

Effects of Races 0 and 1 of *Exserohilum turcicum* on Sweet Corn Hybrids Differing for *Ht*- and Partial Resistance to Northern Leaf Blight

J. K. PATAKY, Department of Plant Pathology, University of Illinois, Urbana 61801

ABSTRACT

Pataky, J. K. 1994. Effects of races 0 and 1 of *Exserohilum turcicum* on sweet corn hybrids differing for *Ht*- and partial resistance to northern leaf blight. Plant Dis. 78:1189-1193.

Field studies were done in 1988, 1989, and 1990 to compare the effects of races 0 and 1 of *Exserohilum turcicum* on sweet corn hybrids with different levels of *Ht*- and partial resistance to northern leaf blight (NLB). NLB decreased yields of susceptible hybrids in spite of chlorotic lesions, conditioned by *Ht*-resistance; but NLB had little effect on yields of hybrids with partial resistance. High levels of partial resistance in hybrids with or without *Ht* genes limited disease severity. Yields of partially resistant *Ht*-hybrids were within 10% of and not different from noninoculated controls. Yields of partially resistant hybrids without the gene *Ht1* were not different from controls except for one comparison. Severity of NLB was less than 20% on partially resistant hybrids with or without *Ht*-genes and greater than 19% for hybrids that did not have partial resistance. Yields of susceptible hybrids without the gene *Ht1* were 6–51% less than controls and did not differ between plots inoculated with races 0 or 1. Yields of susceptible hybrids with the gene *Ht1* were 6–18% less than controls and did not differ between plots inoculated with races 0 or 1.

Additional keywords: *Helminthosporium turcicum*, maize, northern corn leaf blight, *Zea mays*

Northern leaf blight (NLB), caused by *Exserohilum turcicum* (Pass.) K.J. Leonard & E.G. Suggs (teleomorph *Setosphaeria turcica* (Luttrell) K.J. Leonard & E.G. Suggs), is one of the most prevalent diseases of sweet corn (*Zea mays* L.). Epidemics of NLB occur regularly in the spring in south Florida

on *sh2* hybrids grown for shipping. Although the occurrence of NLB epidemics is variable on sweet corn grown in the midwestern United States, NLB can be severe in wet years such as 1992 and 1993. The reactions to NLB of some 500 commercially available sweet corn hybrids range from highly resistant to highly susceptible, with both *Ht*- (single gene) and partial resistance being expressed (20–23).

Early in the 1960s, the gene *Ht* was identified from Ladyfinger popcorn and a dent corn inbred, GE440 (5,32). The gene, presently referred to as *Ht1*, conditioned a previously unreported type of chlorotic-lesion resistance (4). Subsequently, additional sources of chlorotic-lesion resistance were reported (7,8,24,

27,35). The gene *Ht2* was identified in NN14, an inbred from Australia (6), and the gene *Ht3* was backcrossed into *Z. mays* from *Tripsacum floridanum* (10). In most backgrounds, the genes *Ht2* and *Ht3* conditioned a lower level of chlorotic-lesion resistance than did *Ht1* (10). A source of monogenic resistance (*HtN*) that prolonged incubation and latent periods without causing chlorotic lesions was identified in South Africa from the Mexican variety Pepitilla (3).

The gene *Ht1* was used extensively in dent corn grown in North America in the 1970s. A new biotype of *E. turcicum* virulent against *Ht1* had been designated race 2 (virulence formula: 0/*Ht1*) when it was first observed in Hawaii in 1974 (1). Virulence of race 2 on corn with the gene *Ht1* was inherited as a monogenic trait (17). In 1978, this biotype was reported from the cornbelt of the United States (31). Subsequently, biotypes virulent on corn with the genes *Ht2*, *Ht3*, and *HtN* were reported (28,29). Presently, the system of racial nomenclature for *E. turcicum* designates races based on virulence formulae (16), e.g., race 0 (*Ht1*,*Ht2*,*Ht3*,*HtN*/0), race 1 (*Ht2*,*Ht3*,*HtN*/*Ht1*), race 23 (*Ht1*,*HtN*/*Ht2*,*Ht3*), race 23N (*Ht1*/*Ht2*,*Ht3*,*HtN*), etc. Several previously unreported combinations of virulence in *E. turcicum* were observed in greenhouse evaluations of progeny from matings of races 1 and 23N (2), and previously unreported combinations of virulence were found among isolates collected in China, Mexico, Uganda, and Zambia (34).

