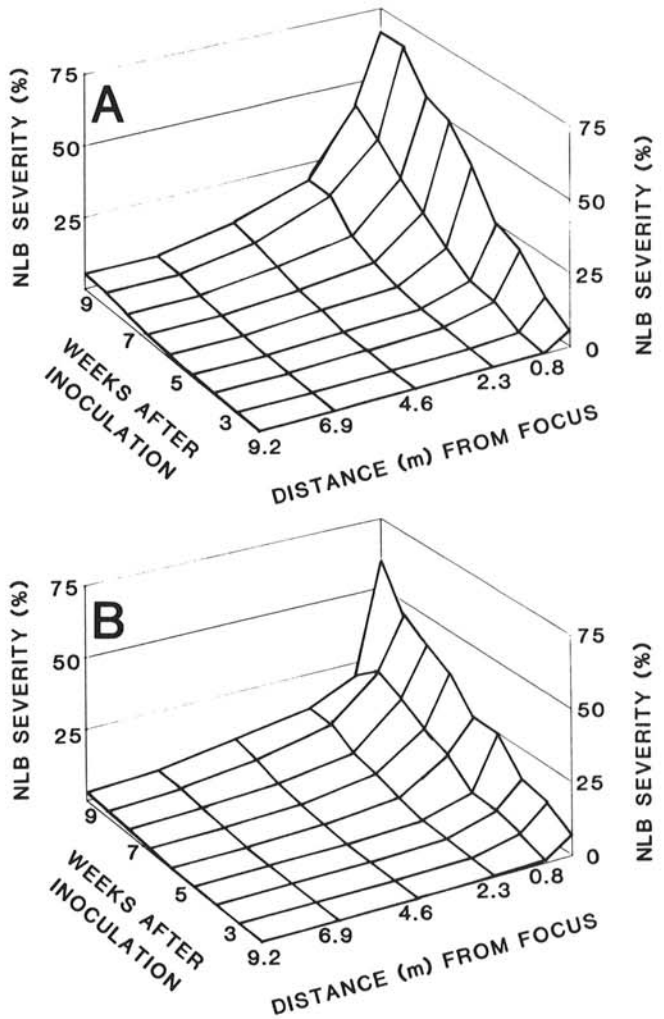

Fig. 5. Spatial and temporal development of northern leaf blight for two maize hybrids that were susceptible to *Exserohilum turcicum* race 1 in 1985: **A,** A619 × A632, and **B,** A619Ht_2 × A632.

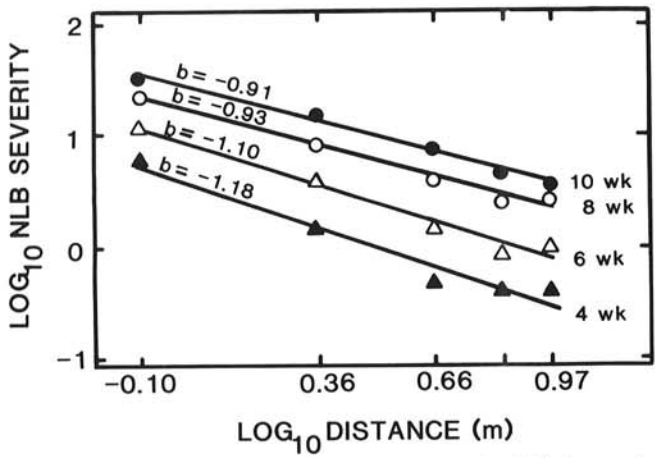

Fig. 6. Gregory's (4) models to describe northern leaf blight severity gradients from a focus of infection for susceptible maize hybrids in 1985.

hybrid, it was consistent and similar to significant results reported previously (15). In A619 × A632, which possesses little quantitative resistance to NLB, the *Ht*1 gene was effective against race 1 but had no detectable effect on race 2.

The *Ht*2 gene in A619 × A632 did not affect the development or spread of NLB caused by race 1 or 2. The *Ht*2 gene has been effective against race 2 in most genotypes (22,24) although identification of *Ht*2 resistance has been difficult in segregating backcross generations of some inbred lines, such as A632 (J. M. Perkins, *unpublished*). Possibly, modifier genes affect the expression of *Ht*2 in certain genotypes.

The small and subtle effects of *Ht*1 on NLB observed on inoculated plants in other studies were not detected. The *Ht*1 gene did not detectably affect the development of NLB caused by race 2 on A619 × A632 in this study; however, Leath and Pedersen (15) observed small differences in lesion size and number of lesions caused by race 2 among versions of A619 × A632 nearly isogenic for *Ht*1. Also, *Ht*1 and quantitative resistance were similar in effect on NLB spread and development in this study, whereas in other studies the degree of resistance conveyed by *Ht* genes has varied slightly in different genetic backgrounds (7,20). For example, Hooker and Kim (7) observed F_2 population mean leaf blight ratings (0 to 5) of 4.3, 2.2, 2.7, and 0.9 for B14 × 187-2, B14*Ht*1 × 187-2*Ht*1, B37 × H55, and B37*Ht*1 × H55*Ht*1, respectively. Thus, the effect of *Ht*1 appeared to be additive to the quantitative resistance in B37 × H55. A619*Ht*1 × A632*Ht*1 inoculated with race 1 was not different than either version of B73 × Mo17 in our study; therefore, additivity between quantitative and qualitative resistance was not observed. Nonetheless, of the five hybrid-race combinations considered resistant in 1985, mean NLB severity was consistently (but not significantly) higher on A619*Ht*1 × A632*Ht*1 inoculated with race 1 than on B73*Ht*1 × Mo17*Ht*1. Possibly, evaluations made under the severe disease pressure associated with inoculations allow for the detection of subtle differences that were not observed when disease developed from inoculum spread from foci.

