

Evaluation of Fungicide Seed Treatments for *Shrunken-2* ("Supersweet") Sweet Corn

D. O. WILSON, JR., S. K. MOHAN, and E. A. KNOTT, University of Idaho, Parma 83660, and B. SHAFII, Statistical Programs, College of Agriculture, University of Idaho, Moscow 83843

ABSTRACT

Wilson, D. O., Jr., Mohan, S. K., Knott, E. A., and Shafii, B. 1993. Evaluation of fungicide seed treatments for *shrunken-2* ("supersweet") sweet corn. *Plant Dis.* 77:348-351.

Shrunken-2 ("supersweet") sweet corn hybrids suffer serious stand losses due to seed rot, damping-off, and seedling blight caused by various seedborne and soilborne pathogens. To identify an optimal fungicide seed treatment to improve *shrunken-2* sweet corn seedling stand in the field, we evaluated 30 treatments in 1989 and 11 in 1990 through a network of cooperators at 32 and 19 locations during 1989 and 1990, respectively. Treatment with a mixture of captan, thiram, metalaxyl, and benomyl (CTMB) was usually the best. In 1989 final stand with this mixture ranged from 13 to 86% (median 72%), compared to 1-72% (median 54%) for the untreated control. In 1990 treatment with CTMB produced stands of 8-91% (median 65%), compared to 0-89% (median 36%) for the control. Addition of other fungicides or insecticides to the CTMB mixture did not improve the final stand. Fungicides were identified that could substitute for certain components of the optimal mixture without loss of efficacy, but omission of any of the components usually resulted in reduced efficacy. The results indicate that an effective seed treatment for *shrunken-2* sweet corn should include a broad-spectrum protectant fungicide such as thiram or captan-thiram, metalaxyl, and a systemic fungicide with activity against *Penicillium* and *Fusarium*, such as imazalil or a benzimidazole fungicide.

Additional keywords: chemical control, *Fusarium moniliforme*, *Penicillium oxalicum*, *Pythium ultimum*

Most commercial sweet corn hybrids in the United States carry one of two mutant endosperm genes, sugary (*su*) or *shrunken-2* (*sh2*). These recessive alleles interfere with starch synthesis in the endosperm, so that sugars accumulate in the kernel (15). Many newer *su* hybrids also bear the sugary extender (*se*) gene, which increases sugar levels in the presence of *su*.

The *sh2* hybrids, also known as "supersweets," have the highest level of sweetness and retain it long after harvest. This facilitates shipment of fresh ears to distant markets with minimal loss of eating quality. These traits have made *sh2* hybrids attractive to fresh market producers, shippers, and consumers. In 1991 about 33% of the sweet corn seed acreage was devoted to production of *sh2* hybrids.

Shrunken-2 hybrids, however, generally show poor germination, low vigor, and low ability to establish stands in the field compared to *su* hybrids (15). The thin and often cracked pericarp may allow rapid microbial colonization of the seed after planting. Fungi of several genera (e.g., *Fusarium*, *Penicillium*, *Rhizopus*) can often be found growing and sporulating profusely in the ears

before seed harvest. In addition to surface contaminants, several fungi are internally seedborne (1,2,5,20). Some of the fungi commonly found on seed, like *Fusarium moniliforme* J. Sheld. and *Penicillium oxalicum* Currie & Thom, have been reported to be pathogenic to sweet corn seedlings (2,5,8,11,14,20). These pathogens can reduce seed germinability and cause seed rot, seedling blight, and low seedling vigor (2,5,8,11,18,20). The presence of fungal pathogens on and in the seed, in addition to the inherent weakness of seeds of this genotype, leads to poor field performance (3,10). Soilborne pathogens (e.g., *Pythium ultimum* Trow, *Fusarium* spp., *Rhizoctonia solani* Kühn, *Rhizopus* spp.) can further reduce the stand (3,6,8,12,13,19), depending on geographic location, soil type, soil moisture, and temperature during seed germination and seedling emergence.

The sweet corn industry relies on seed treatment with mixtures of fungicides to obtain satisfactory stands in the field with *sh2* seeds. Berger and Wolf (3) found that seeds of the *sh2* hybrid Florida Staysweet treated with captan-benomyl yielded consistently higher stands than seeds treated with several other fungicides or mixtures of fungicides. Results from our previous work (*unpublished*) at Parma, ID, indicated that treatment with a mixture of captan, thiram, metalaxyl, and benomyl (CTMB) increased seedling stand of *sh2* hybrids. However, the effectiveness of the treatments and the relative importance of the

components under diverse soil and weather conditions were not known. This study was conducted to identify optimum seed treatment mixtures for improving seedling stand of *sh2* sweet corn across diverse planting environments.

