Reactions of Sweet Corn Hybrids with Resistance to Maize Dwarf Mosaic

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ABSTRACT

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Maize dwarf mosaic (MDM), caused by the sugarcane mosaic subgroup of potyviruses, can substantially reduce yield and ear quality of sweet corn (*Zea mays*). The effects of strain A of maize dwarf mosaic virus (MDMV-A) and strain MDMV-B of sugarcane mosaic virus (SCMV-MB) on resistant sweet corn hybrids were evaluated. Incidence of symptomatic plants, types of symptomatic responses, yield, and ear quality were compared among 20 hybrids in 1993 and 14 hybrids in 1994. Incidence of symptomatic plants measured at the beginning of harvest ranged from 4 to 100% in 1993 and 1 to 100% in 1994 and differed among hybrids inoculated with MDMV-A and/or SCMV-MB. Hybrids BiGuard, Dallas, HMX 9352, and Topacio had low (<20%) incidences of symptomatic plants when inoculated with MDMV-A, SCMV-B, or MDMV-A/SCMV-MB. Some hybrids had moderate responses, with incidence varying around 25%. Some hybrids appeared to have greater resistance to MDMV-A than to SCMV-MB. The incidence of sectoring plants (i.e., distinct bands or sectors of asymptomatic and symptomatic tissue) was higher for hybrids with high levels of resistance and ranged from 0 to 14% in 1993 and 0 to 83% in 1994. Ear weight of inoculated hybrids, expressed as a percentage of the non-inoculated control, ranged from 84 to 109% in 1993 and from 84 to 105% in 1994. Slope coefficients from regressions of percent ear weight on incidence ranged from –0.085 to –0.15. Butt blanking was associated with high incidence of MDM-symptomatic plants. None of the commercially available MDM-resistant sweet corn hybrids were 100% asymptomatic, but levels of resistance in most of these hybrids were adequate to minimize the effects of MDM on yield.

Yield and ear quality of sweet corn (*Zea mays* L.) can be reduced by maize dwarf mosaic (MDM). When Janson and Ellett (6) first reported the disease from southern Ohio, they observed nearly total losses in fields of late-planted sweet corn.

Yield reduction due to MDM and symptom expression are affected by the growth stage at which sweet corn is infected (5,12, 18,25). When late plantings of sweet corn are exposed to large populations of viruliferous aphids, plants can be infected at early stages of host growth. Infection at juvenile growth stages reduces plant height, delays maturity, decreases ear diameter and length, reduces ear weight, and increases the number of missing kernels in the basal end of the ear, which is commonly called butt blanking (5,17,18). Butt blanking results from retarded growth of pollen germ tubes on silks of infected plants (19), and it reduces the marketability of sweet corn used for fresh market and the weight of kernels cut from the cob that are used for processing.

Yields of susceptible sweet corn hybrids can be reduced as much as 40% by MDM (5,17,18,21). Reductions of up to 12% were observed when two sweet corn hybrids were inoculated at various growth stages with maize dwarf mosaic virus (MDMV-A) (5). Yield was reduced as much as 36% when hybrids were inoculated with sugarcane mosaic virus (SCMV-MB) at the four- to five-leaf stage (17). Reductions of 15% occurred when hybrids were inoculated at various growth stages with a mixture of MDMV-A and SCMV-MB (17). Yield reductions were as large as 40% when hybrids were inoculated at the three- to four-leaf stage with MDMV-A or SCMV-MB/MDMV-A (21).

Planting MDM-resistant hybrids is the most feasible method of controlling MDM, because control of aphid vectors is not practical. Resistance to MDM is thought to be expressed through restriction of cell-to-cell movement of the virus (11,12,15), interference with systemic movement (14), lower concentrations of virus (1,7,10,15, 21), and hypersensitive reactions that localize viral infection to a few cells (15). Hybrids and inbreds with genes for resistance to MDM do not always exhibit asymptomatic responses after infection (7,12,21, 25). Scott and Rosenkranz (25) suggested that symptomatic reactions may be induced by microenvironmental influences, expression of resistance at late growth stages, inoculum concentrations above a specific threshold, or a combination of these factors. Jones and Tolin (7) observed an unusual reaction in resistant hybrids expressed as distinct sectors of symptomatic and asymptomatic tissue. Concentration of virus was high in chlorotic, symptomatic sectors, but virus was not detected in asymptomatic sectors (7). Sectoring may be the result of restricted cell-to-cell movement of a virus due to a mechanism of resistance (7,12).