Research supported by the Illinois Agricultural Experiment Station, University of Illinois, Urbana, as part of project 68-0323 and by the United States-Israel Binational Agricultural Research and Development Fund (BARD), project US 1213-86.

Accepted for publication 9 September 1994.

© 1994 The American Phytopathological Society

Prior to the discovery of monogenic *Ht*-resistance, much of the research on NLB focused on partial (i.e., polygenic) resistance, which was highly heritable (11–13). In some cases, resistance was controlled by many genes, although some genes had major effects (12,13). In other cases, partial resistance was conditioned by relatively few genes (11). The expression of *Ht* genes was influenced considerably by genetic backgrounds when compared in genotypes with different levels of partial resistance (9,15,24–26). Ullstrup (33) reported a greater average reduction in yield due to NLB on *Ht*-resistant hybrids compared to partially resistant hybrids, presumably because of the extensive chlorosis associated with gene expression in extremely susceptible backgrounds.

In sweet corn, reactions of commercial hybrids to NLB vary from highly resistant to highly susceptible (20–23). Many hybrids have *Ht*-gene and/or partial resistance, although some hybrids with chlorotic-lesion reactions were rated susceptible based on more than 25% symptomatic leaf area (20,23).

Yield of sweet corn was reduced from 8 to 14% when severity of NLB was about 25% (18,22); thus, yield reductions may occur on sweet corn hybrids with extremely susceptible backgrounds despite having *Ht*-genes. Likewise, race 1 of *E. turcicum* is prevalent in many areas where sweet corn is grown (14,19); and thus, the gene *Ht1* may be of limited value in deterring development of NLB and in preventing reductions in yield. This paper reports on the effects of races 0 and 1 of *E. turcicum* on sweet corn hybrids with different levels of partial and *Ht*-resistance.

MATERIALS AND METHODS

Experiments were done at the University of Illinois Agronomy/Plant Pathology South Farm in 1988, 1989, and 1990.

Soils were a Flannigan silt loam in 1988 and 1989, and a Drummer silt loam in 1990. Planting dates were 27 May 1988, 8 June 1989, and 30 May 1990.

A total of eleven sweet corn hybrids were grown. Hybrids were selected based on reactions to NLB. Florida Staysweet, Sch 5009, and SummerSweet 7210 have both *Ht*- and partial resistance to NLB. Honey n Frost and XPH 2670 have partial resistance but do not have the gene *Ht1*. Sch 4055, Sch 4064, and Shield Crest have the gene *Ht1* with relatively little partial resistance. FMX 235, Stylish, and Shield Crest are susceptible. Based on 3:1 ratios of resistant:susceptible reactions to race 0 in the F₂ generation, the *Ht*-resistant hybrids appeared to be heterozygous for *Ht1*, except for SummerSweet 7210, for which all F₂ plants were resistant.

The experimental design was a split plot of a randomized complete block with four replicates. The treatment design was a factorial of hybrids and three inoculation treatments. Six hybrids were grown in 1988, and 10 hybrids were grown in 1989 and 1990. The three inoculation treatments were races 0 and 1 of *E. turcicum* and a noninoculated control. Hybrids were planted in main plots, and inoculation treatments were applied to subplots. Each experimental unit consisted of an eight-row plot with the middle two rows treated and the outer six rows as borders. Each row was approximately 3.5 m long with 16 plants per row. Rows were spaced 0.76 m apart.

Plants were inoculated by spraying a conidial suspension (approximately 10³ conidia per milliliter) of race 0 or race 1 isolates directly into leaf whorls when plants were at the 3- to 7-leaf stages. Inoculation dates were 29 June and 1 July 1988; 26 and 28 June and 3 and 6 July 1989; and 21, 25, and 28 June and 2 and 5 July 1990. Plants were inoculated several times in 1989 and 1990, until

sporulating lesions were observed on lower leaves. Several isolates of race 0 or race 1 were used each year for inoculum, to be as heterogeneous within races as possible. Isolates were cultured and inoculum was prepared as described previously (22).