MATERIALS AND METHODS

Seed treatments. In 1989, we used seed of the *sh2* hybrid Ssupersweet 8701W, provided by Abbott & Cobb Co. In 1990, we used seed of two *sh2* hybrids, Supersweet Jubilee, provided by Rogers NK Seed Co., and Ssupersweet 8701W. A sugary enhanced (*su,se*) genotype, Snowbelle, provided by Asgrow Seed Co., was also included in 1990.

Thirty treatments were included in the 1989 trial. A complete factorial of four fungicides—captan plus thiram, benomyl, metalaxyl, and imazalil—formed the core of the treatment design. Six products were evaluated as possible additions to CTMB: iprodione, carboxin, PCNB, TCMTB, and two insecticides, thiodicarb and chlorpyrifos. Eight more treatments were included to examine the effect of deletion of or substitution for components of the CTMB mixture: captan-metalaxyl-benomyl, thiram-metalaxyl-benomyl, PCNB-metalaxyl-benomyl, PCNB-metalaxyl-imazalil, TCMTB-metalaxyl-benomyl, iprodione-metalaxyl-benomyl, PCNB-iprodione-metalaxyl-benomyl, and captan-thiram-metalaxyl-thiabendazole. An untreated control was included.

In view of the possible loss of captan for use as a seed treatment, and based on the 1989 results, thiram alone was used as the basic broad-spectrum protectant fungicide in the 1990 treatment design. The 1990 treatments were thiram, thiram-metalaxyl, thiram-benomyl, metalaxyl-benomyl, thiram-metalaxyl-benomyl, thiram-metalaxyl-imazalil, CTMB, thiram-metalaxyl-benomyl-carboxin, thiram-metalaxyl-benomyl-triadimenol, captan-metalaxyl-*Trichoderma harzianum* Rifai (F-Stop, Eastman Kodak, Rochester, NY), and the untreated control.

For each treatment, the fungicides were applied as a premixed slurry by Gustafson Inc. at their facility in Plano, TX. Treatments were applied at the label rates for sweet corn seed. Products not labeled for sweet corn were applied at rates typical for other large-seeded vegetable crops. The rates used (g a.i./kg of seed) were: thiram (Gustafson 42-S), 1.25; metalaxyl (Apron FL), 0.15;

benomyl (Benlate 50WP), 1.25; imazalil (Flo-Pro Imz FF), 0.07; thiabendazole (Mertect LSP), 0.31; captan 400D, 1.25; carboxin (Vitavax 34), 0.78; iprodione (Epic 30FL), 0.65; chlorpyrifos (Lorsban 50), 0.83; thiodicarb (Magnum 3.2FL), 1.00; PCNB (RTU-PCNB), 0.52; triadimenol (Baytan 30FL), 0.62; and TCMTB (Nusan 30), 0.60. *T. harzianum* was applied at a rate of 4.16 g of formulation per kilogram of seed together with captan at 0.50 and metalaxyl at 0.75 g a.i./kg.

Treated seeds were sent to cooperators at various locations and were planted according to local practices. In general, four replicates of each treatment were planted, with 100 seeds per plot (fewer seeds were planted at a few locations). The experimental design was a split-plot randomized complete block with hybrid as the main plot and seed treatment as the subplot. Stand counts taken at the five- to six-leaf stage, approximately 6 wk after planting for most locations, were considered the final stand and were expressed as percentages.

Location of sites. The sites for the 1989 trials were as follows: Honolulu, HI (HI); Hokkaido, Japan (Japan); Belle Glade, FL (FL1 and FL2); Hollister, Indio, and Davis, CA (CA1, CA2, and CA3, respectively); Tifton, GA (GA1 and GA2); Nyssa and Ontario, OR (OR1 and OR2); Farmington and Waseca, MN (MN1 and MN2); LeSueur, MN (MN3 and MN4); Fruita, Brighton, and Henderson, CO (CO1, CO2, and CO3); Champaign and Urbana, IL (IL1 and IL2); Rochester, NY (NY1 and NY2); Nampa, ID (ID1, ID2, and ID3); Buhl, ID (ID4); Parma, ID (ID5 and ID6); Sun Prairie, WI (WI1 and WI2); Johnston, IA (IA); and Elizabethtown, PA (PA). Some trials were planted at the same location but on different planting dates. GA2, NY2, MN4, and ID6 refer to later plantings at the same locations. Other trials (FL1 and FL2, WI1 and WI2, and ID1, ID2, and ID3) were planted in the same general area but by different cooperators.

