Influence of Host Resistance and Growth Stage at the Time of Inoculation on Stewart's Wilt and Goss's Wilt Development and Sweet Corn Hybrid Yield

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

ABSTRACT

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Stewart's and Goss's bacterial wilts were similar in symptomatology, disease development, and effects on sweet corn yield. The severity of the two bacterial wilts and their effects on sweet corn yield depended on the level of host resistance and on the host growth stage at which plants were inoculated. Disease development was greatest when sweet corn hybrids at the three- to five-leaf stage were inoculated with *Erwinia stewartii* and *Clavibacter michiganense* subsp. *nebraskense*. Disease severity was less when plants were inoculated at later growth stages, although disease development was affected less by plant age in susceptible and moderately susceptible hybrids than in resistant and moderately resistant hybrids. Yields of susceptible and moderately susceptible hybrids were significantly reduced by both diseases when plants were inoculated at the three- to five-leaf or the five- to seven-leaf stage. The yield of a moderately resistant hybrid inoculated at the three- to five-leaf stage was reduced in 2 of 3 yr. The yield of a resistant hybrid was not significantly affected by either disease. A damage threshold of approximately 40% severity (i.e., a disease rating above 6, which corresponds to systemic infection and stunting) 1 wk before harvest was observed for both diseases on the susceptible and moderately susceptible hybrids. Beyond this threshold, sweet corn yield decreased 17 and 19%, by weight and number of ears, respectively, for each 10% increase in severity.

Stewart's bacterial wilt, caused by *Erwinia stewartii* (Smith) Dye, and Goss's wilt, caused by *Clavibacter michiganense* subsp. *nebraskense* (Schuster et al) (8), are important bacterial diseases of corn (*Zea mays* L.) (10,12,18,19,25,26). Stewart's and Goss's wilts can affect corn plants at any stage of plant growth. Young plants infected by either bacterium may die or become stunted and fail to produce ears (5,10, 14,22). Plants infected at later growth stages may have typical leaf blight symptoms, which are similar for the two diseases.

Present address of first author: Sukamandi Research Institute for Food Crops, Balittan Sukamandi, Sukamandi, Subang, Java Barat, Indonesia.

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The most pronounced symptoms of Stewart's wilt are wilting and stunting, although two phases of the disease are recognized (4,10,22,24). Water-soaked, streaked leaf lesions and stunting are symptomatic of infected seedlings. Irregular, lengthy local lesions occur between leaf veins of plants infected after anthesis (4,10,22). Similarly, the most pronounced symptoms of Goss's wilt are wilting and blight (18,19,25,26), but unlike Stewart's wilt, Goss's wilt produces characteristic symptoms of dark green to black spots, which resemble freckles, within wilted tissue. Wilted, water-soaked tissue often results in leaf scorching symptoms.

Both bacterial diseases are of increasing concern to sweet corn producers. Stewart's wilt of susceptible sweet corn hybrids was severe in cooperative disease observation nurseries from 1983 to 1988 in Delaware, Pennsylvania, New Jersey, New York, and Illinois (USDA, *unpublished*). Also, unusual outbreaks of Stewart's wilt have recently occurred in the Lake Ontario region of New York (H. Dillard, *personal communication*) and as far north as Ontario, Canada, even when winter temperature indices have predicted less disease (1). Goss's wilt is a relatively new disease of corn, which was reported first in northern Nebraska in 1969 (18,27) and shortly thereafter in other areas of Nebraska, Colorado, Iowa, Kansas, South Dakota, Illinois, Minnesota, and Wisconsin (19,25,26).

In the 1930s, when Stewart's wilt was epidemic in the corn belt, many sweet corn cultivars were observed to be more susceptible than dent corn (11). Presently, commercial sweet corn hybrids vary in their resistance and susceptibility to *E. stewartii* and *C. m.* subsp. *nebraskense*

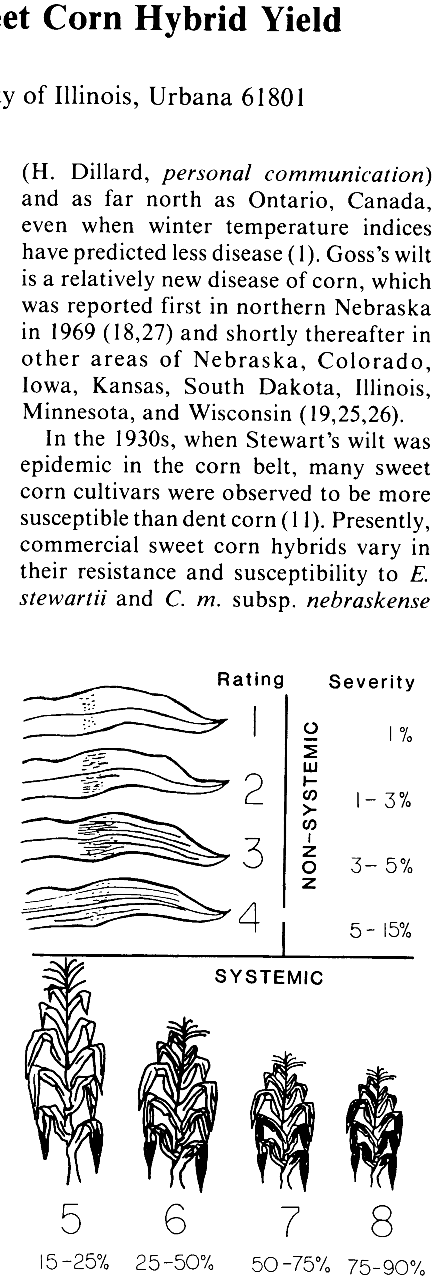

Fig. 1. Rating scale (0–9) for Stewart's and Goss's wilts when leaves were inoculated in plant whorls. A rating of 9 indicates that plants were dead. See text for descriptions of other categories.