Severity of NLB was rated within 1 wk of harvest each year by the author and another person. Assessments of severity were based on necrotic leaf tissue. Chlorosis resulting from reactions of race 0 on *Ht1* hybrids was not included as diseased tissue. All leaves from five representative plants in each experimental unit were rated separately using a disease assessment diagram reported previously (18). The net blotch program of DISTRAIN (30) was used as a training tool prior to ratings. Within 1 wk of harvest, leaf area was measured from five healthy plants in the border rows of each control plot. Leaves were removed from plants, labeled for position from the primary ear, and measured on a Li-Cor leaf area meter. The percentage of the total leaf area comprised by leaves at each position from the primary ear was calculated for each hybrid. Severity of NLB was then calculated as a weighted sum: $\sum_{i=1}^n (NLB_i \times LA_i)$, in which NLB_i = severity of NLB on the *i*th leaf, LA_i = proportion of the total leaf area accounted for by the *i*th leaf; and n = the total number of leaves. Plot ratings of severity also were done using a scale modified (20) from that of Elliott and Jenkins.

Primary ears were harvested by hand at fresh market maturity (about 20 days after mid-silk) from 10 consecutive plants in each of the two treatment rows per experimental unit. Ears were weighed after husking. Harvest dates varied among hybrids due to maturity differences, beginning 2 August 1988, 18 August 1989, and 15 August 1990 and ending 12 August 1988, 25 August 1989,

Table 1. Total leaf area (cm²) within 1 wk of harvest and percent leaf area accounted for by each leaf of sweet corn hybrids

| Resistance to NLB ^a Hybrid | Leaf area | Leaves below primary ear | | | | | E ^b | Leaves above primary ear | | | | | |
|--|--------------------|--------------------------|----|----|----|----|----------------|--------------------------|----|----|----|----|----|
| | | B5 | B4 | B3 | B2 | B1 | | T1 | T2 | T3 | T4 | T5 | T6 |
| <i>Ht</i> and partial resistance | | | | | | | | | | | | | |
| Florida Staysweet | 4,478 ^c | 2 ^d | 6 | 9 | 12 | 13 | 13 | 13 | 11 | 10 | 7 | 4 | 1 |
| Sch 5009 | 4,095 | 2 | 4 | 8 | 10 | 12 | 12 | 12 | 12 | 11 | 9 | 6 | 1 |
| SummerSweet 7210 | 3,749 | <1 | 3 | 7 | 11 | 12 | 14 | 14 | 13 | 11 | 9 | 5 | 1 |
| Partial resistance | | | | | | | | | | | | | |
| Honey n Frost | 3,557 | <1 | 4 | 8 | 10 | 12 | 13 | 14 | 13 | 11 | 8 | 4 | 2 |
| XPH 2670 | 3,245 | 1 | 3 | 7 | 11 | 12 | 14 | 14 | 13 | 12 | 9 | 4 | <1 |
| <i>Ht</i> -resistance | | | | | | | | | | | | | |
| Sch 4055 | 3,042 | 1 | 5 | 8 | 11 | 12 | 13 | 12 | 14 | 11 | 7 | 5 | 1 |
| Sch 4064 | 3,597 | 1 | 4 | 7 | 9 | 11 | 13 | 13 | 12 | 11 | 9 | 6 | 3 |
| Shield Crest | 3,695 | 3 | 6 | 9 | 10 | 11 | 12 | 12 | 11 | 9 | 8 | 6 | 2 |
| Susceptible | | | | | | | | | | | | | |
| FMX 235 | 2,515 | 2 | 5 | 5 | 11 | 13 | 16 | 17 | 15 | 12 | 7 | 2 | <1 |
| Stylish | 3,244 | 1 | 5 | 5 | 8 | 13 | 14 | 15 | 14 | 12 | 10 | 6 | 1 |
| Sunset | 3,063 | 2 | 5 | 5 | 10 | 14 | 15 | 14 | 14 | 12 | 9 | 4 | <1 |

^aNorthern leaf blight.

^bE-leaf at the primary ear.