Some of the same locations were used for the 1990 trial (Japan, GA1, ID1, ID2, ID3, ID6, CA3, MN2, MN3, MN4, OR1, OR2, CO2, NY2, WI1, IA, and IL2), and two new sites, Fort Collins, CO (CO4), and Sun City, FL (FL3), were added. Only one site (MN3 and MN4) had two planting dates (MN4 was the later planting date).

Statistical analysis. The 1989 data were subjected to combined analysis of variance using SAS PROC ANOVA (16). The terms in the model included location, treatment, block within location, and location \times treatment. The 1990 data were unbalanced because of missing plots, and analysis of variance was attempted using SAS PROC GLM. The analysis could not be performed on the whole data set because of computer memory limitations. Hence, random subsets of the data

were subjected to analysis of variance. The terms in the model included treatment, hybrid, location, block within location, hybrid \times treatment, location \times hybrid, and treatment \times location.

Significant treatment \times location interactions ($P < 0.0001$) in these initial analyses suggested that the data could not be simply averaged across locations. Because a large number of locations or location \times hybrid combinations were included in the experiment, a method was sought to detect clusters of locations with uniform behavior. If such groups could be identified, they would exhibit smaller treatment \times location effects, which would allow calculation of treatment means across locations within the groups and thus make it easier to summarize the results. The groupings might also be used to classify locations with regard to fungicide seed treatment responses.

To cluster locations, we decomposed the interaction as described by Gabriel (9) and Bradu and Gabriel (4). Briefly, treatment means averaged over blocks were subjected to analysis of variance, including only main effects (treatment, location) in the model. The matrix of residuals was subjected to principal component analysis (PCA) (4,9). This method extracts orthogonal coordinate axes, called principal components, that describe the structure of the interaction (17). Although many principal components can be calculated, the first few generally account for most of the interaction variance (7,17). In our analyses, only the first two principal components were retained; jointly they accounted for 49.6 and 50.8% of the interaction variability in the 1989 and 1990 data, respectively.

PCA could not be applied directly to the 1990 data set because the available methods are inherently two-dimensional, whereas the 1990 experiment had a three-

way treatment design. Because the hybrid \times treatment interaction was significant ($P = 0.001$), we could not average over hybrids to achieve a two-way classification. Instead, each location \times hybrid combination was treated as if it were a separate location for purposes of clustering locations. This was appropriate because our main goal was to identify optimum seed treatments applicable to hybrids in general.

The principal component scores derived from the interaction terms were used to construct biplots (4,7,9) (Figs. 1 and 2). Each axis of the plots in Figures 1 and 2 represents a linear combination of the residuals from each mean. The two axes are orthogonal. Points near the center of the graphs represent locations that were consistent in their responses to treatments. Those farther from the main axes show unusual patterns of responses to the treatments and contributed the most to the interaction variance (17).

Instead of distinct groups of locations, only one central group with outliers was discernible each year. Locations that fell outside the central cloud of points in the graphs (Figs. 1 and 2) were removed from the full data sets for individual analysis and consideration. Data from the remaining locations were pooled to form the main data sets, and analysis of variance was again conducted on the original replications within each location group. The model for the main data set in 1989 included location, treatment, and block within location. The 1990 model included location/hybrid, block within location/hybrid, and treatment. Data from locations (1989) or location/hybrids (1990) set aside for special consideration were analyzed individually using a model that included treatment and block. Fisher's protected least significant difference was used to perform mean separation within the two main data sets and individually