(9,13,16,17). Reactions of hybrids to Goss's wilt and Stewart's wilt have been highly correlated (15,17). Even though many commercial sweet corn hybrids are resistant to these diseases, several moderately susceptible and susceptible hybrids are widely grown, because of their superior horticultural or agronomic qualities, such as improved germination, seedling vigor, ear shape, kernel depth, and cut kernel weight.

Comparatively little is known about the effect of Stewart's and Goss's wilts on sweet corn yield. Both bacteria are primarily vascular pathogens and usually do not infect ears but might affect the amount of dry matter stored in grain. Estimates of yield reduction due to Stewart's and Goss's wilts of dent corn have ranged from 20 to 50% but generally have not been based on experimental data (12,13,24,26). Ayers et al (2) reported that a susceptible sweet corn hybrid, Sugar and Gold, had only three to 15 ears per 50 plants when heavily infected with Stewart's wilt. Goth and Stienke (9) screened 22 sweet corn hybrids for Stewart's wilt reactions and reported 40–90% stand reductions and no marketable yield among susceptible genotypes, 10–40% stand reductions and some yield among tolerant genotypes, and less than 10% stand reductions and negligible yield reductions among resistant genotypes. Pataky et al (17) observed yield reductions as high as 60% for susceptible sweet corn hybrids infected with either of the two bacterial wilt pathogens. Yield reductions due to the bacterial wilts decreased drastically as resistance levels increased.

Quantitative data that relate levels of disease development to yield reductions are necessary for the management of these diseases. Accurate estimates of yield loss will establish economic thresholds and identify management strategies for susceptible, intermediate, and resistant genotypes.

The objectives of this research were to evaluate the influence of host resistance and the time of inoculation on the development of Stewart's and Goss's wilts and to determine the relationships between disease severity and sweet corn yield. Preliminary results have been reported (23).

Table 1. Disease ratings for Stewart's and Goss's wilts 2–3 wk after inoculation of sweet corn hybrids at different growth stages

Pathogen[a]	Disease onset[b]	1985[c] M	1985 GC	1985 J	1986 M	1986 GC	1986 HC	1986 J	1987 M	1987 GC	1987 HC	1987 J
Es	CK	1.9	1.1	1.5	1.0	1.1	2.2	2.9	...	...	...	...
	3–5	2.3	3.8	7.5	2.2	3.5	4.2	6.3	...	...	...	...
	5–7	2.3	2.6	5.4	2.1	3.1	3.9	5.5	...	...	...	...
	7–9	2.4	2.6	6.2	1.5	1.9	3.7	4.6	...	...	...	...
$FLSD_{0.05}$[d]			0.75			0.72						
Cmn	CK	1.0	1.1	1.8	1.0	1.1	2.5	3.3	1.0	1.0	1.3	2.1
	3–5	3.1	4.9	6.7	1.4	3.8	3.4	5.6	2.4	6.1	5.1	8.0
	5–7	1.3	1.9	4.4	1.1	3.6	3.7	4.6	1.7	2.6	3.2	4.8
	7–9	2.0	2.6	3.8	1.1	2.2	3.7	4.2	1.6	2.1	2.7	3.6
$FLSD_{0.05}$			1.23			0.84				0.82		

[a] *Es* = *Erwinia stewartii*; *Cmn* = *Clavibacter michiganense* subsp. *nebraskense*.
[b] CK = check plants (not inoculated). In other treatments, plants were inoculated at the three- to five-leaf (3–5), five- to seven-leaf (5–7), or seven- to nine-leaf (7–9) stage.
[c] Hybrids: M = Miracle; GC = Gold Cup; HC = Honeycomb; J = Jubilee.
[d] $FLSD_{0.05}$ = Fisher's least significant difference ($P<0.05$). Values are for comparison of hybrid-treatment combinations.

Table 2. Disease severity 1 wk before harvest for Stewart's and Goss's wilts of sweet corn hybrids inoculated at different growth stages

Pathogen[a]	Disease onset[b]	1986[c] M	1986 GC	1986 HC	1986 J	1987 M	1987 GC	1987 HC	1987 J
Es	CK	1	1	1	18	...	...	...	...
	3–5	1	15	43	68	...	...	...	...
	5–7	3	4	35	53	...	...	...	...
	7–9	1	4	18	35	...	...	...	...
$FLSD_{0.05}$[d]			12.0						
Cmn	CK	1	1	6	33	1	1	5	22
	3–5	1	28	28	68	5	57	57	97
	5–7	2	40	28	50	4	25	28	53
	7–9	1	7	11	25	8	11	18	35
$FLSD_{0.05}$			15.2				8.4		

[a] *Es* = *Erwinia stewartii*; *Cmn* = *Clavibacter michiganense* subsp. *nebraskense*.
[b] CK = check plants (not inoculated). In other treatments, plants were inoculated at the three- to five-leaf (3–5), five- to seven-leaf (5–7), or seven- to nine-leaf (7–9) stage.
[c] Hybrids: M = Miracle; GC = Gold Cup; HC = Honeycomb; J = Jubilee.
[d] $FLSD_{0.05}$ = Fisher's least significant difference ($P<0.05$). Values are for comparison of hybrid-treatment combinations.