^cLeaf area (cm²) averaged from five plants per experimental unit from the control treatment in 1988, 1989, and 1990.

^dPercent total leaf area accounted for by each leaf according to leaf position from the primary ear.

and 23 August 1990. Yields from plots inoculated with races 0 and 1 were converted to a percentage of the noninoculated control treatment of the same hybrid.

Data were analyzed by analysis of variance (ANOVA), and appropriate mean separation tests (BLSD) were used to compare hybrids, races, and combinations of hybrids and races. The ANOVA for actual yield included the three inoculation treatments, but other ANOVAs (i.e., those for disease measurements and percent yield) did not include the control treatment because of lack of variation (i.e., by definition, percent yield was 100% for control treatments). Correlations between NLB severity and percent yield were done on means from experimental units.

RESULTS

Leaf area. Total leaf area differed slightly among years, probably due to differing environments. Averaged over years, leaf areas ranged from 2,515 cm² for FMX 235 to 4,478 cm² for Florida Staysweet (Table 1). The percentage of the total leaf area accounted for by leaf position was relatively similar among the 11 hybrids. The three lowermost and uppermost leaves comprised only 8–18% and 10–18% of the total leaf area, respectively; whereas the three leaves nearest the primary ear (the primary ear leaf and the leaves immediately above and below the primary ear) accounted for 35–46% of the total leaf area (Table 1). Five leaves nearest the primary ear comprised 56–72% of the total leaf area. Seven leaves nearest the primary ear comprised 74–89% of the total leaf area.

Severity of NLB. Severity of NLB ranged from 2 to 43%, 3 to 56%, and 1 to 49% in the 1988, 1989, and 1990 trials, respectively (Table 2). Main effects of hybrids comprised 80, 99, and 97% of the treatment sums of squares in the ANOVAs of NLB severity for 1988, 1989, and 1990, respectively. Severity was 20% or less for all hybrids with partial resistance. Severity was 19% or above for all hybrids that did not have partial resistance.

The hybrid-by-race interaction term was significant at the 0.01 level in each trial. In 1988 and 1990, NLB severity resulting from race 1 was 6–24% greater than that from race 0 on susceptible hybrids with the *Ht* gene (i.e., Sch 4055, Sch 4064, and Shield Crest). Severity of NLB from race 1 also was 5–6% greater than that from race 0 for two of the five comparisons of partially resistant *Ht*-hybrids (SummerSweet 7210 in 1988 and Florida Staysweet in 1990). Severity did not differ between races 0 and 1 in 1988 and 1990 for hybrids that did not have *Ht*-resistance (Honey n Frost, XPH 2670, FMX 235, Stylish, and Sunset). In 1989, NLB severity did not differ between races 0 and 1 for hybrids with *Ht*-resis-

tance, but severity resulting from race 0 was 3–6% greater than that from race 1 for three of the five hybrids that did not have *Ht*-resistance (Honey n Frost, XPH 2670, and Sunset). Apparently, the isolates of race 0 used in 1989 were slightly more aggressive than those of race 1; whereas in 1988 and 1990, isolates of the two races had relatively equal aggressiveness, as indicated by equal disease severity on hybrids without the gene *Ht1*.

Severity of NLB calculated from assessments of individual leaves was relatively well correlated with ratings from entire plots. Correlation coefficients ranged from 0.74 in 1988 to 0.94 in 1990. In 1988 and 1989, severity assessments from plot ratings were similar to weighted sums calculated from individual leaves; however, in 1990, plot ratings were about 10–15% higher than the weighted sums even though the correlation coefficient was slightly higher.

Yield. Yields in noninoculated control plots ranged from 2,194 to 2,841 kg per plot, 2,818 to 4,532 kg per plot, and 2,741 to 4,644 kg per plot in 1988, 1989, and 1990, respectively (Table 3). Yields were lower in 1988 as the result of a drought. Yields from plots inoculated with races 0 and 1 ranged from 86 to 102%, 74 to 103%, and 49 to 103% of those from noninoculated plots of the same hybrid in 1988, 1989, and 1990, respectively (Table 4). Coefficients of variation ranged from 8 to 16% and 8 to 21% for actual yields and percent yields, respectively.