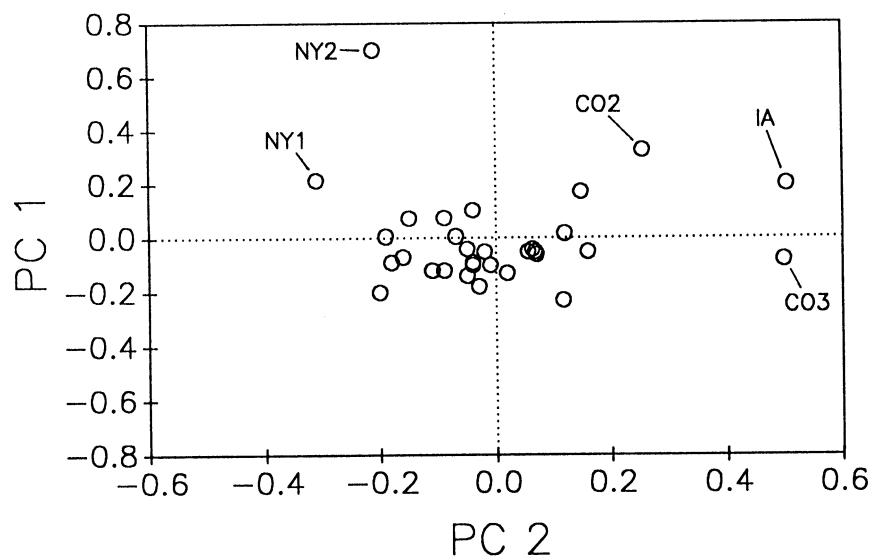


Fig. 1. Biplot of principal component scores for locations from principal component analysis of the location \times treatment interaction in the 1989 *sh2* sweet corn seed treatment trial. Labeled points indicate locations that were removed from the data set for individual analysis.

for each location set aside for special consideration.

RESULTS

Preliminary analysis of variance showed that the location \times treatment

interaction was significant ($P < 0.0001$) in both years. In 1990 the hybrid \times treatment interaction was also significant ($P < 0.001$).

Location means across treatments in 1989 ranged from 18 to 82% (median

67%). In 1990, location/hybrid means ranged from 10 to 91% (median 57%). Treatment means across locations for the untreated control ranged from 1 to 72% (median 54%) in 1989 and from 0 to 89% (median 36%) in 1990. Stands from the CTMB treatment ranged from 13 to 86% (median 72%) in 1989 and from 8 to 91% (median 65%) in 1990. Postemergence stand loss in most locations was below 5%.

In 1989, five locations—NY2, CO2, IA, CO3, and NY1—appeared to lie outside the main group (Fig. 1). Stand means for these locations were 41, 33, 31, 51, and 45%, respectively. In 1990, eight location/hybrid (A = Snowbelle, B = Supersweet Jubilee, C = Supersweet 8701W) combinations—CO2C, CO4A, CO4B, CO4C, MN2A, MN3A, MN3B, and ID2A—were removed from the full data set (Fig. 2). Stand means for these location/hybrids were 36, 57, 40, 59, 63, 73, 48, and 63%, respectively. The other locations were clustered around the main axes, indicating more uniform behavior (Figs. 1 and 2).

In the 1989 main data set, all the single-component treatments significantly increased final stand compared to untreated seed (Table 1). In general, the best seed treatments consisted of three components: captan-thiram or thiram alone, metalaxyl, and a broad-spectrum systemic fungicide such as benomyl, thiabendazole, or imazalil. However, imazalil was not as effective as benomyl in this combination, resulting in a 4% lower final stand in the main data set and 21 and 19% reductions in two of the five locations considered independently (Table 1). Treatments that performed equally well and were never significantly different from CTMB included thiram-metalaxyl-benomyl, captan-thiram-metalaxyl-thiabendazole, and PCNB-iprodione-metalaxyl-benomyl (Table 1). Omission of any component, other than captan, from the CTMB combination caused a 3–8% reduction in the final stand. Addition of other products to CTMB did not increase the final stand (Table 1).

The locations removed from the main group in 1989 differed in treatment responses in several ways. For example, in CO2 and CO3, captan-thiram increased the final stand over the untreated control, but treatment with metalaxyl alone did not increase final stand significantly (Table 1). In IA, captan-thiram-metalaxyl-imazalil was 21% lower than CTMB. The highest ranked treatments at these locations were all combinations of CTMB plus another chemical. However, none of these combinations was significantly better than CTMB (Table 1).

