Effects of Northern Leaf Blight and Detasseling on Yields and Yield Components of Corn Inbreds

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ABSTRACT

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Variation in yield of three corn inbreds infected with *Exserohilum turcicum*, the causal agent of northern corn leaf blight, was best explained by the area under the disease progress curve, whereas variation in number of kernels per plant and in 200 kernel weight was best explained by leaf blight severity assessed just after completion of flowering and at physiologic maturity, respectively. Higher disease severities were correlated with decreased seed size, although disease and seed size were not correlated with seed germinability. Yields were lowered 10% by detasseling by cutting and 1% by detasseling by pulling. The detrimental effects of detasseling and northern leaf blight on yields were additive.

Northern leaf blight (NLB), caused by *Exserohilum turcicum* (Pass.) Leonard & Suggs, is a major disease of corn (*Zea mays* L.) in the United States corn belt (14). Yield losses due to NLB may be as much as 40% when susceptible hybrids are infected before silking and environment favors disease development (16,19). Most commercial hybrids currently grown in the U.S. corn belt have sufficient levels of resistance to prevent major losses due to NLB.

Corn inbreds, grown in hybrid seed production, have been hand- or mechanically detasseled since the 1970 epidemic of southern leaf blight, caused by *Bipolaris maydis* (Nisik.) Shoemaker race T, on Texas cytoplasmic male sterile corn. Detasseling assures hybridization between female and male inbred parents (6) but involves loss of photosynthetic tissue. Leaf area loss in hybrids has been shown to be proportionally related to yield reduction (4,10). The response of inbreds to leaf area loss differs greatly (13), however, and may coincide with genotypic differences in final leaf number (20).

Variable levels of polygenic resistance to NLB are available in corn inbreds, as is monogenic resistance conferred by *Ht* genes. For over 15 yr, *E. turcicum* race 1 has been effectively controlled in hybrids and inbreds by the *Ht*1 gene (11). However, physiologic races of this pathogen have recently developed against which the *Ht*1 gene is ineffective (18) and for which no good sources of monogenic resistance are readily available (15).

Virulent races of *E. turcicum* are found in most corn-producing states (14), and hybrid seed producers regularly apply fungicides to prevent yield losses due to this and other pathogens (6). Hybrid seed corn, produced on inbreds, is of high economic value and fungicidal control of disease is economically feasible. However, the effects of virulent races of *E. turcicum* on inbred yields and seed quality have not been studied. Seed producers need precise data on the effects of NLB in order to evaluate the efficacy of fungicide applications. They also need to know how partial defoliation during detasseling affects yields in order to assess the efficacy of pollen control methods. The interaction between detasseling and NLB, on yields and hybrid seed quality, has not been studied.

This study was initiated: 1) to investigate the effects of *E. turcicum* race 2 infection on yields and yield components of inbred plants, 2) to determine the effect of three detasseling methods on yields of three corn inbreds, and 3) to evaluate the interaction between detasseling and northern leaf blight.

MATERIALS AND METHODS

Field plots and inbred lines. Field experiments were completed at the Agronomy and Plant Pathology South Farm near Urbana and the Northwestern Research Center near Monmouth, Illinois, in 1984, 1985, and 1986. Factorial experiments were arranged in a split-plot design for each of three inbred parental sets, with disease treatments nested within pollen control methods. Experiments were replicated five times in 1984 and four times in 1985 and 1986. Plots consisted of six rows, with the four center rows planted to an ear parent and the two outer rows planted to a pollen parent. Female inbreds were FR632c-cms, FRB73c-cms, and FRMo17c-cms, and male inbreds were FR619, FRMo17rfc, and FR634rfc. (The FR notation indicates that the inbreds are backcross derived lines from Illinois Foundation Seeds, Inc., Urbana, and they carry the *Ht*1 gene.) FR632 matures early and is very susceptible to *E. turcicum* race 2, whereas FRB73 and FRMo17 are intermediate in maturity and moderately and highly resistant, respectively.

Plot rows, 76 cm apart, were 4.6 m long in Urbana and 13.7 m long in Monmouth. Seed was machine-planted between 10 and 17 May at both locations in 1984 and between 29 April and 4 May in 1985 and 1986. Fifty percent of male inbreds were delay-planted 1 to 2 wk later, throughout all male rows, to ensure viable pollen when silks were receptive. In each year, final populations were 52,000 plants per hectare. Growth stages (GS), according to Hanway (9), were noted throughout the growing seasons; two developmental stages of particular importance were GS 4 (tip of tassel visible) and GS 9 (all kernels fully dented).