Table 3. Total husked ear weight (g) per plot of sweet corn hybrids inoculated with the Stewart's and Goss's wilt pathogens at different growth stages

Disease onset[a]	1985[b] M	1985 GC	1985 J	1986 M	1986 GC	1986 HC	1986 J	1987 M	1987 GC	1987 HC	1987 J
CK	5,853	6,317	7,574	4,394	2,845	4,523	4,253	4,867	4,419	4,264	4,441
3–5	5,715	5,762	4,681	4,342	2,825	4,112	2,575	5,031	2,115	2,774	28
5–7	5,998	6,250	6,595	4,422	2,784	4,083	3,558	5,060	4,214	4,026	2,875
7–9	6,177	6,087	7,031	4,434	2,786	4,356	4,041	5,022	3,951	4,314	4,434
$FLSD_{0.05}$[c]		418.9			332.6				707.8		

[a] CK = check plants (not inoculated). In other treatments, plants were inoculated at the three- to five-leaf (3–5), five- to seven-leaf (5–7), or seven- to nine-leaf (7–9) stage.
[b] Hybrids: M = Miracle; GC = Gold Cup; HC = Honeycomb; J = Jubilee.
[c] $FLSD_{0.05}$ = Fisher's least significant difference ($P<0.05$). Values are for comparison of hybrid-treatment combinations.

MATERIALS AND METHODS

Field plots and sweet corn hybrids. Field plots were planted on 8 May 1985, 9 May 1986, and 13 May 1987 at the University of Illinois Agronomy/Plant Pathology South Farm, Urbana. Standard sweet corn production practices were followed. Soil types were a Drummer silty clay, a Flanigan silt loam, and a Drummer silty clay in 1985, 1986, and 1987, respectively. The seeding rate was 86,000 seeds per hectare. Seedlings were thinned to about 42,000 plants per hectare 20 days after emergence. The sweet corn hybrids Miracle, Gold Cup, and Jubilee were planted in all years. In 1986 and 1987, the hybrid Honeycomb was also planted. The four hybrids were consistent in their reactions to Stewart's and Goss's wilts in disease nurseries (16,17): Miracle was resistant, Gold Cup was moderately resistant, Honeycomb was moderately susceptible, and Jubilee was susceptible.

Treatments and experimental designs. The experiments were done as a 3×2×4 factorial in 1985 and as a 4 × 2 × 4 factorial in 1986 and 1987, with four replicates arranged in a split split-plot in which the main plots were in a randomized complete block experimental design. The main plots were hybrids. The subplots were inoculated with *C. m.* subsp. *nebraskense* or *E. stewartii*. The sub-subplots were inoculated at one of three different host growth stages (three- to five-leaf, five- to seven-leaf, or seven- to nine-leaf stage) or were an uninoculated control. Each experimental unit (sub-subplot) was three rows approximately 4.6 m long and spaced 76 cm apart. The second and third rows of each experimental unit were inoculated. The first row was a border.

Inoculum and inoculations. *C. m.* subsp. *nebraskense* and *E. stewartii* were isolated from naturally infected corn leaves in 1984. The isolates were preserved at −80 C as described by Sleesman and Leben (20) and were used throughout the study. Inoculum was prepared by transferring the bacteria to nutrient broth-yeast broth in Erlenmeyer flasks, incubating the transferred cultures at room temperature on shakers for approximately 48 hr, and streaking those cultures onto nutrient broth-yeast extract agar medium in petri plates. Cultures on the plates were incubated at 25 C for about 48 hr and used as inoculum. Cells were suspended in a 0.1 M NaCl buffer solution and adjusted to a final concentration of about 10^7 cfu/ml (A_{590} = 0.05).

Plants at the three- to five-leaf, five- to seven-leaf, and seven- to nine-leaf stages were inoculated on 29 May, 12 June, and 2 July 1985, respectively; 4 June, 16 June, and 1 July 1986; and 5 June, 15 June, and 3 July 1987. Inoculations were done by the pinprick techniques described by Blanco et al (3) and Chang et al (7) with the slight modification of spraying an additional 5–10 ml of diluted inoculum (10^4–10^5 cfu/ml) into plant whorls immediately after wounding.