Sources of resistance to MDM in sweet corn have not been identified or widely used, although some sugary enhancer inbreds may express partial resistance (2). Genes for resistance from dent corn sources, such as Pa405, Ga209, and B68, have been introgressed into commercial and public sweet corn inbreds (1,8,16). The inheritance of resistance to MDM may be controlled by one or more genes, possibly involving modifiers or minor genes (3,4,9,13,14,20,22, 24). A single dominant gene, *Mdm*1, on the short arm of chromosome 6 appears to be involved in resistance to all viral strains causing MDM (15). Louie et al. (14) postulated a common genetic basis of resistance to all strains; however, Scheifele and Wernham (24) observed different genetic systems controlling reactions to MDMV-A and SCMV-MB. Modifier genes may be associated with specific resistance to MDMV-A and SCMV-MB (14) and with the degree of cell-to-cell movement of virus that can result in sectoring symptoms and systemically symptomatic plants in spite of some resistance genes.

Most of the previous research on the effects of MDM on yield evaluated the reactions of susceptible hybrids to MDMV-A or SCMV-MB (5,17,18,21). Although previous studies have demonstrated that MDM reduces yield, it is not apparent that yields of resistant hybrids with varying incidences of symptomatic reactions will be entirely unaffected by infection. The objectives of this study were to compare the incidence of MDM infection and types of symptomatic responses among commercial sweet corn hybrids with resistance to MDM and to determine the effects of MDMV-A and SCMV-MB on the yields of these hybrids.

MATERIAL AND METHODS

Field experiments were done at the Agronomy/Plant Pathology South Farm, Champaign, IL, in 1993 and 1994. The experimental design was a split plot with main plots arranged in a randomized complete block, with two or three replications in 1993 and five replications in 1994. Hybrids were planted in main plots, and viral treatments were applied to subplots. Experimental units were four-row plots with 15 plants per row. Rows were spaced 76 cm apart and were 4.7 m long. Seed was planted on 22 and 23 May 1993 and 19 May 1994.

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The treatment design was a factorial of hybrids and four inoculation treatments: MDMV-A, SCMV-MB, MDMV-A/SCMV-MB, and a noninoculated control. Twenty hybrids were evaluated in 1993. Fourteen hybrids were planted in three replicates, and six were planted in two replicates. Eighteen hybrids were planted in five replicates in 1994. Twelve hybrids were common to both trials. Commercial and experimental hybrids with MDM resistance contributed by one or both inbred parents were evaluated, as were susceptible commercial hybrids that served as checks. One hybrid, Sundance, thought to be tolerant to MDM also was included.

MDMV-A and SCMV-MB were obtained from and confirmed by R. E. Ford (University of Illinois, Urbana) and R. Louie (The Ohio State University, Wooster) and maintained in separate greenhouses on susceptible sweet corn. To increase inoculum, a mixture of MDM-susceptible sweet corn hybrids was inoculated in isolated field plots during early May. Plants inoculated with MDMV-A or SCMV-MB for the purpose of increasing inoculum were separated in the field by at least 30 m, and aphid vectors were not observed visually during May.

Inoculum was prepared by harvesting leaves from field-grown, symptomatic plants that had been mechanically inoculated with MDMV-A or SCMV-MB 14 to 21 days previously. Leaves were added to 0.1 M potassium phosphate buffer (approximately 1 g of tissue per 4 ml of buffer) (pH 7) and blended in a Waring blender for 1 min. The homogenate was filtered through cheesecloth. MDMV-A and SCMV-MB were kept separate at all times during preparation, and separate equipment was used to avoid cross-contamination. Carborundum was added to the inocula at about 5 g/liter. The underside of the youngest, fully expanded leaf of each plant was inoculated mechanically with a painter’s airbrush operated at 5.6 kg/cm^2 on 9, 17, and 24 June 1993 and 13 and 17 June 1994. Plants were at the two- to three-leaf stage when first inoculated.