Inoculations and fungicide treatments. Inoculations were done with leaf tissue collected from infected plants grown in the preceding year. Approximately 1.1 g (50 cc) of dried, ground tissue was placed into the whorls of plants in the center two rows of each plot between GS 2 and GS 3 in each year. The presence of susceptible NLB lesions on uninoculated male inbreds (carrying the *Ht*1 gene) indicated that *E. turcicum* race 2 was the predominant pathogen in inoculation material in all years of this study.

In 1984, five treatments were imposed for establishing different levels of disease: Uninoc (not inoculated or treated with fungicides), NoFung (no fungicidal control after inoculation), early mancozeb (bimonthly applications of mancozeb [Dithane M-45] at 1.34 kg a.i/ha, initiated 3 wk after inoculation), mid-mancozeb (initiated 5 wk after inoculation), and late mancozeb (initiated 7 wk after inoculation).

In 1985 and 1986, mancozeb was used on two treatments and reapplied weekly. Fungicide applications for the early mancozeb treatment were initiated 2 wk

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after inoculation (GS 3 in FR632) in 1985 and 4 wk after inoculation (GS 4 in FR632) in 1986, before 1% disease was observed. Late mancozeb applications were initiated 4 and 7 wk after inoculation in 1985 and 1986, respectively, when significant disease differences were observed between early mancozeb and NoFung. A systemic fungicide, propiconazole (Tilt 3.6EC) was applied at 125 g a.i./ha in 1985 and at 60 g a.i./ha in 1986 for two additional treatments. Propiconazole applications were initiated 2 and 5 wk after inoculation in 1985 and 1986, respectively, and reapplied at 3-wk intervals for the early propiconazole treatment. A single application of propiconazole, applied at 6 and 8 wk after inoculation in 1985 and 1986, respectively, was the late propiconazole treatment. Two treatments, Uninoc and NoFung, remained the same as in 1984.

Disease assessment. Disease severity, the proportion of diseased leaf tissue, was visually estimated on plants in the center two rows of each plot, at weekly intervals throughout the season, starting at 15 days after inoculation. Areas under the disease progress curve (AUDPC) were calculated for each treatment as: $\text{AUDPC} = \Sigma_{i=1}^{k-1} \frac{1}{2} (Y_{i+1} + Y_i)(t_{i-1} - t_i)$, where Y = the proportion of diseased tissue at time t, i = day of assessment (starting on the day of inoculation with 0% disease severity), and k = number of assessments. AUDPCs were used as comparative measures of the epidemics in plots (17).

Detasseling. All female inbreds were c-cytoplasmic male sterile to maintain genetic uniformity throughout the study. Three pollen control methods were compared: 1) leaving c-male sterile female plants intact (Whole), 2) cutting off the upper portion of female plants 15–20 cm above the ear at GS 4 (Cut), and 3) removing the tassel by hand, taking as little leaf tissue as possible, from female plants at GS 4 (Pulled). In 1985 and 1986 at Urbana, total plant leaf areas were measured at weekly intervals, from emergence to 2 wk after tasseling, using a Li-Cor (Lincoln, NE) leaf area meter.

Yields and yield components. Ten competitive plants (evenly spaced with neighbors) were hand-harvested from the two innermost rows of female plants (2). Grain moisture at the time of harvest in 1984 was 18–22% at both locations, 30–40% at Urbana and 22–28% at Monmouth in 1985, and 28–36% at both locations in 1986. Ears were dried in hot-air dryers at 32 C for 6 days, and grain moisture was reduced to less than 15%. All grain weights were corrected to 15.5% moisture. In 1985 and 1986, numbers of kernels per ear were estimated by multiplying the number of rows of kernels by the number of kernels in an average row. Numbers of aborted kernels also were counted and subtracted from the estimated number of kernels per ear. In all years, grain from each ear was weighed and bulked by plot for yield, and 200 kernel weights were determined. In 1985 and 1986, plot samples were graded into four seed grades: large rounds, small rounds, large flats, and small flats. Flats were first separated from rounds by working seed through a 0.52 × 1.90 cm slotted screen. Rounds and flats were each sized with 0.79- and 0.64-cm-diameter screens. Seeds that did not pass through the larger screen were graded as large rounds or large flats; seeds that passed through the 0.79-cm-diameter screen but not the smaller screen were small rounds or small flats. Seed quantities in each grade were converted to percentages. Some samples from each seed grade were tested for germinability using the Iowa State cold germ test (1). The 1985 cold germination tests were done by Cargill, Inc., Aurora, IL. The 1986 cold germination tests were done at the University of Illinois.