In the ANOVAs of percent yields, hybrids were the only significant source of variation among treatments. Yields of partially resistant *Ht*-hybrids inoculated with races 0 or 1 were within 10% of, and not significantly different from, the noninoculated controls (Table 4). Yields of inoculated, partially resistant hybrids without the *Ht*-gene were not signifi-

Table 2. Severity of northern leaf blight (NLB) caused by race 0 or 1 of *Exserohilum turcicum* on sweet corn hybrids differing for *Ht*- and partial resistance

| Resistance to NLB Hybrid | Severity of NLB (%) ^a | | | | | |
|------------------------------------|----------------------------------|--------|--------|--------|--------|-----------------|
| | 1988 | | 1989 | | 1990 | |
| | Race 0 | Race 1 | Race 0 | Race 1 | Race 0 | Race 1 |
| <i>Ht</i> - and partial resistance | | | | | | |
| Florida Staysweet | ... | ... | 19 | 20 | 4 | 9* ^b |
| Sch 5009 | 2 | 2 | 3 | 3 | 1 | 2 |
| SummerSweet 7210 | 4 | 10* | 6 | 6 | 2 | 2 |
| Partial resistance | | | | | | |
| Honey n Frost | ... | ... | 11* | 6 | 7 | 5 |
| XPH 2670 | ... | ... | 18* | 15 | 9 | 7 |
| <i>Ht</i> resistance | | | | | | |
| Sch 4055 | 19 | 43* | ... | ... | ... | ... |
| Sch 4064 | 22 | 28* | 49 | 48 | 26 | 39* |
| Shield Crest | 29 | 39* | 44 | 42 | 28 | 34* |
| Susceptible | | | | | | |
| FMX 235 | 19 | 19 | 47 | 46 | 31 | 35 |
| Stylish | ... | ... | 56 | 54 | 49 | 45 |
| Sunset | ... | ... | 48* | 42 | 39 | 35 |
| BLSD <i>k</i> = 100 | 5.3 | | 2.3 | | 4.1 | |

^aSeverity of NLB = percent leaf area infected based on visual assessments of individual leaves and adjusted for the proportion of total leaf area from each leaf (See Table 1 and text).

^b* = Significant difference (*P* = 0.05) between races 0 and 1 compared within hybrids.

Table 3. Yield (kg per plot) from noninoculated control treatments of sweet corn hybrids differing for *Ht*- and partial resistance to northern leaf blight (NLB)

| Resistance to NLB Hybrid | 1988 | 1989 | 1990 |
|------------------------------------|-------|-------|-------|
| <i>Ht</i> - and partial resistance | | | |
| Florida Staysweet | ... | 3,567 | 3,447 |
| Sch 5009 | 2,682 | 4,260 | 3,780 |
| SummerSweet 7210 | 2,633 | 3,862 | 4,262 |
| Partial resistance | | | |
| Honey n Frost | ... | 3,828 | 4,383 |
| XPH 2670 | ... | 4,211 | 4,644 |
| <i>Ht</i> -resistance | | | |
| Sch 4055 | 2,194 | ... | ... |
| Sch 4064 | 2,572 | 2,818 | 3,991 |
| Shield Crest | 2,841 | 4,434 | 4,323 |
| Susceptible | | | |
| FMX 235 | 2,500 | 3,927 | 4,765 |
| Stylish | ... | 4,029 | 2,741 |
| Sunset | ... | 4,532 | 4,500 |
| BLSD (<i>k</i> = 100) | 162.2 | 608.2 | 469.4 |

cantly different from controls, except for XPH 2670 inoculated with race 1 in 1990, for which the yield was 81% of the control. Yields of inoculated, susceptible hybrids with the *Ht*-gene ranged from 82 to 96% of noninoculated controls and were significantly less than controls for 8 of 14 comparisons. Yields of susceptible hybrids ranged from 49 to 94% of controls and were significantly less than controls for 11 of 14 comparisons.

Correlations of percent yield and NLB severity were relatively low: -0.53, -0.22, and -0.51 in 1988, 1989, and 1990, respectively.