Stand counts and incidence of symptomatic plants in the middle two rows of the four-row plots were recorded on 22 June 1993 and 1 July 1994. Incidence also was measured on 2 July 1993. Incidence of systemically symptomatic plants and incidence of sectoring plants (plants with discrete bands of symptomatic and asymptomatic tissue usually following the veins of leaves) were recorded 2 to 4 days prior to the beginning of harvest on 28 July 1993 and 21 July 1994.

Ears were harvested about 21 days after the midsilk growth stage. Due to maturity differences among hybrids, harvest dates ranged from 30 July to 10 August 1993 and from 25 July to 10 August 1994. Ears were harvested from 20 plants from the middle two rows of each experimental unit. The weight of ears with husk leaves, number of marketable ears, and number of ears with butt blanking were recorded. Within hybrids, ear weight was converted to a percentage of the mean of noninoculated control treatments.

Data were analyzed by analysis of variance (ANOVA). Noninoculated control treatments were excluded from ANOVA when the dependent variable did not vary (i.e., incidence and percent ear weight). Main effects of hybrids and viral treatments were compared by Waller-Duncan Bayesian least significant values when the hybrid by treatment interaction was not significant. Percent ear weight was plotted on incidence measured at the beginning of harvest and analyzed by ordinary least squares regressions. The increase in the percentage of ears with butt blanking from that in noninoculated control treatments also was plotted on MDM incidence and was analyzed by regression.

RESULTS

Symptomatic plants were not observed in noninoculated plots. In 1993, mean incidence of symptomatic plants for the 20 hybrids ranged from 0 to 68%, 4 to 79%, and 4 to 100% for the 22 June and 2 and 28 July ratings, respectively. In 1994, mean incidence for the 18 hybrids ranged from 4 to 92% and 1 to 100% for the 1 and 21 July ratings, respectively. The main effects of hybrids and viral treatments and the interaction of hybrids and treatments were significant in the ANOVAs of incidence measured at the beginning of the harvest during both years.

Incidence of systemic symptomatic plants differed among hybrids inoculated with MDMV-A or SCMV-MB (Tables 1 and 2). In 1993, incidence for all three viral treatments was less than 32% for seven hybrids: BiGuard, Dallas, Enforcer, GH2757, HMX 9352, Silverette, and Topacio (Table 1). For Esteem, GH 1209, Sundial, Terminator, and WH 3443, incidence was greater for plants inoculated with SCMV-MB than for plants inoculated with MDMV-A. Incidence of sectoring among symptomatic plants was 11% for GH 1759 and less than 5% for all other hybrids (Table 1).

In 1994, incidence was less than 8% for BiGuard, Dallas, and Topacio inoculated with any of the three viral treatments (Table 2). Incidence was between 6 and 26% for all treatments of Enforcer, GH 1209, GH 2757, HMX 0381, and Sundial. Incidence was less than 35% when Elite, Esteem, HMX 9350, Sundance, and Terminator were inoculated with MDMV-A but more than 50% when these hybrids were inoculated with SCMV-MB or MDMV-A/SCMV-MB. Incidence ranged from 77 to 100% for the susceptible checks: Crisp n Sweet 710-A, Excellency, and Green Giant Code 30. Elite, Esteem, Green Giant Code 29, Green Giant Code 30, HMX 9350, More, Sundance, and Terminator had lower incidences when inoculated with MDMV-A than when inocu-

Table 1. Incidence of maize dwarf mosaic (MDM) and sectoring among symptomatic plants of 20 sweet corn hybrids inoculated in 1993 with maize dwarf mosaic virus (MDMV-A) and/or sugarcane mosaic virus (SCMV-MB)