Data analysis. Analyses of variance (ANOVA) were done on data from each environment to determine differences due to experimental effects. For each inbred, using only data from whole plants, correlation coefficients were computed between yield variables (yield, number of kernels per ear, and 200 kernel weight) and disease variables (each leaf blight assessment and AUDPC). Variables with consistently high correlation coefficients were used for developing loss models by regressing yield on disease. Intercepts of resultant models were considered the best estimates of maximum yield for each inbred in each environment (location by year). Linear regression models with percentage of maximum yield as a function of AUDPC were calculated for each inbred in each environment. F statistics were examined to compare overall significance ($P = 0.10$) of models. Coefficients of determination (r^2) estimated the proportion of variation in yield reduction explained by disease. Coefficients of variation estimated the variation in the data. Residuals were tested for homogeneity, appropriateness of the model, and outliers. Loss models were constructed in the same way for individual components of yield, using disease severity at a single assessment date as independent variables.

RESULTS

NLB development. NLB was observed in inoculated plots in Urbana by 3 wk after inoculation and in uninoculated plots by 5 wk after inoculation in each year. Final disease severity was similar in both NoFung and Uninoc treatments in 1985. In 1985 and 1986, rapid disease development was later at Monmouth than at Urbana; therefore, sprays were applied 1–2 wk later at Monmouth. Differences in disease development among the three inbreds were significant in each year.

Inoculations and fungicide applications were effective at achieving various NLB severities, ranging from 0 to 13.1, 0 to 9.8, and 0 to 0.6 at GS 4, and from 1.4 to 100, 2.8 to 99.6, and 0.4 to 84.5 at GS 9 in 1984, 1985, and 1986, respectively (Table 1).

Relationships between disease and yield. ANOVAs showed significant ($P = 0.05$) differences in yields due to pollen control methods and disease treatments

Table 1. Means and ranges of northern leaf blight severity at the time of tassel appearance and full dent for corn inbreds evaluated at Urbana and Monmouth, IL, in 1984, 1985, and 1986

Inbred	1984				1985				1986			
	Urbana		Monmouth		Urbana		Monmouth		Urbana		Monmouth	
Growth stage	$\bar{x}$	Range	$\bar{x}$	Range	$\bar{x}$	Range	$\bar{x}$	Range	$\bar{x}$	Range	$\bar{x}$	Range
FRMo17												
Tassel	0.4	0.0–0.7	1.2	0.6–1.4	1.4	0.1–2.2	0.5	0.0–0.8	0.0	0.0–0.0	n.d.[z]	n.d.
Dent	2.6	1.4–3.3	4.5	3.6–6.7	21.0	7.2–16.3	5.8	2.8–8.0	3.0	0.4–6.1	1.6	1.4–2.1
FRB73												
Tassel	2.4	0.1–3.1	2.5	0.9–3.8	1.8	0.0–2.8	2.2	0.3–2.8	0.2	0.0–0.3	0.0	0.0–0.1
Dent	19.6	7.8–25.0	57.4	53.0–64.0	41.5	29.7–55.4	39.5	29.6–49.6	46.8	38.8–60.0	55.6	49.2–60.0
FR632												
Tassel	4.4	2.2–5.6	8.5	1.0–13.2	7.4	0.7–9.8	3.1	0.0–4.3	0.4	0.0–0.6	0.3	0.3–0.5
Dent	96.2	93.5–100	98.6	93.5–100	91.6	72.7–99.6	82.0	71.2–90.8	72.0	55.4–84.5	63.9	51.7–70.8

[z] n.d. = Not done. Disease notes were not taken on FRMo17 at Monmouth in 1986.

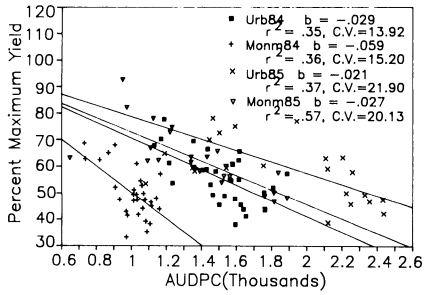

Fig. 1. Regressions of percentage of maximum yield from the corn inbred FR632 on the area under the disease progress curve (AUDPC) of northern leaf blight, in four environments.