DISCUSSION

Northern leaf blight decreased yields of susceptible sweet corn hybrids in spite of chlorotic-lesion *Ht*-resistance, but NLB had little effect on yields of hybrids with partial resistance. Yields of susceptible sweet corn hybrids inoculated with race 0 or 1 of *E. turcicum* were as much as 50% less than those of noninoculated control treatments. Susceptible hybrids with the *Ht1* gene for chlorotic-lesion resistance yielded 4-18% less than controls when inoculated with race 1, and 6-17% less than controls when inoculated with race 0. Yields of partially resistant hybrids were not significantly different from the controls except for XPH 2670 inoculated with race 1 in 1990.

High levels of partial resistance, with or without *Ht*-genes, appear to be an extremely effective method to limit the effects of NLB on sweet corn yield. Severity of NLB on partially resistant hybrids was consistently less than half of that on susceptible hybrids, regardless of the presence or absence of *Ht1*. As in a previous study (18), yields were not affected greatly by NLB when severity was less than about 15%. Approximately 9% (34 of 377) of the commercial hybrids evaluated in a sweet corn disease nursery in 1993 were rated resistant to NLB, with

less than 18% severity, compared to a trial mean of 31% and a range of 6-88% severity (23). Based on assessments of yields in this and previous studies (18,22), it is doubtful that yields of these hybrids would be affected substantially even under conditions conducive to severe NLB. Levels of partial resistance similar to those of the most resistant hybrid in this trial, Sch 5009, appear adequate to prevent yield reductions due to NLB under normal conditions in North America, even though higher levels of partial resistance exist in some sweet corn lines, and particularly in exotic (i.e., tropical) germ plasm.

Ht-resistance had a significant effect on NLB severity. Severity of NLB on *Ht*-resistant hybrids differed considerably (from 5 to 24%) when plants were inoculated with races 0 or 1 in 1988 and 1990; particularly when *Ht*-resistance was present in a susceptible background. However, yields of these hybrids were not significantly different between the race 0 and race 1 treatments. As Ullstrup suggested (33), extensive chlorosis associated with the expression of *Ht1* in extremely susceptible backgrounds may limit the benefits of this type of resistance when abundant secondary inoculum results in many infections. When secondary inoculum is less abundant, *Ht*-resistance may perform better by limiting sporulation (25) and subsequent infection. Also, yields were more variable than severity in these trials, so effects of races 0 and 1 on yield were more difficult to detect than their effects on disease severity.

Many sweet corn breeders are using simply inherited resistances, such as the genes *Rp1d* to control common rust (caused by *Puccinia sorghi* Schwein.), *Rpp9* to control southern rust (caused by *Puccinia polysora* Underw.), *Mdm1* to control maize dwarf mosaic virus, and the *Ht*-genes to control NLB. Used in

sweet corn in North America, these resistances may have a prolonged period of effectiveness. Approximately 300,000 ha of sweet corn grown in North America may not exert sufficient pressure to select for specific virulence in pathogen populations that reproduce primarily on approximately 30,000,000 ha of field corn. Single-gene sources of resistance usually are not used on a widespread basis in field corn. Although a prolonged usefulness of simply inherited resistance in sweet corn is conceivable, chlorotic-lesion resistance to *E. turcicum* conveyed by the genes *Ht1*, *Ht2*, and *Ht3* is of questionable value in the extremely susceptible backgrounds of some of the most popular sweet corn varieties. Since adequate levels of partial resistance are available in adapted sweet corn germ plasm and usually are highly heritable (11-13), a more logical approach would be to use those lines as sources from which to improve the resistance of susceptible lines. Hence, yields would not be affected by extensive areas of chlorotic tissue, and new combinations of virulence would not affect NLB resistance. Conversely, the gene *HtN* may be useful if selection for virulence does not occur rapidly and *HtN* is more effective in susceptible backgrounds than chlorotic-lesion *Ht*-resistance genes.

ACKNOWLEDGMENTS

I thank John Gantz, Payam Fallah Moghaddam, Shaun Zimmerman, and Shane Zimmerman for technical assistance; and Duane Jeffers (Ferry Morse Seed Company), Bryant Long (Abbott and Cobb, Inc.), Steve Marshall (Asgrow Seed Company), Pat Mosely (Illinois Foundation Seeds, Inc.), and Eric Sandsted (SeedWay, Inc.) for seed of sweet corn hybrids.