Disease and yield assessments. Disease severity was rated 2–3 wk after inoculation in all 3 yr and 1 wk before harvest in 1986 and 1987. The ratings 2–3 wk after inoculation were ratings of individual plants. Each plant in a plot was rated on a 1–9 scale (Fig. 1) in which 1 = no spread or symptoms; 2 = little spread, with chlorosis and necrosis within 5 cm of inoculation; 3 = limited spread, with water-soaking, chlorosis, and necrosis toward the tip end of inoculated leaves only; 4 = abundant spread, with water-soaking, chlorosis, and necrosis toward both ends of inoculated leaves or slight systemic infection of plants; 5 = limited systemic infection of entire plants, with chlorosis, water-soaking, and limited necrosis of uninoculated leaves; 6 = severe water-soaking, wilting, chlorosis, and necrosis of 25–50% of the total leaf area, with stunting; 7 = severe chlorosis, necrosis, and wilting of 50–75% of the total leaf area, with severe stunting; 8 = severe chlorosis and necrosis of 75–90% of the leaf area; and 9 = death of plants. Individual plant ratings were converted to plot means. The ratings 1 wk before harvest were ratings of plots on the 1–9 scale. Ratings were converted to percentage severity, expressed as the mean percentage in each category. For example, if a plot rating was 6 (25–50% severity), disease severity in that plot equaled 37.5%.

Ears were harvested from 20 plants per experimental unit. The ear weight of unhusked and husked ears, the number of marketable and unmarketable ears, ear diameter, and ear length were measured independently for primary and secondary ears immediately after harvest.

Statistical analyses. The effects of treatments were assessed by analysis of variance (ANOVA). Main effects and first- and second-order interactions were determined. ANOVAs were not combined over years because of differences in the number of hybrids and the success of inoculations. For each hybrid in each year, the total husked ear weight and the total number of marketable ears were regressed on percentage disease severity 1 wk before harvest by ordinary least squares methods. For regressions, F statistics ($P<0.05$) were used to evaluate the significance of the model and independent variables. Coefficients of determination were calculated to determine the variation explained by the model. The intercept (b_0) of the regression of the total husked ear weight and the total number of marketable ears on disease severity estimated maximum yield for a hybrid in an experiment. Yields were then converted to percentages

Table 4. Total number of marketable ears per plot of sweet corn hybrids inoculated with the Stewart's and Goss's wilt pathogens at different growth stages

Disease onset[a]	1985[b] M	1985 GC	1985 J	1986 M	1986 GC	1986 HC	1986 J	1987 M	1987 GC	1987 HC	1987 J
CK	21	29	28	20	20	20	20	20	23	20	20
3–5	21	27	17	20	20	20	17	20	15	16	1
5–7	21	31	24	20	20	20	20	20	24	20	17
7–9	22	29	26	20	18	20	20	20	22	20	20
$FLSD_{0.05}$[c]	2.8			0.7				3.1			

[a] CK = check plants (not inoculated). In other treatments, plants were inoculated at the three- to five-leaf (3–5), five- to seven-leaf (5–7), or seven- to nine-leaf (7–9) stage.
[b] Hybrids: M = Miracle; GC = Gold Cup; HC = Honeycomb; J = Jubilee.
[c] $FLSD_{0.05}$ = Fisher's least significant difference ($P < 0.05$). Values are for comparison of hybrid-treatment combinations.

Table 5. Percentage of marketable primary ears of sweet corn hybrids inoculated with the Stewart's and Goss's wilt pathogens at different growth stages

Disease onset[a]	1985[b] M	1985 GC	1985 J	1986 M	1986 GC	1986 HC	1986 J	1987 M	1987 GC	1987 HC	1987 J
CK	96	98	98	91	91	68	83	93	93	80	95
3–5	94	92	71	88	96	62	33	96	61	41	0
5–7	96	98	93	92	91	53	51	96	95	70	54
7–9	95	98	97	89	89	68	75	98	93	89	94
$FLSD_{0.05}$[c]	7.3			13.5				15.5			

[a] CK = check plants (not inoculated). In other treatments, plants were inoculated at the three- to five-leaf (3–5), five- to seven-leaf (5–7), or seven- to nine-leaf (7–9) stage.
[b] Hybrids: M = Miracle; GC = Gold Cup; HC = Honeycomb; J = Jubilee.
[c] $FLSD_{0.05}$ = Fisher's least significant difference ($P < 0.05$). Values are for comparison of hybrid-treatment combinations.

Table 6. Regression coefficients and coefficients of determination for regressions of total husked ear weight (kg) and total number of marketable ears per plot on Stewart's and Goss's wilt severity 1 wk before harvest for four sweet corn hybrids

Hybrid	Year	Ear weight[a] b_0	b_1	b_2	r^2	Marketable ears b_0	b_1	b_2	r^2
Miracle	1986	4.52	ns	ns	0.09	19.95	ns	ns	0.09
	1987	4.88	ns	ns	0.31	20.00	ns	ns	0.18
Gold Cup	1986	2.83	ns	ns	0.02	19.96	ns	ns	0.01
	1987	4.56	−0.04	ns	0.54	22.12	0.21	−0.06	0.55
Honeycomb	1986	4.50	−0.01		0.23	20.04	ns	ns	0.10
	1987	4.24	0.009	−0.0006	0.79	19.80	0.05	−0.002	0.87
Jubilee	1986	3.94	0.02	−0.0005	0.43	17.92	0.16	−0.002	0.41
	1987	4.88	−0.008	−0.0004	0.93	19.22	0.14	−0.003	0.93

[a] Regression coefficients (b_0, b_1, and b_2) and coefficients of determination (r^2) for the equation $Y = b_0 + b_1(X) + b_2(X)^2$, where Y is sweet corn yield (ear weight or marketable ears) and X is Stewart's or Goss's wilt severity 1 wk before harvest. ns = Not significant.

of the maximum yield for each hybrid in each experiment. The percentage of the maximum total husked ear weight and the total number of marketable ears were then regressed on disease severity 1 wk before harvest for each experiment and hybrid. A single regression model was developed to estimate the relationship between percentage yield (as the total husked ear weight and as the total number of marketable ears) and disease severity of Stewart's and Goss's wilts.