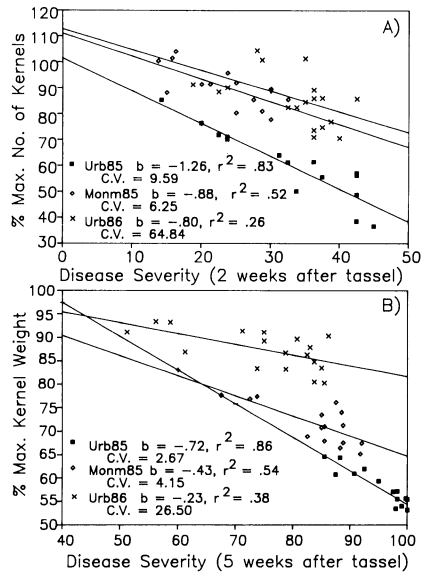

Fig. 2. Regressions of percentage of maximum yield components on northern leaf blight disease severities after full tassel for corn inbred FR632. **(A)** Number of kernels per ear on disease severity 2 wk after tassel appearance and **(B)** 200 kernel weight on disease severity 5 wk after tassel appearance in three environments.

in three inbreds at Urbana in 1985 and on FR632 at both locations in 1984. Only pollen control methods affected yields in FRMo17 at Monmouth in 1985 and FR632 at Monmouth in 1986, and only disease treatments had an effect on yields of FR632 at Monmouth in 1985.

Correlation coefficients were highest between yields and AUDPCs for whole plants. Effects of disease treatments on yields in FRMo17 and FRB73 in any environment, or in FR632 in 1986, were not significant, so loss models were constructed only for FR632 at both locations in 1984 and 1985. AUDPC accounted for 35–57% (r^2) of the variation in percent maximum yield of FR632 in these models (Fig. 1). Each of the models was very highly significant ($P = <0.001$) and regression coefficients were significantly different from zero.

Correlation coefficients between yield components (number of ears per plant, number of kernels per ear, and 200 kernel weight) and disease variables were low from two inbreds, FRMo17 and FRB73. However, correlation coefficients between yield components and disease variables on FR632 showed consistent patterns. Number of ears per plant and disease severities early in the season (GS 3 to GS 4) on FR632 had high negative correlations. Midseason assessments of disease severity (GS 4 to GS 6) were highly negatively correlated to numbers of kernels per ear, whereas 200 kernel weights were negatively correlated to later assessments (GS 8 to GS 9).

Loss models were constructed for components of yield from FR632 in 1985 and 1986. Critical point models, with disease severity at a single assessment date as independent variables, were the best predictors of reduction in individual yield components. Adding quadratic terms did not improve the fit of these models. Regression of disease severity at GS 3 on number of ears per plant did not produce a significant model. Disease severity at 2 wk after tassel appearance on FR632 in three environments accounted for 26–83% of the variation in seed numbers per ear (Fig. 2A). At GS 8, disease severity accounted for 38–86% of the variation in seed weight of FR632 in three environments (Fig. 2B). Percent of seed samples grading out as large also decreased with increasing AUDPC on FR632 in 1985 at two locations (Fig. 3).

Detasseling effects. Pulling the tassel off by hand, with inadvertent removal of leaf tissue, resulted in 3.0–11.9% defoliation and cutting the tassel off removed 13.6–24.8% of the leaf tissue from inbreds grown at Urbana in 1985 and 1986 (Table 2). Detasseling by pulling or by cutting resulted in a yield reduction ($P = <0.05$) at Urbana in 1985, whereas only cutting FR632 decreased yields in 1986. Yields at Monmouth showed trends similar to those observed at Urbana, but differences among pollen control methods were not significant at Monmouth. Number of ears per plant was consistently lower from plants that had been detasseled by cutting than from plants detasseled by pulling or from whole plants (Table 3). Number of kernels per ear also tended to be lower from plants that had been detasseled by cutting. In 1985, 200 kernel weight was lower from plants that had been detasseled by cutting, but no consistent effects due to detasseling were observed on 200 kernel weight in 1986.