			Incidence[b] of MDM in treatments				
Hybrid	**Endosperm**	**Kernel color[a]**	**MDMV-A (%)**	**SCMV-MB (%)**	**MDMV-A/ SCMV-MB (%)**	**$\bar{x}$[c] (%)**	**Sectoring[d] (%)**
BiGuard	su	B	0	8	11	6	2
Dallas	su	Y	1	12	4	6	0
Eliminator	su	Y	17	43	15	28	1
Elite	se	Y	68	76	68	70	4
Enforcer	su	Y	8	30	28	22	1
Esteem	su	Y	14	70	27	37	1
GH 1209	su	Y	13	58	23	31	11
GH 1759	su	Y	47	51	41	46	0
GH 2535	su	Y	9	38	33	24	3
GH 2681	se	Y	56	57	40	51	0
GH 2757	se	Y	4	19	15	13	2
HMX 9352	sh_2	Y	8	0	3	4	0
HMX 9373	su	Y	…	100	100	100	0
More	su	Y	70	98	97	88	1
Silverette	su	W	24	29	13	22	3
Sundance	su	Y	51	100	96	83	0
Sundial	se	Y	5	38	2	15	1
Terminator	se	Y	19	65	20	30	1
Topacio	se	Y	4	18	9	10	0
WH 3443	su	W	9	40	3	19	1
$\bar{x}$[e]			28	46	36		
BLSD[f]				32			

[a] B = bicolor, Y = yellow, and W = white.

[b] (Symptomatic plants/total plants) × 100, rated 28 July 1993.

[c] Means of hybrids.

[d] Percentage of symptomatic plants sectoring (i.e., distinct bands of asymptomatic and symptomatic tissue).

[e] Means of viral treatments.

[f] Bayesian least significant difference for comparisons of combinations of hybrids and viral treatments.

lated with SCMV-MB. More than half of the symptomatic plants of BiGuard, Dallas, HMX 0381, and Sundial sectored (Table 2). Incidence of sectoring symptoms ranged from 10 to 50% for Elite, Enforcer, GH 1209, GH 2757, HMX 9350, and Terminator.

Weights of 20 ears with husk leaves ranged from 3.5 to 7.2 kg among the combination of 20 hybrids and four treatments in 1993 and from 3.9 to 7.2 kg among the 18 hybrids and four treatments in 1994. Hybrid means ranged from 4.0 to 6.8 kg in 1993 and from 4.1 to 6.7 in 1994.

Hybrid means for ear weight as a percentage of controls ranged from 84 to 109% in 1993 (Table 3). Viral treatment means of 97, 95, and 95% for the MDMV-A, SCMV-MB, and MDMV-A/SCMV-MB treatments did not differ. In 1994, hybrid means for percent ear weight ranged from 84 to 105% (Table 4). Viral treatment means of 99, 96, and 93% for the MDMV-A, SCMV-MB, and MDMV-A/SCMV-MB treatments differed. The hybrid by treatment interaction was not significant in either year.

Yields (percent ear weight) of Esteem, HMX 9373, More, Sundance, and Sundial were reduced 8% or more by MDM in 1993 (Table 3). Yields of Dallas, Elite, and GH 2535 were reduced from 4 to 8% by MDM in 1993. In 1994, yields of Crisp n Sweet 710A, Excellency, Green Giant Code 30, More, and Sundance were reduced 10% or more due to MDM (Table 4). Yields of Esteem, GH 1209, and HMX 9350 were reduced from 5 to 10% by MDM.

Because the main effect of viral treatments and the hybrid by viral treatment interaction were not significant in the ANOVA of percent yield in 1993, hybrid means were used in the regression of percent yield (ear weight) on incidence (Fig. 1). The slope coefficient was –0.15, and the coefficient of determination was 0.44. In 1994, when the main effect of viral treatments was significant, percent yield of each hybrid was regressed on incidence within each MDM treatment (Fig. 2). Slope coefficients were –0.085, –0.14, and –0.15, and coefficients of determination were 0.29, 0.62, and 0.63 for the MDMV-A, SCMV-MB, and MDMV-A/SCMV-MB treatments, respectively. The slope for the MDMV-A treatment differed from the SCMV-MB and MDMV-A/SCMV-MB treatments. The slopes for the SCMV-MB and MDMV-A/SCMV-MB treatments did not differ.