In the 1990 trials, thiram-metalaxyl-benomyl (TMB) was considered the standard treatment, and the other treatments were compared against it. The

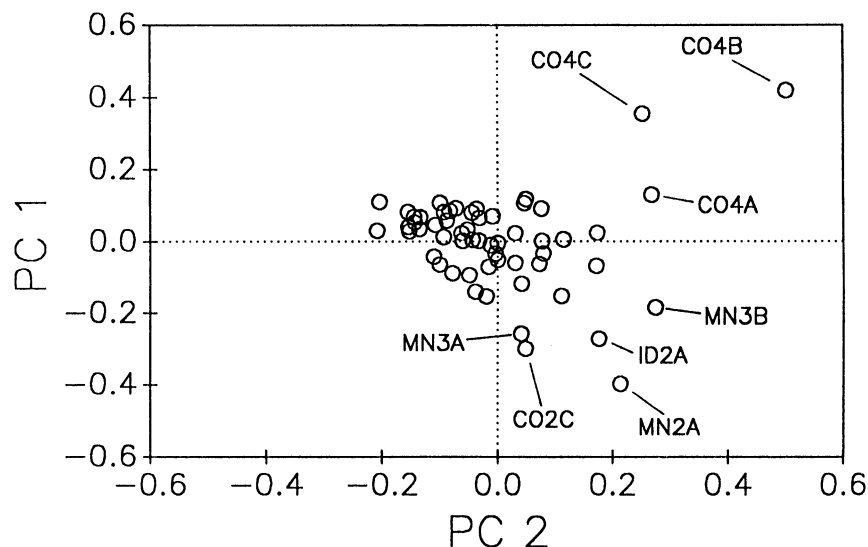


Fig. 2. Biplot of principal component scores for location/hybrids from principal component analysis of the location/hybrid \times treatment interaction in the 1990 *sh2* sweet corn seed treatment trial. Labeled points indicate location \times hybrid combinations that were removed from the data set for individual analysis.

Table 1. Final stand means (%) for the pooled locations and for locations analyzed individually in the 1989 sweet corn seed treatment trial

Treatment code ^a	Main data set ^b	Locations analyzed individually ^c				
		CO2	CO3	NY2	IA	NY1
U	53	8	36	7	13	26
M	58	20	38	18	15	40
I	59	17	49	7	21	26
B	60	14	52	6	18	20
BI	62	24	50	11	27	36
MBE	62	42	50	48	38	50
MIP	62	17	47	23	23	45
MI	63	19	47	29	26	34
CTM	63	29	40	41	23	46
CT	65	38	49	46	17	32
MB	65	26	39	49	20	58
CTI	66	22	50	30	33	45
MBI	66	45	59	48	46	52
MBP	66	22	52	46	16	45
MBN	67	18	43	46	23	50
CTB	67	25	57	36	31	48
CTMI	67	37	50	40	24	31
CMB	68	41	46	56	24	51
CTBI	69	39	63	19	35	47
CTMBV	69	41	63	57	44	43
CTMZ	69	31	59	49	34	50
MBEP	69	37	52	58	42	50
CTMBP	69	52	58	59	43	51
CTMBE	69	48	57	68	52	50
TMB	69	51	40	56	39	56
CTMBD	69	45	52	59	41	60
CTMBN	69	39	58	51	36	51
CTMB	71	41	58	59	45	50
CTMBI	71	46	66	59	46	54
CTMBL	71	43	57	53	39	67
LSD _(0.05) ^d	2	16	13	15	11	17

^aU = untreated control, T = thiram, M = metalaxyl, B = benomyl, I = imazalil, Z = thiabendazole, C = captan, V = carboxin, E = iprodione, L = chlorpyrifos, D = thiodicarb, P = PCNB, and N = TCMTB (Nusan).

^bMeans across 27 locations.

^cLocations considered individually are denoted by the state code. Multiple locations within states are indicated by the state code followed by a number.

^dLeast significant difference ($P < 0.05$).

final stand with the biocontrol treatment captan-metalaxyl-*Trichoderma* (46%) was higher than the untreated control (37%), but not as high as TMB (60%) (Table 2). Treatment with thiram-metalaxyl-imazalil resulted in a stand 4% lower than treatment with TMB in the main data set. In two of the eight location/hybrid combinations considered independently, use of imazalil in place of benomyl in this combination lowered the final stand by 19% (Table 2). No component added to TMB increased final stand significantly, and again, removal of any component from the optimum combination of TMB decreased final stand.