Disease and detasseling. Yields and yield components of FR632 were reduced by detasseling and by disease in 1984 at both locations and in 1985 at Urbana, but no interaction between detasseling and disease was statistically significant. However, the detrimental effects of detasseling and disease appear to be additive in reducing numbers of kernels per plant and total grain weight. Figure 4 shows the effect of disease severities at GS 5 on numbers of kernels per plant in whole, pulled, and cut plants. Similar effects of AUDPC are shown on grain weight per plant in Figure 5. Although differences were not always significant, trends in the data were consistent.

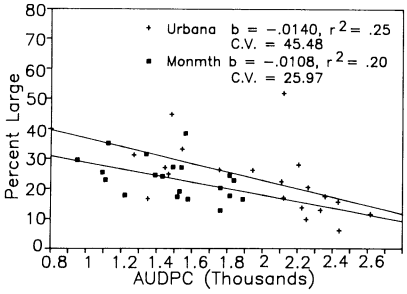

Fig. 3. Regressions of percentage of large seed on the area under the disease progress curve (AUDPC) of northern leaf blight at two locations, Urbana and Monmouth, IL, on corn inbred FR632 in 1985.

Table 2. Percentage of leaf tissue removed with detasseling and corresponding yield reduction in three corn inbreds at Urbana, IL, in 1985 and 1986[y]

		1985		1986	
Inbred	Treatment	% Defoliation	% Yield	% Defoliation	% Yield
FR632	Whole	···	100 a	···	100 a
	Pulled	9.9	97 b	4.7	99 a
	Cut	24.8	82 b	13.6	88 b
FRB73	Whole	···	100 a	···	100
	Pulled	4.4	96 b	4.7	108
	Cut	18.2	83 b	21.5	97
FRMo17	Whole	···	100	···	n.d.[z]
	Pulled	3.0	94	11.9	n.d.
	Cut	22.8	87	16.4	n.d.

[y] Data are means of four replicates and when followed by the same letter are not significantly different according to Fisher's least significant difference ($P = 0.05$).
[z] n.d. = Not done. FRMo17 was not harvested in 1986 because of poor pollen set.

Seed grades and germination. In 1985, flat seed grades from FRMo17 and FRB73 had higher ($P = 0.05$) germination percentages than round seed grades. Large flats of FRMo17 had an average germination of 91.6%, compared with 82.3% germination of large rounds. In FRB73, small flats averaged 90.5% germination and small rounds averaged 87.2%. No other differences in germinability due to grade of seed or disease severities of parental plant were observed.

DISCUSSION

Although no plots were maintained free from disease in any year, significantly different disease severities occurred within each inbred. Maximum loss in yield potential due to *E. turcicum* race 2 in the highly susceptible inbred FR632 was 44% in 1984, 32% in 1985, and 37% in 1986. Previous studies on yield losses due to NLB (16,19) showed similar losses due to NLB in susceptible hybrids. Inbreds with higher levels of polygenic resistance, FRB73 and FRMo17, did not become severely diseased in any year, and NLB on these two inbreds did not consistently affect yields or yield components.

Components of yield are most affected by environmental stresses, such as drought, during flowering (8). Claasen and Shaw (5) and Egharevba et al (7) found reductions in kernel numbers when drought stress occurred during silking and pollination; stress during or after silking reduced kernel weight. Our results support a previous study (8) showing that stress during flowering reduced the number of secondary ears that set grain. Our data show that near the time of tassel emergence (GS 4), disease severity reduced number of ears per plant, whereas later disease first affected number of kernels per ear, then 200 kernel weight.

Yield loss models for components of yield (numbers of kernels and 200 kernel weight) had similar proportions of variation in the data explained by the model (r^2) over all environments. As with the overall loss model (total yield = f[AUDPC]), the r^2 was lowest for 1986 data. This may be due to the low disease severities observed in 1986. Regardless of differences in the r^2, slopes of the lines describing losses in any yield variable are similar between environments. For example, kernel numbers decreased from 0.8 to 1.2% for every 1% increment in disease severity, and total yields decreased 2.1–5.9% for every 100 units AUDPC, across all environments.

According to Craig (6), seed size (large round, small flat, etc.) is dictated primarily by the seed parents and therefore would be unaffected by disease levels. However, while there was no consistent effect of disease on seed shape (flat or round), there was a negative correlation between AUDPC and the percentage of seed samples that were graded as large. This supports a previous study (4) showing that loss in leaf tissue reduces grain fill, resulting in smaller kernel size and lower kernel weight. Increasing amounts of AUDPC also appeared to have a positive effect on seed germinability. This may be due to a decrease in the number of kernels of marginal quality and vigor with increasing disease severities.