Butt blanking varied between years and hybrids. In 1993, the main effect of hybrids was significant, but treatment and the interaction term were not. In 1994, the main effect of hybrids and treatments and the interaction of hybrids and treatments were highly significant. Hybrid means for percentage of butt blanking in 1993 ranged from 7 to 61% (Table 3). In 1994, percentages of butt blanking ranged from 3 to 68% for combinations of hybrids and viral treatments. Treatment means in 1994 were 23, 26, 30, and 19% for MDMV-A, SCMV-MB, MDMV-A/SCMV-MB, and the noninoculated control.

In 1993, butt blanking was less than 10% for BiGuard and Terminator (Table 3). Butt blanking was between 13 and 18% for Eliminator, GH 1209, and Silverette. In 1994, butt blanking for many hybrids did not differ between viral treatments and the noninoculated treatment, including BiGuard, Dallas, and Terminator, for which butt blanking was less than 17%, GH 1209, HMX 0381, and HMX 9350, for which butt blanking was from 12 to 36%, and Enforcer, GH 2757, Sundial, and Topacio, for which butt blanking was from 23 to 45% (Table 4). For three hybrids, Crisp n Sweet 710A, More, and Sundance, the percentage of ears with butt blanking was higher for all three viral treatments than for the noninoculated control. For five hybrids, Elite, Esteem, Excellency, Green Giant Code 29, and Green Giant Code 30, the percentage of ears with butt blanking was greater for the SCMV-MB/MDMV-A treatment than for the noninoculated treatment, but butt blanking did not differ between the MDMV-A and noninoculated control treatments (Table 4).

Incidence of MDM was more than 60% for 14 of 15 hybrid/viral treatments for which butt blanking was greater than the noninoculated control in 1994 (Tables 2 and 4). Similarly, of 21 hybrid/viral treatments with MDM incidence of 50% or more in 1994, 14 had butt blanking that was significantly greater than the noninoculated treatment.

Table 2. Incidence of maize dwarf mosaic (MDM) and sectoring among symptomatic plants of 18 sweet corn hybrids inoculated in 1994 with maize dwarf mosaic virus (MDMV-A) and/or sugarcane mosaic virus (SCMV-MB)

			Incidence[c] of MDM in treatments				
Hybrid[a]	Endo-sperm	Kernel color[b]	MDMV-A (%)	SCMV-MB (%)	MDMV-A/ SCMV-MB (%)	$\bar{x}$[d] (%)	Sectoring[e] (%)
BiGuard	su	B	1	6	6	4	50
CnS 710A	sh_2	Y	99	100	99	99	0
Dallas	su	Y	1	5	8	5	82
Elite	se	Y	32	73	74	60	24
Enforcer	su	Y	9	16	25	17	19
Esteem	su	Y	34	59	66	53	7
Excellency	su	Y	100	100	100	100	0
GG Code 29	su	Y	61	87	92	80	1
GG Code 30	su	Y	77	99	100	92	0
GH 1209	su	Y	8	20	12	13	39
GH 2757	se	Y	10	19	26	18	48
HMX 0381	sh_2	B	14	25	19	19	55
HMX 9350	su	Y	22	56	62	47	13
More	su	Y	61	87	96	93	0
Sundance	su	Y	23	97	95	72	0
Sundial	se	Y	6	18	11	11	85
Terminator	se	Y	35	51	58	48	14
Topacio	su	Y	0	2	1	1	0
$\bar{x}$[f]			35	51	53		
BLSD[g]				6			

[a] CnS = Crisp n Sweet and GG - Green Giant.

[b] B = bicolor and Y = yellow.

[c] (Symptomatic plants/total plants) × 100, rated 21 July 1994.

[d] Means of hybrids.

[e] Percentage of symptomatic plants sectoring (i.e., distinct bands of asymptomatic and symptomatic tissue).

[f] Means of viral treatments.

[g] Bayesian least significant difference for comparisons of combinations of hybrids and treatments.