RESULTS

Stewart's and Goss's bacterial wilts affected sweet corn plants similarly. Symptom development was less in resistant than in susceptible hybrids, and both diseases were more severe in plants inoculated at the three- to five-leaf stage, compared to plants inoculated at later growth stages. In the resistant hybrid, the two diseases did not become systemic (i.e., all disease ratings were below 4). Localized symptoms developed, and tip ends of infected leaves became necrotic. In the susceptible hybrid, disease symptoms were observed about 4 days after inoculation, as leaves emerged from the whorl. Inoculated leaves began to wilt 2–3 days later. Chlorotic streaks appeared at the inoculation points and advanced toward the tip and basal ends of the leaves. Within 2–3 wk after inoculations, inoculated leaves were predominantly necrotic, and symptoms were systemic throughout the plants. Plants died or remained severely stunted. Disease development in the moderately resistant and moderately susceptible hybrids was intermediate to that of the resistant and susceptible hybrids. Both diseases were severe in the susceptible hybrid in 1985 and 1986; however, Stewart's wilt did not develop despite repeated inoculations in 1987.

Stewart's and Goss's wilt development was affected differently among hybrids at different times of inoculation (Tables 1 and 2); however, there were no significant differences between the pathogens. Disease ratings 2–3 wk after inoculation were significantly greater than that of the check treatment for all hybrids inoculated at the three- to five-leaf stage except for Stewart's wilt on Miracle in 1985 and Goss's wilt on Miracle in 1986 (Table 1). When plants were inoculated at the five- to seven-leaf or seven- to nine-leaf stage, disease ratings for the moderately resistant, moderately susceptible, and susceptible hybrids were significantly greater than that of the check treatment except for Goss's wilt on Gold Cup at the five- to seven-leaf stage in 1985. For the resistant hybrid, Miracle, inoculated at the five- to seven-leaf or seven- to nine-leaf stage, disease ratings 2–3 wk after inoculation were not significantly different from that of the check except for Stewart's wilt at the five- to seven-leaf stage in 1986. One week before harvest, disease severity was relatively low in the check plots except for Jubilee (Table 2). Within hybrids, disease severity was greatest for plants inoculated at the three- to five-leaf stage except for Gold Cup inoculated with *C. m.* subsp. *nebraskense* at the five- to seven-leaf stage in 1986 (Table 2).

Total husked ear weight was affected differently among hybrids at different times of inoculation (Table 3). The total husked ear weight of Miracle was not significantly reduced by inoculation. For the moderately resistant Gold Cup, inoculation at the three- to five-leaf stage reduced total husked ear weight about 9 and 50% in 1985 and 1987, as compared to the check treatment. When plants were inoculated at the three- to five-leaf stage, reductions in total ear weight were about 9 and 35% for the moderately susceptible Honeycomb in 1986 and 1987 and about 38, 39, and 99% for the susceptible Jubilee in 1985, 1986, and 1987. Inoculation at the five- to seven-leaf or seven- to nine-leaf stage did not significantly affect the total husked ear weight of the resistant and moderately resistant hybrids. When plants were inoculated at the five- to seven-leaf stage, the total husked ear weight of Honeycomb was reduced by about 10% in 1986, and that of Jubilee was reduced by about 13, 16, and 35% in 1985, 1986, and 1987. The total husked ear weight of Honeycomb and Jubilee inoculated at the seven- to nine-leaf stage was not significantly reduced; means were 7 and 5% lower for Jubilee than for the check treatments in 1985 and 1986. In 1985, when the pathogen main effect term was significant, the treatment means of total husked ear weight were 6,306 and 6,034 g for plants infected with Stewart's wilt and Goss's wilt, respectively.

The total number of marketable ears per plot was affected by the hybrid and the growth stage at which plants were inoculated (Table 4). The total number of marketable ears of Miracle was not affected by inoculation. For Gold Cup, the total was reduced only in 1987, from 23 ears in uninoculated check plots to 15 ears in plots inoculated at the three- to five-leaf stage. Similarly, the total number of marketable ears of Honeycomb was reduced only for plants inoculated at the three- to five-leaf stage in 1987. For Jubilee, reductions in the total number of marketable ears occurred in all years for plants inoculated at the three- to five-leaf stage and in 1985 for plants inoculated at the five- to seven-leaf stage.