The limited spread of NLB from the foci in resistant and susceptible hybrids was extremely similar to results of Raymundo who evaluated the spread of NLB caused by race 1 on A619 × A632 in 1975 and 1976 (21). Raymundo did not observe NLB severity above 20% at distances further than 0.8 m from a strip source of inoculum until 67 days after inoculation. Slope values from Gregory's model ranging from −0.829 to −1.167 in 1975 and from −1.081 to −1.629 in 1976 were similar to those observed in this study. In A619*Ht*1 × A632*Ht*1, NLB severity was not above 7% at any distance from the strip source at any time. Thus, our results and those of Raymundo suggest that the spread of NLB in hybrids with quantitative or qualitative resistance is limited primarily to neighboring plants and would not reach levels that cause significant yield reduction (8). In susceptible hybrids, NLB severity did not reach damaging levels at distances further than 4 m in Raymundo's and our studies. Effective airborne dispersal of *E. turcicum* conidia in fields of maize hybrids appeared to be considerably shorter than spore dispersal of several other foliar pathogens. Turner and Hart also have suggested that the spread of *E. turcicum* conidia is localized and that spore production is affected significantly by host genotype (23). Consequently, large amounts of initial inoculum or conditions that result in excessive secondary inoculum production and dissemination are probably necessary for severe NLB epidemics.

LITERATURE CITED

1. Berger, R. D. 1970. Forecasting *Helminthosporium turcicum* attacks in Florida sweet corn. (Abstr.) Phytopathology 60:1284.
2. Elliott, C., and Jenkins, M. T. 1946. *Helminthosporium turcicum* leaf blight of corn. Phytopathology 36:660-666.
3. Gevers, H. O. 1975. A new major gene for resistance to *Helminthosporium turcicum* leaf blight of maize. Plant Dis. Rep. 59:296-299.
4. Gregory, P. H. 1968. Interpreting plant disease dispersal gradients. Annu. Rev. Phytopathol. 6:189-212.
5. Hooker, A. L. 1963. Monogenic resistance in *Zea mays* to *Helminthosporium turcicum*. Crop Sci. 3:381-383.
6. Hooker, A. L. 1977. A second major gene locus in corn for chlorotic-lesion resistance to *Helminthosporium turcicum*. Crop Sci. 17:132-135.
7. Hooker, A. L., and Kim, S. K. 1973. Monogenic and multigenic resistance to *Helminthosporium turcicum* in corn. Plant Dis. Rep. 57:586-589.
8. Hooker, A. L., and Perkins, J. M. 1980. Helminthosporium leaf blights of corn—the state of the art. Pages 68-87 in: Proc. 35th Annu. Corn and Sorghum Res. Conf., Chicago, IL.
9. Jenkins, M. T., and Robert, A. L. 1952. Inheritance of resistance to the leaf blight of corn caused by *Helminthosporium turcicum*. Agron. J. 44:136-140.
10. Jenkins, M. T., Robert, A. L., and Findley, W. R., Jr. 1954. Recurrent selection as a method for concentrating genes for resistance to *Helminthosporium turcicum* leaf blight in corn. Agron. J. 46:89-94.
11. Jordan, E. G., Perkins, J. M., Schall, R. A., and Pedersen, W. L. 1983. Occurrence of race 2 of *Exserohilum turcicum* on corn in the central and eastern United States. Plant Dis. 67:1163-1165.
12. Kiyosawa, S., and Shiyomi, M. 1972. A theoretical evaluation of the effect of mixing a resistant variety with a susceptible variety for controlling plant diseases. Ann. Phytopathol. Soc. Jpn. 38:41-51.
13. Leach, C. M., Fullerton, R. A., and Young, K. 1977. Northern leaf blight of maize in New Zealand: Release and dispersal of conidia of *Drechsler turcica*. Phytopathology 67:380-387.
14. Leath, S., and Pedersen, W. L. 1984. An inoculation technique to detect the *Ht*N gene in inbred lines of corn under greenhouse conditions. Plant Dis. 67:520-522.
15. Leath, S., and Pedersen, W. L. 1986. Differences in resistance between maize hybrids with or without the Ht_1 gene when infected with *Exserohilum turcicum* race 2. Phytopathology 76:257-260.
16. Leath, S., and Pedersen, W. L. 1986. Effects of the *Ht*, *Ht*2, and/or *Ht*3 genes in three maize inbreds on quantitative resistance to *Exserohilum turcicum* race 2. Plant Dis. 70:529-531.
17. Levy, Y., and Cohen, Y. 1980. Sporulation of *Helminthosporium turcicum* on sweet corn: Effects of temperature and dew period. Can. J. Plant Pathol. 2:65-69.
18. Levy, Y., and Cohen, Y. 1983. Biotic and environmental factors affecting infection of sweet corn with *Exserohilum turcicum*. Phytopathology 73:722-725.
19. Miles, J. W., Dudley, J. W., White, D. G., and Lambert, R. J. 1980. Improving corn population for grain yield and resistance to leaf blight and stalk rot. Crop Sci. 20:247-251.
20. 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.
21. Raymundo, A. D. 1978. Epidemiology of northern leaf blight as affected by host resistance and yield losses following simulated epidemics. Ph.D. thesis, University of Illinois, Urbana-Champaign. 110 pp.
22. Smith, D. R., and Kinsey, J. R. 1980. Further physiologic specialization of *Helminthosporium turcicum*. Plant Dis. 64:779-781.
23. Turner, M. T., and Hart, K. 1975. Field spore production of *Helminthosporium turcicum* on *Zea mays* with and without monogenic resistance. Phytopathology 65:735-736.
24. 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.