Table 3. Weight of ears with husk leaves as a percentage of the noninoculated control and incidence of butt blanking for 20 sweet corn hybrids inoculated in 1993 with maize dwarf mosaic virus (MDMV-A) and/or sugarcane mosaic virus (SCMV-MB)

Hybrid	Ear weight[a] (%)	Butt blanking[b] (%)
BiGuard	97	7
Dallas	96	30
Eliminator	100	13
Elite	94	61
Enforcer	98	31
Esteem	87	32
GH 1209	98	18
GH 1759	99	28
GH 2535	95	24
GH 2681	100	29
GH 2757	100	37
HMX 9352	97	38
HMX 9373	86	45
More	86	35
Silverette	102	16
Sundance	84	34
Sundial	92	37
Terminator	98	8
Topacio	102	36
WH 3443	109	23
BLSD[c]	4	11

[a] Mean of three viral treatments (i.e., MDMV-A, SCMV-MB, and combination of both) as a percentage of the noninoculated control for each hybrid.

[b] Percentage of ears with poor kernel fill at the basal portion of the ear.

[c] Bayesian least significant difference value (k = 100) for comparisons of hybrid means.

Thus, an increase in butt blanking from that which occurred in the noninoculated treatment was associated with a high incidence of MDM. In both years, there was a significant linear relationship between the increase in the percentage of ears with butt blanking (from that in the noninoculated control treatment) and incidence of MDM. Regression coefficients ranged from 0.13 to 0.27, and coefficients of determination ranged from 0.38 to 0.85.

DISCUSSION

Resistance to MDM in commercial sweet corn hybrids significantly lowered the incidence of symptomatic plants and reduced the adverse effects of MDM on yield, but it did not prevent all infections. None of the hybrids in this study were 100% asymptomatic when inoculated with MDMV-A and/or SCMV-MB. Hybrids differed in the effectiveness of MDM resistance. Incidence of symptomatic plants was below 20% for all viral treatments (MDMV-A, SCMV-MB, and MDMV-A/SCMV-MB) in both years for four hybrids: BiGuard, Dallas, HMX 9352, and Topacio. These hybrids appeared to have greater resistance than the other hybrids evaluated in this study, based on a lower incidence of symptomatic plants. A second group of hybrids had moderate levels of resistance to MDM based on an incidence of symptomatic plants that was greater than the most resistant hybrids but lower than the susceptible hybrids. Incidence of symptomatic plants was 30% or less for Enforcer and GH 2757 in both trials, for Silverette in the 1993 trial, and for HMX 0381 in the 1994 trial. Some hybrids (i.e., Eliminator, Elite, Esteem, GH 1209, HMX 9350, Terminator, and WH 3443) appeared to have greater resistance to MDMV-A than to SCMV-MB, based on significant differences in incidence of symptoms when plants were inoculated with either of the two viruses.

Even though reactions to MDMV-A and SCMV-MB differed among the resistant sweet corn hybrids in this study, all of the resistant hybrids probably carry the *Mdm-1* gene, because widely used sources of resistance in sweet corn were derived from Pa 405 or B68. Differences among MDM-resistant sweet corn hybrids in our trial may be due to the specific source of resistance or whether both inbred parents contributed resistance genes to the F_1 hybrids.

Although the homozygous condition is not necessary for asymptomatic, resistant reactions in F_1 hybrids, as is evident in several studies in which F_1 hybrids with Pa405 as a resistant parent were asymptomatic (15,20,23), it is conceivable that two resistant inbreds may produce an F_1 hybrid with higher levels of MDM resistance, because each resistant inbred may carry different modifiers of the *Mdm*1 gene. Assuming that Pa405 has the full compliment of modifiers (because F_1 hybrids with this line usually are asymptomatic), the lack of modifiers of *Mdm*1 in some MDM-resistant sweet corn inbreds may limit the degree to which MDM-resistance restricts viral movement, especially when inoculum is introduced in close proximity to meristematic tissues. Thus, incidence of symptomatic plants in hybrids produced from these resistant inbreds is less than that for susceptible hybrids but greater than other MDM-resistant materials. Similarly, reactions to MDMV-A and SCMV-MB may be affected by modifiers of *Mdm-1*.