The percentage of marketable primary ears was affected similarly (Table 5). Inoculation had no affect on the resistant hybrid, Miracle. Inoculation at the three- to five-leaf stage reduced the percentage of marketable primary ears of the susceptible Jubilee by 27, 50, and 95% in 1985, 1986, and 1987, respectively, whereas reductions of 32 and 39% occurred only in 1987 on the moderately resistant Gold Cup and the moderately susceptible Honeycomb, respectively. Inoculation of Jubilee at the five- to seven-leaf stage reduced the percentage

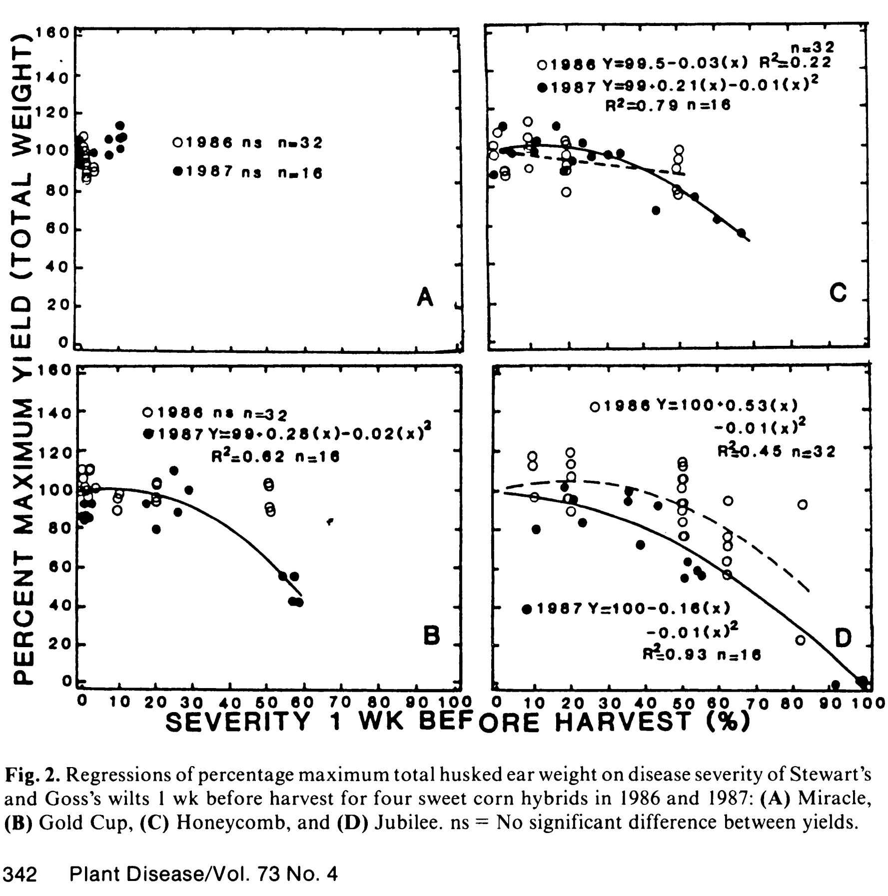

Fig. 2. Regressions of percentage maximum total husked ear weight on disease severity of Stewart's and Goss's wilts 1 wk before harvest for four sweet corn hybrids in 1986 and 1987: **(A)** Miracle, **(B)** Gold Cup, **(C)** Honeycomb, and **(D)** Jubilee. ns = No significant difference between yields.

of marketable primary ears by 32% in 1986 and by 41% in 1987. No significant effects were observed for any of the hybrids inoculated at the seven- to nine-leaf stage.

Significant differences in ear diameter and length followed the general pattern of other yield variables, with no effect of the bacterial wilts on Miracle or Gold Cup inoculated at any growth stage or on Honeycomb or Jubilee inoculated at the seven- to nine-leaf stage. Slight reductions in ear length and diameter were observed for Honeycomb inoculated at the three- to five-leaf stage and Jubilee inoculated at the five- to seven-leaf stage. Ear diameter was reduced by 4–44% and ear length by 7–50% for Jubilee inoculated at the three- to five-leaf stage.

Variation in total yield (the total husked weight and the total number of marketable ears) was explained when the independent variable of regression models was Stewart's or Goss's wilt severity 1 wk before harvest (Table 6). Both bacterial wilts decreased the total husked ear weight of Gold Cup, Honeycomb, and Jubilee, as indicated by significant slope coefficients, but had no effect on total ear weight of Miracle. The total number of marketable ears of Gold Cup, Honeycomb, and Jubilee also decreased as a function of Stewart's and Goss's wilt severity 1 wk before harvest, as indicated by slope coefficients (Table 6). Stewart's and Goss's wilts did not reduce the total number of marketable ears of Miracle.

Regressions of percentage maximum yield (as total husked ear weight) on disease severity 1 wk before harvest were significant for Gold Cup in 1987 and Honeycomb and Jubilee in 1986 and 1987 (Fig. 2). Disease severity on Miracle ranged from 1 to 11% (ratings of 1–4.1), and slope coefficients were not significant. For Gold Cup, Honeycomb, and Jubilee, quadratic regressions gave the best fit to the data when severity was a significant independent variable, thus indicating a severity threshold below which damage did not occur, except for Honeycomb in 1986, for which the regression was linear. Regressions of percentage maximum yield (as the total number of marketable ears) on disease severity 1 wk before harvest (Fig. 3) were similar to those for percentage total husked ear weight.