LITERATURE CITED

- Bergquist, R. R., and Masias, O. R. 1974. Physiologic specialization in *Trichometasphaeria turcica* f. sp. *zeae* and *Trichometasphaeria turcica* f. sp. *sorghii* in Hawaii. Phytopathology 64:645-649.
- Fallah Moghaddam, P., and Pataky, J. K. 1994. Reactions of isolates from matings of races 1 and 23N of *Exserohilum turcicum*. Plant Dis. 78:767-771.
- Gevers, H. O. 1975. A new major gene for resistance to *Helminthosporium turcicum* leaf blight of maize. Plant Dis. Rep. 59:296-299.
- Hooker, A. L. 1961. A new type of resistance in corn to *Helminthosporium turcicum*. Plant Dis. Rep. 45:780-781.
- Hooker, A. L. 1963. Monogenic resistance in *Zea mays* L. to *Helminthosporium turcicum*. Crop Sci. 3:381-383.
- Hooker, A. L. 1977. A second major gene locus in corn for chlorotic-lesion resistance to *Helminthosporium turcicum*. Crop Sci. 17:132-135.
- Hooker, A. L. 1978. Additional sources of monogenic resistance in corn to *Helminthosporium turcicum*. Crop Sci. 18:787-788.
- Hooker, A. L., Hilu, H. M., and Wilkinson, D. R. 1964. Additional sources of chlorotic-lesion resistance to *Helminthosporium turcicum* in corn. Plant Dis. Rep. 48:777-780.
- Hooker, A. L., and Kim, S. K. 1973. Monogenic and multigenic resistance to *Helminthosporium turcicum* in corn. Plant Dis. Rep. 57:586-589.
- Hooker, A. L., and Perkins, J. M. 1980. Helminthosporium leaf blights of corn - the state of the art. Pages 68-87 in: Proc. Annu. Corn Sorghum Res. Conf., Am. Seed Trade Assoc., Chicago, IL.

Table 4. Yield (%)^a of sweet corn hybrids differing for *Ht*- and partial resistance to northern leaf blight (NLB) following inoculation with races 0 and 1 of *Exserohilum turcicum*

| Resistance to NLB Hybrid | 1988 | | 1989 | | 1990 | |
|------------------------------------|--------|--------|--------|--------|--------|-----------------|
| | Race 0 | Race 1 | Race 0 | Race 1 | Race 0 | Race 1 |
| <i>Ht</i> - and partial resistance | | | | | | |
| Florida Staysweet | ... | ... | 97 | 93 | 103 | 92 |
| Sch 5009 | 97 | 102 | 103 | 95 | 97 | 95 |
| SummerSweet 7210 | 98 | 101 | 96 | 97 | 94 | 90 |
| Partial resistance | | | | | | |
| Honey n Frost | ... | ... | 94 | 102 | 87 | 92 |
| XPH 2670 | ... | ... | 89 | 92 | 88 | 81 ^b |
| <i>Ht</i> -resistance | | | | | | |
| Sch 4055 | 87* | 85* | ... | ... | ... | ... |
| Sch 4064 | 93 | 86* | 91 | 96 | 83* | 82* |
| Shield Crest | 94 | 88* | 90 | 83* | 89 | 83* |
| Susceptible | | | | | | |
| FMX 235 | 90* | 88* | 94 | 92 | 85* | 90 |
| Stylish | ... | ... | 80* | 74* | 49* | 56* |
| Sunset | ... | ... | 83* | 77* | 72* | 84* |
| BLSD <i>k</i> = 100 | 7.7 | | 16.3 | | 14.1 | |

^aYield (%) as a percentage of noninoculated control treatments of the same hybrid.

^b* = Significant difference (*P* = 0.05) between noninoculated controls (100%) and treatments inoculated with races 0 or 1.