In MN2A and MN3B, location/hybrid combinations removed from the main data set in 1990, thiram-metalaxyl-imazalil was equal to TMB. At the Fort Collins (CO4) location, the thiram-metalaxyl-benomyl-carboxin treatment resulted in anomalously high counts (> 100%) for all three hybrids. We suspect that the seed packets for this treatment at this location were miscounted. Aside from this apparent error, the highest ranked treatments from CO4 included CTMB and TMB.

DISCUSSION

Most of the locations that were removed from the main data sets in both years were below average in final stand means. The collaborators at some of these locations described the weather as unusually cold or wet after planting. Physical stress may have altered the response to the treatments.

The results suggest that an effective formulation for *sh2* sweet corn seed treatment should include three components: a protectant such as captan-thiram or thiram, metalaxyl, and a broad-spectrum systemic such as one of the benzimidazoles or imazalil. The results are similar to those of Berger and Wolf (3), who found that seed treatment with mixtures of captafol (a broad-spectrum protectant) and benomyl produced consistently high stands of *sh2* sweet corn.

Although TMB and CTMB were the best combinations in our study, benomyl is no longer registered for use on sweet corn seed. Imazalil is currently available under an emergency exemption for treatment of sweet corn seed in Idaho.

The addition of other fungicides or insecticides to CTMB or TMB did not increase the stand. Effective substitutes could be found for all the components of CTMB except metalaxyl. For example, captan-thiram could be replaced by thiram alone or by PCNB-iprodione, and benomyl could be replaced by thiabendazole. Imazalil was not an equivalent substitute for benomyl in these mixtures and usually resulted in slightly lower stands.

Previous studies (1,3,6,7) have shown

Table 2. Final stand means (%) for the pooled location × hybrid combinations and for location × hybrid combinations analyzed individually in the 1990 sweet corn seed treatment trial

Treatment code ^a	Main data set ^b	Location × hybrid combinations analyzed individually ^c							
		MN2A	CO4A	ID2A	MN3A	CO4B	MN3B	CO4C	CO2C
U	37	9	28	16	33	16	11	42	5
T	45	51	56	46	67	18	22	49	20
CM- <i>Trich</i>	46	59	32	57	77	11	28	46	28
TM	50	79	55	70	83	26	50	42	49
BM	51	58	47	63	75	36	58	60	32
TB	53	47	54	59	65	42	31	60	28
TMI	56	76	60	81	73	30	61	53	46
TMBBay	59	75	61	67	77	42	63	60	36
TMB	60	81	69	72	92	49	67	65	56
CTMB	61	78	65	80	83	52	62	61	58
TMBV	62	80	101	79	81	114	71	113	37
LSD _(0.05) ^d	3	12	13	24	13	10	19	16	15

^aU = untreated control, T = thiram, M = metalaxyl, B = benomyl, I = imazalil, C = captan, V = carboxin, Bay = triadimenol, *Trich* = *Trichoderma harzianum*.

^bMeans across 49 location × hybrid combinations.

^cLocation × hybrid combinations considered individually are denoted by a state code followed by a number showing which trial within the state, followed by a letter denoting the hybrid (A = Snowbelle, B = Supersweet Jubilee, C = Supersweet 8701W).

^dLeast significant difference ($P < 0.05$).

that several types of fungi, both seed-borne and soilborne, can incite disease in sweet corn during seed germination, seedling emergence, and stand establishment. Anderegg and Guthrie (1) found that infection by *F. moniliforme* was independent of the level of seedborne inoculum when sweet corn seedlings were grown in fields in Caldwell, ID, but was correlated with levels of seedborne inoculum in plants grown in Moscow, ID. They concluded that soilborne *F. moniliforme* was important in sweet corn seedling infection. Our results also suggest the need to control both seedborne and soilborne fungi.

ACKNOWLEDGMENTS

We would like to thank Bob Trent (Crookham Co.); J. L. Brewbaker (University of Hawaii); Joe Kojima (Sakata Seed America); Lee Schweitzer (Asgrow Seed Co.); Ken Christensen and Tom Natti (Harris Moran Seed Co.); Jim Watkins, Kent Keim, Mike Cain, and Steve Grier (Sunseeds Genetics Inc.); Jim Berry (Rogers NK Seed Co.); D. R. Sumner (University of Georgia); Dennis Larsen (Campbell Foods); Vince Fritz (University of Minnesota); Clint Shock (Oregon State University); Bill Brown (Colorado State University); Pat Mosley (Illinois Foundation Seed); Duane Jeffers and Rob Gehin (Ferry Morse Seed Co.); Don Miles (Pioneer Seed Co.); Jerald Pataky (University of Illinois); Dean Cotton (Agway, Inc.); George Klacken (Green Giant); and R. T. McMillan, Jr. (University of Florida) for their cooperation. The help received from Kyle Rushing and Sue Shen of Gustafson Inc. is gratefully acknowledged. We would like to thank William Price for valuable help with the statistical analysis.