Single models for the moderately susceptible and susceptible hybrids were developed over 2 yr for percentage maximum total husked ear weight (Fig. 4) and percentage maximum total number of marketable ears (Fig. 5). A damage threshold was observed at about 40% severity, which corresponds to the point at which plants begin to be stunted because of systemic infection (disease rating of 6.1). Disease severity was above 40% primarily for plants inoculated at the three- to five-leaf or five- to seven-leaf stage (Table 2 and Figs. 4 and 5); thus, the late-season phases of the two bacterial wilts had little effect on yield, even for the susceptible hybrids. Beyond this disease threshold, linear models fit the yield data, although variation about the predicted value was substantial: $Y = 174.1 - 1.71(X)$ and $Y = 204.7 - 1.89(X)$, where X is disease severity 1 wk before harvest and Y is percentage yield, as percentage total husked ear weight and percentage total number of marketable ears, respectively.

DISCUSSION

The development of Stewart's and Goss's wilts and the effects of the bacterial wilts on sweet corn yield were extremely similar, as indicated by the general absence of significant main effects of pathogens and interactions involving pathogens in the ANOVAs of most of the independent variables. Previously, correlations between sweet corn hybrid reactions to Goss's wilt and Stewart's wilt had been reported to range from 0.48 to 0.88 (15,17). Thus, because of the similarity of the two bacterial wilts in terms of genotype response, disease development, and effects on sweet corn yield, management decisions should be similar for these two diseases, except for the few hybrids that respond differently to the two pathogens.

The development of the bacterial wilts varied by hybrid and the host growth stage at which plants were inoculated. Disease severity was greatest when susceptible sweet corn hybrids were inoculated at the three- to five-leaf stage, as compared to plants inoculated at the five- to seven-leaf or seven- to nine-leaf stage. Nevertheless, both diseases were severe on the susceptible hybrid, Jubilee, inoculated at all plant growth stages. Hybrids having moderate levels of resistance, Gold Cup and Miracle, were not severely affected when inoculated at the later growth stages. Significant hybrid-by-treatment interactions reflected the difference between resistant and susceptible genotypes with respect to the responses of plants at different ages. Calub et al (5) previously reported a significant role of plant age in the development of Goss's wilt. Two-week-old dent corn seedlings were most susceptible; however, the plant age response was not the same for all genotypes, primarily because the response of susceptible hybrids was affected very little by plant age at inoculation.

The age-dependent responses observed in these experiments may have important implications for Stewart's wilt management in plantings that are staggered in time, a common sweet corn production practice. In years for which forecasts indicate favorable Stewart's wilt development (6,21), inoculum and vectors are predicted to be abundant in mid- to late-season plantings, when moderately susceptible and susceptible hybrids are at the critical susceptible growth stages (three- to seven-leaf stages), resulting in substantial yield reductions if plants are infected. To avoid yield losses in those

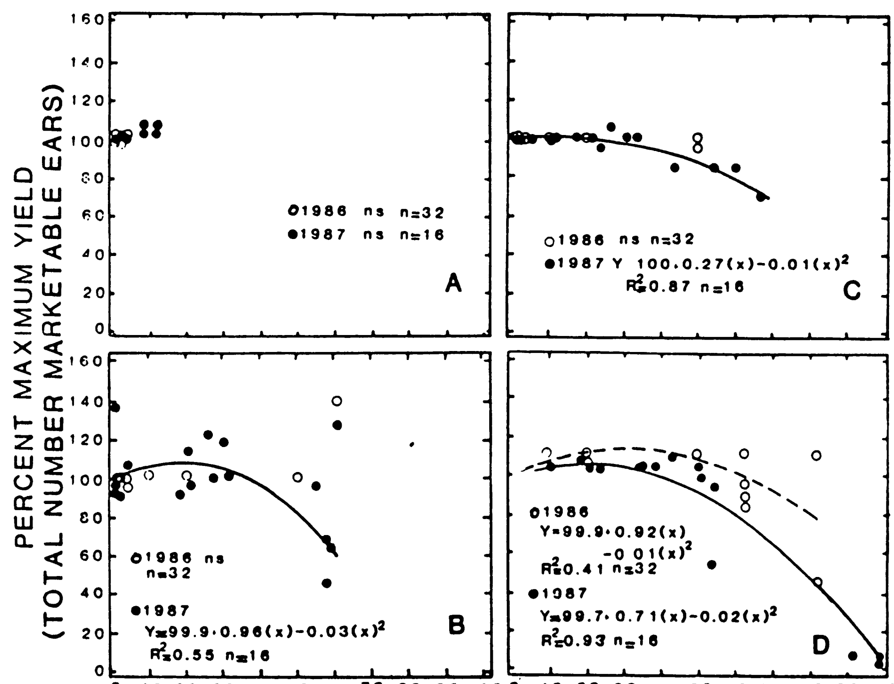

Fig. 3. Regressions of percentage maximum total number of marketable ears on disease severity of Stewart's and Goss's wilts 1 wk before harvest for four sweet corn hybrids in 1986 and 1987: **(A)** Miracle, **(B)** Gold Cup, **(C)** Honeycomb, and **(D)** Jubilee. ns = No significant difference between yields.

years, moderately resistant and resistant hybrids should be grown at all plantings except for the very earliest. The age-dependent responses of sweet corn hybrids may also affect strategies for the use of insecticides to control Stewart's wilt, if seedling applications of insecticides can maintain disease severity below the threshold level of 40% (ratings of approximately 6, which correspond to systemic infection and stunting). Two to five applications of insecticides such as carbaryl, carbofuran, diazinon, and methoxychlor, beginning immediately after emergence, with an interval of 3–5 days, have been recommended to control the corn flea beetle (2).