11. Hughes, G. R., and Hooker, A. L. 1971. Gene action conditioning resistance to northern leaf blight. *Crop Sci.* 11:180-184.
12. 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.
13. Jenkins, M. T., Robert, A. L., and Findley, W. R., Jr. 1952. Inheritance of resistance to *Helminthosporium turcicum* leaf blight in populations of F3 progenies. *Agron. J.* 44:438-442.
14. 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.
15. Leath, S., and Pedersen, W. L. 1986. Effects of the *Ht*, *Ht2*, and/or *Ht3* genes in three maize inbreds on quantitative resistance to *Exserohilum turcicum* race 2. *Plant Dis.* 70:529-531.
16. Leonard, K. J., Levy, Y., and Smith, D. R. 1989. Proposed nomenclature for pathogenic races of *Exserohilum turcicum* on corn. *Plant Dis.* 73:776-777.
17. 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.
18. Pataky, J. K. 1992. Relationships between yield of sweet corn and northern leaf blight caused by *Exserohilum turcicum*. *Phytopathology* 82:370-375.
19. Pataky, J. K., Carson, M. L., and Mosely, P. R. 1991. Race 23N of *Exserohilum turcicum* in Florida. *Plant Dis.* 75:863.
20. Pataky, J. K., and Eastburn, D. M. 1993. Reactions of sweet corn hybrids to common rust, Stewart's wilt, northern leaf blight, and common smut. Pages 143-155 in: *Midwestern Vegetable Variety Trial Report for 1993*. J. E. Simon et al, eds. Purdue Univ. Agric. Exp. Stn. Bull. No. 680.
21. Pataky, J. K., and Eastburn, D. M. 1993. Using hybrid disease nurseries and yield loss studies to evaluate levels of resistance in sweet corn. *Plant Dis.* 77:760-765.
22. Pataky, J. K., Headrick, J. M., and Suparyono. 1988. Classification of sweet corn hybrid reactions to common rust, northern leaf blight, Stewart's wilt, and Goss's wilt and associated yield reductions. *Phytopathology* 78:172-178.
23. Pataky, J. K., Nankam, C., Kerns, M. R., Fallah Moghaddam, P., and Gantz, J. W. 1993. Sweet corn hybrid disease nursery - 1993. Pages 156-166 in: *Midwestern Vegetable Cultivar Report for 1993*. J. E. Simon et al, eds. Purdue Univ. Agric. Exp. Stn. Bull. No. 680.
24. 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.
25. Raymundo, A. D., and Hooker, A. L. 1982. Single and combined effects of monogenic and polygenic resistance on certain components of northern corn leaf blight development. *Phytopathology* 72:99-103.
26. Raymundo, A. D., Hooker, A. L., and Perkins, J. M. 1981. Effect of gene *HtN* on the development of northern corn leaf blight epidemics. *Plant Dis.* 65:327-330.
27. Simone, G. W. 1978. Inheritance of resistance in fifteen corn selections to *Helminthosporium turcicum*. Ph.D. thesis. University of Illinois, Urbana.
28. Smith, D. R., and Kinsey, J. G. 1980. Further physiologic specialization in *Helminthosporium turcicum*. *Plant Dis.* 64:779-781.
29. Thakur, R. P., Leonard, K. J., and Jones, R. K. 1989. Characterization of a new race of *Exserohilum turcicum* virulent on corn with resistance gene *HtN*. *Plant Dis.* 73:151-155.
30. Tomerlin, J. R., and Howell, T. A. 1988. DISTRAIN: A computer program for training people to estimate disease severity on cereal leaves. *Plant Dis.* 72:455-459.
31. 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.
32. Ullstrup, A. J. 1963. Sources of resistance to northern corn leaf blight. *Plant Dis. Rep.* 47:107-108.
33. Ullstrup, A. J. 1970. A comparison of monogenic and polygenic resistance to *Helminthosporium turcicum*. *Phytopathology* 60:1597-1599.
34. Welz, H. G., Wagner, R., and Geiger, H. H. 1993. Virulence variation in *Setosphaeria turcica* populations collected from maize in China, Mexico, Uganda, and Zambia. *Phytopathology* 83:1356.
35. Wilson, G. F., and Rhodes, A. M. 1970. Similarity of five sources of chlorotic-lesion resistance to *Helminthosporium turcicum* in a sweet corn background. *Plant Dis. Rep.* 54:896-897.