LITERATURE CITED

- Anderegg, J., and Guthrie, J. W. 1981. Seedborne *Fusarium moniliforme* and seedling infection in hybrid sweet corn. *Phytopathology* 71:1196-1198.
- Aulakh, K. S., Grewal, R. K., and Goel, R. K. 1976. Detection of seed borne fungi of maize and their role in causing seed rot and seedling infections. *Indian Phytopathol.* 29:241-245.
- Berger, R. D., and Wolf, E. A. 1974. Control of seedborne and soilborne mycoses of 'Florida Sweet' corn by seed treatment. *Plant Dis. Rep.* 58:922-923.
- Bradu, D., and Gabriel, K. R. 1978. The biplot

as a diagnostic tool for models of two-way tables. *Technometrics* 20:47-68.

- Caldwell, R. W., Tuite, J., and Carlton, W. W. 1981. Pathogenicity of *Penicillia* to corn ears. *Phytopathology* 71:175-180.
- Callan, N. W., Mathre, D. E., and Miller, J. B. 1990. Bio-priming seed treatment for biological control of *Pythium ultimum* preemergence damping-off in *sh2* sweet corn. *Plant Dis.* 74:368-372.
- Dunn, G., and Everitt, B. S. 1982. *An Introduction to Mathematical Taxonomy*, pp. 46-58. Cambridge Press, New York.
- Futrell, M. C., and Kilgore, M. 1969. Poor stands of corn and reduction of root growth caused by *Fusarium moniliforme*. *Plant Dis. Rep.* 53:213-215.
- Gabriel, K. R. 1971. The biplot graphic display of matrices with application to principal component analysis. *Biometrika* 58:453-467.
- Halfon-Meir, A., and Solel, Z. 1989. Control of seed-borne *Penicillium oxalicum* in sweet corn by seed treatment. *Z. Pflanzenkrankh. Pflanzenschutz.* 96:636-639.
- Halfon-Meir, A., and Solel, Z. 1990. Factors affecting seedling blight of sweet corn caused by seedborne *Penicillium oxalicum*. *Plant Dis.* 74:36-39.
- Ho, W. C. 1944. Soil-inhabiting fungi attacking the roots of maize. *Iowa Agric. Exp. Stn. Res. Bull.* 332:402-446.
- Koehler, B. 1957. Pericarp injuries in seed corn: Prevalence in dent corn and relation to seedling blights. *Bull. Agric. Exp. Stn. Ill.* 617.
- Lawrence, E. B., Nelson, P. E., and Ayers, J. E. 1981. Histopathology of sweet corn seed and plants infected with *Fusarium moniliforme* and *F. oxysporum*. *Phytopathology* 71:379-386.
- Marshall, S. W. 1987. Sweet corn. Pages 431-445 in: *Corn: Chemistry and Technology*. S. A. Watson and P. E. Ramstad, eds. American Association of Cereal Chemists, St. Paul, MN.
- SAS Institute. 1987. *SAS/STAT Guide for Personal Computers*, Version 6 ed. SAS Institute, Cary, NC.
- Shafii, B., Mahler, K. A., Price, W. J., and Auld, D. L. 1992. Genotype by environment interaction effects on yield and oil content of winter rapeseed. *Crop Sci.* 32:922-927.
- Singh, D., and Singh, T. 1977. Location of *Fusarium moniliforme* in kernels of maize and disease transmission. *Indian J. Mycol. Plant Pathol.* 7:32-38.
- Sumner, D. R., and Bell, D. K. 1982. Root diseases induced in corn by *Rhizoctonia solani* and *Rhizoctonia zeae*. *Phytopathology* 72:86-91.
- Wilson, D. O., Jr., and Mohan, S. K. 1991. Seedling blight of sweet corn. *Idaho Agric. Exp. Stn. Curr. Inf. Ser.* 879.