Our results agree with those of Scheifele and Wernham (24), who found that hybrids can differ in reactions to MDMV-A and SCMV-MB. Incidence differed significantly between MDMV-A and SCMV-MB for 5 and 11 hybrids in 1993 and 1994, respectively. In each case, incidence was lower for plants inoculated with MDMV-A. In another study (9), we observed a higher incidence of MDM due to MDMV-A than SCMV-MB in backcrosses of sweet corn inbreds when Pa 405 was the source of resistance. Another explanation for these results may be that our isolates of SCMV-

Table 4. Weight of maize ears with husk leaves as a percentage of the noninoculated control and incidence of butt blanking for 18 sweet corn hybrids inoculated in 1994 with maize dwarf mosaic virus (MDMV-A) and/or sugarcane mosaic virus (SCMV-MB)

		Butt blanking[b] in MDM treatments			
Hybrid	Ear weight[a] (%)	MDMV-A (%)	SCMV-MB (%)	MDMV-A/ SCMV-MB (%)	CK[c] (%)
BiGuard	103	3	4	7	8
Crisp n Sweet 710A	88	41	39	51	15
Dallas	105	17	9	10	17
Elite	96	49	66	68	39
Enforcer	101	29	27	37	39
Esteem	93	15	17	29	11
Excellency	90	11	21	31	6
Green Giant Code 29	100	23	30	36	14
Green Giant Code 30	89	5	14	23	3
GH 1209	93	12	15	18	22
GH 2757	99	30	30	36	34
HMX 0381	96	17	13	17	22
HMX 9350	94	24	30	19	25
More	89	33	36	37	10
Sundance	84	25	36	28	7
Sundial	100	30	23	36	29
Terminator	97	5	13	14	2
Topacio	102	45	39	43	35
BLSD	5[d]		16[e]		

[a] Mean of three viral treatments (i.e., MDMV-A, SCMV-MB, and combination of both) as a percentage of the noninoculated control for each hybrid.

[b] Percentage of ears with poor kernel fill at the basal portion of the ear.

[c] Noninoculated control plot.

[d] Bayesian least significant difference (BLSD) value ($k = 100$) for comparisons of hybrid means.

[e] BLSD for comparison of hybrid-treatment combinations.

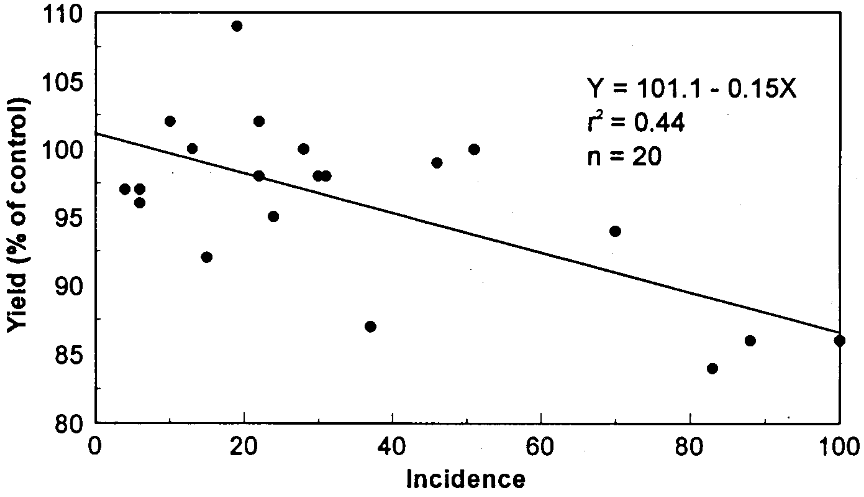

Fig. 1. Yield (ear weight) as a percentage of noninoculated controls of 20 sweet corn hybrids inoculated with maize dwarf mosaic virus (MDMV-A), sugarcane mosaic virus (SCMV-MB), or MDMV-A/SCMV-MB regressed on incidence of maize dwarf mosaic-infected plants measured at the beginning of the harvest (28 July 1993). Each data point represents a hybrid mean of three replicates of three viral treatments (MDMV-A, SCMV-MB, and MDMV-A/SCMV-MB).