Because hybrid seed corn is generally sold on a per-unit basis (80,000 kernels per unit), seed corn producers are interested in producing maximum numbers of kernels. Since kernel numbers are established at anthesis, minimization of stress at that stage is important. If a stress, such as disease, is severe at anthesis, disease control later in host development may not contribute to minimizing losses due to disease. The loss models presented may be used by seed producers as guidelines in making decisions concerning disease control. If 5% disease severity 2 wk after tassel appearance causes 0–5% loss in kernel

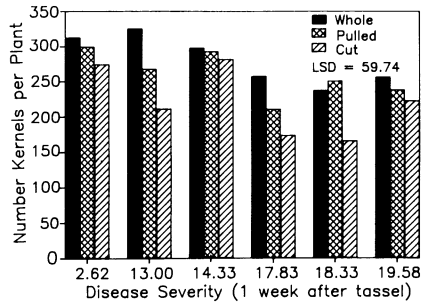

Fig. 4. Effects of three pollen control methods and disease severities 1 wk after tassel appearance on numbers of kernels per plant from inbred FR632 at Urbana in 1985, where LSD ($P = 0.05$) between disease severities is 2.93.

Table 3. Effects of three pollen control methods on corn yield components from three inbreds, planted as females in hybrid production fields, where "pulled" and "cut" are two methods of detasseling[y]

Location Inbred	Pollen control method	1985 Number of ears	1985 Number of kernels	1985 200 Seed weight (g)	1986 Number of ears	1986 Number of kernels	1986 200 Seed weight (g)
Monmouth, IL							
FR632	Whole	1.25	267	75.9	1.58 a	279	75.4
	Pulled	1.26	259	76.0	1.59 a	289	79.5
	Cut	1.20	253	74.7	1.40 b	244	74.7
FRB73	Whole	1.23	218	97.5	1.26	389	58.7
	Pulled	1.15	231	97.2	1.29	383	60.8
	Cut	1.12	227	97.6	1.15	398	60.1
FRMo17	Whole	1.11 a	284	106.4	n.d.[z]	n.d.	n.d.
	Pulled	1.13 a	264	104.4	n.d.	n.d.	n.d.
	Cut	0.99 b	250	100.2	n.d.	n.d.	n.d.
Urbana, IL							
FR632	Whole	1.07	311 a	93.3	1.34 a	354 a	76.3
	Pulled	1.04	284 b	94.5	1.35 a	345 a	78.7
	Cut	1.02	236 c	94.4	1.15	303 b	77.0
FRB73	Whole	1.08	483 a	64.7 a	0.97	419	59.8
	Pulled	1.12	492 a	64.3 a	1.00	436	59.4
	Cut	1.04	425 b	61.6 b	1.01	404	60.1
FRMo17	Whole	0.96	346	90.0 b	n.d.	n.d.	n.d.
	Pulled	0.91	325	95.3 a	n.d.	n.d.	n.d.
	Cut	0.88	333	88.7 b	n.d.	n.d.	n.d.

[y] Data are means of four replicates and when followed by the same letter are not significantly different according to Fisher's least significant difference ($P = 0.05$).
[z] n.d. = Not done. FRMo17 was not harvested at either location in 1986 because of poor pollen set.

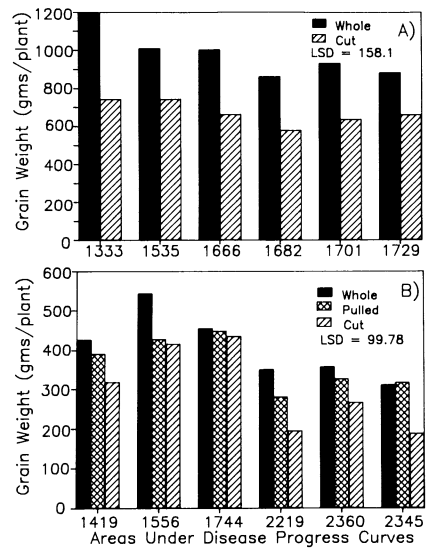

Fig. 5. Effects of pollen control methods and areas under disease progress curves (AUDPC) on grain weight from corn inbred FR632 at Urbana in **(A)** 1984, where LSD between AUDPCs is 77.5, and **(B)** 1985, where LSD is 88.77.

numbers but does not affect other yield components (particularly if the environment does not remain favorable for disease development), control action is not warranted. If 20% disease is observed at anthesis, however, greater than 3.2% loss in kernel numbers and a decrease in kernel weight (especially if there is any further increase in disease) can be expected, and control action may be economically feasible. Loss functions for each of the yield components can be used to check potential losses as the season progresses.