The effect of the two diseases on sweet corn yield gradually increased as susceptibility increased and as the age at which plants were inoculated decreased. For example, the yield of the resistant hybrid, Miracle, was not significantly affected by inoculation. The moderately resistant hybrid, Gold Cup, was sometimes affected when plants were inoculated at the three- to five-leaf stage. The yield of the moderately susceptible hybrid, Honeycomb, was always reduced when plants were inoculated at the three- to five-leaf stage and occasionally at the five- to seven-leaf stage. Yield reductions for the susceptible hybrid, Jubilee, inoculated at the three- to five-leaf or five- to seven-leaf stage were greater than those of the moderately resistant and moderately susceptible hybrids. Thus, comparable responses might be expected for a susceptible hybrid inoculated at a later growth stage (seven- to nine-leaf stage) and a moderately resistant or moderately susceptible hybrid inoculated at an early growth stage (three- to five-leaf or five- to seven-leaf stage, respectively), although responses are likely to vary among environments.

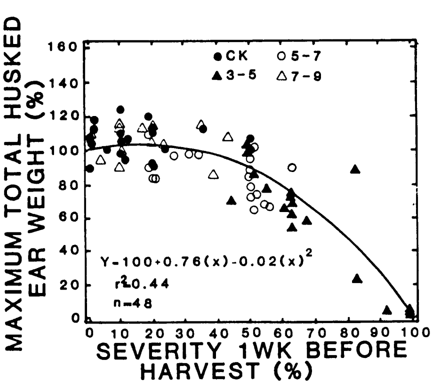

Fig. 4. Regression of percentage maximum total husked ear weight on disease severity of Stewart's and Goss's wilts of two susceptible sweet corn hybrids, Honeycomb and Jubilee, 1 wk before harvest over 2 yr. In the check treatment (●), plants were not inoculated. In other treatments, plants were inoculated at the three- to five-leaf (▲), five- to seven-leaf (○), or seven- to nine-leaf (△) stage.

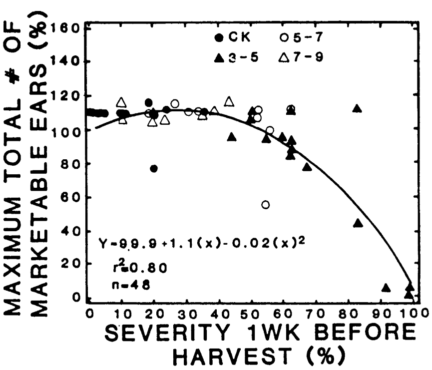

Fig. 5. Regression of percentage maximum total number of marketable ears on disease severity of Stewart's and Goss's wilts of two susceptible sweet corn hybrids, Honeycomb and Jubilee, 1 wk before harvest over 2 yr. In the check treatment (●), plants were not inoculated. In other treatments, plants were inoculated at the three- to five-leaf (▲), five- to seven-leaf (○), or seven- to nine-leaf (△) stage.

In the absence of individual models for specific hybrids and years, the general regression models derived from our data provide an estimate of damage due to Stewart's and Goss's wilts. The models were similar for hybrids grown for fresh market, for which yield was measured as the number of marketable ears, and for hybrids grown for processing, for which yield was measured as ear weight. A damage threshold of approximately 40% disease severity (rating of approximately 6) 1 wk before harvest was observed. Plants with these severity ratings were systemically infected, whereas infection of plants with ratings below 6 (severity below 37.5%) was usually not so severe as to cause stunting. Beyond this threshold, a linear regression of sweet corn yield on disease severity estimated a 17 and 19% decrease in yield by weight and number of ears, respectively, for every 10% increase in disease severity. Damage thresholds of about 0, 15, and 20% were observed previously for these bacterial wilts in a sweet corn hybrid disease nursery (17). The difference between those damage thresholds and the 40% threshold in this study may be attributed to the lack of consideration of secondary ears in the previous study or the various times of inoculation in this study. All plants in the disease nurseries (17) were inoculated at the three- to five-leaf stage, when plants were most affected by the bacterial wilts. Thus, various levels of severity at harvest were due to host reactions in the nurseries, whereas in this study various levels of severity 1 wk before harvest were due to host reactions and the time of inoculation. Possibly, equal levels of disease severity 1 wk before harvest do not have equal effects on yield, depending on the time of disease onset. For example, an intermediate hybrid infected at the three- to five-leaf stage and a susceptible hybrid infected at the seven- to nine-leaf stage may both display more than 40% disease severity 1 wk before harvest; however, yield reduction may be greater in the intermediate hybrid as a result of disease onset at an earlier growth stage. The 40% damage threshold in this study applies to the moderately susceptible and susceptible hybrids, with differences in severity primarily due to the time of inoculation. At disease levels of less than 40%, which were observed in the check treatments and in the seven-to-nine-leaf-stage treatments, sweet corn yields were not substantially decreased. Beyond 40% disease severity, which was due to early disease onset (at the three- to seven-leaf stages) and resulted in systemic infection, yields were decreased significantly.

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