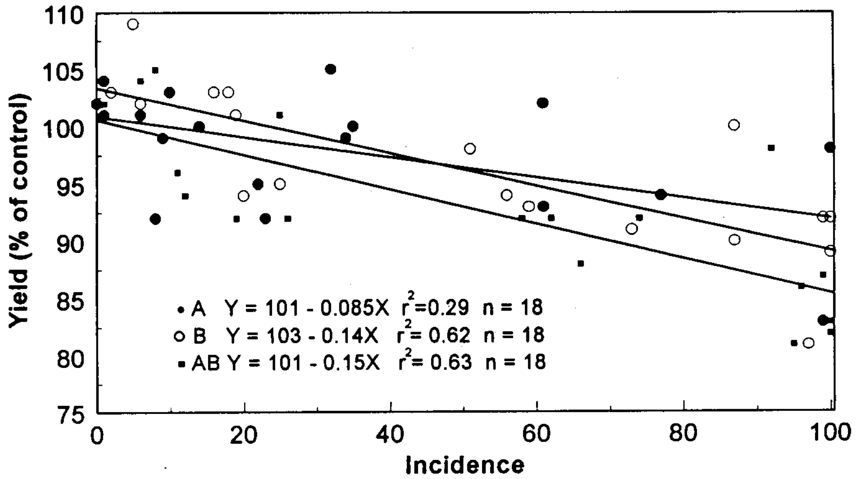

Fig. 2. Yield (ear weight) as a percentage of noninoculated controls of 18 sweet corn hybrids inoculated with maize dwarf mosaic virus (MDMV-A), sugarcane mosaic virus (SCMV-MB), or MDMV-A/SCMV-MB regressed on incidence of maize dwarf mosaic-infected plants measured at the beginning of the harvest (21 July 1994). Each data point represents the mean of five replicates of three viral treatments (MDMV-A, SCMV-MB, and MDMV-A/SCMV-MB).

MB had a greater ability to cause symptoms than did our isolates of MDMV-A.

Plants displaying sectoring symptoms were more prevalent in 1994 than in 1993. In 1994, sectoring symptoms were observed in half or more of the symptomatic plants of four hybrids: BiGuard, Dallas, GH 2757, and HMX 0381. Jones and Tolin (7) suggested that sectoring is an expression of resistance, so it is not surprising that these symptoms were most prevalent in hybrids for which incidence of symptomatic plants was relatively low. Sectoring is a categorical measurement of response to viral infection. We did not attempt to make a numerical measurement (e.g., severity) of MDM symptoms, because previous research has indicated a lack of association between symptom severity and titer (21).

Yields of hybrids with some resistance to MDM were not affected greatly by MDM. Yields were lowered by 10 to 20% compared to the noninoculated control plots for only four hybrids in 1993 and 1994. Slope coefficients of –0.15 in 1993 and from –0.085 to –0.15 in 1994, which correspond to an approximately 15% yield reduction when MDM infection was 100%, were similar to the lower end of the range of yield losses observed in previous studies (5,17, 21). None of the hybrids with higher levels of MDM resistance had more than 18% incidence, and yields were not affected for these hybrids. Butt blanking also was lower for hybrids with low incidence of symptomatic plants. Although slope coefficients from regressions of butt blanking on incidence were slightly more negative than those from regressions of percent yield (ear weight) and incidence, the effects of MDM on ear weight and butt blanking were similar to those observed previously (21).

In this study, plants were inoculated two to three times beginning at the three-leaf stage, which may be comparable to late-planted sweet corn under moderate pressure from viruliferous aphids. If populations of viruliferous aphids are as low as usual in most years in the major sweet corn-producing regions of the Midwest, yield of currently available MDM-resistant hybrids probably will not be adversely affected by this disease, although some symptomatic plants may be seen. In areas with higher populations of viruliferous aphids and where johnsongrass (*Sorghum halapense*) occurs (i.e., the Ohio River Valley), incidence of MDM on resistant sweet corn may be higher than desired, but hybrids with resistance should perform better than susceptible hybrids.

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