Disease and detasseling both reduce photosynthetic leaf area (3), thereby reducing the plants' yield potential in an additive manner. However, the detrimental effect on total yields due to detasseling is through cumulative and statistically insignificant effects on individual components of yield. Disease had a measurable detrimental effect on yield components and may increasingly damage a plant throughout the season. Plants may compensate for small losses in leaf tissue due to detasseling, actually making more efficient use of remaining leaf area with a resultant increase in yields (12), but not when additionally damaged by disease.

Management practices aimed at reducing stress and loss of photosynthetic area (through disease or detasseling) during flowering are recommended for maximizing yields of inbreds. Data from this study show that early disease control, especially on more susceptible lines, can maximize yields. The critical-point models presented can provide guidance in early control decisions and for predicting yield losses early in corn growth.

LITERATURE CITED

1. Anonymous. 1984. Seedsman's Handbook: Corn. 11th ed. M. Brayton Seeds, Inc., Ames, IA. 55 pp.
2. Balko, L. G., and Russell, W. A. 1980. Response of maize inbred lines to N fertilizer. Agron. J. 72:723-728.
3. Boote, K. J., Jones, J. W., Mishoe, J. W., and Berger, R. D. 1983. Coupling pests to crop growth simulators to predict yield reductions. Phytopathology 73:1581-1587.
4. Camery, M. P., and Weber, C. R. 1953. Effects of certain components of simulated hail injury on soybeans and corn. Pages 465-504 in: Iowa Agric. Home Econ. Exp. Stn. Res. Bull. 400.
5. Claasen, M. M., and Shaw, R. H. 1970. Water deficit effects on corn. II. Grain components. Agron. J. 62:652-655.
6. Craig, W. F. 1977. Production of hybrid seed corn. Pages 671-719 in: Corn and Corn Improvement. G. F. Sprague, ed. American Society of Agronomy, Inc., Madison, WI. 774 pp.
7. Egharevba, P. N., Horrocks, R. D., and Zuber, M. S. 1976. Dry matter accumulation in maize in response to defoliation. Agron. J. 68:40-43.
8. Hall, A. J., Lemcoff, J. H., and Trapani, N. 1981. Water stress before and during flowering in maize and its effects on yield, its components, and their determinants. Maydica 26:19-38.
9. Hanway, J. J. 1963. Growth stages of corn (*Zea mays* L.). Agron. J. 55:487-492.
10. Hicks, D. R., Nelson, W. W., and Ford, J. H. 1977. Defoliation effects on corn hybrids adapted to the northern corn belt. Agron. J. 69:387-390.
11. Hooker, A. L. 1961. A new type of resistance in corn to *Helminthosporium turcicum*. Plant Dis. Rep. 45:780-781.
12. Hunter, R. B., Daynard, T. B., Hume, D. J., Tanner, J. W., Curtis, J. D., and Kennenberg, L. W. 1969. Effect of tassel removal on grain yield of corn (*Zea mays* L.). Crop Sci. 9:405-406.
13. Hunter, R. B., Mortimore, C. G., and Kannenberg, L. W. 1973. Inbred maize performance following tassel and leaf removal. Agron. J. 65:471-472.
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*, *Ht*2, and/or *Ht*3 genes in three maize inbreds on quantitative resistance to *Exserohilum turcicum* race 2. Plant Dis. 70:529-531.
16. Raymundo, A. D., and Hooker, A. L. 1981. Measuring the relationship between northern corn leaf blight and yield losses. Plant Dis. 65:325-327.
17. Tooley, P. W., and Grau, G. R. 1984. Field characterization of rate-reducing resistance to *Phytophthora megasperma* f. sp. *glycinea* in soybean. Phytopathology 74:1201-1208.
18. 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.
19. Ullstrup, A. J., and Miles, S. R. 1957. The effects of some leaf blights of corn on grain yield. Phytopathology 47:331-336.
20. Vasilas, B. L., and Seif, R. D. 1985. Pre-anthesis defoliation effects on six corn inbreds. Agron. J. 77:831-835.